ANSC/FSTC 607
Biochemistry and Physiology of Muscle as a Food
MYOFIBRIL SYNTHESIS AND MUSCLE FIBER PLASTICITY

I. Conversion of myotubes to myofibers

A. Fusion of myoblasts → myotubes

1. Large increase in transcription, translation of myofibrillar proteins

2. Later migration of myofibrillar proteins (e.g., desmin) to Z-lines

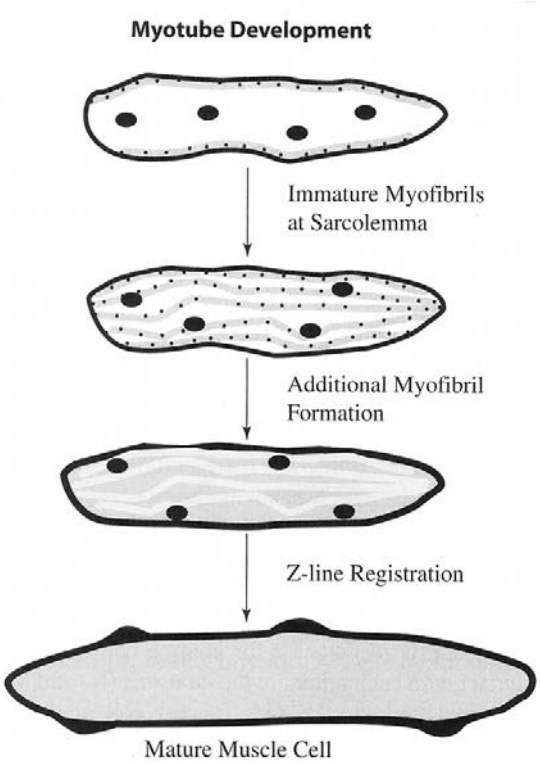
4. Cytoplasm and nuclei in core of myotube.

