

# INHERITED MUSCLE ABNORMALITY IN THE DOMESTIC FOWL

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**M**ANY abnormalities, both lethal and nonlethal, are now known in the domestic fowl (Hutt<sup>4</sup>, Jull<sup>6</sup>, and others). Of these more than half involve the skeleton, while few are known to affect the muscles. However, the domestic fowl does exhibit variation in conformation of the breast and the shape of the breast musculature as shown, for example, by the Cornish as contrasted to most other breeds.

Muscular dystrophies of man are diseases of hereditary bases and are characterized by weakness and altered size of skeletal muscles (Adams, *et al.*<sup>1</sup>). Some of these diseases are characterized by gross enlargement of the involved muscles followed by atrophy. A hereditary muscular abnormality in New Hampshires, with features similar to certain muscular dystrophies of man, is the subject of this report. Genetic aspects are emphasized; however, a preliminary report of observations on the anatomy of the condition is included.

## Material and Methods

Eggs from stock known to be heavily muscled on the breast, with some birds unable to raise their wings, were obtained from a New Hampshire breeder in March 1953. Of these, 72 were from a selection designated (by the breeder) Broad Line and 48 were from a Lock Wing selection; 48 of the former and 28 of the latter hatched, and subsequently eight and five respectively (16.7 and 17.9 percent of the birds classified) were found to be unable to raise their wings. The stock constitutes Strain 3 of the New Hampshire breed used by the California Agricultural Experiment Station.

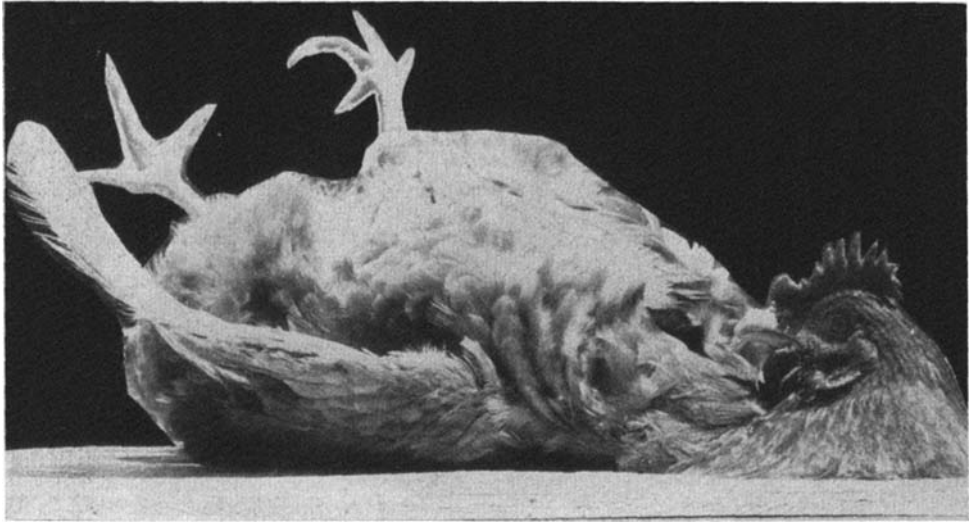
The chicks have been routinely weighed when hatched and at four and

eight weeks of age. Some were weighed at 12 and/or 20 weeks and at maturity. The length of the shank (tarso-metatarsus), the keel of the sternum, and the width of the breast 1 cm. above the ventral surface of the keel and near its anterior end were measured at eight or 12 weeks. Some mature birds were also measured. The length of the shank was measured with a special instrument (Burmester and Lerner<sup>3</sup>), the keel with calipers, and the width of breast with soldering wire which was fitted to the breast and then transferred to lined paper.

