INTRODUCTION

This presentation will cover some of the recent information available on postnatal muscle growth. Three related areas will be discussed: (1) postnatal muscle growth, (2) genetic and endocrine control of muscle growth, and (3) management and nutritional factors which influence muscle growth. In the past few years, the subject of muscle growth and development has been discussed at this conference. Information covered in these reviews should be consulted for a more comprehensive coverage of postnatal growth (Zobrisky, 1965; Berg, 1968; Bailey, 1969; Cassens et al., 1969; Hegarty, 1971; and Topel, 1971).

Postnatal Muscle Growth Span

It is generally assumed among investigators, who use histometric approaches to muscle growth, that the prenatal growth of muscle is chiefly due to an increase in the number of fibers (hyperplasia) while postnatal growth is due to growth in length and circumference of already existing fibers (hypertrophy). On the other hand, researchers using chemical approaches believed that prenatal muscle growth proceeds entirely by cell division (DNA). The DNA, weight, and protein all increase proportionally resulting in a constant cell size expressed either by weight or protein per nucleus. Postnatal growth is achieved both by nuclei hyperplasia (DNA) and increase in protein/DNA ratio which is a measure of cell size (hypertrophy). Histometric hypertrophy can be understood in terms of chemical hyperplasia if it is recognized that the increase in fiber diameter is mainly due to the increase in the number of nuclei (DNA) in the muscle during the postnatal life. Therefore, while little or no hyperplasia occurs during postnatal muscle growth histometrically, there is both hyperplasia and hypertrophy of muscle cells (DNA) during postnatal growth biochemically. The use of DNA per unit in muscle is a functional concept which assumes that each nucleus within the fiber has jurisdiction over a finite mass of cytoplasm. Since muscle tissue contains only diploid nuclei, which have constant amount or DNA (about 6.2 picogram per nucleus), this DNA increment represents increases in the number of nuclei within muscle fibers.

At birth, muscle tissue constitutes 25% of human and rat body weight (Elliott and Cheek, 1968). At maturity, the muscle forms 45% of the body weight of mammals regardless of their size (Young, 1970). Therefore, there is a substantial increase in the proportion of muscle tissue during postnatal life.

Postnatal growth in rats is recognized to follow three phases of growth as put forward by Winick and Noble (1965): hyperplasia of the constituent cells, hyperplasia plus hypertrophy and hypertrophy alone. They observed that the transition from one phase to another was entirely dependent on slowing down and finally on cessation of DNA synthesis. In rat muscle, between 2 and 5 weeks of age, there is an increase of 4.5 times in mass and there is a simultaneous 3-fold increase in the total amount of DNA (Enesco and Puddy, 1964). From 5-12 weeks, there is further increase of DNA by about 50% whereas muscle weight continues to increase by a factor of 2.5. In a similar study using chickens, Moss (1968) showed that during normal muscle growth, the weight of the gastrocnemius muscle and the number of nuclei almost maintained a linear relationship (figure 1) and also that during the entire growth period studied, the mean cross-sectional area of the fibers is proportional to the total number of nuclei in the muscle (figure 2).

There is cytological evidence of a modest increase in fiber number during the early postnatal period for species such as rat and mouse (Chiakulus and Pauly, 1965; Goldspink, 1962), but the greater part of the increase in cross-sectional area of the muscle during growth is achieved by uniform increase in the diameter of the existing fibers. Postnatal muscle growth is also achieved through growth in length of the fibers. This is accomplished mainly by the addition of new sarcomeres at the end of the fibers rather than an increase in size of the existing sarcomeres (Goldspink, 1968, 1972). During this longitudinal growth, the nuclei per muscle fiber is bound to increase. However, the mechanism by which new sarcomeres are added remains to be elucidated. Legato (1970) suggested that in the cardiac muscle, the Z-disks are the centers for the production of new sarcomeres. New sarcomere formation begins with the hypertrophy of the Z-disk. The Z-disk grows, occupying similar area as the sarcomere length; at this stage the Z-substance is gradually replaced by thick and thin filaments until a new sarcomere, bordered on both sides by the formerly hypertrophied Z-disk, has been formed. More studies are needed in this area before the mechanism by which new sarcomeres are added to the ends of muscle fibers are fully understood.

