A CHARACTERIZATION OF MYODEGENERATION

SYNDROME IN PORCINE MUSCLE

By

STANLEY NATHAN FALK

Bachelor of Science

University of Arkansas at Little Rock

Little Rock, Arkansas

1970

Submitted to the Faculty of the Graduate College of the Oklahoma State University in partial fulfillment of the requirements for the Degree of MASTER OF SCIENCE May, 1972



OKLAHOMA STATE UNIVERSITY IRPARY

SEP 1 1972

A CHARACTERIZATION OF MYODEGENERATION

SYNDROME IN PORCINE MUSCLE

Thesis Approved:

ichan Thesis Adviser in 11 66

Dean of the Graduate College

ACKNOWLEDGEMENTS

The author wishes to express deep appreciation to Dr. R. L. Henrickson, Professor, for his encouragement, patience, guidance, and assistance throughout the course of this study.

Appreciation is also extended to Dr. R. J. Panciera and Dr. C. V. Maxwell for their advice, assistance, and suggestions in the preparation of this manuscript, Mr. Curtis Kastner for his assistance, advice, and most importantly his friendship, Mr. Leroy Kimbrell, Mr. Albert Rutledge and Mr. J. L. Marsden for their help during the slaughter operations, and Mr. R. W. Givens for his assistance with the histological study.

A very special thank you goes to my parents for their faith, encouragement and love; and to my wife, Terrie, whose sacrifices, tears, and abiding love have made this possible.

TABLE OF CONTENTS

Chapter	Page
I. INTRODUCTION	I
II. REVIEW OF LITERATURE	3
Muscle of the Normal Animal	3
Fiber Types	3 4 5
Metabolism	7 9
Muscle of the Abnormal Animal	11
Myodegeneration Syndrome and Pale, Soft, Exudative Pork Environmental Influences. Electrical Stimulation. Hormonal Influences Fiber Distribution. Metabolism. Ultrastructure. Muscle Quality. Slaughter. pH Histochemical Procedure. Histological Samples	11 13 15 16 20 21 24 28 30 30 31 31 31 31 33
Fiber Diameter Degree Rigor (Percent Kinkiness) Carcass Evaluation Carcass Evaluation Percent Moisture Ether Extract (Percent Fat) Shear Force Analysis of Data IV. RESULTS AND DISCUSSION.	33 34 34 36 37 37 38 38
pH	39 41

TABLE OF CONTENTS (Continued)

Chapter	Page
Carcass Evaluation	44 47
V. SUMMARY AND CONCLUSIONS	65
LITERATURE CITED	68
APPENDIX	75

- 4

LIST OF TABLES

.

.

Table		Page
I.	Procedure for Calculation of Percent Kinkiness	35
II.	Listing of Parameters Measured for Animals With Low Initial pH	76
III.	Listing of Parameters Measured for Animals With Intermedi- ate Initial pH	77
IV.	Listing of Parameters Measured for Animals With High Ini- tial pH	78
V .	Listing of Averages of Parameters Measured for Animals in the Three Initial pH Ranges	79

LIST OF FIGURES

,

Figure		Page
1.	Change in pH for Porcine Muscle	40
2.	A Comparison of Intramuscular Temperature With Porcine Muscle pH	43
3.	A Comparison of Live Weight With Porcine Muscle pH	45
4.	A Comparison of Chilled Side Weight With Porcine Muscle pH	45
5.	A Comparison of Carcass Lean Cut Yield With Porcine Muscle pH	46
6.	A Comparison of Live Lean Cut Yield With Porcine Muscle pH	46
7.	A Comparison of Carcass Length With Porcine Muscle pH	48
8.	A Comparison of Loin Eye Area With Porcine Muscle pH	48
9.	A Comparison of Longissimus dorsi Weight With Porcine Mus- cle pH	49
10.	A Comparison of Three Quality Characteristics With Porcine Muscle pH	50
11.	A Comparison of Ether Extract and Moisture Values With Porcine Muscle pH	53
12.	A Comparison of Fiber Diameter With Porcine Muscle pH	55
13.	A Comparison of Degree Fiber Rigor and Muscle Shear Values With Porcine Muscle pH	56
14.	The DPNH-TR Staining Reaction Showing How Electrons are Transferred to the Nitro-Blue Tetrazolium Salt	59
15.	A Comparison of Fiber Distribution and Arrangement With Porcine Muscle pH	61
16.	A Comparison of Fiber Types With Porcine Muscle pH	62

•

CHAPTER I

INTRODUCTION

In recent years, increased death rate of swine has resulted from stresses incumbent in medical treatment, weighing, lot movement, exercise, and hauling. This death rate increase has been attributed to a muscle abnormality known as Myodegeneration Syndrome. The purpose of this research was to characterize the Syndrome on a gross and microscopic level.

This muscle abnormality has become a major problem in today's meat industry as a result of the efforts of breeders to satisfy the consumers demands for not only meatier but higher quality pork both in the raw and cooked form. Realizing the scope of the problem, the swine producer has managed to provide animals with not only more but a higher quality of meat through selective breeding, enriched feeds, or by a combination of the two. As a general rule, his efforts have succeeded, except where the Myodegeneration Syndrome is noted, for it appears that the meatier the animals, the more susceptible they are to the disease and the lower their quality. Topel et al. (1968) reported a similar abnormality termed Porcine Stress Syndrome in which the conditions that were expressed could result in the death of the animal before slaughter or in meat that was Pale, Soft, and Exudative (PSE) post-mortem. The incidence of losses due to death from the abnormality was reported to be about 33% of the animals studied. Another survey by Forrest et al. (1968) of 15,000 hams

which passed through a processing plant in a year's period expresses, in part, the magnitude of the problem caused by PSE or the Myodegeneration Syndrome for the meat industry. This survey showed that the condition occurred in approximately 18 - 20% of the hams but could go as high as 40%. In addition Bray (1966) indicated that the cooking and shrinkage loss was some 15% greater in PSE muscle than normal muscle. In the processing procedures, hams were noted to yield from 6 - 10% less. Other research has shown that the nutrient loss is greater in PSE muscle (Meyer et al., 1963). From the consumer's point of view such muscle is extremely unacceptable both from the standpoint of its unattractiveness and low palatability.

Therefore it is obvious that the Myodegeneration Syndrome is costing the meat industry countless dollars due to difficulty in processing and merchandizing. It was hoped that this investigation would afford some characterization of the Syndrome so that the problem might be alleviated, both in the processing plant and the breeding stock.

CHAPTER II

LITERATURE REVIEW

Muscle of the Normal Animal

Fiber Types

Because of the close relationship of fiber type to Myodegeneration Syndrome and PSE, this portion of the review will be concerned with various studies of the ultrastructure, biochemistry, and histochemical characterization of skeletal muscle fibers. Bear in mind that these studies will deal with normal muscle tissue and the second part of this chapter with the abnormal aspects.

Lorenzini in 1678 (Ciaccio, 1898) was perhaps the first investigator to report a distinct difference in muscles, as he was able to differentiate them as being either red or white upon inspection with the unaided eye. Ranvier (1874) noted that fibers comprising various skeletal muscles differed in their microscopic appearance. By the early 1960's histochemical techniques had developed sufficiently to allow investigators to distinguish fiber types on the basis of their reaction with various enzyme stains. At first, fibers were categorized as being only red or white but eventually a third, intermediate fiber, emerged. Red fibers were found to be aerobic while white fibers were anaerobic metabolizing. The intermediate fiber on the other hand could metabolize by either pattern depending upon conditions which existed within the

muscle. The distribution of these three fiber types may vary from muscle to muscle or within any given muscle. In the rat diaphragm for example, it was calculated that the red fibers constituted 60% of the total fiber population while the white and intermediate fibers contributed only 20% each to the total fiber area (Gauthier, 1970). The porcine Longissimus dorsi has a distinctly different distribution as the red and intermediate fibers make up 30% of the total area while the white fibers comprise 70% of all the fibers (Cooper et al., 1969). The amount and type of fibers becomes important when one considers their role in muscle color and metabolism. Various investigators have found that muscles which are uniform in color usually have a metabolism related to their color intensity; red muscles are equipped for an aerobic type of metabolism whereas white muscle depends largely on anaerobic metabolism (Beatty et al., 1963; Needham, 1926; Ogata, 1960). The reason for such results is quite obvious as those muscles designated as red or aerobic, such as the heart, will contain a very high percentage of red fibers. White or anaerobic muscles will have a predominance of white fibers such as the Longissimus dorsi.

Ultrastructure

The ultrastructural characteristics of muscle fibers give additional support to the distinct metabolic types. Gauthier (1970) observed that mitochondrial content was inversely related to the diameter of the fibers. Thus, a red fiber which has a small diameter would be rich in mitochondria whereas the white fiber with its larger fiber diameter has a low mitochondrial content. Differences have been found in the mitochondria themselves, for in the red fiber they had a circular and fila-

mentous profile on the interior while subsarcolemmal aggregations consisted of large, closely packed mitochondria. These were seen to have abundant cristae in the form of parallel sheets. Similar mitochondria were arranged in parallel rows between the myofibrils where triglyceride droplets were often associated with them. Paired mitochondria encircled the myofibrils at the I bands. In the white fiber, the mitochondria were almost entirely filamentous in profile, reflecting paired mitochondria at the I bands. These were evidently present in all three fiber types. It is interesting to note that the Z lines were narrowest in the white fiber type. Intermediate fibers were quite similar to the red type except that the mitochondria were somewhat smaller with less closely packed cristae. The Z lines were thinner in the red fiber, but somewhat more dense than in the white. The greater abundance of mitochondria in the red fibers reflect a higher oxidative capacity and enzyme activity of those systems involved in aerobic metabolism. Lastly, the distribution of the sarcoplasmic reticulum was about the same in the three fiber types with the exception of the transverse network in the region of the H band.

Innervation

Szent-Gyorgi (1953) developed the classic postulate that white muscle was capable of rapid but brief contraction, utilizing glycolysis for energy production whereas red muscle, which was slower contracting, relied chiefly on oxidative mechanisms. Henneman et al. (1965b) found that the Soleus m., which has a majority of red fibers, contracted tonically and supported aerobic metabolism. The Gastrocnemius, conversely, contracted tetanically under less efficient anaerobic conditions. These

differences in contraction suggest that there may be variations in nervous control. In fact, evidence now points to the idea that the nervous system may control the differentiation of the fiber types. Buller et al. (1960) demonstrated that by crossing the nerves leading to fast and slow muscles, the contraction velocity could be reversed. During the procedure, the nerves were sectioned and then interchanged before reconnecting. Those nerve fibers which were disconnected from their cell bodies. degenerated and the space left by them was gradually replaced by axons from the other nerve. Interestingly enough, what was once a fast muscle became nearly indistinguishable from a slow muscle with respect to its contractile characteristics. The slow muscle, in turn, became a little faster but did not reach the velocity of the fast muscle. Another series of experiments demonstrated that there were three categories of muscle fibers in the cat with respect to contraction velocity, fatigueability, and demonstrable or presumable metabolic characteristics. Secondly, the motor units appeared to consist of only one type of muscle fiber. Lastly, motor units of differing characteristics were physiologically activated in certain sequences and by certain rules as to the size and characteristics of the unit and its motor neuron (Henneman et al., 1965a, b). Increasing evidence was added to the function of the nerve in fiber differentiation by Doyle et al. (1969) who observed that the nerve supply of a muscle arose from cell bodies in the anterior horn of the spinal cord. An axon from a single anterior horn cell branched peripherally to supply many muscle fibers and was termed the motor unit. The number of fibers in any given unit was found to vary from a few dozen fibers to several hundred. Their work showed that the fiber type was related to the nerves supplying the muscle fibers and therefore it was not surpris-

ing to find that all muscle fibers of a given motor unit stained identically. Karpati et al. (1967) observed that in some animals at birth. the Soleus was a mixed muscle consisting of both type I (red) and type II (white) fibers, but during the early development, the number of type II fibers diminished until the muscle was composed of predominantly type I fibers. When the Soleus was denervated before such differentiations took place, further differentiation was prevented and the muscle remained mixed. The author concluded that this evidence suggested that the histochemical as well as physiological properties of muscle fibers were influenced by the nerve supply. Such an influence may be due to either a direct trophic action of the nerve on the muscle or an indirect action due to the nature of the contraction pattern induced by the firing of the nerve. This is supported by Padykula et al. (1970) who found that there were distinct differences in the neuromuscular junctions of mammalian red, white, and intermediate skeletal muscle. It was further interesting to note that nuclei were aggregated in the sarcoplasm of the neuromuscular junction. The nuclear surface was invaginated into deep clefts which faced toward the junctional folds thus directing a greater nuclear surface area toward this specialized area of the neuromuscular junction. It would therefore, not be inappropriate to postulate that there may be an interchange of messenger RNA between these two tissue types.