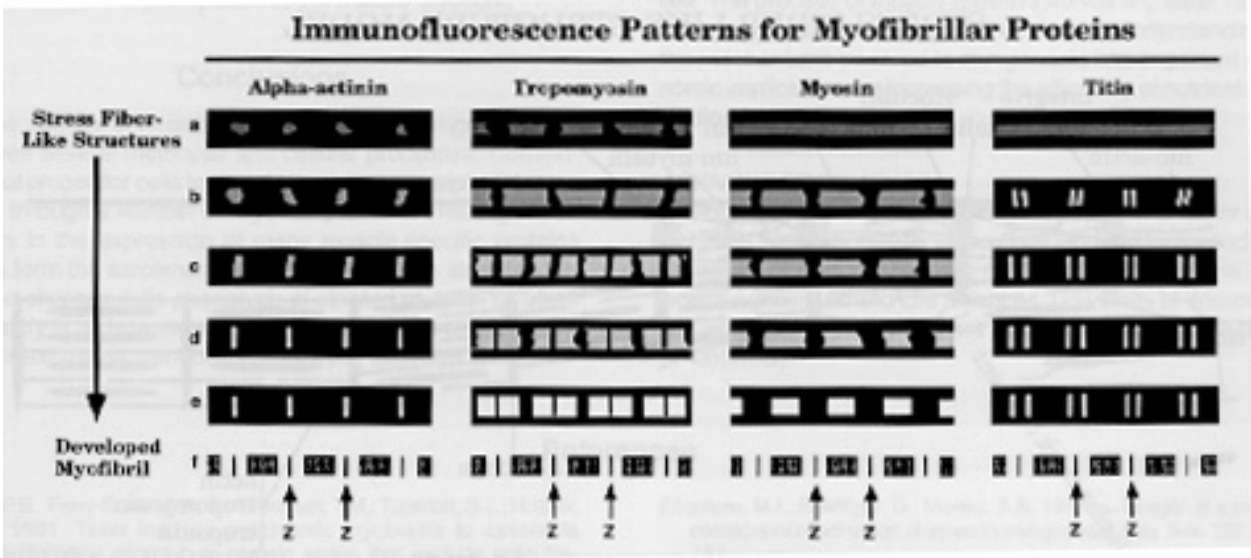
B. Formation of myofibrils

1. Aggregation of Z-line material (α-actinin) around filaments

2. Synthesis of myofilaments, no apparent development of sarcomeres

3. Synthesis of sarcomeres and appearance of striations

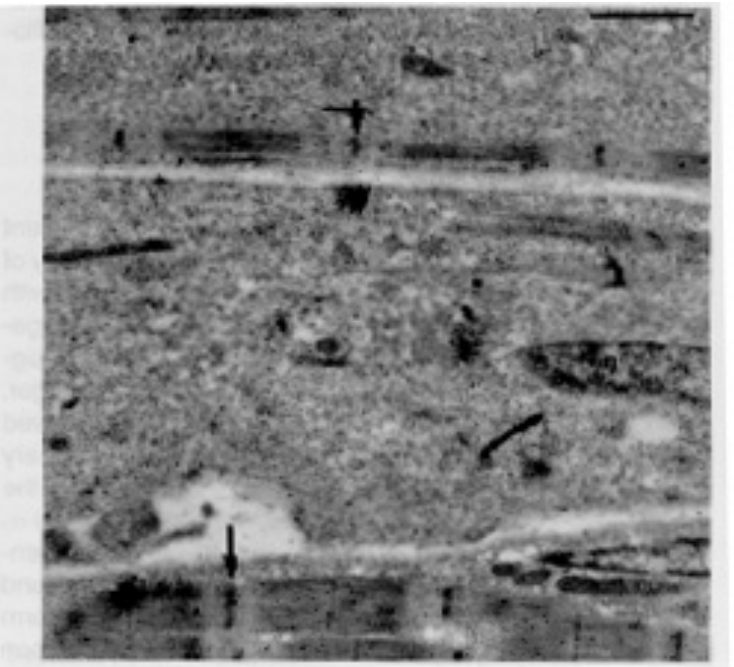




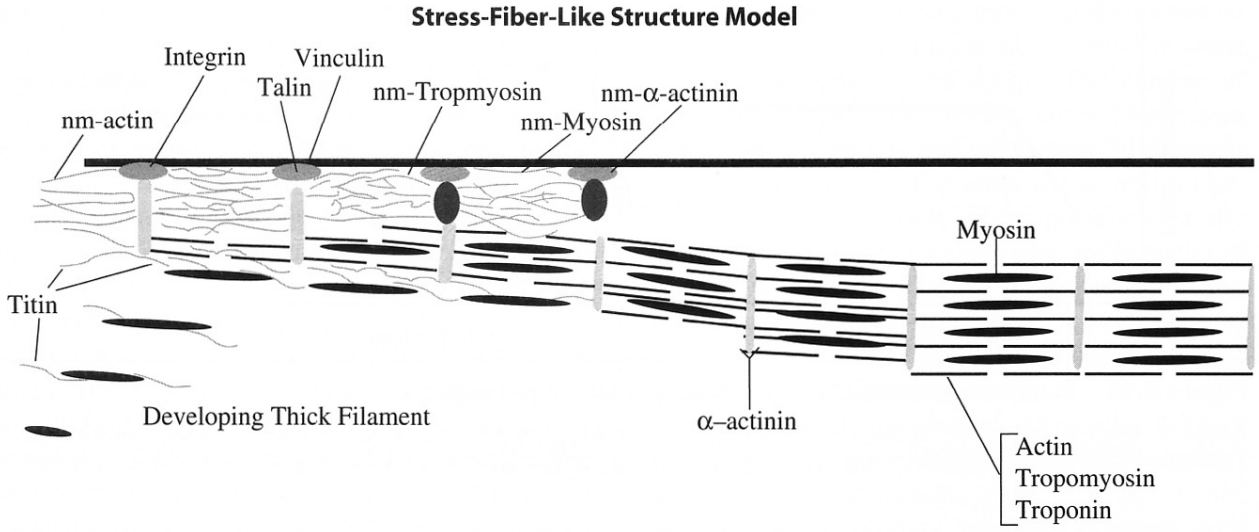
II. Myofibril assembly

A. Stress fiber model

1. A combination of nonmuscle (nm) and muscle-specific myofibrillar proteins aggregate immediately under the sarcolemma of the developing myofiber.
2. Muscle-type myofibrillar proteins and thick filaments are added adjacent to the stress fiber structure, using the original structure as a template.
3. The myofibril separates from the sarcolemma, and is added to the pool of myofibrils.
4. Sarcomere length *does not* increase in length as the myofibril develops.