Information from rats and mice indicates that postnatal muscle growth is a process of nuclear multiplication (DNA) followed by hypertrophy of the cells (protein/DNA). Fiber hypertrophy is a result of the increase in size of the constituent cell (DNA). A graph showing the increase in total DNA with age in rat's gastrocnemius, taken from studies by Gordon et al. (1966) is shown in figure 3. Total DNA rose slowly at first from 43-60 days, then rather steeply and finally reached a plateau between 80 and 90 days. In their studies, they noticed no increase in cell weight per unit DNA during the period of nuclear proliferation. However, after 90 days, hypertrophy alone continued to occur, demonstrated by the absolute rise in myofibrillar and sarcoplasmic protein content.
Fig. 1. Relationship between the number of nuclei and the weight of the pectoral muscle during growth (0-266 days). From Moss (1968)
Fig. 2. Relationship between the cross-sectional area of the fibres and the number of nuclei in the pectoral muscle during growth (0-266 days). From Moss (1968)
Fig. 3. Total DNA in quadriceps of growing rats. From Gordon et al. (1966)
The gain in total DNA represents neural, endothelial, fibroblastic, and other elements along with muscle fiber DNA. Enesco and Puddy (1964) determined that 35% of the nuclei in samples of four different rat muscles lay outside the muscle fibers in male Sherman rats at 16, 36, 86 days of age. Since the percentage of nuclei outside the fibers did not change over the span studied, an increase in DNA content even if uncorrected for non-fiber nuclei, will proportionately reflect growth in muscle cell nuclei.

**Satellite Cells and Postnatal Increase in Muscle Cells (DNA)**

The question arises as to what might be the origin of the new nuclei that appear in the muscle fibers, since nuclei within the muscle fibers have ceased to divide (Bischoff and Holtzer, 1969; Stockdale and Holtzer, 1961). Mauro (1961) describes the presence of small, mononucleated fusiform cells which he called satellite cells, lying between the basement membrane and the plasmalemma of muscle fibers. The studies of Ishikawa (1966) and Enesco and Leblond (1962) have confirmed the existence of satellite cells and their capability of mitotic division. Studies of Moss and Leblond (1971) show that satellite cells possess the ability to fuse with mature skeletal muscle cells and thereby add on more nucleus to the skeletal muscle fiber. Therefore, it is probable that satellite cells are the progenitors of new muscle nuclei during growth.

As in the rats and mice, the basic mechanisms of muscle growth in pigs appears to be similar. Staun (1963) reported that muscle development during postnatal period in pig is solely due to increases in the cross-sectional area of the fibers already existing at birth.

Muscle protein synthesis is preceded by increases in DNA and RNA synthesis. A study in skeletal muscle of pigs shows a continuous decrease in DNA concentration from time of birth indicating a relatively constant amount which is being diluted by the increasing accumulation of protein (Tsai et al., 1973). RNA concentration shows an immediate rapid increase followed by a decline to a rather constant level. The initial increase in RNA precedes the increasing protein accumulation. The changes in protein, DNA, and RNA concentration during muscle development as reported by Tsai et al. (1973) is shown in figure 4. Since RNA synthesis precedes rapid accumulation of protein and since DNA is responsible for the elaboration of all species of RNA involved in protein synthesis, RNA/DNA ratio will be a good measure of the muscle protein synthesis.