Metabolism

Earlier it was stated that the red fiber was primarily aerobic while the white fiber was basically anaerobic in its metabolism. If these statements are true, one would then expect that there would be differ-

ences in concentrations of métabolites and enzymes, as well as, differences in enzymatic activities. Beatty et al. (1963) has shown that lactate production was greater in white than in red muscle fibers. Furthermore white muscle, since it has less oxidative capacity, may rely on glycolytic metabolism and energy reserves that are immediately available such as creatine phosphate. These statements were based on the greater disappearance of glycogen and higher lactate production. Acetoacetic acid uptake was shown to be 30 - 35% higher in red than in white muscle, thus giving support to the evidence of higher oxidative capacity in red muscle. Beecher et al. (1968) observed that myoglobin concentration, percent red fibers, and succinic dehydrogenase activity was more than twice as high in the Semitendinosus dark than in the light portion. Glycogen concentrations were similar in the two portions. Lactic acid concentrations were significantly (P < .01) higher in the light portion. The Semitendinosus light portion had generally higher concentrations of ATP and creatine phosphate. Amounts of ADP and inorganic phosphate were similar in both portions, however, AMP was higher in the dark. There was a significantly higher (P < .05) phosphorus content in the lighter portion. The ionic content may be summarized as follows: levels of potassium were similar while significantly higher concentrations of sodium were found in the dark portion. Concentrations of zinc, iron, and copper were higher in the dark portion while calcium, nickel, and boron were similar. The higher concentration of iron was most likely the result of myoglobin content whereas the higher content of calcium was due to the cytochrome system, as both were present to a greater extent in the dark portion. Finally, the moisture content was significantly lower and lipid significantly higher in the Semitendinosus light portion than in the dark. Another study of porcine Semitendinosus found that the dark muscle portions contained higher initial values for pH and glycogen, and exhibited a shorter delay phase of rigor mortis (Beecher et al., 1965a). This agreed well with Schmidt et al., (1970a) who reported that white muscle had a shorter time course for rigor mortis than did red muscle. Kirsten et al. (1969) have shown that ATP decreased only in red muscle while phosphoryl creatine decreased about equally in red and white muscle during tetanization. Along with the increased glycolysis of the white fiber and oxidative processes of the red, a positive correlation was found to exist between the amount of muscle pigment, myoglobin, and the Glutamic-Oxalacetic as well as the Glutamic-Pyruvic transaminase activity. Therefore, red muscles have higher transaminase activity due to the aerobic metabolism or presence of the citric acid cycle (Hamm et al., 1969).

Histochemistry

Capitalizing on the metabolic patterns which exist in the various fiber types, investigators have attempted to classify fibers on the basis of their histochemical reactions. In general the alkaline phosphatases, ATPases, and amylophosphorylases are used to histochemically identify white fibers, whereas, the dehydrogenases, NADH-TR, and esterases are primarily useful for differentiation of the red fiber types. Dubowitz et al. (1960) was among the early investigators who differentiated fibers into two major types using alkaline phosphatase and several dehydrogenase stains. The type I fiber was described as being rich in dehydrogenase and poor in phosphorylase while the type II fiber was rich in phosphorylase and poor in dehydrogenases. These investigators also

found intermediate fibers but did not classify them as a specific type. Stein et al. (1960) defined three fiber types on the basis of the succinic dehydrogenase reaction. These fibers were designated as A, B, or C. The type A corresponded to the white or type II fiber while the B and C were equivalent to the red or type I fiber. Romanul (1964) used a wide variety of enzyme stains such as diphorase, various dehydrogenases, phosphorylases, etc., and then correlated the relative activities of all the enzymes in the individual fibers. By this technique, he was able to recognize eight possible fiber types. These fibers were scaled from strictly anaerobic metabolizing one one end, to completely aerobic on the other end of the scale. The intermediate fiber was recognized as a distinct fiber type in the work of Moody et al. (1968) who used DPNH-TR and the amylophosphorylase stain. The typical red fiber was positive for DPNH-TR but negative for amylophosphorylase while the white fiber exhibited just the opposite staining pattern. Red fibers were described as being in clumps of five to seven and were surrounded by intermediate fibers. The white fibers were located on the periphery of the fasciculus. These investigations showed that the number of red fibers from the amylophosphorylase stain did not correlate well with the DPNH-TR technique. It was found that the intermediate fibers were responsible for the differences in the red fiber content of the two staining methods as the number of these fibers was much higher for the DPNH-TR reaction than for amylophosphorylase. Furthermore, there were many more intermediate fibers present in the Longissimus dorsi than in the Trapezius. Also, the authors felt that these fibers should be considered as a type separate from the classical red and white fibers. In a subsequent article by Cooper et al. (1969) it was stated that the reaction to DPNH-TR should

indicate the oxidative potential of the fiber. Fibers which reacted positively to this stain showed a uniform deposition of diformazan. Such heavy, uniform deposits were typical of true red fibers, whereas dense, subsarcolemmal deposits with light centers were typical for intermediate fibers. The importance of these fiber types in the development of Myodegeneration Syndrome and PSE will be discussed in a later section dealing with their distribution and metabolism in these abnormalities.

Muscle of the Abnormal Animal

Myodegeneration Syndrome and Pale, Soft, Exudative Pork

Because of little past research with the Myodegeneration Syndrome, the author has chosen a related abnormality, pale, soft, exudative pork (PSE) to describe conditions which exist in affected muscle. The author feels that the conditions may be different expressions of the same abnormality with Myodegeneration Syndrome being the more severe of the two, In general, there have been seven areas of research in this respect: Effects of the environment, electrical stimulation, the endocrine glands and their relation to blood supply and metabolism, fiber distribution, biochemistry, ultrastructure, and quality. It must be remembered at all times by the reader that each is intricately related to the other and can by no means be separated. Topel et al. (1968) gave an excellent description of the abnormality in a step by step manner. Deaths from the "disease" occurred without warning but in most cases the history indicated a recent stressing experience such as a change in temperature, atmospheric condition, or fighting. In some cases, hogs died after two minutes exposure to such stress. The signs of Porcine Stress Syndrome

(PSS) developed rapidly with the first indication being a rapid tremor of the tail when the animal was aroused or excited. Dypshea occurred with the affected animals developing open-mouth breathing. The body temperature was elevated during this phase with irregularly shaped, alternating areas of blanching and erythema developing on the skin. Finally, the pigs became reluctant to move and would collapse and die if the stress was continued. Associated with the PSS condition was a rapid glycolytic rate which occurred post-mortem in the stress-prone pigs. The initial muscle pH values were reported to be 5.97 and 6.20 in control animals while the stress-susceptible pigs had muscle with pHs of 5.33 and 5.85 one-hour post-mortem. The muscle temperature for the groups was almost the same throughout the chilling period, but the color of the Longissimus dorsi differed considerably in that the muscle from the stress-prone pigs was pale at one-hour while that from the control animals was normal in color. At four and 24 hours, the muscle from stress-prone pigs was very pale, soft, and exudative while controls were normal in all aspects. Not all muscles exhibited the PSE condition, for example, only the Gluteus medius and Biceps femoris were effected in the ham. There was also evidence of edema as some muscles were noted to separate from the subcutaneous fat indicating that there was some connective tissue alternation. Blood pH in normal pigs stressed for five minutes decreased from 7.52 to 7.20 which compared to 7.57 and 6.97 in stress-susceptible animals, giving some indication that the normal pigs had a greater ability to metabolize acid waste substances resulting from muscular activity. The rapid rate of post-mortem glycolysis caused a decrease in muscle pH at temperatures above 38°C which appeared to be responsible for the PSE condition. Characteristic of both PSE and

Myodegeneration Syndrome was that all animals which exhibited the abnormality were very muscular and had small backfat thicknesses. They were all from outstanding swine herds and exhibited a rapid growth rate with good feeding efficiency.

Environmental Influences

Judge et al. (1967) stated that PSE could be the result of a genetic defect which prevents animals from adapting to their environment. They further postulated that some animals have defense mechanisms which are capable of restoring physiological balance in muscle tissue during severe stress, whereas others do not possess these mechanisms and the response to stress conditions became progressively more severe. Many investigators have observed that environmental variations tend to produce higher incidences of PSE. Folk (1966) stated that "in its broadest sense, the term environmental stress encompasses any stressful situation. On the other hand, there is a plethora of terms with subtle and pronounced differences in meaning which are used by environmental physiologists to describe the responses of animals to their surroundings." Wismer-Pedersen et al. (1961) found that it appeared to be possible to produce pale, soft, exudative tissue by retaining body heat in the carcass for an extended period. They found that temperatures to 40°C were not critical if pH values were above 6.20. Temperatures above 35°C appeared to be influencing factors if the muscle tissue possessed a pH value below 5.90. This suggested that if the pH declined below 5.90, the muscle structure would undoubtedly be altered. Similarly, a temperature above 30°C is critical if the pH value is below 5.6. These data reveal the importance of understanding the acidity-temperature relationships during

post-mortem chilling for it was found that chilling the tissues rapidly prevented the extreme variation in pH and muscle structure alterations. Sayre et al. (1961) placed animals in cold water $(0.5^{\circ}C)$ for 30 - 40 minutes in an attempt to simulate severe environmental change. The stress caused by the change from a warm to cold temperature decreased the initial glycogen levels which resulted in decreased lactic acid concentrations and increased color intensity of the chilled muscle. The water binding capacity was not severely affected. Forrest et al. (1965) found that respiration and heart rate tended to increase as muscle temperature increased. These increased rates were associated with rapid rates of post-mortem pH decline, low post-mortem pH, and PSE musculature. In experiments conducted by Addis et al. (1967) differences were found in the mean scores for color and gross morphology of the Longissimus dorsi, suggesting that low or fluctuating humidity, when combined with a warm temperature in the growing environment, may produce a slightly greater incidence of PSE. It was suggested that this may have been due to an imbalance of trophic hormones from the pituitary due to alternating humidity levels. This topic will be discussed more completely in a later section. The influence of the environment on the incidence of PSE was clearly demonstrated by a survey conducted by Forrest et al. (1968) in a processing plant over a 12-month period. This survey included over 15,000 hams. Temperature variations as well as variations in incidence were recorded throughout the year. During cold weather a low incidence was observed but sharp increases were noted during periods when the temperature fluctuated from warm to cold. In such cases, the incidence of PSE would elevate to 30 - 40% of the hams examined. The incidence of PSE in the hams averaged from 18 - 20%. In

the laboratory it was found that a ten minute exposure to a 42° C environment caused increases followed by sharp reductions of respiratory rate, heart rate, and cardiac output. It was also reported that the partial pressure of CO₂ increased while that of O₂ decreased as did the pH of the venous blood. Furthermore, those pigs designated as being stress-resistant were able to withstand longer durations of heat stress with only minor changes in the gases and pH of the blood. In agreement with the work done by Forrest, Sayre (1962) observed that there were wide variations in the ultimate pH, color, and gross morphology between days or periods representing major environmental changes. Such experiments serve to show that the PSE animal was unable to make the necessary adaptations to its environment.

Electrical Stimulation

Other investigators have tried to show that PSE is related to conditions within the animal which resulted in muscle that was of undesirable quality. The first of these which will be discussed is the effect of electrical stimulation. Lewis et al. (1962) has shown that stress from periodic electrical stimulation prior to slaughter increased the pH, tenderness, texture, and juiciness of the Psoas major and Quadriceps femoris muscles, while it decreased the evaporation due to cooking, total water and dry matter loss, time for rigor to set in, and the two-toned score of the ham. In general this work indicated that such stress had no detrimental effect on pork eating quality. Just the opposite was demonstrated by Hallund et al. (1965) who observed that the slow fall of pH in muscle could be converted to a quick fall by subjecting excised samples to a short tetanus. Such evidence was only observed in normal

muscle but the rapidly glycolyzing muscle did not exhibit such a change. These high rates of pH fall were invariably associated with the occurrence of watery meat which was determined to a large extent by long term after effect nervous stimuli which reached the muscle during the sticking process. In pigs which were successfully immobilized before death by injection with myanesin, which inactivates the motor horn cells, the rates of pH fall were the lowest. Forrest et al. (1966) also observed the response of an excised muscle to electrical stimulus was greatly associated with the post-mortem muscle properties. It was found that the excitability threshold was high in muscle which had a short time course of rigor mortis, fast post-mortem glycolysis, and PSE gross morphology. In those muscles with a long time course of rigor, slow post-mortem glycolysis and ultimately normal color morphology, the excitability threshold was low. The duration of contractility was longer in the normal muscle.

Hormonal Influences

Other investigators have concentrated efforts in the area of hormonal control in the development of PSE. Judge (1969) found marked differences in the endocrine status of porcine animals, and presumably these differences play important roles in the responses of the animals to environmental stress. The stress-susceptible animal may therefore represent the result of intense selection for economically important traits with a concominant loss of defense mechanisms originally necessary for survival. Current research depicted the stress-susceptible animal as an individual whose physiological adjustments during stress were poorly coordinated and augmented by the endocrines; an animal whose

muscle underwent extremely rapid rates of post-mortem glycolysis with the accompanying development of a pale, soft, exudative condition; and whose carcass was frequently well muscled. Topel (1968) studied adrenal secretions, namely plasma 17-OHCS and found that pigs with lower than normal levels of this hormone had problems with accelerated carbohydrate metabolism and conversion of lactate to other metabolic substances, such as glycogen when they were stressed. Judge et al. (1968) found a significant relationship between 17-ketosteroid levels and the color of the Gluteus medius muscle. These data indicated that adrenocortical hormones may prevent the development of PSE muscle by reducing lactic acid accumulation in the immediate ante-mortem period. The data also suggested that catecholamine production may influence post-mortem muscle properties since the duration of muscle contraction is regulated by this hormone and is positively correlated with the time course of rigor mortis and ultimate color. It was also shown that the adrenal steroids had a direct effect on blood pressure and circulation. Howe et al. (1969) found that stress-susceptible animals had a loss of functional capacity of the adrenal gland due to lipid accumulation in the zona reticularis. In agreement with Judge et al. (1968), Schayer (1964) found that all of the actions of the glucocorticoids could be related to their control of the arterioles and capillaries. Romanul (1965) stated that the capillary supply of the muscle fibers has significance in terms of the energy metabolism of the fibers and that a directly proportionate relationship was present between the density of the capillary network around each fiber and the oxidative potential of the fibers as measured by cytochrome oxidase and succinic dehydrogenase activity. Morita et al. (1969) reported that red fibers have a much greater capillary supply than do

white. These observations agree well with Cooper et al. (1969) who investigated the probable connection of the circulatory system to the PSE condition. However, no difference in capillary to fiber ratio was found between PSE muscle and normal muscle, but the number of capillaries per unit area of red and intermediate fibers were less in PSE muscle. In conjunction with the circulatory system which carries oxygen to the muscle tissue it would now be appropriate to discuss the role of myoglobin in oxygen storage and its relationship to fiber type. Lawrie (1952) reported that the function of myoglobin, with its power of reversible combination with oxygen and its low loading tension, is to assist in ensuring a constant supply of oxygen to the muscle oxidase system. Those animals with a low myoglobin content in skeletal muscle have an excellent supply of oxygen from the blood. Furthermore, the more myoglobin in skeletal muscle, the greater appears to be its capacity for respiratory metabolism and the less its power for carrying out glycolytic processes. Chinoy (1963) observed that the red narrow fibers with their high concentration of oxidative enzymes are shown to possess practically all of the myoglobin in comparison with the broad white fibers which contain very little. Therefore, one is led to believe that the Myodegeneration Syndrome animal with its pale muscle possesses a great many more white and intermediate fibers and thus it would have much less capacity for oxygen transport as well as oxygen storage. Judge (1969) stated "an inadequate circulatory system would not maintain physiological conditions locally in muscle after a massive discharge of nervous impulses such as occur upon death. These circumstances would explain the immediate buildup of lactic acid that occurred in some pigs after stunning." Therefore, one may see that the hormonal control of circulation, the capillary

supply, and myoglobin content all have a complex relationship and may be one of the causative agents of Myodegeneration Syndrome.

