Developmental and Maturational Aspects of Inherited Avian Myopathies (43061)

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Abstract. **Three inherited abnormalities of muscle growth of poultry are an inherited muscular dystrophy of the chicken, deep pectoral myopathy of turkeys and broilers, and focal myopathy of turkeys. The major features of each are described and compared. Cellular and molecular bases of dystrophy of the chicken and treatments to alleviate the disorder are discussed. The pathologic progression and anatomical basis for deep pectoral myopathy are presented. Evidence is given that focal myopathy of the turkey is a growth-dependent disorder. The implications of the idea that such disorders are partly consequences of selection are discussed in the context of the future needs of a poultry industry emphasizing processing of poultry meat.** [P.S.E.B.M. 1990, Vol 194]

The development of the complex multinucleated musculature of vertebrates affords many opportunities for congenital abnormalities. The processes by which single cells fuse to form multinucleated myotubes, neurons innervate embryonic fibers, and embryonic and neonatal muscle fibers mature into adult muscle fiber types are some of the steps that are vulnerable to genetic or epigenetic influences during development.

Inherited Muscular Dystrophy of the Chicken

Since genetic muscular dystrophy (GMD) of the chicken was first described in 1956 (1), hundreds of reports have been published on the abnormality.[1] The disorder is caused by a single gene; whether it is a deletion or a point mutation is not known. GMD was first noted in a New Hampshire strain of chickens; a similar abnormality was described in a Cornish strain (2).

Muscles with fast twitch α-white muscle fibers such as the superior pectoralis and biceps are most affected by the disorder. Slow, tonic "red" fibers, such as the anterior latissimus dorsi and the lateral adductor, are not involved, at least at an early age.

Experiments in which embryonic limb buds (3) and muscles (4) were transplanted from one genotype to another demonstrated that the presence of the dystrophic gene in the muscle tissue was sufficient for the dystrophy to be expressed. Dystrophic muscles grafted to a normal host and its nerve were phenotypically dystrophic, and normal muscles transplanted to a dystrophic host were normal.

GMD is classified as a dystrophy because the muscle fibers eventually atrophy and are replaced by fat and connective tissue, there are irregular shaped rounded fibers that are both larger and smaller than normal, and there is no evidence of primary nerve damage (5). The early appearance of the dystrophy in fast twitch but not in tonic fibers suggests its expression is restricted to a particular myogenic lineage (6).

Major features of dystrophic muscles from young chicks include enlarged transverse tubule/sarcoplasmic reticulum regions, myotonia, embryonic acetycholinesterase (AChE) and neonatal myosin forms, high mitochondrial activity, cellular hypertrophy and hyperplasia, relatively high fat and low protein content, and high serum creatine kinase, regardless of the genetic backgrounds of the lines examined (Table I). Symptoms of the disorder appear during late embryogenesis and neonatal muscle maturation. Many of the properties of dystrophic muscle resemble those of embryonic and adult slow, tonic red muscle. Others seem distinctive to the abnormality itself, and some are probably nonspecific consequences of muscle damage.

The irregularly shaped, abnormally large and small diameters fibers of dystrophic muscle are shown in Figure 1B. Unlike other muscle abnormalities, there

[1] A bibliography of research on the dystrophic chicken is available from the author.

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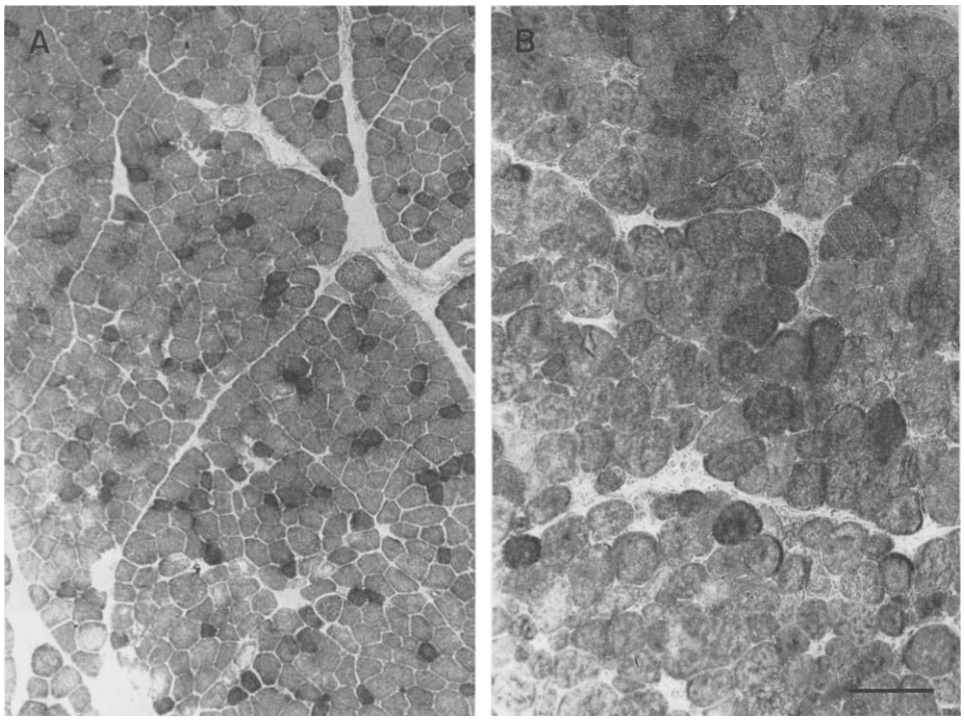