The differential muscle growth observed in different strains of pigs indicates that this may be caused by altered rate of growth of lean and/or altered duration of growth phase. Allen (1974) reported a significant difference in the rate of carcass protein and water deposition between two strains of pigs with different propensities to fatten. In another study, Hegarty et al. (1973) showed that miniature pigs have
Fig. 4. Change in protein, DNA, and RNA concentrations during muscle development. From Tsai et al. (1973)
compressed growth curve and small muscle mass and suggested that this may be due to the fact that they have fewer muscle fibers than the normals. This has the effect of shortening the time necessary for the muscle fiber diameter to reach a maximum size. Once this maximum size is reached, further increases in muscle are limited and the animal becomes fat.

From a review of the literature, it appears that as more muscular type pigs are developed, the period of muscle development is extended well beyond the conventional market weight of 90 kg. Hammond reported in 1933 that muscle growth in pigs reaches a peak around 60 kg. This is no longer true of our modern pigs. Table 1 shows the various body weights after which muscle growth is reported to decline in swine.

Table 1. Period of muscle growth in the pig

<table>
<thead>
<tr>
<th>Species</th>
<th>Period of muscle deposition</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swine</td>
<td>Birth - 75 kg</td>
<td>Hammond (1933)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 79 kg</td>
<td>Hiner (1971)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 82 kg</td>
<td>Allen (1974)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 91 kg</td>
<td>Richmond and Berg (1971)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 105 kg</td>
<td>Powell and Aberle (1975)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 130 kg</td>
<td>Doornenball (1971)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 136 kg</td>
<td>Witte and Stringer (1969)</td>
</tr>
<tr>
<td></td>
<td>&quot; - 194 kg</td>
<td>Gobble et al. (1975)</td>
</tr>
</tbody>
</table>

It would seem that as more muscling is being selected for in pigs, more time is needed for the muscle to reach its potential for growth. This is further borne out by recent studies by Powell and Aberle (1975) in which growth in low and high muscle pigs were compared. They found that while the weight of biceps femoris and semitendinosus muscle almost leveled off in low muscle pigs by the age of 145 days (68.6 kg); the high muscle pigs continued increasing up to the age of 210 days or 105 kg when the experiment was terminated. It is interesting to note that muscle DNA concentration (μg DNA per g of tissue) was slightly higher in high muscle pigs but was not statistically significant. However, at 105 and 145 days high muscle pigs have significantly higher muscle DNA concentrations. Total DNA, RNA and protein increased markedly during growth up to 210 days of age and are consistently higher in the high muscle pigs. Therefore, the rate and duration of DNA proliferation during postnatal muscle growth may be a more reliable determinant of potential for muscle development. In another experiment by Witte and Stringer (1969) in which two groups of hogs were fed to heavy weights up to 300 lbs., it was shown that meat type hogs can be taken to heavier weights without substantially increasing fat trim or decreasing the
percent four lean cuts (table 2). The small changes in percent lean cuts and percent fat trim indicates that these hogs have the capacity for growth of lean tissue and have not reached the point where weight is increasing principally by the production of fat.

Table 2. Means of percent four lean cuts of the cold carcass by experiment and weight group

<table>
<thead>
<tr>
<th>Weight group</th>
<th>Experiment I</th>
<th>Experiment II</th>
</tr>
</thead>
<tbody>
<tr>
<td>220</td>
<td>50.45(^{a})</td>
<td>52.94</td>
</tr>
<tr>
<td>240</td>
<td>50.32(^{b})</td>
<td>51.42(^{a})</td>
</tr>
<tr>
<td>260</td>
<td>49.16(^{c})</td>
<td>51.65(^{a})</td>
</tr>
<tr>
<td>280</td>
<td>48.61(^{b})</td>
<td>51.01(^{a})</td>
</tr>
<tr>
<td>300</td>
<td>48.01(^{b})</td>
<td>50.56(^{a})</td>
</tr>
</tbody>
</table>

\(^{a,b}\) All percentage means which bear common superscripts do not differ significantly (\(P < .05\)). From Witte and Stringer (1969).