The birds were classified as normal on the basis of their ability to raise their wings and rise from a flat surface when laid on their backs (Figure 25). Most classifications were made at 8 weeks of age or later. Some were observed to be abnormal at 4 weeks. The age at which the birds show this abnormality varies and may occur for some individuals later than 8 weeks of age. It should also be noted that some Strain 3 birds and birds out of crosses of Strains 3 and 2 have stiff wings that apparently cannot be raised as high as those of Strain 2 birds, yet are "normal" in the sense that they can rise from a flat surface when laid on their backs.

In order to determine how the muscular abnormality is inherited, the abnormal and normal birds of New Hampshire Strain 3 were mated among themselves and to hens of New Hampshire Strain 2. The latter strain (N.H. Strain 2) has never exhibited muscular symptoms. The wider breasted normal birds of Strain 3 were selected for breeding. The abnormal and Strain 2 birds were not selected except to eliminate sick and obviously unthrifty birds.

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**THE EFFECTS OF MUSCULAR DYSTROPHY**

Figure 25

Muscular dystrophy prevents this hen from rising from a flat surface when placed on her back.

Several abnormally winged birds kept on the floor fell on their backs and died before they were noticed. The abnormal hens are unable to fly to nests off the ground. Because of these handicaps, abnormal birds kept for breeding were transferred to individual cages before maturity, and all matings involving them have been by artificial insemination.

Birds have been submitted for anatomical study periodically since 1953. In 1955 a systematic study was undertaken in which birds of normal and abnormal stocks were autopsied at 1, 4, 12, and 20 weeks of age. Body measurements and muscle weights were recorded and samples of involved muscles were taken for microscopic study.

**Results**

Typical hatching results are shown in Table I. Since all the abnormal birds bred to date

are out of Strain 3, it is evident that the hatch of fertile eggs from matings of birds within this strain is lower than that of eggs from crosses between strains 3 and 2. This suggests that Strain 3 is inbred and that the improved hatch from crossing reflects hybrid vigor. Were a lethal (or lethals) associated with the muscle abnormality involved, the lowest hatch would have been expected from abnormal birds, which is not the case. Embryos that failed to hatch were examined and no gross morphological deviations that might suggest a lethal were found.

According to limited data, abnormal hens of Strain 3 are slower maturing and lay at a somewhat lower rate than those of Strain 2. Until data are obtained under strictly comparable conditions these data must be considered indicative and not conclusive.

Gain in weight is a little slower for abnormal birds of Strain 3 than for normal birds of Strain 2, but the mature weight is about the same. The abnormal birds have slightly shorter bones, as shown by the shanks (tarsometatarsi) and keels of the sternum, and these differences are found in both young, growing

TABLE I. Hatching results, 1954

Type of mating*	Dam	Fertile		Percent of fertile			
		Percent	No.	Dead Embryos			Hatched
Site				1st week	2nd week	3rd week	
A	A	78	52	14	4	12	71
A	N3	81	17	6	12	12	71
A	N2	57	90	9	2	6	83
N3	A	85	28	7		25	68
N3	N3	75	66	16	3	16	64
N3	N2	87	41	7	2	5	85

\* A is for abnormal, N for normal of strains 3 or 2.

PARENTS SIRE   DAM	GENOTYPED PROGENY	BREAST WIDTH IN CENTIMETERS:	
		FEMALE	MALE
A   A	am   am	3.37	3.86
A   N3	am   am	3.10	3.22
A   N3	Am   am	2.65	2.91
A   N2	Am   am	2.36	2.90
N2   N2	Am   Am	2.28	2.29

**BREAST WIDTH**

Figure 26

Effect of the *am* gene on breast width of eight-week-old birds. The width was corrected for differences between average body weights of groups of birds of the same sex. Note that the males were slightly wider breasted than the females.

birds and mature birds. For eight-week-old birds these differences in weight and bone length, while not large, are statistically significant (Table II).

The abnormal birds have much wider breasts, on the average, than normal birds, (Figure 26). Both normal and abnormal birds vary considerably in width (Figure 27), and there is an obvious overlapping of normal birds from Strain 2 on normal from Strain 3 and of progeny between the two strains. Similarly, the narrower breasted abnormal overlap on the wider breasted normal.