Figure 1. Light microscopy of normal (A) and dystrophic (B) chicken superficial pectoralis muscle. Eight-week-old normal line 412 and dystrophic line 413. Ten-micrometer cryostat cross-sections stained for succinic dehydrogenase. Bar is 100 μm.

Table I. Early Differences between Normal and Dystrophic Muscles

Sarcoplasmic reticulum	Myotonia
AChE Forms	Myosin forms
Mitochondria	Fiber size/shape
High fat	Low protein
Serum creatine kinase	

are few regions of fiber degeneration or regeneration in GMD muscle.

Enlarged sarcotubular regions are characteristic of dystrophic muscles regardless of background genes (Fig. 2B). They are not a property of normal embryonic or red muscle. However, similar structures are found in other muscle abnormalities and may be associated with calcium defects (Franzini-Armstrong, personal communication). Accompanying the anatomical sarcotubular defect is a relative decrease in the calcium-pumping proteins of the sarcoplasmic reticulum (7). The swollen, irregular shaped muscle fibers are dramatically reduced in dystrophic chickens deprived of water, suggesting that the characteristic abnormal morphology of the muscles is related to ion and water balance (8).

GMD muscles are myotonic, that is they respond to a single mechanical stimulus with repetitive discharges, indicative of an instability in bioelectrical membrane properties. Episodes often last 30 sec or longer (Fig. 3). Electrophysiologic investigations of the muscles show potassium and chloride abnormalities comparable to other myotonic disorders (9).

The maturation of dystrophic muscle differs from its normal counterpart. For example, the relative decrease in the level of mitochondrial activity that starts shortly before hatching in normal fast twitch muscle does not occur in dystrophic fast twitch muscle, resulting in an abnormally high level of oxidative metabolism in the adult muscles (10). Afflicted muscles from chicks from all dystrophic lines have higher levels of free amino acids, higher fat, and lower protein than their normal counterparts, resembling the composition of red muscle (11).

Indeed, many of the metabolic abnormalities of dystrophic muscle are normal properties of embryonic and tonic muscles and are known to be under neural influence. These include the high mitochondrial activity mentioned above, low lactic dehydrogenase activity, and the presence of globular forms of AChE in dystrophic muscle. However, this does not mean that the problem with dystrophic muscle is a faulty set of neural instructions. Genotypically, dystrophic nerves did not induce dystrophy in genotypically normal muscle transplants (3). If neural influences are involved in GMD, it is likely it is the dystrophic muscle that is unable to respond properly to signals from its phenotypically normal nerve.

The development and regulation of the multiple molecular forms of AChE of normal and dystrophic muscle illustrate the complexity of the problem (Fig. 4). Embryonic muscle fibers contain both low molecular weight globular 4-7S and 11S and a larger collagen-tailed 20S AChE form. After hatching the globular