Genetic and Endocrine Control of Muscle Growth

Evidence for genetic control of muscle growth can be found in laboratory animals (Zucker and Zucker, 1961; Luff and Goldspink, 1967; Robinson and Bradford, 1969) as well as in the pig (Topel, 1971) and cattle (Bendall and Vogle, 1967; Ouhayoun and Beaumont, 1968). Our observations have been mainly in the pig and laboratory animals. In the pig we have made preliminary studies in the Yorkshire and Ossabaw pig (Martin et al., 1973). Laboratory animals used include mice selected for postweaning growth rate (Bradford, 1972) and Zucker obese rat (Zucker and Zucker, 1961). These animals represent extremes in the pattern of growth and growth rate and in the composition of body gain. As such they offer us the opportunity to determine the key regulatory mechanisms or factors influencing muscle growth and development.

Ossabaw vs. Yorkshire Pig. Muscle development in the Ossabaw (obese) pig is inferior to that of the Yorkshire pig (lean). Data recently reported (Ezekwe and Martin, 1975) showed that the smaller semitendinosus muscles of the obese pig was lower in total DNA and had a lower RNA to DNA ratio indicating inferior capacity to synthesize muscle protein. We are in the process of determining fiber number and size to characterize the mechanism by which the muscle growth differential is achieved in these animals. While these types of measurements are helpful in seeing what changes are taking place in muscle tissue itself, a more integrated look at other physiological processes which may influence the development of muscle is also underway. The
metabolic pattern (tissue enzyme levels and in vitro tracer studies) has been described (Martin et al., 1973, and Martin and Herbein, 1975). This will be summarized later. The endocrine status is known to influence muscle growth hormone, insulin and glucose tolerance (Wangsness and Martin, 1974; Wangsness and Martin, 1975). Insulin release in response to arginine is elevated in the obese pig while growth hormone release is depressed. These hormone changes are compatible with the observed changes in tissue growth. Insulin is known to increase adipose tissue mass (Wagner and Scow, 1957) and growth hormone injections increase muscle size and decrease adipose tissue weight (Engel and Kostyo, 1964). Further work is needed to provide adequate proof that the genetic difference for muscle growth in these animals is mediated through the regulation of hormone secretion.

Zucker Obese Rat. This animal model develops spontaneous obesity in which energy is diverted from muscle tissue to adipose tissue for storage in the form of fat. The muscle development is impaired and spontaneous activity is decreased. Energy and nitrogen balance studies indicate that the efficiency of protein utilization is decreased and energy storage is increased (table 3). We have not found any differences in total muscle DNA content or fiber number (Martin, 1975). Therefore, it appears that the block in muscle protein deposition occurs somewhere between RNA synthesis and the final synthesis of protein.

Table 3. Energy and nitrogen balance in pair-fed Zucker obese rats

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein intake*</td>
<td>94.0 ± 4.0</td>
<td>93.8 ± 4.1</td>
</tr>
<tr>
<td>Protein gain*</td>
<td>21.9 ± 1.6</td>
<td>8.0 ± 0.9</td>
</tr>
<tr>
<td>Energy intake**</td>
<td>2073 ± 87</td>
<td>2050 ± 89</td>
</tr>
<tr>
<td>Energy gain**</td>
<td>282 ± 17</td>
<td>627 ± 38</td>
</tr>
</tbody>
</table>

* g of protein over 4-week period
** kcal over a 4-week period

The overall pattern for nitrogen utilization in the animal that has impaired muscle growth and improved fat deposition is shown in figure 5. From our studies, it appears that amino acids normally utilized for muscle protein synthesis is diverted to the liver for catabolism. In the liver, carbon skeletons are used for either glucose synthesis or fatty acid synthesis.
Figure 5. Protein metabolism in animals with inferior development of muscle tissue. There is an apparent shunting of amino acids away from muscle protein synthesis and toward greater amino acid catabolism by the liver.
Table 4. Immunoreactive growth hormone and insulin levels in genetically lean and obese rats