**Anatomy**

A detailed report on the anatomy of the abnormal birds will be presented elsewhere. Preliminary studies indicate that most of the major muscles of the bird are affected by the development of this abnormality. The breast muscles, muscles of all segments of the wings, and muscles of the legs are affected. When enlargement is first detected, the involved muscles are more opaque and somewhat lighter in color than normal. The grossly visible striated nature of skeletal muscle is accentuated in the affected muscles. This is due to enlargement of each fasciculus, which in turn is due to enlargement of each individual muscle fiber. Some of the muscles are more extensively involved than others. As an example, the superficial pectoral muscle (pectoralis major) is more enlarged than its antagonist, the deep pectoral (pectoralis minor); however, gross and microscopic alterations are detected in both. Each muscle apparently has a different pattern of onset and progress of the abnormal state which is superimposed

upon its individual normal growth pattern. A major microscopic feature of the enlarged muscles is an increase in number of muscle nuclei, many of which are centrally located in the muscle fibers.

Atrophy appears to be a sequel of muscular enlargement. This phase of the process is variable. Limited data suggest that only enlargement occurs in some animals; atrophic changes, if any, are long delayed. In others atrophy rapidly follows muscular enlargement. Profound microscopic alterations can be demonstrated in the grossly atrophied muscles. These include deposition of fat, degeneration of muscle fibers, and fibrosis. The time of onset of microscopic changes is to be correlated with gross alterations of the muscles. These should result in a fuller appreciation of the full spectrum of manifestations of the condition and should aid in evaluating genetic data.

Although anatomical studies are in preliminary stages, it is evident that the condition of chickens described above has many similarities to the muscular dystrophies of man. The sequence of gross alterations in size of the muscles (hypertrophy and atrophy), the centrally placed nuclei in hypertrophied fibers, and the degenerative changes stated above have their counterparts in muscular diseases of human beings.

**Genetics**

The data available for various matings are summarized in Table III. Progeny were not obtained from abnormal birds until 1954, partly as noted above, because of the inability of the hens to get into the trapnests and perhaps partly because of their lower egg production.

In 1953-54 mixed semen from several males was used for insemination, whereas the sire and the dam of all birds hatched in 1955 were known. It is not certain that all birds suspected of being abnormal in 1954 were tested by laying them on their backs; however this test was applied to all progeny of abnormal sires mated to abnormal dams in 1955 with the exception of two early dead and 10 birds picked at random for dissection when a week old. A few birds were similarly picked for dissection from other matings in 1955 when they were one week old. Since all chicks that subsequently became abnormal appeared normal when one week old, the birds removed for this dissection are omitted from the table.

The 1953-54 data suggested that abnormal muscling might be a simple autosomal recessive to normal for which the symbol *am* will

TABLE II. Means with standard errors of weights and measurements at eight weeks of age, 1955, of abnormal (Strain 3) and normal (Strain 2) New Hampshire

Classification	Sex	No. of birds	Weight kilos.	Length of shank cm.	Length of keel cm.	Width of breast cm.
Abnormal	male	14	1.02 ± .02	9.17 ± .07	8.11 ± .08	3.67 ± .10
"	female	30	.80 ± .02	8.28 ± .07	7.61 ± .07	3.33 ± .09
Normal	male	123	1.09 ± .01	9.60 ± .04	8.42 ± .04	2.92 ± .04
"	female	109	.86 ± .01	8.68 ± .04	7.63 ± .04	2.67 ± .03