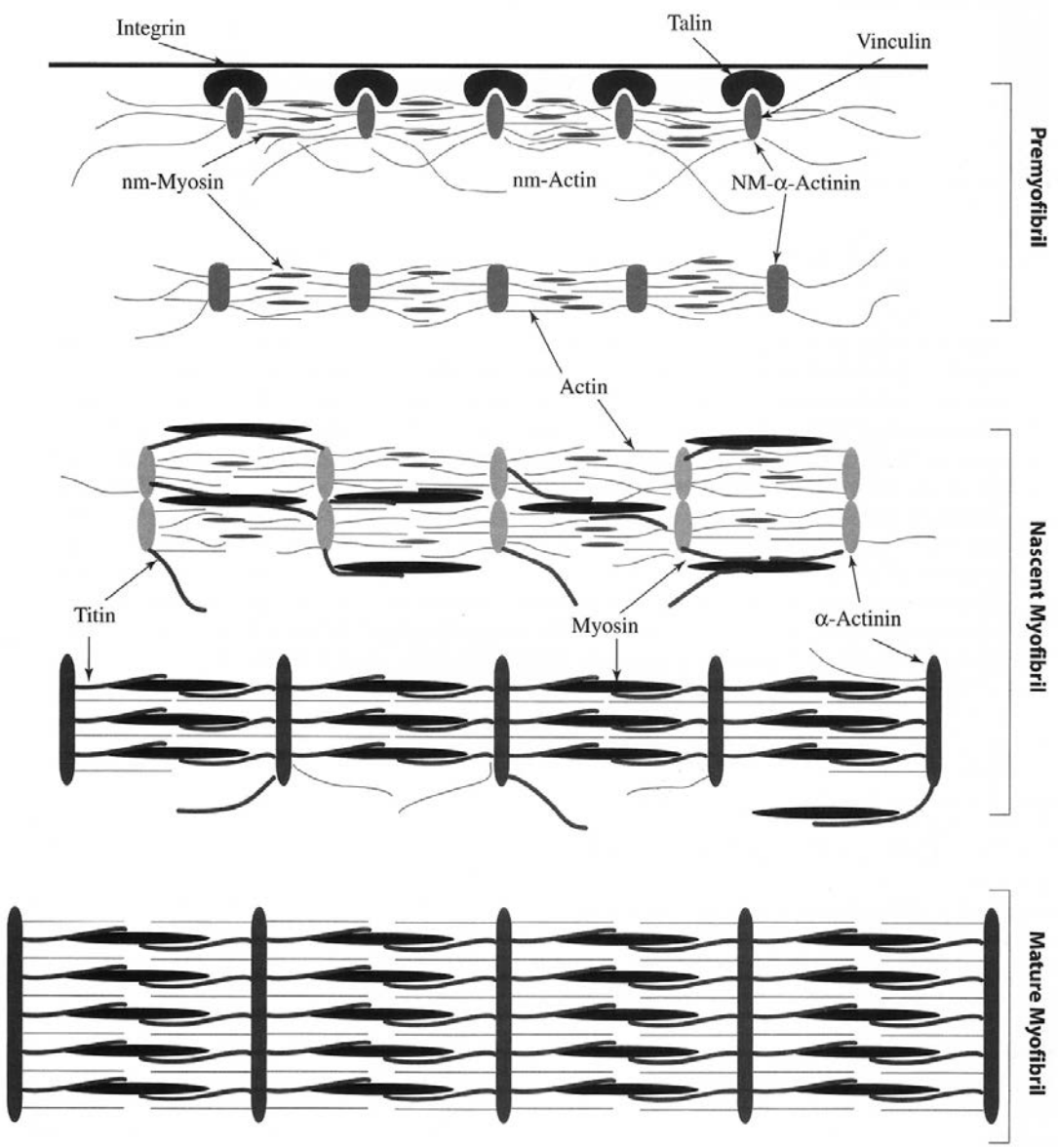


Subsarcolemmal myofibril formation in post-natal muscle. The square area marked in Figure 4 was enlarged to better illustrate the close apposition of the immature myofibril (arrow head at Z-line) to the sarcolemma in a poorly-developed myotube. This can be contrasted with the well-developed myotube in the lower part of the micrograph (arrow at Z-line). Scale is 1 micron and n = nuclei.



B. Premyofibril model

1. A premyofibril containing only nonmuscle proteins forms under the sarcolemma.
2. This detaches from sarcolemma and serves as the template for the developing myofibril.
3. Nonmuscle proteins are gradually replaced with muscle-type myofibrillar proteins.
4. There is a *lengthening* of the sarcomere, which does not occur in the stress fiber model.