<table>
<thead>
<tr>
<th>Animals</th>
<th>Growth hormone (ng/ml)</th>
<th>Insulin (μU/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Study I</td>
<td>Study II</td>
</tr>
<tr>
<td>Lean (6)</td>
<td>284 ± 68</td>
<td>240 ± 71</td>
</tr>
<tr>
<td>Obese (6)</td>
<td>31 ± 12</td>
<td>81 ± 36</td>
</tr>
</tbody>
</table>

Growth Strain Mice. The selection for rapid post-weaning growth rate results in an increase in mature body size (Bradford, 1971). Our studies indicate that this selection procedure results in greater muscle size and increased fiber number, fiber size, fiber length, and total DNA content of the semitendinosus (table 5). Other workers have found similar results with mice selected for large body size versus small body size (Hanrahan et al., 1973). It should be noted that not all strains selected for rapid postweaning growth rate respond in the same manner. It has been shown that in three separate families, all selected for rapid postweaning growth rate, two show an increase in body fat and one shows no significant change in body fat (John White, Personal communication). Therefore, a comparison of these animals in which genetic selection has increased growth rate and had a differential effect on body composition would provide fundamental data on the interaction of muscle and adipose tissue growth. A preliminary study of insulin levels in these mice is shown in figure 6. It can be seen that in the animals that have increased growth rate and fat content (H₁ and H₂), serum insulin levels are elevated whereas in animals with increased growth rate and no change in composition of gain (H₄), insulin levels are only slightly increased.
Figure 6. Serum insulin levels in mice with varying propensities for growth rate and fat deposition.
Table 5. Body weight, muscle weight, and physical characteristics of skeletal muscle from growth and control strains of mice (Ezekwe and Martin, 1975)

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Strain</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Growth</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>Body weight (g)</td>
<td>51.4 ± 2.1a</td>
<td>38.9 ± 1.7</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>Muscle weight (mg)</td>
<td>126.7 ± 8.4b</td>
<td>96.9 ± 7.6</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Muscle length (mm)</td>
<td>19.9 ± 0.4</td>
<td>17.1 ± 0.4</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>Fiber diameter (μ)</td>
<td>54.8 ± 0.9</td>
<td>45.7 ± 0.8</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>Fiber number (X10³)</td>
<td>16.8 ± 0.4</td>
<td>12.8 ± 0.9</td>
<td>&lt;.005</td>
</tr>
</tbody>
</table>

Values represent the mean ± SEM of 7 animals.

Nutritional and Management Practices Which Influence Muscle Growth

Obviously, nutritional adequacy is required for optimum expression of genetic potential. However, because of the cost of certain feed ingredients, marginal diets are fed to economize on existing market situations. Pigs fed a high protein diet from weaning to market weight will develop a higher proportion of lean cuts (Smith et al., 1967). By reducing the protein content in finishing rations, the producers realize an economic advantage. This practice is also based on the fact that the energy value of the protein deposited as a percentage of total caloric intake decreases with age. This is caused by a decrease in the ability of swine to retain nitrogen with increasing age. The ability to retain nitrogen over longer periods of time is under genetic control as noted earlier. Consequently, the level of dietary nitrogen fed should be recommended on the basis of genetic potential for postnatal muscle growth. If larger market weights for swine are desirable for more economical production of lean meat, perhaps a longer period of feeding a high protein diet would be beneficial.

The influence of early nutritional experiences on growth and development has received considerable attention in recent years. The permanent changes in tissue cellularity caused by altering neonatal nutrition have been extensively investigated in the rat (Chow and Lee, 1964; Winick and Noble, 1966; Knittle and Hirsch, 1968). But little information on this subject is available in the pig (Pond, 1973).