be used. The criterion for abnormality was, as stated above, the inability of the bird to right itself when placed on its back on a flat surface. There is no indication of sex-linkage. Assuming that the difference is determined by a single gene, the 1954 data show these discrepancies: one progeny classified as normal from abnormal parents and three progeny out of normal (N2) dams classified as abnormal when only normal were expected. For these two types of matings (both parents abnormal or one parent out of Strain 2) the 1955 progeny that were classified agree well with expectation. If all the progeny in Table III from segregating families out of normal Strain 3 parents are combined, there were 155 normal to 41 abnormal, which agrees well with expectation of 147 to 49 on the basis of a ratio of 3 : 1. Similarly, the progeny from all segregating families of N3 × A or A × N3 numbered 40 normal to 40 abnormal in agreement with expectation of 1:1, although the 12 progeny from N3 × A parents in 1954 deviate from expectation ( $p = .016$ ). On the other hand, the number of abnormal among the F<sub>2</sub> progeny (original cross, abnormal males mated to Strain 2 females) is less than expected. As noted above, the age at which the birds become abnormal varies and the degree of abnormality may also vary. These variables could well account for the apparent discrepancies in the data. It will, however, be necessary to make further test matings to estimate the effect of gene interaction on the expression of the trait as measured by the ability to raise the wings.

Some of the effects of the *am* gene on conformation and muscles are apparent from the

measurements on live birds. Thus, the data in Table II and Figure 26 indicate that the *am* gene influences width of breast, a polygenic trait, mostly determined by additively acting genes and much influenced by body weight<sup>7,8,2</sup>. When the variation normally found in breast width is superimposed on the effect of the *am* gene, the result for older birds is as shown in Figure 27. It will be observed that a few of the abnormal birds were quite wide, thus making this group the homozygous recessives much more variable than the homozygous normal (Strain 2). Detailed anatomical studies now in progress on the 1955 hatch and anticipated for the 1956 hatch of birds may contribute toward a clarification of this situation.

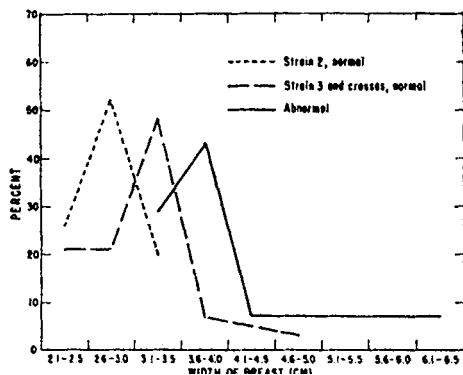
If the *am* gene affects width of breast in the homozygous and heterozygous combinations, the effect being additive so that  $am\ am > Am\ am$ , then data for progeny from some of the matings should illustrate this relationship. Figure 26 shows, as does Figure 27, that abnormal birds are wider breasted than normal birds out of the same parents (compare *am am* with *Am am* progeny out of A × N3 parents) and that heterozygous (*Am am*) normal birds are wider breasted than homozygous (*Am Am*) birds. Likewise, backcross progeny from heterozygotes on abnormal or homozygous normal should be more variable, since they are a mixture of two genotypes, than the progeny of the latter, which are all heterozygous. To test this point, in Figure 28 data for the 1955 male progeny of normal Strain 2 females out of abnormal males are compared with progeny of Strain 2 females mated to Strain 3 heterozygous normal males. In agreement with expectation, the latter are more variable;

**TABLE III.** Summary of results of matings of abnormal and normal muscled birds in 1953, 1954 and 1955. Expected numbers, based on the assumption that abnormal muscling is a simple autosomal recessive, and shown only for segregating families. Progeny classified at eight weeks except as noted in text