Other work on adrenal secretions was done by Shafrir et al. (1960) who found that subcutaneous injection of dogs with epinephrine mobilized both the free fatty acids and lipoproteins and caused increases in plasma free fatty acid levels that persisted from one to three hours. Similar work by Cunningham et al. (1963) on pigs indicated that subcutaneous injections of epinephrine (approximately 0.15 mg/kg of body weight in the flank region per day) may enhance nitrogen retention in porcine animals at all ages up to market weight. The injections increased the growth rate of older pigs, but restricted growth rate in younger animals. Decreased fat storage was also noted in younger animals but there was no apparent effect in the older group.

Other investigators have explored the role of the thyroid gland in the development of the PSE condition. Kastenschmidt et al. (1965) showed that animals which exhibited rapid post-mortem glycolysis tended to have a larger thyroid-adrenal ratio which was due primarily to lighter adrenals. Just the opposite was found by Judge et al. (1968) who observed that PSE pigs had low levels of thyroid iodine, small thyroid glands, and elevations of plasma protein-bound iodine as compared to normal animals. In a similar aspect, thyroidectomy has been shown to result in a marked reduction in the growth rate and Basal Metabolic Rate (BMR) of the whole animal, as well as, lowered mitochondrial respiration and phosphorylation per unit of protein. Administration of the thyroid hormone, thyroxine, to these animals over a period of three weeks nearly doubled the rates of growth and considerably increased the BMR and oxidative metabolism in isolated mitochondria (Gustafsson et al. 1965). These ex-

periments serve to illustrate that the thyroid and adrenal hormones may be important in the expression of both quantitative and qualitative characteristics of porcine muscle (Judge et al. 1968).

Fiber Distribution

Cooper et al. (1969) has done some very interesting work with the distribution of the fiber types and their relationship to PSE musculature. By using a histochemical technique involving the DPNH-TR staining reaction they found that there were considerably fewer red fibers and more intermediate fibers in muscle from stress-susceptible animals which became PSE than in muscle from stress-resistant pigs that remained normal in color and gross morphology. There was also an increased intensity to ATPase and phosphorylase reactions in the muscle fibers from stresssusceptible animals which became PSE and this increased intensity was especially evident in the large intermediate fibers. It therefore appeared that it was both the number and nature of the intermediate fibers which were the key contributors to the stress-susceptibility of the animal and to the development of PSE characteristics in the musculature. It also seemed that the response of a stress-susceptible animal to anoxia in its skeletal muscle was due to the large number of intermediate fibers which were dependent upon aerobic metabolism, but unlike typical red fibers, had especially high ATPase and phosphorylase activity, breaking down ATP and accelerating glycolysis to trigger a rapid glycolytic rate in the entire muscle. The regular white fibers also had a rather intense ATPase and phosphorylase activity and further contributed to the acceleration of these metabolic phenomena. Lister et al. (1970) has also studied the fiber characteristics of PSE and normal pig muscle using Sudan black B, cytochrome oxidase, and succinic dehydrogenase. These workers concluded that PSE fibers were larger than muscle fibers from normal muscle and that there was a greater area of red fibers per bundle in PSE muscle. However, it is important to note that the DPNH-TR reaction was not used and therefore the differentiation of intermediate fibers was not made. Cassens et al. (1969) reported the occurrence of a giant fiber which appeared to be related to the PSE condition. The typical giant fiber was round and usually larger than the surrounding fibers, having a diameter of some 180 microns. These giant fibers often appeared at the periphery of a fasciculus and had compressed the surrounding fibers to conform to their circular shape. These fibers displayed a variable reaction to DPNH-TR, a negative reaction to phosphorylase, and a positive reaction to ATPase. Thus, the giant fiber did not correspond to either the classical red or white fiber. Such entities were found to compose less than one percent of the total fiber population, but were noted to occur frequently in the muscle of stress-susceptible pigs and rarely in muscle from normal animals. Fibers such as these may reflect some sort of pathological change in the muscle.

Metabolism

The distribution of the different fiber types is important in Myodegeneration Syndrome and PSE when one considers their role in influencing the rate of post-mortem metabolism. Kastenschmidt (1970) stated that it is the variable rate of post-mortem metabolism which will determine the ultimate usefulness of muscle as a food. Briskey et al. (1961a) found that there were at least four distinct types of post-mortem pH patterns in pork muscle as determined by continuous recording from pork

carcasses. These patterns were: a) a slow gradual decrease to an ultimate pH of 5.7 to 6.3; b) a gradual decrease to approximately 5.7 at eight hours, with an ultimate pH of 5.3 to 5.7; c) a relatively rapid decrease to 5.5 at three hours with an ultimate pH of 5.3 to 5.6; d) a sharp significant decrease to a pH of 5.1 to 5.4 at one and one-half hours, and a subsequent elevation to 5.3 to 5.6. It was the last group of animals which exhibited the "violent nature of anaerobic glycolysis," for it was observed that the glycogen concentrations were unusually high but only a limited percentage of the glycogen was readily soluble in cold 10% TCA. While the muscle temperature remained at its living level, the pH decreased to 5.1 which was followed by a rapid depletion of labile phosphorous. This action simultaneously initiated the immediate and "complete production of lactic acid" and a sharp increase in the quantity of loose water. Furthermore, the total calories evolved during chilling was found to be comparable in all groups, but in the last series (d) there was a sudden and drastic release of heat. It was therefore concluded that the high muscle temperature and low pH was responsible for the PSE condition. This is also in agreement with Sayre et al. (1961), Briskey (1964), Bendall et al. (1963), Wismer-Pedersen et al. (1961), and Koch (1969) who demonstrated that PSE pigs were characterized by a rapid rate of post-mortem glycolysis with subsequent decreases in ATP and Creatine phosphate. The findings of such drastic decreases in high energy metabolities is interesting in relationship to observations by Hess et al. (1961), Henson et al. (1966), and Vester et al. (1968) who demonstrated that an elevation of Creatine phosphokinase appeared to exist for most diseases of skeletal muscle. The enzyme creatine phosphokinase is responsible for the transfer of a high energy phosphate from

creatine phosphate to ADP thus yielding ATP. Kastenschmidt et al. (1966) have shown that PSE muscles were fast glycolyzing and that they were in an oxygen deficient state prior to the time of death. This information ties in well with reports of poor capillary supply and myoglobin contents. Furthermore, the stress-susceptible animals were either more easily made anoxic or responded to anoxia to a greater extent. Kastenschmidt et al. (1968) observed that during the first 60 minutes post-mortem levels of Glucose-1-Phosphate (G-1-P), Glucese-6-Phosphate (G-6-P), and Fructose-6-Phosphate (F-6-P) were lower in slow glycolyzing muscles than in fast glycolyzing muscles. Levels of intermediates from Fructose diphosphate to Phosphoenolpyruvate were higher in slowglycolyzing muscles. Also, the initial levels of ATP and ADP were about 40% higher and AMP was about 50% lower in the slow-glycolyzing muscle. Levels of total pyridine nucleotides were higher in stress-resistant pigs but declined much more rapidly in those that were stress-susceptible. The higher levels of ATP and phosphocreatine suggested a more efficient maintainance of energy levels. Data suggesting a lower G-1-P, G-6-P, and F-6-P in slow-glycolyzing muscles support a concept that increased phosphorylase activity in fast glycolyzing muscle is responsible for a large part of the increased glycolytic rate, in that they apparently play a large role in the regulation of phosphorylase activity. Both ATP and G-6-P counteract the activating influences of AMP and P_i on phosphorylase. ADP can also counteract these influences. On the basis of a 40% higher ATP and ADP level in slow-glycolyzing muscle combined with a 50% lower level of AMP appears to explain the decrease in phosphorylase activity in this muscle type. Schmidt et al. (1970b) found that serum from stress-susceptible Poland China pigs had significantly

(P < .01) greater calcium and inorganic phosphorous, significantly (P < .01) greater sodium concentrations, and significantly (P < .01) greater calcium to magnesium ratios than did serum from stress-resistant Chester White pigs. There was no difference detected in either magnesium or potassium concentrations. The serum did have significantly (P < .01)greater concentrations of alkaline phosphatase, lactic dehydrogenase, glutamic oxaloacetic transaminase, and blood urea nitrogen than did serum from the stress-resistant Chester White pigs. There were no differences observed in glucose, total protein, or in cholesterol content. This observation agreed well with Sink et al. (1967) who noted that there were no significant differences between normal and PSE muscles in total lipid or fatty acid composition. Schmidt et al. (1970b) also found that electrolyte differences were small and difficult to interpret. Briskey et al. (1961b) have shown that stress-susceptible pigs possessed a smaller pyruvic acid pool and a greater lactate concentration. It was furthermore asserted that the inferior musculature which appeared to occur in more muscular pigs could be construed to imply that maturation of the enzyme systems had not kept pace with the growth of the muscle mass.

Ultrastructure.

Besides causing a drastic visual change, the metabolic characteristics of PSE musculature are responsible for some interesting ultrastructural alterations. Cassens et al. (1963) conducted the first study of post-mortem structural changes in porcine muscle utilizing the electron microscope. They observed that muscle which underwent a normal change post-mortem exhibited a gradual disruption of the sarcoplasmic components with little if any change in the myofibrils. However, muscle that underwent a very rapid change post-mortem revealed a very rapid disruption of sarcoplasmic components and some apparent disruption of the myofibrils. Greaser et al. (1969b) observed some distinct differences in mitochondrial, heavy and light sarcoplasmic reticulum, and myofibrillar fractions isolated from normal and PSE muscles. It was found that the myofibrils 24 hours post-mortem had more granular appearing filaments and wider Z lines in PSE than in normal muscle. The Z-lines were obscured by an accumulation of material that could have been precipitated sarcoplasmic protein. The filaments were also granular due to changes in the conformation and properties of the myofibrillar proteins or a precipitation of sarcoplasmic protein. It is this author's view that it was due to the sarcoplasmic proteins since Bendall et al. (1962), using titration studies showed that the sarcoplasmic proteins would denature and precipitate on the structural proteins preventing the extractability of the latter protein group, and furthermore, there was no evidence of change in the structural proteins. The authors further felt that the changes in the sarcoplasmic proteins may result in the loss of water binding capacity and decreased ATPase activity. This is in agreement with Cassens et al. (1963) and Hamm (1960).

Another important aspect of the sarcoplasmic reticulum is its function in the release and uptake of the calcium ion. Ebashi et al. (1969) illustrated that the excititory process lead to a liberation of calcium which in some manner activated the contraction process by supressing an inhibitory interaction. Furthermore, the active state of contraction is slowed and subsequently terminated by a rebinding of calcium to the sarcoplasmic membrane. Ebashi et al. (1962) found that the relaxing

factor consisted of fragments of the sarcoplasmic reticulum which had the ability to remove calcium from solution by an ATP-dependent transport process. Ikemoto et al. (1968) have shown that the sarcoplasmic reticulum consisted of tiny particles which resembled the elementary particles of mitochondria. These subunits consisted of a globular head to which one or more tails were attached. The authors found that the calcium uptake and ATPase activity was localized in the globular portion of the vesicles. The ATPase activity was in the greatest strength at the junction of the tail and globular portion. This supports an earlier work by Engel et al. (1966) and Tice et al. (1966) who isolated a microsomal fraction from the rabbit Psoas muscle. The fraction was found to contain a magnesium-dependent ATPase which had activity present throughout the sarcoplasmic reticulum but was found to be absent in the T-system.

The uptake and release of calcium is very much dependent upon what is known as the calcium pump. Such a mechanism has been postulated to be composed of proteins which comprise the sarcoplasmic reticulum and act like swinging doors. That is, they swing out and bind to the calcium, and then swing in to release the ions within the vesicles. The efficiency of the calcium pump is the greatest when there is an ATP regeneration system (such as phosphocreatine) to maintain a high level of ATP and to keep levels of ADP, which is an inhibitor of the calcium pump, at low levels (Hasselbach et al. 1962).