Nutritional stress imposed after birth affects the tissues in relation to the phase of cellular growth of the tissue at the time. There is permanent stunting of the tissues in which hyperplastic growth is still taking place (Winick and Noble, 1966). The musculature is one of the later developing tissues and, therefore, may be affected by
Fig. 7. Summary of interaction of factors affecting postnatal muscle growth.
nutritional deprivation imposed during cellular hyperplasia. Robinson (1969) reported that undernutrition during pregnancy does not affect muscle cell number in the pig while stress during pregnancy and lactation caused muscle cellular hyperplasia to terminate much earlier than in the controls. In similar studies, Gilbreath and Trout (1972) showed that the weight of longissimus dorsi and its protein content did not differ when nutritional restriction is imposed on pigs before weaning. However, extending the restriction period to two weeks or four weeks post-weaning, resulted in significant decreases in muscle weight and protein content. When the pigs were repleted, significant decreases in muscle weight and muscle DNA were observed in all restricted pigs. However, the pigs were sacrificed at 12 weeks of age, and this probably does not allow enough time to determine if the stunting was permanent or reversible. Recently, we reported a study to determine the effect of neonatal nutritional treatments on the carcass composition and tissue cellularity of pigs and the reversibility of these responses after a period of realimentation. When pigs were fed a low energy milk diet the first four weeks, Semitendinosus muscle growth was not affected by either low protein or low energy rations. The RNA/DNA ratio was lowest for the low energy group indicating lower rates of protein synthesis. In another study pigs from small and large litters sacrificed at seven weeks of age showed a significant difference in muscle weight and muscle DNA and RNA (table 6). By six months of age, these effects were reversed. Carcass evaluations by the PSU Meat Lab revealed no significant differences in carcass characteristics. The ability of the muscle from porcine animals to recover from neonatal nutritional stress was in contrast to the findings in rodents which indicate that preweaning malnutrition has a permanent effect on muscle growth (Winick and Noble, 1966; Review by Trenkle, 1974). The difference between muscle response to early nutrition in pigs and mice may be due to the relative immaturity of muscles in rodents. The muscle fiber numbers in rats and mice have been shown to increase postnatally while no such increase has been shown in pigs. Also muscle DNA has been shown to increase in pigs up to 100 days of age (Robinson, 1969) and 210 days (Powell and Aberle, 1975). Therefore, nutritional stress of longer duration may be needed to cause an impairment in muscle cellularity in pigs.

Exercise

Physical exercise leads to muscle hypertrophy while inactivity causes decreased muscle growth. Christensen and Crampton (1965) have shown that in one-year old rats, forced exercise during the year causes an increase in DNA, RNA and protein in the gastrocnemius muscle. Exercise has been shown to increase muscle diameter (Walker, 1966; Carrow et al., 1967; Goldspink, 1970). The effect, however, depends on the type and duration of exercise. Goldberg (1969) found that work-induced muscle hypertrophy reduced the rate of protein catabolism which permits greater protein accumulation. We have found that exercise increases the protein deposited in animals that have a decreased capacity for muscle growth (table 7). A more detailed discussion of
Table 6. Effects of litter size on organ weights and tissue metabolic characteristics of pigs at 7 weeks of age

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Large litter</th>
<th>Small litter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>14.3 ± 0.6</td>
<td>23.6 ± 2.3**</td>
</tr>
<tr>
<td>Liver weight (g)</td>
<td>370.0 ± 55.0</td>
<td>528.0 ± 78.0**</td>
</tr>
<tr>
<td>Kidney weight (g)</td>
<td>31.6 ± 1.5</td>
<td>52.8 ± 3.6**</td>
</tr>
<tr>
<td>Muscle weight (g)</td>
<td>52.9 ± 3.8</td>
<td>90.5 ± 11.4*</td>
</tr>
<tr>
<td>Muscle DNA (g)</td>
<td>35.6 ± 1.8</td>
<td>56.2 ± 4.8**</td>
</tr>
<tr>
<td>Muscle RNA (g)</td>
<td>50.3 ± 4.1</td>
<td>89.2 ± 6.6**</td>
</tr>
<tr>
<td>Adipose soluble protein (mg/g tissue)</td>
<td>13.0 ± 1.6</td>
<td>10.7 ± 0.8*</td>
</tr>
<tr>
<td>Adipose tissue enzymes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Citrate cleavage enzyme</td>
<td>57.2 ± 15.6</td>
<td>103.0 ± 14.9*</td>
</tr>
<tr>
<td>Malic enzyme</td>
<td>332.8 ± 29.9</td>
<td>116.6 ±116.6**</td>
</tr>
</tbody>
</table>