III. Changes in fiber type distributions during growth

A. Type I myofibers

1. Even at birth, type I myofibers (lightly stained) make up only a small proportion of total myfibers.
2. Change in muscle mass is caused by an increase in myofiber diameter.

B. Type II myofibers

1. Most myofibers in the neonate are type II.
2. The proportion of type IIB increases and IIA decreases.

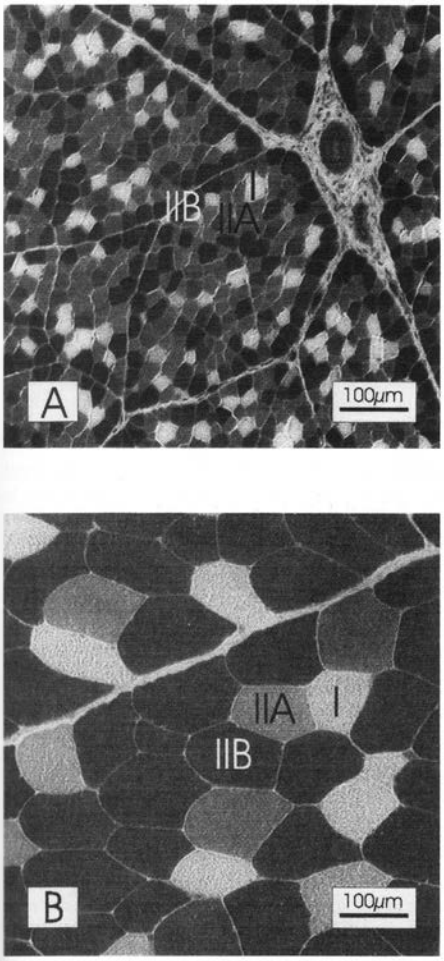


Figure 1. Cross-sections of semitendinosus muscle from (A) newborn and (B) 24-mo-old Holstein Friesian bulls. Histochemical staining of fiber types with ATPase.

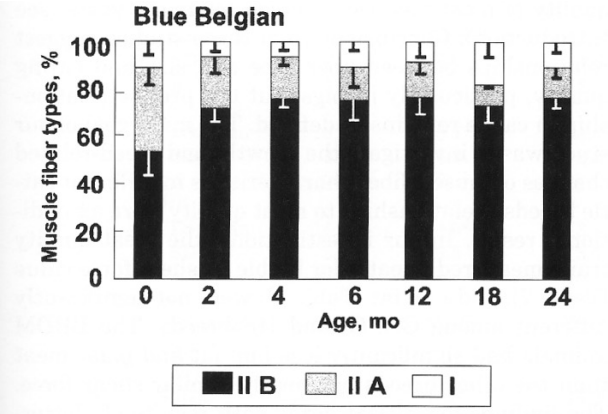


Figure 2. Changes of fiber type frequencies during growth. Results are expressed as least squares means (columns) and standard deviation (bars).