--- Matings* ---			Progeny†						
Sires	Dams	Year		Males	Normal Females	Total	Males	Abnormal Females	Total
A	A	1954	Obs.	1		1	9	16	25
"	"	"	Exp.						26
A	N3	1954	Obs.	3	7	10		2	2
"	"	"	Exp.			6			6
A	N2	1954	Obs.	30	40	71	1		1
"	"	"	Exp.			71			0
N3	A	1954	Obs.	3	1	4	1	2	3
"	"	"	Exp.			3.5			3.5
N3	N3	1953-4	Obs.	71	62	133	15	20	35
"	"	"	Exp.			126			42
N3	N3	" "	Obs.	36	30	66			
N3	N2	1954	Obs.	13	17	30		2	2
"	"	"	Exp.			32			0
A	A	1955	Obs.				19	17	36
A	N3	1955	Obs.	11	12	23	13	12	25
"	"	"	Exp.			24			24
N3	A	1955	Obs.	2	1	3	6	4	10
"	"	"	Exp.			6.5			6.5
N3	N3	1955	Obs.	14	8	22	3	3	6
N3	N3	1955	Exp.			21			7
A	N2	1955	Obs.	22	17	39			
N3	N2	1955	Obs.	30	24	54			
F <sub>1</sub>	F <sub>1</sub>	1955	Obs.	38	42	80	5	5	10
"	"	"	Exp.			67.5			22.5

\* See footnote Table I.

† Birds that died or were taken for dissection before they could be classified (e.g. when 1 week old) are not included.



### ABNORMAL BIRDS ARE WIDER BREASTED

Figure 27

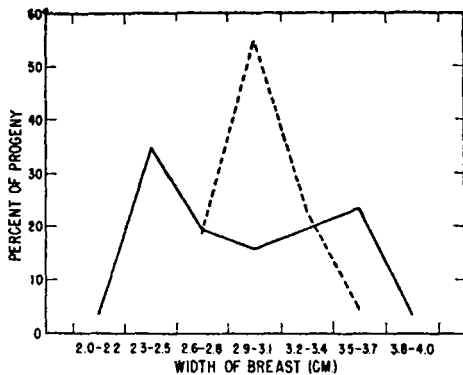
Width of breast in centimeters, one cm. above the ventral surface of the keel of the sternum. Data for 20-week-old males and females combined.

about one third are narrower and some wider, than any of the heterozygous progeny of the abnormal  $\times$  Strain 2 mating. While it presumably would not be possible to segregate homozygous from heterozygous normal on the basis of width of breast, the results illustrated in Figure 28 suggest that the narrowest breasted birds from a backcross population (Figure 28, broken line) should be homozygous normal while the widest breasted are heterozygous. Again it should be emphasized that anatomical data may help to clarify some of the genetic information. As stated above, the superficial pectoral (pectoralis major) muscle exhibits the greatest alteration in size of the muscles that contribute to width of breast. An evaluation of dimensions and masses of individual muscles shows promise of contributing toward a solution of these problems.

The simplest explanation for the data so far available is that abnormal muscling, as shown by the inability of the birds to raise their wings, is a simple autosomal recessive and behaves as such within Strain 3 of the New Hampshire breed. The possibility exists that genes carried by other strains and breeds may modify the age at which this trait appears and its severity of expression to a greater extent than those in Strain 3. The gene also affects width of breast and other traits, but here its effect is modified, though not obliterated, by many other genes. Further work on the genetics of the trait is under way.

### Summary

A muscle abnormality, which prevents the birds from raising their wings and rising from a flat surface when laid on their backs, has occurred in New Hampshires. When compared with normal birds of this breed but of



### BREAST WIDTH OF MALE PROGENY

Figure 28

Width of breast of eight-week-old birds: broken line— $F_2$  male progeny of abnormal ( $am\ am$ ) males and homozygous normal ( $Am\ Am$ ) Strain N2 females; solid line—male progeny of normal heterozygous ( $Am\ am$ ) Strain 3 males and Strain N2 females.

a different strain (Strain No. 2), in which muscular symptoms have not occurred, the abnormal birds were found to be wider breasted and to have shorter bones.

Preliminary anatomical studies show that most of the muscles of the bird are involved. Gross and microscopic alterations of the muscles are comparable to recorded changes in muscular dystrophies of man.

The data, from various types of matings, indicate that abnormal birds are homozygous for an autosomal gene ( $am$ ) which is recessive to normal.

Heterozygous (normal) birds have wider breasts than homozygous normal birds of the New Hampshire breed.

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