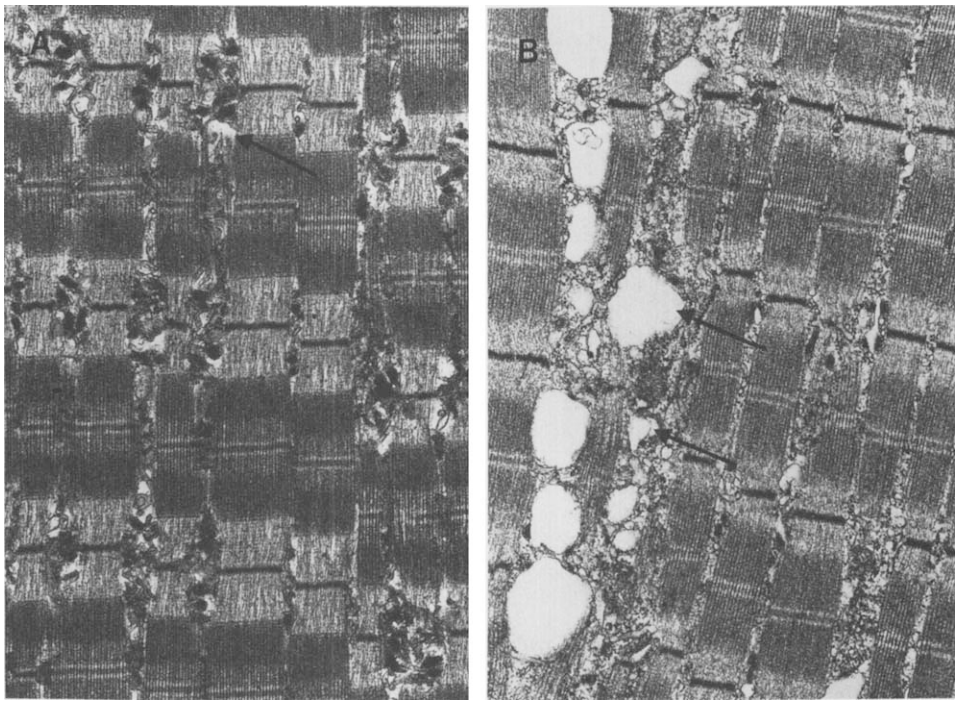

Figure 2. Transmission electron microscopy of normal (A) and dystrophic (B) chicken superficial pectoral muscle. Five-week-old normal line 412 and dystrophic line 413. Muscle tissue fixed with glutaraldehyde, stained with osmium, and photographed with an AEI EM-80 microscope (original magnification, ×18,000). Arrows indicate sarcotubule regions (data adapted from Ref. 5).

forms disappear in fast twitch muscles (12) and the 20S form eventually disappears in slow tonic muscles (13). Dystrophic fast twitch muscles maintain the embryonic forms of AChE as well as the large 20S form.

Denervation leads to an increase in AChE activity outside the motor end plates, the disappearance of the 20S form, and reappearance of the small 4-7S forms in normal fast twitch fibers. And, when denervated, the AChE pattern of dystrophic muscle becomes similar to that of normal denervated muscle (12). It is as if dystrophic muscle is unable to respond properly to signals given by the nerve that control maturation of AChE, signals that are lost when a nerve is cut.

Study of the AChE system focused attention on the later stages of muscle development as a time of activity of the dystrophic gene. Studies of the maturation of myosin isoforms suggest that the developmental defect is active during neonatal maturation. Different myosins progressively appear in embryonic, neonatal, and adult muscle. Bandman (14) found that embryonic myosin isoforms disappear but neonatal myosin forms are maintained in dystrophic muscle (Fig. 5).

A clue to the developmental origin of GMD is its resemblance to the complexus (hatching) muscle of the neck. Shortly before hatching, the complexus muscle greatly enlarges. Its fibers become swollen, rounded, and electrophysiologically myotonic (15). After hatching, the fibers shrink, the muscle atrophies, becoming indistinguishable from other long muscles in the neck (16). The swollen, rounded, myotonic fibers of the normal complexus muscle much resemble those of the dystrophic chicken. It is as if the developmental program of dystrophic muscles has become partly confused with that of the complexus.

Inbred lines of chickens are important in studying the relationships between the characteristics of dystrophic muscle and the genetic background of the animals. These are lines in which dystrophic chickens were repeatedly backcrossed to normal inbred New Hampshire or White Leghorn lines (5, 17), producing lines of homozygous dystrophic chickens with closely related genetically normal counterparts.

Examination of these lines revealed that gross size of the dystrophic muscle is affected greatly by background genes. Afflicted muscles from dystrophic White Leghorn chicks show early relative atrophy; they grow at rates slower than normal. Muscles from dystrophic New Hampshire chicks show early hypertrophy; they grow faster than normal and then atrophy. Muscles from crosses of dystrophic White Leghorn and dystrophic New Hampshire chickens exhibit neither relative atrophy nor hypertrophy, growing at rates similar to their genetically normal counterparts.

Several treatments affect GMD. One of the first discovered was to maintain chicks in an elevated oxygen atmosphere (18). Serum enzymes and mitochondrial activity remained normal for the several weeks the treatment was maintained. Whether or not treatments