Reviewing the literature concerning the metabolism of PSE muscle it is obvious that there is a rapid depletion of creatine phosphate and ATP, a rapid accumulation of ADP, as well as drastic decreases in muscle pH. Realizing that these conditions are conducive to the loss of functional-

ity of the calcium pump, it might be possible to relate the rapidly. glycolyzing muscle to a massive release of calcium, since this ion is controlling factor of myofibrillar ATPase activity. Greaser et al. (1969c) found that treatment of the sarcoplasmic reticulum with a pH of 5.6 at 37°C for one hour almost completely abolished the reticular uptake of calcium. They concluded that low muscle pH and high temperatures may be responsible for the inactivation. In a subsequent experiment Greaser et al. (1969a) measured differences in calcium uptake in a solution containing ⁴⁵Ca of normal and PSE muscle. It was found that the calcium accumulating ability of subcellular fractions declined five to ten fold between 0 and 24 hours post-mortem. The main portion of the decline occurred one hour after death in the fractions from PSE muscle but was more gradual in normal fractions. The ATPase activities did not differ significantly, increasing with time post-mortem in most of the normal fractions, but decreased in those from PSE animals. There did appear to be a close relationship of post-mortem pH decline in muscle and a loss of calcium accumulation ability which may have been due to: a) an inactivation of the relaxing factor leading to a more rapid myofibrillar ATPase activity which depleted the creatine phosphate and ATP reserves; b) the increasing acidity of the muscle had a specific inactivating effect on the calcium accumulating ability of the relaxing factor; c) a partial inactivation of the relaxing factor which lead to a more rapid pH decline, which in turn accelerated inactivation. In one other experiment by Greaser et al. (1969d) the accumulating ability of purified sarcolasmic reticulum was found to be greater than that of similar preparations from PSE muscle. Furthermore, it was interesting that no evidence was obtained to indicate that the accumulating ability

declined between biopsy and death in the crude fractions. These results suggested that the differences observed between normal and PSE preparations were inherent and might not reflect the resultant effects of a more rapid post-mortem change.

Muscle Quality

Perhaps the most obvious effect such abnormal metabolism and structural alterations have on porcine muscle is a definite lowering of quality in both the raw and cooked forms. Bray (1966) stated that quality to the meat scientist concerns those factors associated with the palatibility of fresh and cured products and economic losses during processing and distribution. To the consumer it means tenderness, juiciness, and flavor of the cooked product. Summarizing the literature on PSE pork, he stated that the fresh and cured meat from PSE animals is less desirable by palatability tests than normal pork muscle. The cooking loss and shrinkage was higher in PSE pork being some 15% greater than that of normal muscles. During processing PSE hams were found to shrink from 3 - 5% or more due to exudation. The gelatinous cookout was from 4 - 8%higher while yields were about 3% lower in fully cooked hams, 6% in canned hams, 10% in Canadian bacon, and 2% for smoked butts. The PSE retail cuts exuded large quantities of juice, resulting in an undesirable accumulation of liquid. Beecher et al. (1965b) found that in longitudinal view, the Longissimus dorsi would appear quite normal in the middle while the ends would frequently be PSE in gross morphology. In cases such as this, the transition from a normal to PSE character would be very gradual or quite abrupt. This type of situation is indeed critical in view of all PSE products because the muscle will not take on an ac-
ceptable cured meat color due to the lack of myoglobin. PSE pork is on the average, less tender than normal muscle, and some investigators have attempted to characterize the connective tissue in order to explain this fact. McClain et al. (1969) found normal epimysial connective tissue was lower in salt soluble collagen than was PSE tissue. This was also true of acid soluble collagen, but this value was nonsignificant. The authors explained that the greater content of salt soluble or newly synthesized tropocollagen in PSE muscle could indicate increased collagen anabolism.

CHAPTER III

MATERIALS AND METHODS

Forty market weight Yorkshire hogs of similar nutritional and genetic background were used in this characterization of the Myodegeneration Syndrome. The hogs were obtained from the Oklahoma State Agricultural Experiment Station and sorted on the basis of their responses to exercise on a treadmill moving at one mile per hour. The treadmill was designed to produce a stress situation in order to induce the clinical disease in susceptible animals.

Slaughter

At the end of the exercise period, the hogs were transported to the meat laboratory and slaughtered. During the procedure each animal was stunned with a Cervin electrical tool (220 volt), shackled by one leg, raised from the floor, and bled in the usual manner. As soon as the animal died, it was lowered to the floor and skinned in order that intramuscular temperature would not be affected by immersion in the scalding vat. Once the skinning operation was completed the hogs were raised, eviscerated, and rapidly split. As soon as splitting was completed a mercury thermometer was inserted into the Longissimus dorsi muscle at the level of the second lumbar vertebra. The thermometer remained in position for five minutes at which time the temperature was read and recorded. During this period the carcasses were rapidly weighed and placed

зU

into the holding cooler at a temperature of 34°F.

Sampling

As soon as the hogs were placed in the holding cooler, the sampling procedure was initiated. Chops were taken from the Longissimus dorsi beginning at the sixth thoracic vertebra for the zero hour (30 minutes post-mortem) sample and subsequently from every other vertebra, working posteriorly, at one, two, three, five, and 24 hours post-mortem. This procedure was followed in order to prevent dehydration or other physiological alteration of the loin due to excessive exposure to the exterior. Sides were also alternated on every other animal.

pН

The pH of the Longissimus dorsi muscle was determined with a Model 10 Beckman pH meter and a Corning semimicro combination electrode. As discussed above, samples were taken at zero hours (30 minutes), one, two, three, five, and 24 hours post-mortem. A representative sample of muscle was removed from the excised loin chop and comminuted. A ten gram aliquot of muscle was weighed and placed in 50 milliliters of distilled water. The solution was mixed thoroughly and allowed to stand for three minutes at which time mixing was repeated. Before reading the pH, the Beckman instrument was calibrated using two pHydrion buffers of pH 5.60 and 7.00. This same procedure was used for each of the sampling periods.

Histochemical Procedure

As soon as the zero hour sample was excised, two, one-half inch cores were removed from the Longissimus dorsi using a bore. Extreme care was taken to insure that the samples were representative and good cross sections. From the two, one-half inch cores were cut three, one-eighth inch slices which were mounted on metal specimen holders with Cryoform adhesive. This entire apparatus was then immersed in liquid nitrogen $(-170^{\circ}C)$ and retained there until all bubbling ceased. The samples were then placed in a SLEE type R Cryostat, covered with glass jars to prevent dehydration, and allowed to equilibrate to $-20^{\circ}C$. After the equilibration period the samples were transversely sectioned at a thickness of 16 microns while at $-20^{\circ}C$. The sections were mounted on glass slides and retained in cold tris buffer pH 7.4 until stained.

The staining procedure used in this investigation was described in detail by Engel (1967) and Cooper et al. (1969). Basically the procedure employed the Nicotinamide-Adenine Dinucleotide-Tetrazolium Reductase (NADH-TR) reaction. The technique called for reduced NADH₂ and Nitroblue tetrazolium both of which were obtained from Sigma Chemical Company. The two reagents were mixed in tris buffer, pH 7.4 and incubated at 36° C. Upon reaching the specified temperature, the slides were immersed in the reaction medium and incubated at 36° C for a period of thirty minutes at which time they were removed and run through an acetone series to remove excessive reagent. The slides were finally washed in distilled water and cover-slipped with glycerol jelly.

Fiber types were identified according to Cooper et al. (1969). In order to calculate the percent red, white, and intermediate types, fibers in three primary fasciculi were counted, using an American Optical microscope at 100 magnifications. To insure accuracy the fibers within the fasciculi were counted twice.

Histological Samples

Samples for degree rigor and fiber diameter were taken from the loin chops, excised five hours post-mortem, by means of a one-half inch bore. These were immediately placed in 10% buffered formalin. The cores were taken at five hours due to the fact that many of the muscles were at or near their ultimate pH and it was hoped that the best estimate of degree rigor could be obtained at this time. At 24 hours, the buffered formalin was changed to insure adequate penetration. All samples fixed in formalin were stored at 34°F until used.

Fiber Diameter

A small longitudinal section of the fixed sample was removed with extreme care, placed in a fresh 10% buffered formalin solution, and blended at a slow speed for one minute in a Waring Blender to dislodge, but not break the muscle fibers. After isolation each sample was checked using the microscope to insure that little or no damage occurred to the fibers during the isolation. The resulting suspension was placed in a glass bottle and maintained at $34^{\circ}F$ until the fibers could be measured. At the time of observation, a portion of the fiber suspension was poured into a two-inch diameter petri dish. The fibers were allowed to settle to the bottom and the petri dish was placed on the American Optical Microscope which was equipped with an ocular micrometer and a built-in light source.

The diameter of 10 fibers was measured for the one petri dish, then it was emptied and the process repeated until a total of 25 fibers had been measured. This entire process was again repeated, with the second 25 fibers constituting a duplicate. Therefore, a total of 50 fibers were measured for each sample. The only fibers measured were those which appeared in the field of a constant course and were at least the length of the field. All fibers were measured at their widest point.

Degree Rigor (Percent Kinkiness)

The degree rigor or percent kinkiness was observed at the same time as fiber diameter was measured. While the fibers were being microscopially observed, a subjective score for kinkiness was assigned to each fiber using a scale ranging from 1-7 depending upon the condition of the fiber. A weighted score for the 25 fibers was then calculated and converted to a percentage kinkiness as illustrated in Table I (Cagle et al., 1970).

Carcass Evaluation

The alternate side of pork not used for sampling was utilized for quality evaluation. Sides were removed from the holding cooler after 48 hours and weighed to determine chilled side weight. Carcass length was then determined with a tape measure running from the forward edge of the first rib to the tip of the puble bone. After these measurements had been taken, the side was broken down into its wholesale cuts. In order to determine the percent live and carcass lean cut yield, the ham, shoulder, and loin were closely trimmed of fat (one-eighth inch) and weighed. The loin was then cut at the tenth rib and a tracing was made to evaluate loin eye area. This parameter was measured with a compensating polar planimeter and the value recorded in square inches. Other evaluations of the loin included subjective appraisal of color, firmness, and marbling. Each descriptive term was associated with a number or

NO. OF FIBERS	CONDITION	SCORE	SUBTOTAL
1	STRAIGHT	1	1
8	STRAIGHT*	2	16
4	WAVY	3	12
6	WAVY*	4	24
2	TWISTED	. 5	10
1	TWISTED	6	6
3	KINKY	7	21
TOTAL 25			90
PERCENT KINKINE	$SS = \frac{90}{175^a} \times$	100 = 51	%

TABLE I

PROCEDURE FOR CALCULATION OF PERCENT KINKINESS

score. Color was evaluated in the following manner: Extremely pale (1), pale (2), slightly pink (3), moderately pink (4), bright pink (5), slightly dark (6), and dark (7). Firmness was evaluated in a similar manner: very soft (1), soft (2), slightly soft (3), average (4), slightly firm (5), firm (6), and very firm (7). Finally the degree of marbling was scored by the range: devoid (1), scant (2), slight (3), average (4), moderate (5), well (6), and abundant (7).

Percent Moisture

A one-inch steak was cut from the loin at the tenth rib comminuted and then homogenized in an Omnixer to acquire a uniform sample. During the homogenization procedure, the sample container was constantly cooled with ice to avoid leaching of fat. The homogenized sample was used for both percent moisture and fat determinations. Samples were placed in a tightly capped small mouth jar and stored at 34°F until used.

To determine the percent moisture, a two gram sample was weighed on a Mettler type H-5 balance into a tared planchet. Weights were determined to the nearest ten thousandth gram, however, the last digit was an estimate. Samples were than placed in a drying oven pre-set at 105°C and allowed to dry for 24 hours. Samples were then pre-weighed and the percent moisture determined by the following equation:

Wet Sample Weight x 100 = Percent Moisture

In every sample, care was taken to insure that the planchets were dried and that they were handled with forceps. Duplicates were also run to assure accuracy.

Ether Extract (Percent Fat)

Samples for ether extract (percent fat) were from the same sample as used for moisture. In this case a four gram sample was weighed by means of the Mettler balance into a glass thimble. Each thimble had the bottom opening stuffed with cotton to prevent sample loss and was dried for 24 hours to prevent error in weighing due to moisture. After the sample was placed in the thimble the top was covered with cotton and placed in a tared beaker to dry overnight. After drying, the thimbles were placed in a desiccator to prevent moisture absorption by the sample. The beakers were filled to one-fourth their volume with ether and then placed on the ether extraction unit (Goldfisch apparatus) with the thimbles. As the ether was heated, it evaporated and subsequently condensed, passing through the thimble, and extracting the fat in the sample. The apparatus was left in operation for 24 hours at which time the thimbles were removed and the ether was evaporated and collected in small vessels. The fat was left behind in the beaker which was subsequently dried to ensure total evaporation of the ether. The beakers and dried fat were again weighed on the Mettler balance and the percent fat calculated in the following manner:

<u>Amount of Fat in Sample</u> x 100 = Percent Fat Sample Weight

As in the percent moisture, duplicates were run to assure accuracy.

Shear Force

Two, two-inch chops were cut from the region of the tenth rib during the evaluation of each carcass. These chops were tagged with a number and immersed in Frymax oil at 250° F. The chops were cooked to an internal temperature of 150° F as determined by a meat thermometer placed in the geometric center of each chop. The samples were then removed to a 34° cooler and retained there for 24 hours to assure that they were of a uniform temperature. Two, one-inch cores were removed from each chop by means of an electric drill fitted with a one-inch bore. Each core was sheared three times using the Warner-Bratzler shear instrument and the value recorded in pounds of shear force.