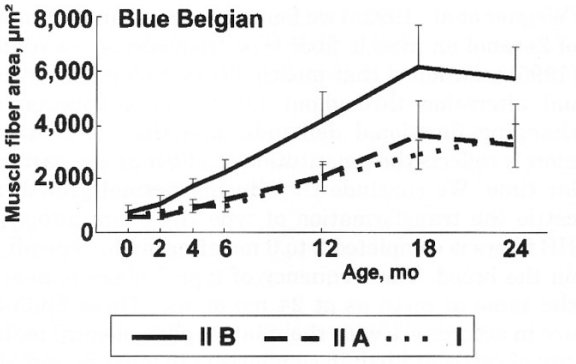
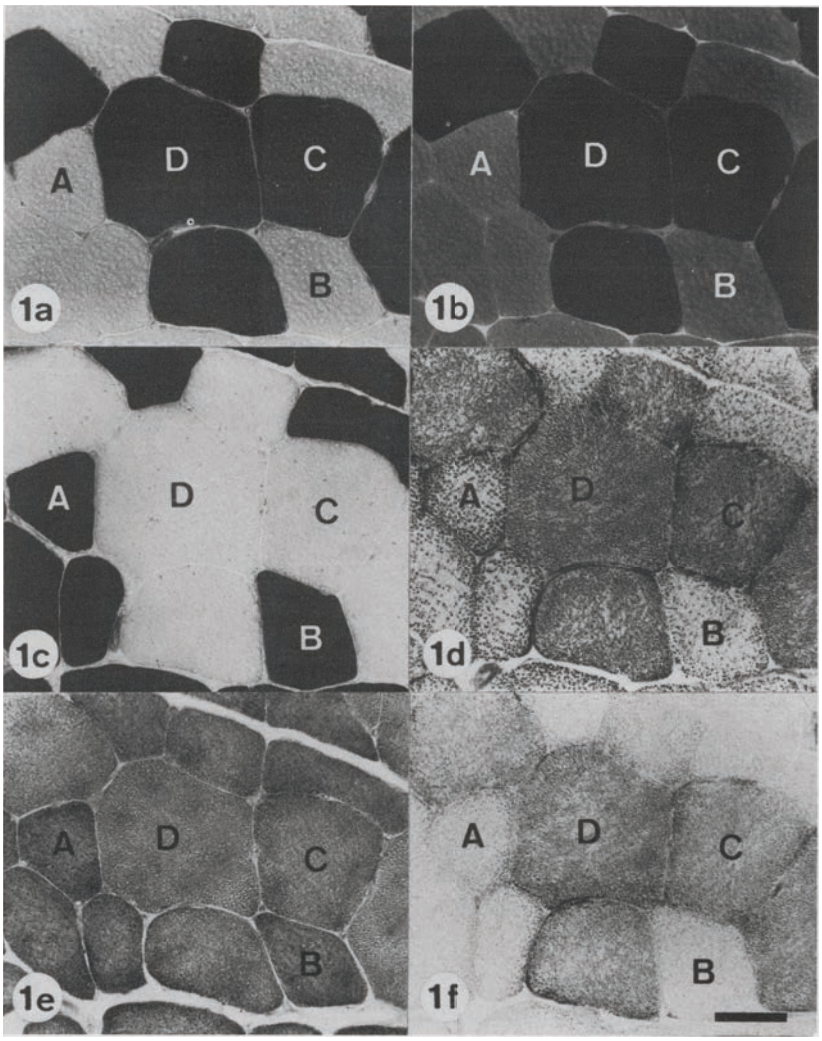


Figure 3. Changes of fiber area during growth. Results are expressed as least squares means (columns) and standard deviation (bars).

IV. Conversion of type II myofibers to type I myofibers in early postnatal muscle

A. In adult muscle, there is a clear distinction between type I and type II myofibers.

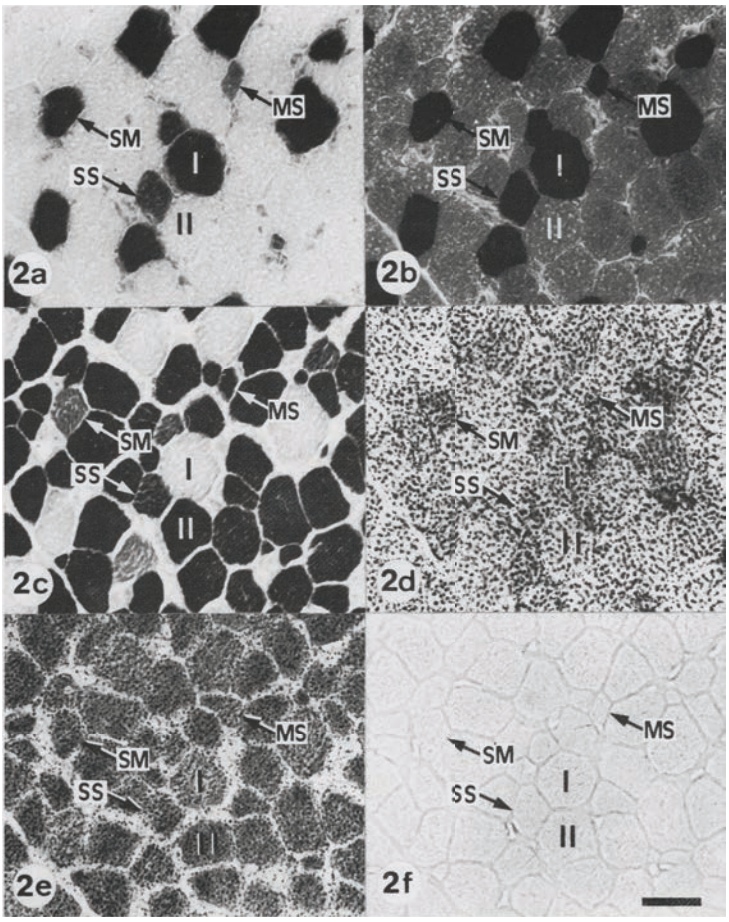
1. Type I myofibers stain strongly for acid-stable ATPase.
2. Type I myofibers stain strongly for mitochondrial activities.
3. Type II myofibers stain strongly for alkali-stable ATPase.