* (P < .05).
** (P < 0.1), significantly different from control values.
From Martin et al. (1974).

Table 7. Effects of exercise on body composition

<table>
<thead>
<tr>
<th>Experimental group</th>
<th>Protein</th>
<th>Lipid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lean</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exercise</td>
<td>22.8 ± 0.34a</td>
<td>6.4 ± 0.49a</td>
</tr>
<tr>
<td>Exercise</td>
<td>23.5 ± 0.23a</td>
<td>4.9 ± 0.41a</td>
</tr>
<tr>
<td>Obese</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exercise</td>
<td>14.8 ± 0.28b</td>
<td>39.0 ± 0.81b</td>
</tr>
<tr>
<td>Exercise</td>
<td>17.3 ± 0.62c</td>
<td>27.7 ± 1.43c</td>
</tr>
</tbody>
</table>

1 Mean ± SEM for six rats in all groups.
a,b,c Means in the same column with different superscripts are significantly different (P < 0.05).
this subject will be presented later in this meeting. The adoption of
exercise is limited because of the reduced feed intake accompanying
such forced exercise. Morrison et al. (1968) reported that exercised
pigs gained at a significantly slower rate as a result of decreased
feed intake and use of energy for exercise.

SUMMARY

Postnatal muscle growth occurs in mammals between birth and
maturity. The timing of various events taking place during muscle
growth depends on the animals potential for muscle growth.

Growth in number of muscle fibers does not seem to contribute very
significantly to the increase in muscle size; however, this may contrib-
ute to growth in rodents, postnatally. Growth by addition of new
sarcomeres to the ends of the fibrils plays a vital role in muscle growth
and this may continue as long as new nuclei are being added to the muscle
fibers. The increased cellular hyperplasia is followed by increment in
RNA and protein. The muscle nuclei proliferation appears to be the
factor which determines the weight or age at which muscle growth stops.
The functional DNA unit is influenced by nutrition, hormones, genetics,
and exercise. Data presented here support the concept of interactions
between genetic and endocrine control and need for "optimal" nutrition
for maximum growth performance. Because of different potential for
muscle growth possessed by different species of livestock, it may be of
economic advantage to give a longer feeding period to the animals with
greater ability for muscle growth. This may mean that animals with
high potential will be maintained longer on the farm than is presently
practiced.

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Dale Zinn: Thank you, Roy. Are there any questions you'd like to ask of Dr. Martin?

Gene Allen: Do you have studies on cellularity of the VPI mice on the fat cells?

R. G. Martin: We don't have any cellularity data yet on these mice. We do have some on the ones from California. I guess you are aware of that. But we have the animals and we have some muscle tissues that are in fixative and some that are in the process of being analyzed for DNA this week. Those animals are very interesting in that they had segregated into two different groups. They both had a high growth potential, but one of them had the potential for fat deposition and the other one for lean deposition. This was under conditions where those mice were selected for a parameter that we are familiar with in animal production practices.

C. E. Allen: What is the basis of the selection? What was the cause of the segregation?

R. G. Martin: This isn't the point. They were always selected for the post-weaning growth rate between three to six weeks of age. It was just circumstances that allowed one family of mice to develop a greater fat deposition than other ones.