Analysis of Data

In order to best analyze the data from the 40 hogs, the products were grouped by their zero hour (30 minutes) pH readings. Animals having muscles with low initial pH ranged from pH 5.00 - 5.99 were placed in Group I, muscles having intermediate initial pHs ranging from 6.00 -6.39 were placed in Group II and finally muscles having high initial pHs of 6.40 - 6.82 were placed in Group III. A similar arrangement was used by Kastenschmidt et al. (1966). Data may be studied on the individual animals within the three groups as given in the Appendix (Tables II, III, and IV). Animals with a low initial pH were termed rapid glycolyzing, abnormal, or extremely Myodegeneration Syndrome susceptible. Those with intermediate initial pHs may be called intermediate or Myodegeneration Syndrome susceptible while those having a high initial pH were identified as being normal. In addition, all parameters measured for each carcass were averaged in the three groups and reported in the Appendix (Table V).

CHAPTER IV

RESULTS AND DISCUSSION

pH

Seven carcasses having a zero hour pH ranging from 5.00 - 5.99 were designated as having low initial pH and termed "abnormal or extremely susceptible to Myodegeneration Syndrome". Sixteen carcasses with zero hour pH ranging from 6.00 - 6.39 were termed "intermediate or Myodegeneration Syndrome susceptible" and designated as having intermediate initial pH values (Figure 1). Finally, seventeen carcasses with zero hour pH ranging from 6.40 - 6.82 were termed "normal" and designated as having high initial pH. Tissue from carcasses termed intermediate and normal exhibited a normal pH fall after death; however, tissue from carcasses with a high initial pH took an average of three hours to reach a pH below 6.00. Muscle from carcasses designated intermediate exhibited a faster pH decline, reaching a pH below 6.00 in less than one hour. Ultimate pH was lower for intermediate carcasses (5.51) than normal carcasses (5.63) at 24 hours post-mortem.

The hogs termed "extremely Myodegeneration Syndrome susceptible" gave evidence of a rapidly glycolyzing muscle which was illustrated by a drastic pH fall, occurring shortly before, or immediately after death. The pH at only 30 minutes post-mortem (zero hour) had an average of 5.60, however, one animal in this group (16-7) had a pH of 5.35. The evidence of an abnormally rapid glycolysis is consistent with the findings of

Ja



Figure 1. Change in pH for Porcine Muscle

Briskey et al. (1961a), Sayre et al. (1961), Wismer-Pedersen (1961), Briskey (1964), Bendall et al. (1963), and Koch (1969) in their investigations with PSE pork.

Because of the extremely low readings, the pH curve of the abnormal muscle tended to resemble a straight line, with the greatest drop in pH being between one and two hours post-mortem. In intermediate and normal muscle, the most rapid pH fall was between zero and one and one-half hours. The ultimate pH in many cases, was reached at two hours postmortem, with only a slightly lower value at 24 hours. In some muscles, the pH tended to rise slightly at three hours (Appendix, Table II), accounting for the higher average at this time. These findings agreed well with Topel et al. (1968) and Briskey et al. (1961b).

Pre- and Post-Slaughter Characteristics

Those animals with an intermediate and high initial muscle pH exhibited "normal" attributes both before and during slaughter. However, carcasses with extremely low muscle pH values immediately postmortem were characterized by symptoms described by Topel et al. (1968). These included gasping, open-mouth breathing, tail quivering, and elevated temperatures. Such symptoms would occur whenever the animal was exposed to stress, which needed to be no more severe than lot movement, loading, or medical treatment. The animals soon became reluctant to move and if the stress was continued, the hogs would die. Most of these symptoms occurred, from start to finish, in less than five minutes.

It is postulated that the gasping, open-mouth breathing is an attempt by the animal to overcome anoxic conditions (Kastenschmidt et al., 1966) in the tissues caused by a reduced blood supply (Cooper et

al., 1969; Romanul, 1965, Schayer, 1964; Kastenschmidt et al., 1968) and/ or poor oxygen storage due to a lack of myoglobin (Lawrie, 1952; Chinoy, 1963). The anoxic condition also accounted for the drastic drop in pH (Judge, 1963), for without oxygen, the animals are forced to metabolize anaerobically. Essentially, this means that glycogen will be converted to lactic acid, rather than the end products of glycolysis during aerobic conditions, such as pyruvate, which are shuttled to the oxidative pathways for synthesis of adenosine triphosphate (ATP). It is then, this rapid and immediate production of lactic acid which is responsible for the drastic fall in pH.

Shortly after death, the carcasses with the lowest pH (see Appendix, Table II) displayed extensive twitching of the muscle tissue, further aiding the buildup of lactate. These data are consistent with the findings of Judge (1969), Hallund et al. (1965), and Forrest et al. (1966). This led to a rapid and complete rigor mortis, developing 15 minutes post-mortem. Intramuscular temperatures taken at 15 minutes post-mortem revealed that carcasses with low initial pH had temperatures ranging from 106.3 to 106.9, with an average temperature of 106.6°F (Figure 2). Elevated temperatures such as these have also been reported by Topel et al. (1968). Carcasses with intermediate initial pH had a mean muscle temperature of 105.3, while those with high initial pH had an average temperature of 105°F. It is difficult to explain the elevated temperatures of the abnormal animals; however, the author postulates that they may be due to an underdeveloped circulatory system in proportion to muscle mass, and thus would not be able to dissipate the metabolic heat. There is also the possibility that the elevated temperatures may reflect inflammation caused by a degradation of the muscle tissue due to repeated



Figure 2. A Comparison of Intramuscular Temperature With Porcine Muscle pH

depressions of pH following subjection to stress situations. Such low pH could result in damage to the muscle because of protein denaturation, especially the sarcoplasmic proteins (Cassens et al., 1963; Greaser et al., 1969b; Bendall et al., 1962).

Carcass Evaluation

The average live weights of the three groups were as follows: Low initial pH, 219.17; intermediate initial pH, 212.31; and high initial pH, 226.00 pounds (Figure 3). There does not appear to be any relationship of live weight to the susceptibility to Myodegeneration Syndrome. Chilled side weight of the three groups also did not slow any relationship (Figure 4). An examination of the average carcass lean cut yield in Figure 5 shows that both the abnormal and intermediate groups had higher yields than did the normal group. All yields were based on closely trimmed hams, boston butts, picnics, and loins. Averages for carcass lean cut yields were 63.44%, 62.40%, and 57.43% for carcasses with low, intermediate, and high initial pH values, respectively. Although averages were naturally lower, almost the identical result occurred when the live lean cut yields were compared (Figure 6). Carcasses with low initial pH averaged 42.74%; intermediate initial pH, 42.11%; and high initial pH, 39.17%. These data serve to illustrate that the Myodegeneration Syndrome susceptible animals possess more total muscling in their lean cuts than do "normal" animals. This is in agreement with Topel et al. (1968) who found that all animals which were stress-susceptible were very muscular. Not only did carcasses with low and intermediate initial pH have a larger percentage of lean, but as shown in Figure 7, they also possessed a smaller carcass length. Carcasses with



Figure 3. A Comparison of Live Weight With Porcine Muscle pH



Figure 4. A Comparison of Chilled Side Weight With Porcine Muscle pH



Figure 5. A Comparison of Carcass Lean Cut Yield With Porcine Muscle pH



Figure 6. A Comparison of Live Lean Cut Yield With Porcine Muscle pH

low initial pH had an average length of 29.92 inches; intermediate, 30.33 inches; and high, 32.10 inches.

Evaluation of the Longissimus dorsi

The average loin eye area measured at the tenth rib was larger in carcasses with low and intermediate initial pHs than in carcasses with a high initial pH. As shown in Figure 8, area means were 5.58, 5.53, and 4.78 square inches for carcasses with low, intermediate, and high initial pH values. This parameter was again indicative of a larger muscle mass in the abnormal and intermediate animals. It was further interesting that there was little, if any, variation in the weight of the Longissimus dorsi, although averages were slightly higher for carcasses with high and intermediate initial pH values (Figure 9).

Quite an obvious difference was observed in the subjective evaluation of the three groups which involved color, firmness, and marbling. The carcasses were scored on each quality attribute with a number ranging from one to seven. The lower the number, the less desirable the meat, from a visual standpoint. Each number corresponds to a degree of color, firmness, or marbling. These scores were determined by evaluation of the Longissimus dorsi at the tenth rib. As a general guideline, pork is the most desirable when it has a pink color, average firmness, and average to modest marbling.

Although carcasses with low initial pH had a larger lean cut yield and loin eye area, the musculature was of a very poor quality, reflecting the effects of a high temperature and low pH. Meat from these carcasses scored an average two for color, two for firmness, and 1.5 for marbling (Figure 10). These values correspond to the descriptive terms



Figure 7. A Comparison of Carcass Length With Porcine Muscle pH



Figure 8. A Comparison of Loin Eye Area With Porcine Muscle pH



Figure 9. A Comparison of Longissimus dorsi Weight With Porcine Muscle pH



Figure 10. A Comparison of Three Quality Characteristics With Porcine Muscle pH

pale color, soft texture, and very scant marbling. In addition, meat from this group exuded a large quantity of fluid, both in the raw and thawed form. The loss of water holding capacity further reflects damage to the muscle tissue resulting from the rapid pH fall (Hamm, 1960; Briskey et al., 1961a; Bendall et al., 1962). The pale coloration was not uniform along the length of the loin, as in some animals the center would appear relatively normal, while the ends would display an extremely pale color. This finding agreed well with Beecher et al. (1965b). Needless to say, pork from these abnormal carcasses would be undesirable to the consumer. However, the problem is further magnified by the meat's inability to develop a cured meat color due to the lack of myoglobin. Therefore, the merchandising of meat from Myodegeneration Syndrome animals in an acceptable, as well as, in a profitable form becomes extremely difficult.

Meat from carcasses with intermediate initial pH was somewhat better in quality and may be described as being moderately pink in color (4), average in texture (4), and slightly marbled (3.37). With a high initial pH, meat was of excellent quality, being moderately pink in color, slightly firm in texture, and slightly marbled. A comparison of "intermediate and normal animals" (Figure 10) shows that carcasses with high initial pH had a higher color and marbling score. Again these data serve to illustrate the undesirable effect of a drastic change in internal environment on the musculature as opposed to a more gradual alteration. The results were so dramatic, that from the data presented here, it was possible to predict the gross morphology of the musculature solely by initial pH.

Chemical analysis of the Longissimus dorsi at the tenth rib was

executed to determine the percent fat and moisture. In each instance, duplicates were run to check accuracy. A four gram homogenized sample was used for ether extract (Goldfisch apparatus) or percent fat and a two gram homogenized sample for percent moisture which was determined by oven drying for 24 hours. The average percent fat and moisture values for each group is illustrated in Figure 11. Both the "abnormal" and "intermediate" groups had a lower percentage of fat and a higher percentage of moisture than the "normal" animals. Values were 1.73, 1.88, and 2.32% fat and 74.56, 74.14, and 74.04% moisture for carcasses with low, intermediate, and high initial pH values, respectively. The indirect relationship of fat content to moisture was expected as it is a well established fact that as the percentage of fat increases, the moisture decreases due to the low moisture content of the fat. One must bear in mind that ether extract is a reliable measure of intramuscular fat or marbling. Therefore, reviewing the data illustrated in Figure 10 one can see that the percent fat agrees well with the visual appraisal of marbling. These data do not agree well with the finding of Sink et al. (1967) who found that there were no differences between normal and PSE muscle in total lipid or fatty acid content. It is hard to believe that no differences were found in light of the degree of anaerobic metabolism which occurred in the abnormal muscles. Realizing that the synthesis of fat is an oxidative process, an anaerobic environment such as occurs in the abnormal muscles, would limit the synthesis of lipid. In addition, any fat that was synthesized would most likely be utilized by aerobic fibers (those in close association with capillaries) for energy production. There is also the possibility of a hormonal imbalance in which a release of epinephrine (adrenaline) resulted in a mobilization



Figure 11. A Comparison of Ether Extract and Moisture Values With Porcine Muscle pH

of free fatty acids and lipoproteins (Shafrir et al., 1960; Cunningham et al., 1963).

Histological examinations of the Longissimus dorsi on each carcass were performed. Samples were again taken at the level of the tenth rib and placed in a 10% buffered formalin solution in which they were fixed... for 72 hours. Fibers were then isolated using a Waring blender with reversed blades at slow speeds. These precautions were taken to prevent undue damage to the fibers. Muscle fibers were then examined for fiber diameter and degree rigor (percent kinkiness). Data presented in Figure 12 shows that there was no relationship between the susceptibility of the hogs to Myodegeneration Syndrome and fiber diameter. These data do not agree with the findings of Lister et al. (1970) who found that PSE fibers were larger than muscle fibers from normal animals.

An interesting relationship was found to exist between the degree rigor (percent kinkiness), shear force, and the susceptibility of the pigs to Myodegeneration Syndrome. The determination of shear force was run in conjunction with the histological examination since a relationship has been shown to exist between these two parameters. As is presented in Figure 13, carcasses with low and intermediate initial pHs had a greater degree rigor and shear force than carcasses with high initial pH. Values were 22.83, 21.62, and 19.27 pounds required for shearing and 55.31, 52.20, and 43.91 percent for degree rigor for carcasses with low, intermediate, and high initial pH, respectively. These data were anticipated since the rapid lactate production and pH fall are conducive to a high degree of rigor. Therefore carcasses exhibiting low and intermediate initial pH values would be less tender since the higher percent rigor and shear values reflect a decreased tenderness. It may therefore



Figure 12. A Comparison of Fiber Diameter With Porcine Muscle pH



Figure 13. A Comparison of Degree Fiber Rigor and Muscle Shear Values With Porcine Muscle pH

be stated that animals which exhibit the Syndrome possess meat which is less desirable in both the raw and cooked form.