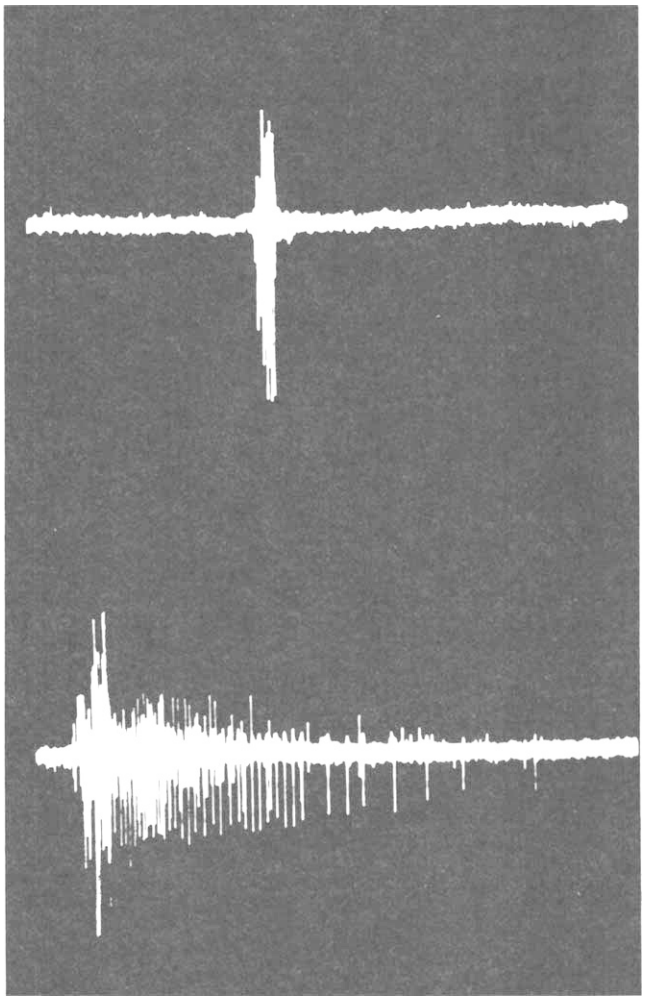

Figure 3. Electromyography of 4-week-old normal line 412 (top) and dystrophic line 413 (bottom) superficial pectoral muscle. Insertion potentials were elicited by a single monopolar electrode (figure courtesy of Dr. R. K. Entrikin; similar to data shown in Ref. 5).

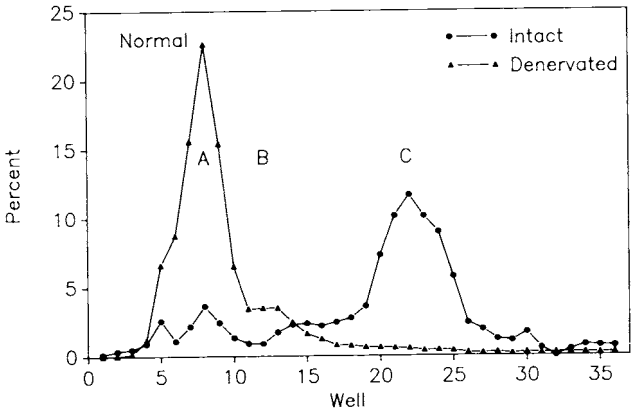

Figure 4. Multiple molecular forms of AChE in intact and denervated normal and dystrophic biceps muscles of the wing, 6-week-old muscles. The left muscle was denervated at 4 weeks and the right muscle used as a contralateral control. Homogenates were prepared as described in Ref. 12 and centrifuged in a 5–20% sucrose, 1 *M* NaCl gradient for 17 hr at 40,000*g* in an SW 40 rotor. (A) Region of 4-7S AChE forms. (B) Region of 11-12S AChE forms. (C) Region of 20S form. Activity presented as percentage of total for each gradient.

that would reduce the oxidative metabolism of the muscles could effectively reverse some of the changes of the dystrophy deserve further study. A constituent of safflower oil reported to prevent the symptoms of dystrophy of the chicken has not been isolated (19).

Recently, Ashmore *et al.* (20) have been studying the effects of passive stretch on the abnormality by placing tubes of various lengths around the growing wing. They found that stretching dystrophic muscles at an early age ameliorated the disorder, but the same treatment brought about symptoms of the dystrophy when performed on older normal birds. They proposed that rigid fiber connections between muscle fibers, as occur with aging, may play a role in the genesis of the dystrophy.

A recent series of papers by Feit *et al.* (21, 22) explore the role of collagen cross-linking in the wing stiffness associated with the dystrophy. The results provide evidence that excess collagen cross-linking may be involved in the progression of the abnormality. Park *et al.* (23) reported beneficial effects of D-penicillamine (a drug affecting collagen formation) on the dystrophy but the treatment did not bring about clear-cut improvements in GMD under other experimental conditions (24).

Other drugs that have been reported to alleviate the symptoms of dystrophy in young chicks include cyproheptadine, methysergide, and, recently, phenobarbital diphenylhydantoin (24–28). Such findings led the Muscular Dystrophy Association to support drug testing with the dystrophic chicken. The largest is a program at the University of California, Davis led by Dr. Richard Entrikin (29). Promising results have been achieved with steroids such as corticosterone-21-acetate (C21A), prednisone, and dexamethasone (30).

Treatment with steroids at an early age alleviates many of the symptoms of the dystrophy; birds are better able to right themselves when placed on their backs, AChE forms and localization become more like controls, serum creatine kinase activity is reduced, muscle lactic dehydrogenase activity increases, myotonia is reduced, and the muscle fibers become more polygonal and less swollen (Table II). To date, the most effective treatments severely reduce the rate of growth of the chicks, as if the synthesis of defective proteins plays a role in expression of the abnormality.

