Variation of Fat Distribution in Pectoral Muscles of Chickens with Hereditary Muscular Dystrophy[1] (35513)

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Excess fat infiltration in pectoral muscles of chickens with hereditary muscular dystrophy (1) was first reported in 1959 by Jordan *et al.* (6). After it was shown that variation in fat content of the pectoralis was a high-heritability trait in muscular dystrophic stock (2), a breeding program was developed to establish high- and low-fat lines at 19 weeks of age. High-fat birds have attained an average of 14% and 20% fat in pectoral muscles of males and females, respectively; averages in the low-fat line are 2.4 and 2.6%, respectively (3). In view of these findings, a consideration was made of lipid concentrations within individual pectoral muscles. Examination of these muscles at necropsy has revealed that fat deposition is not uniform. This study was undertaken to describe the deposition of fat in various areas of the pectoralis and to determine if a consistent pattern exists.

Materials and Methods. Twenty chickens were selected from a line of muscular dystrophic birds produced for large pectoral muscles with high-fat content. This new line (UCD 309) was developed by crossing birds in broad-breasted line 304 with those in high-fat line 307. Details regarding genetic lines and their identification are presented in Table I. The right superficial pectoral muscle was removed from 10 birds (primary experimental group) 32 weeks of age, from 2 birds 16 weeks, and 2 birds 60 weeks of age. The muscles were sealed in individual plastic bags and frozen at —23° for later fat analysis.

Corrosion casts were made of 4 birds, 32 weeks of age, to compare vasculature with fat deposits in the pectoralis. Techniques for the preparation of casts are described by Narat *et al.* (11). Two birds, 32 weeks old, were utilized for acetylthiocholine staining of motor end plates (10).

TABLE I. Identification and Description of Experimental Lines of Muscular Dystrophic Chickens.[a]

Breeding line	Selection
200	Randomly selected normal
304	Early onset of disability[b]
305	Late onset of disability[b]
307	High fat content of pectoralis[c]
308	Low fat content of pectoralis[c]
309	Large pectoralis and high fat content[d]

[a] After Holliday *et al.* (5).
[b] Inability to rise from supine position.
[c] At 12–19 weeks of age.
[d] At 8 weeks of age.

The selection of 10 specific areas to be tested for fat was based on data from analysis of 25 sites in a single pectoral muscle. To insure that samples were taken from corresponding areas of each muscle, the sample sites were etched on a plastic grid and projected onto the individual muscles. A 1- to 2-g sample was removed from each test area.

Analysis for fat was done according to a modified procedure of Jordan *et al.* (6). The samples were weighed, dried to constant weight in a vacuum oven (25 psi) at 65°, and reweighed to obtain water content. The samples were then extracted four times with petroleum ether (30 to 60° bp) at 12-hr intervals, followed by extraction with absolute methanol for 12 hr. The samples were again dried and reweighed. Percentage fat was calculated on the basis of original fresh weight.

Results. Lipids had the same distribution pattern in all muscles tested (Fig. 1). The

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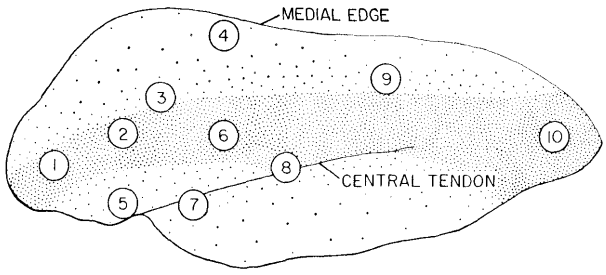

FIG. 1. This high-fat zone in the right superficial pectoral muscle of a muscular dystrophic chicken was delineated through gross fat staining techniques and lipid analysis of samples from sites shown.

fat was concentrated in a zone approximately midway between the central tendon and medial edge (keel) of the muscle, and decreased peripherally from this zone. In 32-week-old birds, there were two- to threefold differences in lipid concentration between sites in the high-fat zone and those located toward the periphery of the muscle.

The sample sites in the primary experimental group were correlated by the Wilcoxon rank method (9). Sites 1, 2, 6, 8, and 10 had high concentrations of fat and were, therefore, in the high-fat zone (Figs. 1 and 2). Sites 3 and 9 appeared to be on the edge of the zone, and had somewhat lower and more variable concentrations. Sites 4, 5, and 7 had relatively low concentrations and were outside the zone.

The relative concentrations of lipid (as described above) remained consistent during the first year, independent of sex or age. A

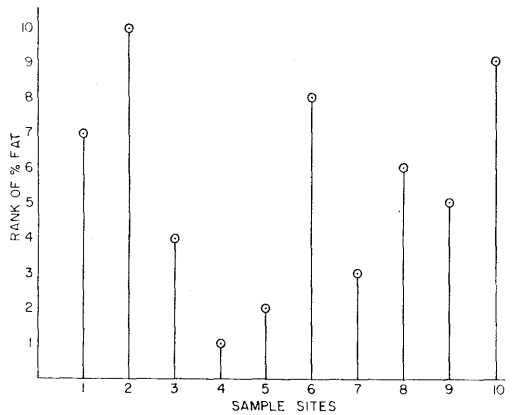

FIG. 2. Wilcoxan rank test showing the relationship between sample sites and lipid content in pectoral muscles from 32-week-old MD chickens.

five- to sixfold increase in fat between 16- and 60-week-old birds was evident. However, the overall increase between sample sites was proportional, thus maintaining a similar distribution pattern between the two age groups. It is possible, therefore, to predict the relative lipid concentration at any site in a given muscle (Line 309), and at any age (to 60 wk) from a single biopsy.

No relationship was found between the arterial system and the fat deposition pattern in pectoral muscles. Also, the results with histochemical staining indicated that motor end plates were located in dispersed groups, with little or no correlation with fat patterns.

Discussion. Fat deposition in the pectoralis seems unrelated to vascular or neurological patterns. Differences in fat concentration, however, may reflect a differential wasting pattern of the muscle fibers. Fat production had long been considered a secondary response of muscle to the disease processes of muscular dystrophy (4). Duchenne, 1868, [in Bonsett (4)] concluded that the amount of adipose tissue was a function of the degenerative state of the disease, and that proliferation of fat and fibrous tissues represents different stages of the same pathologic process (4).

Pectoral muscles of the 309 line produce a consistent lipid pattern which incorporates high- and low-fat concentrations at the same time. The fat which predominates in certain areas of the pectoralis is similar to that of adipose tissue, the principal component being triglyceride, and is directly associated with the loss of muscle tissue (4, 8). Therefore, a model of dystrophic pathology was

produced, illustrating aspects of the disease from "normal" tissue (low-fat areas) to completely destroyed tissue in which fibers had been replaced by fat. Support for this hypothesis is based on work by Gowers, 1879 (in Bonsett), and Bonsett (4), who reported differential wasting patterns in human pectoral muscles. Bonsett suggested that the difference in rate and degree of degeneration of a muscle is an expression of the difference in work load imposed on the muscle. This relationship is difficult to demonstrate in dystrophic chickens because the pectoralis is primarily involved with flight, and these birds have been rendered essentially flightless by the disease and by housing facilities.

Summary. The accumulation of lipids in pectoral muscles of New Hampshire chickens with muscular dystrophy occurred in a centralized zone through the long axis of the muscle. The concentration of fat decreased toward the periphery of the muscle. The fat distribution pattern remained constant in birds 16 to 60 weeks of age, and did not conform to vascular or neurologic patterns in the pectoralis. Fat infiltration appears to be an index of muscle fiber destruction.

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