In order to better understand the nature of the rapidly glycolyzing muscle of the abnormal animals and how it differed from the intermediate and normal animals, a histochemical procedure was used to determine the oxidative potential of the muscle tissue. The method utilized the procedure described by Engel (1967) and Cooper et al. (1969). The technique employs the co-factor Nicotinamide-Adenine Dinucleotide in a reduced form (NADH₂) and 2,2'-di-p-nitropheny1-5,5'-dipheny1-3,3'-(3,3'dimethoxy-4,4'-biphenylene) ditetrazolium chloride, better known as Nitro-blue tetrazolium (TR). The reaction works by taking advantage of the oxidation-reduction reactions which take place in the muscle tissue. Oxidation refers to a loss of electrons, therefore, the substance donating hydrogen atoms or electrons is oxidized and the one accepting is re-These reactions, catalyzed by enzymes (dehydrogenases), occur duced. within the mitochondria and are coupled to a series of electron donors and acceptors which act in a chain-like fashion. Histochemical reactions involving Nicotinamide-Adenine Dinucleotide-Tetrazolium Reductase (NADH-TR) techniques utilize the citric acid cycle or more specifically the following reaction:

Succinic Acid < Succinic dehydrogenase > Fumaric Acid FAD FADH

Here, succinate, catalyzed by the enzyme succinic dehydrogenase, is oxidized to form fumaric acid. In the process, a hydrogen atom or electron is given off and picked up by the oxidized co-factor Flavin Adenine Dinucleotide (FAD) which subsequently becomes reduced (FADH). The func-

tion of the dehydrogenase is to transfer the electron to the co-factor FAD which would, in turn, transfer the hydrogen to molecular oxygen.

The color development of the NADH-TR is due solely to the tetrazolium reductase. These salts, characterized by a heterocyclic ring structure containing one carbon and four nitrogen atoms, are colorless or pale yellow, and are readily reduced to form intensely blue-colored, water insoluble formazans. However, it is important to note that the tetrazolium salts do not accept electrons directly from the dehydrogenases. Since NAD has a higher oxidative potential than FAD, it will pick up the electrons first from the dehydrogenase (Figure 14). These electrons are then transferred to the carrier with the next highest potential, FAD, which in turn, transfers the hydrogen atoms to the tetrazolium salt. The tetrazolium is reduced to form the insoluble, blue diformazan crystals.

Any muscle fibers, then, which were aerobic metabolizing would stain a uniform, intense blue, and thus were designated as red fibers. Intermediate fibers were characterized by a dark, blue, subsarcolemmal deposition of diformazan with a light or clear center. One must keep in mind that intermediate fibers are either aerobic or anaerobic, depending upon conditions within the muscle. Therefore, they would have a smaller amount of succinic dehydrogenase to enter into the reaction. White or anaerobic fibers, naturally would stain negatively (no color development). One may also explain the reaction on the basis of mitochondrial content in light of the finding of Gauthier (1970) who established that red fibers had an abundance of these organelles whereas intermediate fibers had a smaller number than red but more than white fibers. Relatively few mitochondria are found in the white fibers.





Referring to the photomicrographs (Figure 15), it may be seen that in sections of normal muscle (A), the fibers appeared to form a regular mosaic pattern within a fasciculus. Red fibers were in clusters of five to seven and were surrounded by intermediate fibers. This entire complex was surrounded by white fibers. Muscle from intermediate animals did not have the compact clusters as did the normal animals (photomicrograph B). The mosaic in this case was more irregular and the fibers were mixed rather than in definite groups. Rapidly glycolyzing muscle (photomicrograph C) had a very haphazard arrangement of fibers with an indefinite mosaic. In addition, giant fibers having a diameter ranging from 150 to 180 microns were found in all abnormal muscle. These would display a variable staining reaction with NADH-TR, sometimes appearing as intermediate fibers and at other times as white fibers. The giant fibers averaged between one and two percent of the total fiber population, but were found to a lesser extent in "intermediate" animals, and were nearly absent in those termed "normal". The presence of giant fibers was consistent with the findings of Cassens et al. (1969).

Analyzing the staining intensity of each fiber type was simply a matter of counting the amount of each type within a fasciculus. By this method, the percentage of red, white, and intermediate fibers was calculated and thus gave an estimate of the degree of aerobic or anaerobic metabolism in the muscle tissue.

An examination of Figure 16 shows that there were some interesting differences in fiber type among the three pH ranges. As may be noted, the percentage of white fibers was fairly consistent within the three groups with an average value of 65%. Greater differences occurred in the number of red and particularly intermediate fibers. Muscle with low



(A) Normal Muscle; NADH-TR; 32X



(B) Intermediate Muscle; NADH-TR; 32X



Figure 15. A Comparison of Fiber Distribution and Arrangement With Porcine Muscle pH



Figure 16. A Comparison of Fiber Type With Porcine Muscle pH

and intermediate initial pHs had fewer red fibers and more intermediate fibers than muscle with a high initial pH. Averages were 12.66, 13.87, and 16.26% for red fibers and 22.75, 22.01, and 16.40% for intermediate muscle fibers from animals with low, intermediate, and high initial pH values. If one combines the averages of red and intermediate fibers and expresses the sum as percent dark fibers, the averages are quite similar. Those animals with rapidly glycolyzing muscles had 35.41%; intermediate, 35.88%; and normal, 32.66% dark fibers. In actuality, of those fibers which could be classified as dark, only 36% were actually red fibers in muscle with low initial pH and 39% in muscle with intermediate initial In muscle from animals with high initial pH almost 50% of the total pH. dark fibers could be classified as red, but ranges within the group were between 50 and 75%. These data are consistent with the work of Cooper et al. (1969) who concluded that "the response of the stress susceptible animal to anoxia is due to the large number of intermediate fibers which were dependent upon aerobic metabolism, but unlike typical red fibers, they have especially high ATPase and phosphorylase activity, breaking down ATP and accelerating glycolysis to trigger a rapid glycolytic rate in the entire muscle. Additionally, even the regular white, and to a lesser extent, the regular red fibers have rather intense ATPase and phosphorylase activity and further contribute to the acceleration of these metabolic phenomena in the muscle of stress-susceptible animals." In conclusion, the characteristics of Myodegeneration Syndrome which developed in the skeletal musculature were most probably related to the fiber characteristics. This by no means is to say that fiber distribution was the sole contributor to the Syndrome for there may be many other important factors such as capillary distribution, myoglobin content, and hormonal imbalance which play major roles in the development of this abnormality.

-
CHAPTER V

SUMMARY AND CONCLUSIONS

Forty market weight Yorkshire hogs of similar nutrition and genetic background were obtained from the Oklahoma Agricultural Experiment Station herd. The animals were stressed using a treadmill moving at one mile per hour to induce clinical symptoms of Myodegenerations Syndrome in susceptible animals. Animals were then slaughtered in a standard manner. During the dressing procedure, intramuscular temperature was taken. As soon as the hot carcass weight was obtained, a loin chop was excised at the level of the thoracic vertebra for the 0 hour (30 minute) post-mortem pH sample. Two, one-half inch cores were removed from the chop, frozen in liquid nitrogen $(-170^{\circ}C)$, and subjected to the DPNH-TR staining technique for determination of the percent red, white, and intermediate fibers. Other pH samples were then taken at one, two, three, five, and 24 hours post-mortem from every other subsequent loin chop. Sides sampled hot were alternated on every other carcass. Chops sampled after 5 hours were used to obtain samples for measuring degree rigor and fiber diameter. At 48 hours, post-mortem the alternate side, not used for pH sampling, was subjected to evaluation including color, marbling, firmness, and loin eye area. These determinations were made at the tenth rib. Live and carcass lean cut yield was also determined. A one-inch chop was removed from the tenth rib and used for percent moisture and fat (ether extract). A shear evaluation was made using two, two-inch

thick chops from each carcass, taken in the region of the tenth rib. One-inch cores from the cooked chops were then sheared using the Warner-Bratzler shear instrument. Analysis of the data was accomplished by dividing the carcasses into three groups on the basis of 0 hour (30 minute) pH readings. Averages were then computed for each parameter within the three groups.

Carcasses described as having a low initial (30 minute) pH ranged from 5.35 - 5.99; intermediate initial pH 6.00 - 6.37; and a high initial pH 6.40 - 6.82. The pH curves of the three groups differed greatly. The abnormal carcasses also exhibited elevated intramuscular temperatures as compared to both the normal and intermediate groups. No relationships were found among groups in live or chilled side weight, however, both abnormal and intermediate animals had higher carcass and live lean cut yields. Subjective evaluation of the carcass revealed that abnormal animals had a larger Longissimus dorsi area, but that the musculature was of a very poor quality, being pale, soft, and possessing very scant marbling. Intermediate and normal muscle was characterized by a moderate, pink color, firm texture, and slight amount of marbling. The carcasses having muscle with low and intermediate initial pH were also found to have less fat and more moisture in the lean than those with high initial pH. Histological examination of fiber diameter revealed that there was no apparent relationship of this parameter to the Myodegeneration Syndrome. However, characterization of degree rigor (percent kinkiness) and shear force indicated that muscle with low and intermediate initial pH had a higher degree rigor and shear force than did that of high initial pH. These data indicated that Myodegeneration Syndrome susceptible animals had muscle that was less tender than normal animals.

Histochemical analysis revealed that the percentage of white fibers was fairly consistent between the three groups, averaging 65%. The percentage of red fibers was lower in abnormal animals than in normal. On the other hand, the percentage of intermediate fibers was much higher in tissue from animals with low and intermediate initial pH than tissue with high initial pH. When expressed on a total dark fiber percentage basis (red and intermediate fibers), only 36% of the fibers were found to be actually red in muscle with low initial pH, 39% in muscle with intermediate initial pH, and 50% in tissue with high initial pH.

It was not feasible from this study to state that Myodegeneration Syndrome is caused solely by an abnormal fiber distribution, although it certainly appeared to be a major contributor. Other areas must be explored, particularly the influence of the nervous and endocrine systems upon the development and differentiation of these fiber types. Another fruitful area of research may possibly be with the relationship of the circulatory system to the development of Myodegeneration Syndrome. Each of these topics has been discussed thoroughly in Chapter II in the section dealing with the "Muscle of the Abnormal Animal." One fact was apparent, however, Myodegeneration Syndrome had drastic implications upon the quality of the musculature and its ultimate usefulness as a food.

LITERATURE CITED

- Addis, P. B., H. R. Johnson, C. J. Heidenreich, H. W. Jones, and M. D. Judge. 1967. Effect of humidity level in a warm growing environment on porcine carcass composition and quality. J. An. Sci. Vol. 26, p. 705.
- Beatty, C. H., R. D. Peterson, and R. M. Bocek. 1963. Metabolism of red and white muscle fiber groups. Amer. J. Physiol. Vol. 204, p. 939.
- Beecher, G. R., E. J. Briskey, and W. G. Hoekstra. 1965a. A comparison of glycolysis and associated changes in light and dark portions of the porcine semitendinosus. J. Food Sci. Vol. 30, p. 477.
- Beecher, G. R., R. G. Cassens, W. G. Hoekstra, and E. J. Briskey. 1965b. Red and white fiber content and associated post-mortem properties of seven porcine muscles. J. Food Sci. Vol. 30, p. 969.
- Beecher, G. R., L. L. Kastenschmidt, R. G. Cassens, W. G. Hoekstra, and E. J. Briskey. 1968. A comparison of the light and dark portions of a striated muscle. J. Food Sci. Vol. 33, p. 84.
- Bendall, J. R. and J. Wismer-Pedersen. 1962. Some properties of the fibrillar proteins of normal and watery pork muscle. J. Food Sci. Vol. 27, p. 144.
- Bendall, J. R., O. Hallund, and J. Wismer-Pedersen. 1963. Post-mortem changes in the muscle of landrace pigs. J. Food Sci. Vol. 28, p. 156.
- Bray, R. W. 1966. Pork quality-definition, characteristics, and significance. J. An. Sci. Vol. 25, p. 839.
- Bresnick, E. and A. Schwartz. 1968. Functional Dynamics of the Cell. Academic Press. New York and London. p. 41.
- Briskey, E. J. and J. Wismer-Pedersen. 1961a. Biochemistry of pork muscle structure. I. Rate of anaerobic glycolysis and temperature change versus the apparent structure of muscle tissue. J. Food Sci. Vol. 26, p. 297.
- Briskey, E. J. and J. Wismer-Pedersen. 1961b. Biochemistry of pork muscle structure. II. Preliminary observations of biopsy samples versus ultimate muscle structure. J. Food Sci. Vol. 26, p. 306.

Briskey, E. J. 1964. Etiological status and associated studies of pale,

soft, exudative porcine musculature. Adv. Food Research, Vol. 13, p. 89.