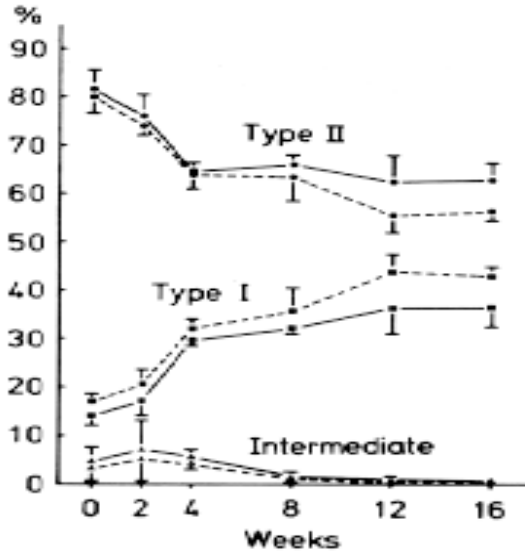
Histochemical profiles of myofiber types of the serratus ventralis thoracis muscle of adult sheep. Myosin ATPase activity after preincubation at pH 4.3 (1a), 4.4 (1b), and 10.3 (1c). NADH-TR, G-3-P, and ß-HBD (1d – 1f).

B. At birth, a small population of myofibers stains both for acid- and alkali-stable ATPase.
   1. SS = strongly staining for both acid- and alkali-stable ATPase.
   2. SM = strong for acid-stable ATPase, medium for alkali-stable ATPase.
   3. MS = medium for acid-stable ATPase, strong for alkali-stable ATPase.

C. These "intermediate" types convert to type I myofibers within weeks after birth.
   1. All "intermediate" types disappear.
   2. Type I myofibers increase in proportion.



Histochemical profiles of myofiber types of the serratus ventralis thoracis muscle 0-week-old sheep. Myosin ATPase activity after preincubation at pH 4.3 (1a), 4.4 (1b), and 10.3 (1c). NADH-TR, G-3-P, and ß-HBD (1d – 1f). Myofiber types are labeled I, II, SM, SS, and MS.



Changes in proportions of the number (solid line) and cross-sectional area (dashed line) in myofibers. The decline in type II myofibers is due to a sharp decline if type IIA (fast-twitch oxidative) to type IIB (fast-twitch glycolytic). Type IIB are completely absent at birth in this muscle.

V. Abnormal muscle growth

A. Double-muscle cattle

1. Semitendinosus and longissimus muscles contain 3.36 and 3.77 x 10^6 myofibers.
2. This is twice as many as in normal cattle.
3. The myostatin gene, which halts myotube formation, is mutated.

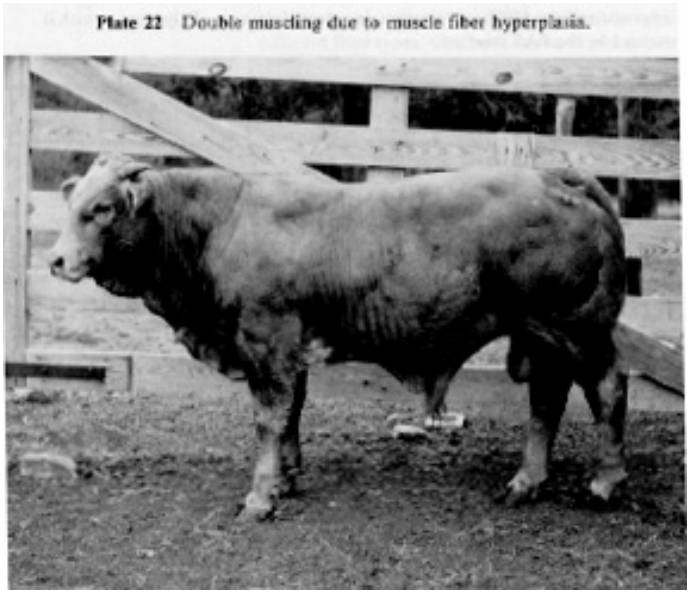


Plate 22 Double muscling due to muscle fiber hyperplasia.

B. Callipyge lambs

1. Larger than normal myofibers
2. Same number of myofibers as normal sheep.
3. Defect is not due to myostatin.