A group of Argentine scientists recently reported (31) that injection of a partially purified antiserum to chicken growth hormone during the first week posthatch enabled treated dystrophic chickens to right themselves more often than untreated birds for up to 9 weeks (Fig. 6). Growth rate of the animals was not affected. Righting ability of normal chickens was not decreased by the treatment; both treated and untreated birds scored at least 19 or 20 "flips." Serum creatine

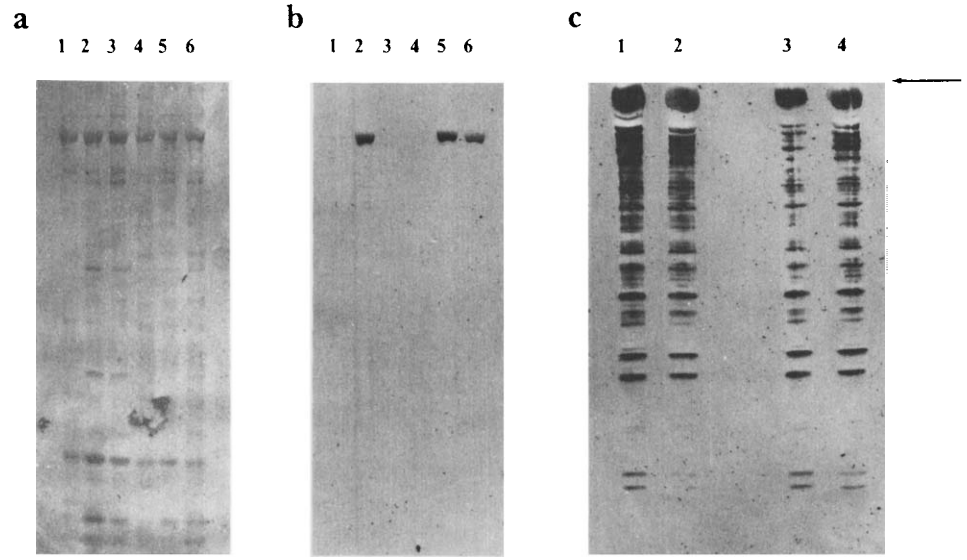

Figure 5. Western blot analysis of myosins from normal and dystrophic chicken tissue with an antibody (2E9A) reacting with myosin from superficial pectoral muscle of 20-day-old chickens but not with myosin from muscle of embryos or 1-year-old chickens. (a) Myosin stained with amido-black. (b) Myosin reacted with antibody. Myosins from 12-day-old normal embryo (Lane 1), 20-day-old normal chick (Lane 2), 1-year-old normal chicken (Lane 3), 12-day-old dystrophic embryo (Lane 4), 20-day-old dystrophic chick (Lane 5), and 1-year-old dystrophic chicken (Lane 6). Reaction with neonatal antibody occurs only in 20-day-old normal chick, 20-day-old dystrophic chick, and 1-year-old dystrophic chick muscles. (c) Myosin heavy chain peptide maps (1, 3) from 20-day-old and 1-year-old (2, 4) chicken muscle cleaved with 25 ng (1, 2) and 50 ng (3, 4) of *Staphylococcus aureus* protease and reacted with antibody (figure and caption adapted from Ref. 14.)

Table II. Genetic Muscular Dystrophy and C21A[a]

Strain	Treatment	Body weight (g)	Pectoralis (% body wt)	AChE	Lactate dehydrogenase	Creatine kinase
HYN	Control	408	6.59	283	789	958
	+C21A	222	7.12	406	749	1022
HYD	Control	402	6.24	2980	177	734
	+C21A	177	6.92	848	449	692

[a] Six and one-half weeks; 20 mg/kg C21A;1 AChE in nmol/min/g; lactate dehydrogenase and creatine kinase in μmol/min/g (adapted from Ref. (17)).

kinase activity of dystrophic chickens was not reduced at 9 weeks by the treatment, but values were not presented for earlier times, when the effect of the antiserum was maximal.

The discovery of and sequencing of dystrophin, the gene associated with Duchenne muscular dystrophy of the human and the mdx dystrophy of the mouse (32, 33), has added a new dimension to the study of muscle abnormalities. Previously, the way to establish the causal chain of events leading from a defective gene to a muscle abnormality was to work backward from the phenotypic events. Now, the road blazed by the elucidation of the gene responsible for Duchenne dystrophy may help us to work forward from a gene defect to unravel its pathologic consequences.

Dystrophin is the largest gene known, coding for almost 4000 amino acids (Table III). Antibody localization studies indicate that the dystrophin protein is on the inner face of the plasma membrane and is probably involved in maintenance of the structure of muscle fibers (34). Whether or not the gene product is defective in inherited muscular dystrophy of the chicken is not known. A recent study of dystrophin in normal chickens (35, 36) indicates that it is closely homologous to the mammal gene (Table IV). The dystrophin protein itself is absent in Duchenne dystrophy and the mdx mouse due to a deletion in the gene. Although a dystrophin protein is present in GMD (37), it is not known whether it is the same as the one in normal chickens.