- Buller, A. J., J. C. Eccles, and R. M. Eccles. 1960. Differentiation of fast and slow muscles in the cat hind limb. J. Physiol. Vol. 150, p. 399.
- Cagle, E. D. and R. L. Henrickson. 1970. Influence of slicing warm porcine muscle on fiber diameter, kinkiness and shear force. J. Food Sci. Vol. 35, p. 270.
- Cassens, R. G., E. J. Briskey, and W. G. Hoekstra. 1963. Electron microscopy of post-mortem changes in porcine muscle. J. Food Sci. Vol. 28, p. 680.
- Cassens, R. G., C. C. Cooper, and E. J. Briskey. 1969. Giant fibers in skeletal muscle of adult animals. Acta Neuropath. Vol. 12, p. 300.
- Chinoy, N. J. 1963. Histochemical localization of myoglobin in the pigeon breast muscle. J. Ani. Morph. and Physiol. Vol. 10, p. 74.
- Ciaccio, V. 1898. La découverte des muscles blancs et des muscles rouges, chez le lapin, revendiquée en taveur de S. Lorenzini. Arch. Ital. Biol. Vol. 30, p. 287.
- Cooper, C. C., R. G. Cassens, and E. J. Briskey. 1969. Capillary distribution and fiber characteristics in skeletal muscle of stresssusceptible animals. J. Food Sci. Vol. 34, p. 299.
- Cunningham, H. M., D. W. Friend, J. W. G. Nicholson, 1963. Effect of epinepherine on nitrogen and fat deposition of pigs. J. An. Sci. Vol. 22, p. 632.
- Doyle, A. M. and R. F. Mayer. 1969. Histochemistry and topology of the motor unit in the cat. Neurol. Vol. 19, p. 296.
- Dubowitz, V, and A. G. E. Pearse. 1960. A comparative histochemical study of oxidative enzyme and phosphorylase activity in skeletal muscle. Histochemie. Vol. 2, p. 105.
- Ebashi, S. and F. Lipmann. 1962. Adenosine triphosphate-linked concentration of calcium ions in a particulate fraction of rabbit muscle. J. Cell Biol. Vol. 14, p. 389.
- Ebashi, S. and M. Endo. 1969. Calcium ion and muscle contraction. Progr. Biophys. Mol. Biol. Vol. 18, p. 123.
- Engel, A. G. and L. W. Tice. 1966. Cytochemistry of phosphatases of the sarcoplasmic reticulum. I. Biochemical Studies. J. Cell Biol. Vol. 31, p. 473.
- Engel, W. K. and M. H. Brooke. 1967. Muscle biopsy as a clinical diagnostic aid. In: <u>Neurological Diagnostic Techniques</u>, Charles C. Thomas, Springfield, Ill.

- Folk, G. E., Jr. 1966. Introduction to Environmental Physiology. Lea and Febiger, Philadelphia.
- Forrest, J. C., L. L. Kastenschmidt, G. R. Beecher, R. H. Grummer, W. G. Hoekstra, and E. J. Briskey. 1965. Porcine muscle properties. B. Relation to naturally occurring and artifically induced variation in heart and respiration rates. J. Food Sci. Vol. 30, p. 492.
- Forrest, J. C., M. D. Judge, J. D. Sink, W. G. Hoekstra, and E. J. Briskey. 1966. Prediction of the time course of rigor mortis through response of muscle tissue to electrical stimulation. J. Food Sci. Vol. 31, p. 13.
- Forrest, J. C., R. F. Gundlach, and E. J. Briskey. 1968. A preliminary survey of the variations in certain pork ham muscle characteristics. Proc. 15th Ann. Res. Conf. Am. Meat Inst. Found. p. 81.
- Gauthier, G. F. 1970. The ultrastructure of three fiber types in mammalian skeletal muscle. In: <u>The Physiology and Biochemistry of</u> <u>Muscle as a Food</u>, Vol. 2 (ed. E. J. Briskey, R. G. Cassens, and B. B. Marsh), the University of Wisconsin Press, Madison, Milwaukee, and London.
- Greaser, M. L., R. G. Cassens, E. J. Briskey, and W. G. Hoekstra. 1969a. Post-mortem changes in subcellular fractions from normal and pale, soft, exudative porcine muscle. 1. Calcium accumulation and adenosine triphosphate activities. J. Food Sci., Vol. 34, p. 120.
- Greaser, M. L., R. G. Cassens, E. J. Briskey, and W. G. Hoekstra. 1969b. Post-mortem changes in subcellular fractions from normal and pale, soft, exudative porcine muscle. 2. Electron microscopy. J. Food Sci., Vol. 34, p. 125.
- Greaser, M. L., R. G. Cassens, W. G. Hoekstra, and E. J. Briskey. 1969c. The effect of pH-temperature treatments on calcium-accumulating ability of purified sarcoplasmic reticulum. J. Food Sci., Vol. 34, p. 633.
- Greaser, M. L., R. G. Cassens, W. G. Hoekstra, E. J. Briskey, G. R. Schmidt, S. D. Carr, and D. E. Galloway. 1969d. Calcium accumulating ability and compositional differences between sarcoplasmic reticulum fractions from normal and pale, soft, exudative porcine muscle. J. An. Sci., Vol. 28, p. 589.
- Gustafsson, R., J. R. Tata, O. Lindberg, and L. Ernster. 1965. The relationship between the structure and activity of rat skeletal muscle mitochondria after thyroidectomy and thyroid hormone treatment. J. Cell Biol. Vol. 26, p. 555.
- Hallund, O. and J. R. Bendall. 1965. The long-term effect of electrical stimulation on the post-mortem fall of pH in the muscles of landrace pigs. J. Food Sci., Vol. 30, p. 296.

Hamm, Reiner, 1960. The biochemistry of meat hydration. Adv. Food Re-

search, Vol. 10, p. 355.

- Hamm, R. and L. Kormendy. 1969. Transaminases of skeletal muscle. 3. Influence of freezing and thawing on the subcellular distribution of glutamicoxaloacetic transaminase in bovine and porcine muscle. J. Food Sci., Vol. 34, p. 452.
- Hasselbach, W. and M. Makinose. 1962. ATP and active transport. Biochem. Biophys. Res. Commun., Vol. 7, p. 132.
- Hedrick, H. B., F. C. Parrish, Jr., and M. E. Bailey. 1964. Effect of adrenaline stress on pork quality. J. An. Sci., Vol. 23, p. 225.
- Henneman, E., G. Somjen, and D. O. Carpenter. 1965a. Functional significance of cell size in spinal motoneurons. J. Neurophysiol. Vol. 28, p. 560.
- Henneman, E., and C. B. Olson. 1965b. Relations between structure and function in the design of skeletal muscles. J. Neurophysiol., Vol. 28, p. 581.
- Henson, J. B. and R. R. Rao. 1966. A comparison of serum creatine phosphokinase and serum glutamic oxalacetic transaminase in skeletal muscle necrosis. Can. J. Comp. Med. Vet. Sci., Vol. 30, p. 157.
- Hess, J. W., R. P. MacDonald, R. J. Frederick, R. N. Jones, J. Neely, and D. Gross. 1961. Serum creatine phosphokinase (CPK) activity in disorders of heart and skeletal muscle. Annals of Internal Med., Vol. 61, p. 1015.
- Howe, J. M., P. B. Addis, R. D. Howard, and M. D. Judge. 1969. Environment-induced adrenocortical lipid in "stress-susceptible" pigs. J. An. Sci., Vol. 28, p. 70.
- Ikemoto, N., F. A. Sreter, A. Nakamura, and J. Gergely. 1968. Tryptic digestion and localization of calcium uptake and ATPase activity in fragments of sarcoplasmic reticulum. J. Ultrastructure Research, Vol. 23, p. 216.
- Judge, M. D., R. G. Cassens, and E. J. Briskey. 1967. Muscle properties of physically restrained stressor-susceptible and stressor-resistant porcine animals. J. Food Sci., Vol. 32, p. 565.
- Judge, M. D., J. C. Forrest, J. D. Sink, and E. J. Briskey. 1968. Endocrine related stress responses and muscle properties of swine. J. An. Sci., Vol. 27, p. 1247.
- Judge, M. D. 1969. Environmental stress and meat quality. J. An. Sci., Vol. 28, p. 755.
- Karpati, G. and W. K. Engel. 1967. Neuronal trophic function. A new aspect demonstrated histochemically in developing soleus muscle. Arch. Neurol., Vol. 17, p. 542.

- Kastenschmidt, L. L., G. R. Beecher, J. C. Forrest, W. G. Hoekstra, and E. J. Briskey. 1965. Porcine muscle properties. A. Alteration of glycolysis by artificially induced changes in ambient temperature. J. Food Sci., Vol. 30, p. 565.
- Kastenschmidt, L. L., W. G. Hoekstra, and E. J. Briskey. 1966. Metabolic intermediates in skeletal muscles with fast and slow rates of post-mortem glycolysis. Nature, Vol. 212, p. 288.
- Kastenschmidt, L. L., W. G. Hoekstra, and E. J. Briskey. 1968. Glycolytic intermediates and co-factors in "fast" and "slow-glycolyzing" muscles of the pig. J. Food Sci., Vol. 33, p. 151.
- Kastenschmidt, L. L. 1970. The metabolism of muscle as a food. In: The Physiology and Biochemistry of Muscle as a Food. (ed. E. J. Briskey, R. G. Cassens, and B. B. Marsh). The University of Wisconsin Press, Madison, Milwaukee, and London. p. 735.
- Kirsten, E., and R. Kirsten. 1969. High energy phosphates in a red and a white muscle of the rat. Pflugers Arch., Vol. 311, p. 209.
- Koch, D. E. 1969. The relationship of some biochemical and physiological factors to post-mortem changes in porcine muscle. Ph. D. Thesis. Michigan State University, East Lansing, Michigan.
- Lawrie, R. A. 1952. Biochemical differences between red and white muscle. Nature, Vol. 170, p. 122.
- Lewis, P. K., Jr., C. J. Brown, and M. C. Heck, 1962. Effect of stress on certain pork carcass characteristics and eating quality. J. An. Sci., Vol. 21, p. 196.
- Lister, D., R. A. Sair, J. A. Will, G. R. Schmidt, R. G. Cassens, W. G. Hoekstra, and E. J. Briskey. 1970. Metabolism of striated muscle of stress-susceptible pigs breathing oxygen or nitrogen. Am. J. Physiol., Vol. 218, p. 102.
- McClain, P. E., A. M. Pearson, J. R. Brunner, and G. A. Crevasse. 1969. Connective tissues from normal and PSE porcine muscle....I. Chemical characterization. J. Food Sci., Vol. 34, p. 115.
- Meyer, J. A., E. J. Briskey, W. G. Hoekstra, and K. G. Weckel. 1963. Niacin, thiamin, and riboflavin in fresh and cooked pale, soft, watery versus dark, firm, dry pork muscle. Food Tech., Vol. 17, p. 485.
- Moody, W. G. and R. G. Cassens. 1968. Histochemical differentiation of red and white muscle fibers. J. An. Sci., Vol. 27, p. 961.
- Morita, S., R. G. Cassens, and E. J. Briskey. 1969. Localization of myoglobin in striated muscle of the domestic pig; Benzidine and NADH₂-TR reactions. Stain Tech., Vol. 44, p. 283.
- Needham, D. M. 1926. Red and white muscle. Physiol. Rev., Vol. 6, p. 1.

- Ogata, T. 1960. The differences in some labile constituents and some enzymatic activities between the red and the white muscle. J. Biochem., Vol. 47, p. 726.
- Padykula, H. A. and G. F. Gauthier. 1970. The ultrastructure of the neuromuscular junctions of mammalian red, white, and intermediate skeletal muscle fibers. J. Cell Biol., Vol. 46, p. 27.
- Ranvier, L. 1874. De quelques faits relatifs a 1 histologie la physiologie des muscles stries. Arc. Physiol. Norm. et Path. 2, Ser. 1:5.
- Romanul, F. C. A. 1964. Enzymes in muscle. I. Histochemical studies of enzymes in individual muscle fibers. Arch. Neurol., Vol. 11, p. 355.
- Romanul, F. C. A. 1965. Capillary supply and metabolism of muscle fibers. Arch. Neurol., Vol. 12, p. 497.
- Sayre, R. N., E. J. Briskey, W. G. Hoekstra, and R. W. Bray. 1961. Effect of preslaughter change to a cold environment on characteristics of pork muscle. J. An. Sci., Vol. 20, p. 487.
- Sayre, R. N. 1962. Glycogen and glycolysis of porcine muscle in relation to its post-mortem physical properties. Ph. D. Thesis. University of Wisconsin, Madison.
- Schayer, R. W. 1964. A unified theory of glucocorticoid action. Perspectives Biol. Med., Vol. 8, p. 71.
- Schmidt, G. R., R. G. Cassens, and E. J. Briskey. 1970a. Changes in tension and certain metabolites during the development of rigor mortis in selected red and white skeletal muscles. J. Food Sci., Vol. 35, p. 571.
- Schmidt, G. R., L. L. Kastenschmidt, R. G. Cassens, and E. J. Briskey. 1970b. Serum enzyme and electrolyte levels of "stress-resistant" chester white pigs and "stress-susceptible" poland china pigs. J. An. Sci., Vol. 31, p. 1168.
- Shafrir, E., K. E. Sussman, and D. Steinberg. 1960. Role of the pituitary and the adrenal in the mobilization of free fatty acids and lipoproteins. J. Lipid Research, Vol. 1, p. 459.
- Sink, J. D., R. W. Bray, W. G. Hoekstra, and E. J. Briskey. 1967. Lipid composition of normal and pale, soft, exudative porcine muscle. J. Food Sci., Vol. 32, p. 258.
- Stein, J. M. and H. A. Padykula, 1960. Histochemical classification of individual skeletal muscle fibers of the rat. Amer. J. Anat., Vol. 110, p. 103.
- Szent-Gyorgyi, A. 1953. <u>Chemical Physiology of Contraction in Body and</u> <u>Heart Muscle</u>. Academic Press, New York, New York, p. 105.

- Tice, L. W. and A. G. Engel. 1966. Cytochemistry of phosphatases of the sarcoplasmic reticulum. II. In situ localization of the Mg-dependent enzyme. J. Cell Biol., Vol. 31, p. 489.
- Topel, D. G. 1968. Relation of plasma glucocorticoid levels to some physical and chemical properties of porcine muscle and the porcine stress syndrome. In: <u>Recent Points of View on the Condition and</u> <u>Meat Quality of Pigs for Slaughter</u>. (ed. W. Sybesma, P. G. van der Wal, and P. Walstra) Zeist, Holland.
- Topel, D. G., E. J. Bicknell, K. S. Preston, L. L. Christian, and C. Y. Matsushima. 1968. Porcine stress syndrome. Mod. Vet. Prac., Vol. 49, p. 40.
- Vester, J. W., G. Sabeh, R. H. Newton, H. B. Finkelhor, G. H. Fetterman, and T. S. Danowski. 1968. Muscle creatine phosphokinase in primary myopathies. Proc. Soc. Exp. Biol. Med., Vol. 128, p. 5.
- Wismer-Pedersen, J. and E. J. Briskey. 1961. Relationship of postmortem acidity and temperature. Food Tech., Vol. 15, p. 232.