J. D. Kemp: In your statement you said that it was wasteful to feed extra feed to animals to get fat, yet some of the processors are telling me now that they don't have enough fat for their processed meat. Do you think we are in danger of getting a population of animals too lean?

R. G. Martin: No!

D. W. Zinn: I think you have a point. At some point in time, we may have circumstances whereby we really don't have enough fat to mix with the lean meat for processed meats, right? Now, I have heard some concern at times that this is true, but I think that we may have a distribution problem.

R. G. Martin: I think that if we consider the changes that have been made in swine, that perhaps there is some indication that part of the stress problem we have in terms of PSE and PSS may be related to
the extremely lean type pig. So possibly, when we were selecting for
that we were not considering the quality aspects as we were making these
changes. And this will be in part the reason for a rather large percents-
tage of this type of pig in today's population.

J. D. Kemp: The reason for my comment, I was talking just this
week to a representative of one of the largest sausage manufacturers.
He said that we are now making wiener's with 5% less fat than formerly
cheaper than they could if they put the 29 to 30% fat in them. They
were looking everywhere and scraping the barrel to get fat to go in
their hot dogs and they weren't able to find it.

R. G. Martin: I don't know where all of these millions of pounds
of fat that are being produced are all going.

J. D. Kemp: The other day we got a phone call from one of the
major meat processors who said that the wholesale price of 50-50 beef
trim in pounds exceeded the price of 70-30 trim.

D. W. Zinn: I think that the situation we're in right now, at
least from the standpoint where I can see it, is that we just have less
fed cattle going to market in relation to the total kill. And we may
be in a shortage of excess fat for processing at the present time. And
we may be in that situation for quite a while until the feedlots crank
back up again. Other questions?

Unidentified: Question not recorded.

R. G. Martin: I think we showed an input level that would be
active for muscle growth. I think the problem with the high level of
insulin becomes a different state where the emphasis is shifted from
muscle growth to adipose tissue growth. But after you reach the required
level of insulin for muscle growth and go beyond that, there is a shift
in the utilization of nutrients. But I want to comment too, that these
are only two of the hormones that we have measured and characterized.
And we haven't done that extensively, but we are looking at several
other growth factors. But the tendency is to shift the metabolic
picture. I don't think I'd like to hang my hat on just insulin as a
regulator of protein and fat deposition. Other than that, it is
required for basal metabolic rates.

Unidentified: Question not recorded.

R. G. Martin: All right, now let me explain that, too. That's a
good point, I'm glad you asked about that. We had really extremes in
muscle growth. I don't think that you achieve this in many of the
animals that you are working with in production processes. I'd like
to point out, too, that we have assays now for a hormone that's been
looked at mostly in patients that are dwarfs. It's called somatamine
and we feel that perhaps some of the lack of correlation between growth
hormones and growth rate isn't due to the fact that intermediate in the
action of growth hormone may be altered. The Lorain dwarf is a good
example; they have normal levels of growth hormone in this dwarf but
the somatamine levels are depressed. And they feel that perhaps
somatamine is a very important regulator of growth.

Unidentified: Finally, what do you think the primary defect is
in these obese animals? Do you think it's the misutilization of amino
acids in muscle or is it the induction of higher catabolic enzymes in
the liver? You can't get them into muscle for growth. So, what do you
think the primary defects are?

R. G. Martin: That's a very good question, I think we have deter-
mined that with the techniques that we have. Because we have to
identify these metabolic changes early in the development process.
In the pigs, we think it's a multiple gene effect. So I don't think
we're going to find one primary event like we would in the leuco rat
that has only one metabolic or only one potential lesion due to genetics.

D. W. Zinn: I would like to take this opportunity to thank the
members of the Meat Animal Growth and Development Committee that are
listed in the back of the program, and particularly the first two
speakers we have had on this program. Harold, for you taking time out
of your very busy schedule to prepare the perspective and throughout
for your time and effort for developing your particular program. We
will now proceed to the papers by Drs. Althen and Fox.