Deep Pectoral Myopathy

Although study of inherited muscular dystrophy of the chicken has revealed much about the growth of broiler muscle, it is not of direct commercial importance and such single gene defects can be kept out of commercial flocks by selection. Deep pectoral myopathy (DPM) of broilers and turkeys is another matter. This polygenic abnormality of the supracoracoideus,

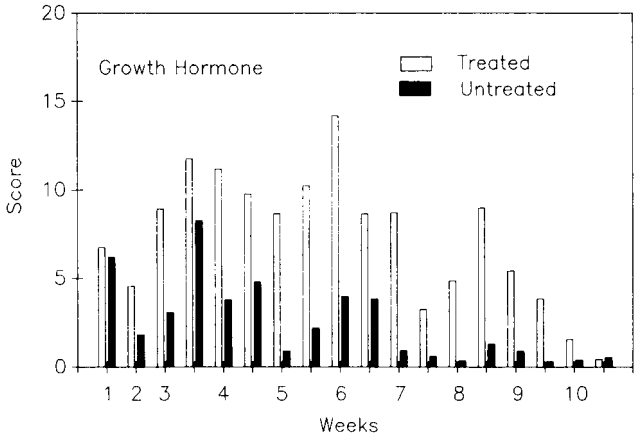

Figure 6. Effect of antiserum to chicken growth hormone on the righting ability of dystrophic chickens. Line 413 chickens were injected with antiserum during the first week posthatch, and their righting ability tested once or twice a week for 10½ weeks. Values are the average number of times chickens were able to right themselves in succession when placed on their backs (adapted from Table 1, Ref. 31).

Table III. Dystrophin (see Refs. 32–34)

Duchenne muscular dystrophy gene
Largest gene known
3685 AA, 400-kDa product
Internal face plasma membrane
Protein-absent Duchenne muscular dystrophy

Table IV. Chicken Dystrophin cDNA (see Refs. 35, 36)

N-Terminal region
(actin-binding domain of α-actinin)
80% Conservation with man
Spectrin-like domain
75% Conservation with man
C-Terminal Region
95% Conservation over 627 AA

the inferior or deep pectoral muscle, was first noted when large portions of breast muscles of birds on the production line were necrotic (38). Harper *et al.* (39) established that the disorder was multigenic and bred a line with high incidence of the muscle damage.

DPM is characterized by death of the midregion of the supracoracoideus muscle of large broilers and marketable turkeys. It is also known as "Green Muscle Disease" and "Oregon Muscle Disease." (A review of Sutherland (40) lumps it with a hereditary muscular dystrophy of the turkey (41), an atrophy of the superficial pectoralis muscle.)

A series of investigations has helped establish the structural basis for the disorder in turkeys and broilers (40–45). Anatomical studies of the subclavian vein and its role in the circulation of the muscle, occlusion experiments, electrical stimulation, and exercise studies showed that the myopathy was caused by an ischemia brought about by swelling of the muscle during exercise (40–45). The combination of an inelastic muscle fascia and a rigid sternum create a situation in which swelling of the muscle during exercise cuts off circulation to its midregion of the muscle, leading to its death.

An example is shown in Table V. Birds from the DPM-sensitive strain of turkeys bred by Harper *et al.* (39) were forced to flap their wings for short periods of time, resulting in fiber damage in more than 80% of the birds. Heterozygotes from a normal × DPM-sensitive strain cross were not afflicted.

Ultrastructural changes can be detected in as little as 15 min when broilers are induced to flap their wings for short periods of time (Table VI). Under these conditions, degeneration sets in before an hour has passed (43).

One solution to the problem is to breed turkeys with better circulation and a different breast muscle configuration. However, food preferences of the public and the economic forces of the turkey industry favor selection for large breasts and, unless steps are taken to select turkeys with better circulation to the breast muscles, deep pectoral muscles predisposed to ischemia may continue to be a problem. Indeed, Siller (45) said that the disorder was a "penalty of successful selection"; ". . .

Table V. Forced Wing Exercise and DPM[a]

Exercise	Turkey line	FWE		% Incidence
		Sec	Number	
0	DPMS 6/M	—	—	16.7
0	DPMS 5/F	—	—	0
+	DPMS 16/M	39	68	81.2
+	DPMS 17/F	39	68	88.2
+	BBB × DPMS 16/M	49	79	0
+	BBB × DPMS 16/F	38	68	6.2

[a] Twenty-week-old turkeys. Mean interval (sec) and number of flaps. Examined at 24 weeks (adapted from Ref. (39)).

Table VI. Deep Pectoral Myopathy (adapted from Ref. 43)

Sequence of Events	
Time	Changes
0	Short bout of flapping
15 min	Muscles pale, swollen, and tense
	Ultrastructural glycogen loss
	Enlarged mitochondria
1 hr	Intramuscular edema separating rounded, enlarged fibers
	Swollen sarcoplasmic reticulum and mitochondria
24 hr	Fibrous exudate
	Mid-region soft and doughy

wild turkeys and less intensely selected old commercial strains are apparently not susceptible to DPM, it is obvious that this disease is man-made."

Focal Myopathy of the Turkey

Turkeys are heir to more ailments than deep pectoral myopathy. For example, major problems are the chondroskeletal and muscular disorders known collectively as "leg weakness." Perhaps related to this is the "focal myopathy" (46) recently studied by my laboratory and Nicholas Turkeys Breeding Farm and by Dr. A. Sosnicki and R. Cassens and their colleagues at Oscar Meyer (47–49). Sosnicki *et al.* (49) histologically described degenerating muscle fibers in several muscles of marketable turkeys and studied muscles from the breeder lines (50, 51). Muscles of leg and breast exhibited rounded and opaque (Fig. 7B), degenerating and digested (Fig. 7C), irregularly shaped, and widely spaced fibers (Fig. 7D). No single fiber type seemed singled out.

We at the University of California, Davis set out with Dr. F. Shultz and Dr. B. Kelly of Nicholas Turkeys Breeding Farms to examine the idea that rapid growth increases muscle degeneration. We examined body and muscle weights, muscle histology, and serum creatine kinase of three rapidly growing selected and one slow growing unselected line of turkeys (Fig. 8). The muscles tended to grow at rates similar to those of the animals themselves; the body weights and muscles of the fast-growing male lines grew most rapidly, those of the unselected line grew more slowly.

In general, the incidence of damaged muscle and levels of serum creatine kinase correlated with age and growth rate. The faster the birds grew, the higher were the serum enzyme levels and the more damage occurred in the muscles (Fig. 9). The results support the hypothesis that a myopathy unrelated to deep pectoral myopathy is associated with rapid growth of turkey muscle. How this myopathy relates to other problems of turkeys such as leg weakness is not known. Sosnicki (personal communication) speculated that the immediate cause of the damage he found was a localized ischemia, in part because of similarities of the histologic findings with those Karpati *et al.* (52) found in experimentally induced ischemia in the rat. One possibility is that selection for rapid growth in turkeys has created muscles that outgrow their life-support systems, and bring about muscle damage when coupled with the conditions used to grow turkeys.

Imagine the following scenario: (i) Rapidly growing turkey muscle fibers outpace the growth of their connective tissue elements and blood supply. (ii) High density in the pens limits the ability of the birds to exercise, leading to a further reduction in muscle tone. (iii) The stress of capturing the birds to send them to market coupled with the other factors might create localized regions of ischemia and/or lead to loss of muscle glycogen, changes in pH, and increased protease

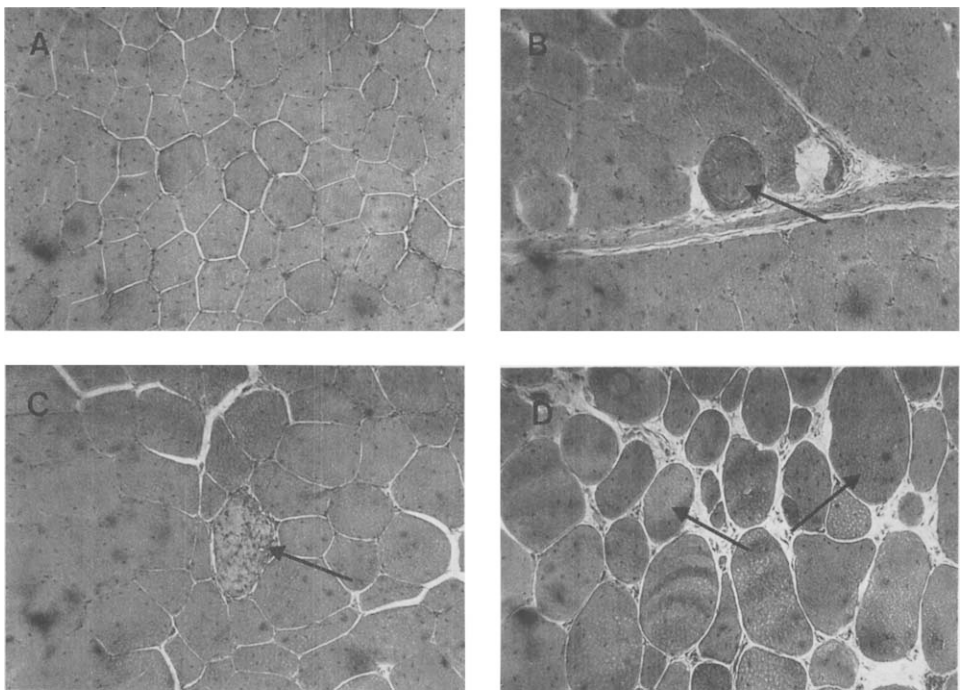

Figure 7. Frozen cryostat cross-sections of 12-week-old turkey superficial pectoralis muscle. Ten-micrometer sections were stained with hematoxylin-eosin. (A) Slowly growing line 91. Note polygonal, closely packed fibers. (B–D) Rapidly growing line 28. (B) Arrow indicates opaque rounded fiber. (C) Arrow indicates degenerating fiber. (D) Arrows indicate irregular shaped rounded fibers. Note loose packing of the muscle fibers (similar to data in Ref. 51).

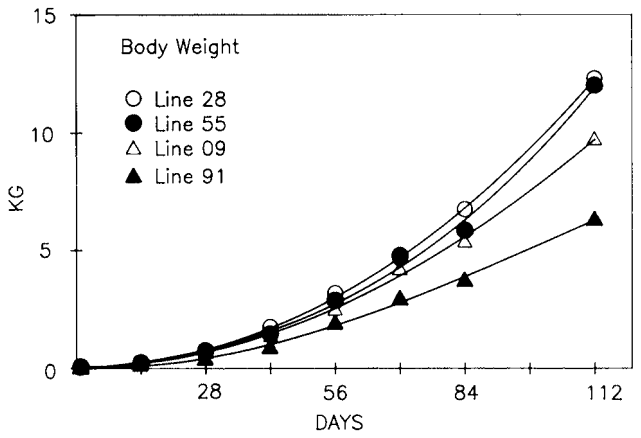

Figure 8. Increase in body weight of four turkey lines from hatching to 112 days (16 weeks). Lines 28, 55, and 09 were selected for rapid growth. Line 91 is an unselected "primitive" line (adapted from data in Ref. 51).

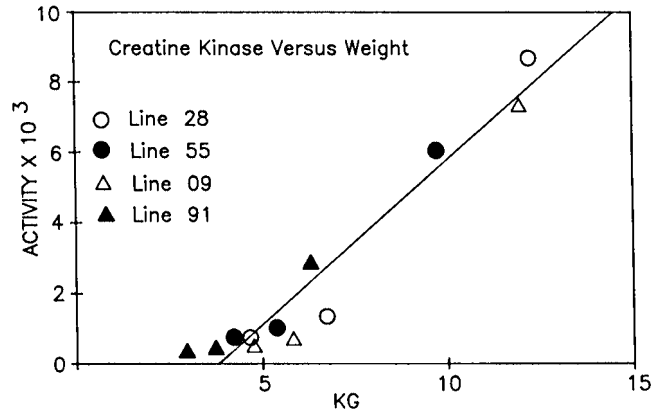

Figure 9. Relationship between plasma creatine kinase activity and body weight of four turkey lines from 12 to 16 weeks of age. Lines 28, 55, and 09 were selected for rapid growth. Line 91 is an unselected primitive line. Values are mean activities expressed as μmol/min/ml plasma (adapted from data in Ref. 51).

activity. (iv) Under such conditions the processed muscle would be low in connective tissue; the muscle fibers would be partly digested by proteases and likely to lose their usual texture.

It is interesting that there is a condition known as "capture myopathy" in which wild turkeys and mammals that are captured in one park and transferred to another arrive paralyzed and with muscle damage (53). Perhaps the turkey is genetically predisposed to such injuries.

Ferket and Sell (54, 55) recently examined the relative growth, organ composition, and extent of leg weakness in turkeys subjected to growth-limiting amino acids and compensatory growth. Consistent with the hypothesis presented here, they found that toms with severe signs of leg weakness decreased more than 60% in weight when dietary protein was restricted during early growth to at least 70% of the NRC recommendation.

The best known nutritionally caused muscle abnormality is vitamin E-deficient dystrophy. The precise mechanisms by which selenium, sulfur amino acid, and vitamin E deficiency cause muscle damage are not known (56). The morphology of the muscle and AChE pattern indicates it is not similar to inherited muscular dystrophy (57). One possibility is that it is due to a circulatory or neural problem resulting in localized regions of damage in the muscle. It is surprising that there is so little work on this disorder. The relationships between nutrition of poultry and muscle fiber growth and meat quality has not been studied to the extent it deserves. Many past studies were restricted to measuring body and organ weights and did not examine the relative growth of intramuscular and extramural protein. It is safe to predict that protein quality will play a larger economic role in the future as animals like the turkey become more important as sources of processed meats.

The Future

Biomedical Models and Economically Important Animals. The mechanisms by which the single gene defect in dystrophy of the chicken causes abnormal development of fast twitch fibers is not known. Future research focusing on the role of the dystrophin gene, establishing expression of the abnormality in cell culture, and studying the factors that regulate maturation of normal muscle are worthy of study. The fact that the dystrophic chicken has a pronounced myotonia as well as many histologic properties found in Duchenne dystrophy of the human suggest that it is a useful experimental animal in the study of specific human abnormalities.

DPM is one of a class of pathologic situations known as "compartment syndromes." One of these is anterior tibialis syndrome of "march gangrene" in which exercise by untrained or susceptible individuals leads to leg muscle damage in humans similar to that in the turkey (58).

Focal myopathy of the turkey does not seem to have a clear cut human counterpart. Its importance may be as a warning of problems to come if selection for rapid growth is continued without modification. It is possible that the damage seen in unprocessed muscles (as shown here) is indicative of more extensive problems to meat quality after processing. Switching from one scheme of selection to another takes time, and the sooner breeding is started to correct such problems the better the meat of the future will be. In the meantime, nutritional studies of the relationship between nutrient quality, growth rate, and muscle growth and composition may well offer a means to phenotypically alter the expression of a potentially dangerous genetic situation.

Many of the advances in poultry research have been accomplished by individual scientists applying

knowledge from their disciplines. Much data has been obtained when birds have been used as experimental animals for basic research funded by the organizations such as the National Institutes of Health and the Muscular Dystrophy Association. However, mounting costs of research, the interdisciplinary and increasingly molecular nature of modern research mean less and less biomedical team research will be done on economically important species and more performed on small experimental animals like rats and mice.

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