Animal No.	Live Weight (15s.)	Chilled Side Wt. (1bs.)	Carcase Lean Cut Yield (X)	Live Lean Cut Tield (X)	Carcass Length (in.)	Muscle Tomp. (Deg. P.)	Loin Eye Area (In. ²)	Longissimus Dorsi WL. (1bs.)	Color Score (1-7)	Firmness Score (1-7)	Marbling Score (1-7)	Ether Extract Fat (%)	Koisture (X)	Fiber Diameter (Microns)	Degree Rigor (X)	Shear Value (1bs.)	NADE-TR Red Fibers (%)	NADH-TR White Fibers (%)	NADE-TR Intermed- iate Fibers (%)	l hr. pH	2 hr. pH	3 hr. pH	5 hr. pH	Initial pë (0 hr.)	Ultimate pH (24 hr.)
16-7	235	82.7	69.22	42.38	30.30	106.4	6.15	5.20	Ext. Pale	V. Soft	Devoid	1.90	74.39	58.80	23,60	16.40	7.40	78.60	14	5.25	5.20	5,25	5.15	5.35	5.21
17–10	226	82.3	57.84	42.12	29.20	106.9	5.51	4.80	(2) Pale	(2) Soft	(2) Scant	2.60	75.69	68.70	38.40	23.40	19.74	69.47	10,53	5.45	5.45	5.45	5.40	5.50	5.38
89-7	216	72.1	66.30	44.26	29.50	106.3	6.11	5.30	(3) S. Pink	(2) Soft	(2) Scent	0.75	74.38	78.50	63.14	30.51	12.90	64.71	22.28	5.50	5.45	5.48	5.48	5.55	5.48
93~5	182	58.5	67.69	43.52	28.70	106.8	4.67	4.00	(2) Pale	(2) Soft	(2) Scant	2.33	74.30	77.10	58.29	27.04	5.98	59.93	34.05	5.47	5.46	5.49	5.45	5.55	5.24
26-8	228	79.0	58.48	40.53	30.10	106.5	5.56	5.10	(2) Pale	(3) \$1. Soft	(2) Slight	1.52	73.80	71.80	81.50	22.14	12.63	75.79	11.57	5.60	5.58	5.54	5.40	5.67	5.37
154	228	81.5	61.10	43.68	31.70	106,4	5.50	5.20	Pale	Soft	Scant	1.30	74.77	79.80	16.70	17.50	12.84	35.69	51.46	5.98	5.45	5.45	5.30	5.99	5.20
11-3	Died												****												

TABLE	II
-------	----

LISTING OF PARAMETERS MEASURED FOR ANIMALS WITH LOW INITIAL pH

TA	BLE	III
114		エエエ

LISTING OF PARAMETERS MEASURED FOR ANIMALS WITH INTERMEDIATE INITIAL PH

Anisel	Live Weight	Chilled Side Wt.	Carcass Lean Cut	Live Lean Cut	Carcass Length	Muscle Zemp.	Loin Eye Areş	Longiesiaus Dorsi Wt.	Color Score	Firmness Score	Marbling Score	Ether Extract	Moisture	Fiber Diameter	Degres Rigor	Shear Value	NADH-TR Red Fibers	NADH-TR White Fibers	MADE-TR Intermed- iate Fibers	l hr. pH	2 hr. pH	3 hr. pH	5 hr. pH	Initial pH	Ultimate pH
No.	(1bs.)	(1bs.)	Yield (X)	Yield (%)	(in.)	(Deg. T.)	(In. ²)	(15)	(1-7)	(1-7)	(1-7) ·	Fat (%)	(%)	(Microns)	(I)	(1bs.)	(%)	(Z)	(%)					(0 hr.)	(24 hr.)
93-2	184	61.4	65.31	43.59	29.00	106.2	5.02	4.05	(4) H. Pink	(5) S1. Firm	A∀g.	1.71	74.60	70.50	52.55	22.83	15.41	57.43	27.15	5.60	5.46	5.50	5.43	6.00	5.40
90-4	200	68.6	62.26	42.40	29.20	106.8	6.13	5.10	(4) M. Piuk	(1) V. Soft	(2) Scant	1.22	74.42	79.60	82.57	18.86	8.44	44.21	43.35	5.45	5.55	5.40	5.40	6.00	5.30
90-7	171	60.2	67.11	47.25	28.00	105.4	6.59	4.80	N. Pink (2)	AVE. (3)	Slight (4)	1.12	74.60	80.70	56.85	25.85	5.17	63.64	31.10	5.72	5,40	5.43	5.00	6.00	5.43
32-4	245	61.40	58.72	39.02	31.20	105.4	5.64	5.40	Pale (3)	S. Soft	Avg.	3.91	73.00	73.70	58.00	20.47	19.33	66.14	14.53	5.71	5.66	5.63	5.60	6.02	5.50
2-7	190	63.00	67.62	44.84	30.20	102.0	4.74	4.60	S. Pink (3)	S. Soft (6)	Slight (4)	0.93	75.19	60.90	28.56	21.98	20.25	54.07	25.69	5.75	5.60	5.50	5.55	6.05	5.50
89-4	194	69.20	64.02	45.67	30.70	103.8	5.79	5.10	S. Pink (6)	Firm (6)	Avg.	1.44	73.74	78,80	82.00	25.44	11.24	58.67	30.85	6.05	6,00	6.10	5.85	6.10	5.55
25-4	214	71.90	61.61	41.40	30.40	105.0	5.61	5.10	S. Dark (4)	Firm (4)	Mod.	2.39	73.60	86.30	76.86	27.92	19.77	66.27	13.53	6.00	5.78	5.72	5.70	6.10	5.65
17-8	242	. 84.00	56.55	39.26	30.80	106.0	5.57	5.20	H. Pink	ATE.	Slight (7)	0.90	73.27	74.30	22.60	14.80	15.40	65.36	19.24	5.40	5.30	5.25	5.15	6.10	5.25
30-4	244	79.40	58.94	38.35	33.30	106.7	6.11	5.65	S. Pink (4)	Avg. (5)	Scant (4)	1.40	73.80	63.66	41.92	23.32	19.79	72.41	13.79	5.85	5.78	5.78	5.70	6.11	5.65
8 9- 1	210	71.20	63.90	43.33	30.30	105.2	6.15	5.55	M. Pink	S. Firm	Avg.	0.97	74.27	83.00	38.29	23.83	13.79	72.41	13.79	6.08	5.65	5.55	5.50	6.15	5.55
86-1	220	77.00	61.82	43.27	31.60	105.0	5.08	4.60	M. Pink (4)	S. Firm (3)	Slight (3)	1.76	73.72	71,70	62.86	21.73	8.83	63.47	27.73	6.10	6.00	5.60	5.60	6.20	5.50
94-5	187	57.80	71.63	44.28	29.50	105.1	5.02	4.00	M. Pink	S. Soft	Slight (2)	0.91	75.89	79.00	71.42	21.44	13.77	70.64	15.15	6.15	6.01	5.84	5.65	6.21	5.65
89-5	200	64.30	63-45	40.80	29.40	104.6	5.40	5.10	S. Pink (3)	Avg. (6)	Scant (6)	0.40	74.47	89.90	39.43	25.11	8.38	62.98	28.61	6.10	5.90	5.85	5.85	6.25	5.52
32-8	236	80.90	60.27	41.27	31.50	106.4	5.77	5.00	S. Pink	Firm (5)	Well (3)	3.85	73.80	65.20	39.43	15.27	13.54	66.94	19.51	6.14	6.04	5.90	5.75	6.32	5.74
25-7	230	77.80	57.07	38.61	30.00	106.3	4.94	4.70	Pink (5)	S. Firm (5)	Slight (5)	3.52	74.00	68,60	52.29	19.50	16.21	70.40	13.39	6.12	5.76	5.70	5.65	6.34	5.60
26-3	230	80.00	58.13	40.34	30.20	105.6	4.87	4.80	Brt. Pink	5. Firm	Slight	2.71	73.80	74.40	29.72	17.59	18.37	70.90	10.63	6.35	6.20	6.15	5.92	6.37	5.37

INDLE IV	
----------	--

LISTING OF PARAMETERS OF MEASURED FOR ANIMALS WITH HIGH INITIAL $\ensuremath{\mathtt{p}}\xspace{\mathsf{h}}$

	Live	Chilled	Carcass	Live	Carcass	Huscle	Loin Eye	Longissimus Dorsi	Color	Firmess	Marbling	Ether		Fiber	Degree	Shear	NADH-TR Red	NADH-TR White	NADH-TR Intermed-	l br.	2 hr.	3 hr.	5 hr.	Initial	Ultimate
Animel No.	Weight (1b∎.)	Side Wt. (1bs.)	Lean Cut Yield (%)	Lean Cut Yield (%)	Length (in.)	Temp. (Deg. Y.)	Area (In. ²)	Wt. (1bs.)	Score (1-7)	Score (1-7)	Score (1-7)	Extract Fat (X)	Hoisture (%)	Diameter (Microns)	Rigor (7)	Value (1bs.)	Fibers (%)	Fibers (X)	iste Fibers (Z)	pH	pH	рЕ	рЦ	рН (0 hr.)	pH (24 hr.)
86-2	190	64.60	62.85	42.74	29.70	104.4	5.09	4.10	(3) S. Pink	(5) S. Firm	(4) Avg.	2.79	74.38	79.40	52.86	22.19	12.96	63.07	23.97	6.29	6.02	5.85	5.77	6.40	5.55
35-1	244	82.60	58.23	39.43	32.50	104.7	5.31	5.40	5. Pink	firm (5)	Scant (2)	1.96	74.90	74.65	67.72	16.15	13.79	72.41	13.79	6.20	5.83	5.80	5,80	6.41	5.60
15-6	232	61.70	53.98	38.02	30.70	104.6	4.38	4.60	N. Pink	S. Firm	Scant (7)	1.30	72.75	73.80	21.00	17.42	14.88	68.03	17.08	5.84	5.55	5.60	5.55	6.42	5.60
26-15	240	80.40	56.09	37.58	31.60	106.4	4.30	4.70	M. Pink (5)	S. Firm (6)	Scant (5)	3.13	73.90	71.20	51.43	19.12	17.14	73.33	9.52	6.17	5.96	5.73	5.69	6.43	5.63
26-11	218	75.90	59.68	41.56	31.00	104-8	5.09	4.80	B. Pink (6)	F1r (7)	Med (3)	3.82	73.80	88.60	80.00	19.58	25.75	60,61	13.64	6.37	6.00	5.87	5.70	6.45	5.68
25-2	220	73,40	55.72	37.18	30,60	105.2	5.38	4.60	S. Dark (4)	J. Firm (5)	Med (2)	2.45	73.40	75.50	83.43	16.19	19.48	67.53	12.99	6.43	6.34	6.20	6.00	6.50	5.84
14-8	222	79.60	56.91	40.81	31.00	104.6	4.18	4.60	M. Pink (4)	S. Firm (4)	Scant (2)	2.02	72.75	70.98	18.60	16.50	13.54	68.37	18.09	5.96	5.83	5.62	5.55	6.52	5.62
28-8	245	79.00	55.06	35.51	32.00	105.3	4.23	4.60	H. Pink (6)	Avg. (6)	Scant (5)	1.20	73.90	74.50	42.00	22.20	14.29	72.22	13.49	6.39	5.93	5.85	5.82	6.54	5.65
25-10	222	73.90	58.05	36.65	31.00	105.4	4.90	5.30	S. Dark (4)	Fir (6)	Hod (5)	2.85	74.00	73.30	40.86	22,10	17.86	/5.00	7.14	6.42	6.33	0.31 4 44	6.31	6.56	5.77
15-7	226	/8.20	55.80	38.67	31.00	105.5	4.64	4.60	(6)	(5)	(5)	2.36	74.52	64 40	70.96	22.27	22 20	65 40	11.61	6.30	6 12	6.05	5 02	6.30	5.85
29-7	220	72.00	63.75	41.73	33.40	105.0	4.77	4.75	5. Dark (6) S. Dark	(6)	(3) Slight	2.34	76-10	76.70	64.00	28.87	10.87	67.39	21.73	5.96	5.95	5.82	5.80	6-60	5.78
20-9	235	78.70	56 47	38 44	37 30	104.0	4 69	A 20	(4) W Pink	(5)	(3) 511ebt	2.10	73.76	75.00	18.00	15.50	11.48	68.89	19.67	6.50	6.00	6.00	5.70	6.65	5.25
20-10	222	78,90	57.54	40.90	31.00	104.3	4.92	4.60	(3) 5. Pink	(4)	(3) Slight	2,80	73.70	68.40	20.40	17.20	67.62	15.35	17.04	6.42	6.25	5.56	5.52	6.65	5.55
18-9	220	75.50	55.10	37.82	31.60	105.0	4.04	3.80	(3) S. Piak	(6) Firm	(3) Slight	2.40	73.00	77.20	25.20	20.5	59.70	17.91	22.39	5.61	5.55	5.35	5.25	6.74	5.25
25-6	240	78.80	56.21	36.92	30.80	104.6	4.73	4.60	(4) B. Pink	(6) Firm	(5) Hod	3.55	73.40	79.40	52.86	15.00	62.23	14.36	28.35	6.67	6.53	6.46	6.44	6.82	5.90

	Live Weight (1bs.)	Chilled Side Wt. (1bs.)	Carcass Lean Cut Yield (X)	Live Lean Cut Yield (X)	Carcass Length (1n.)	Muscle Temp. (Deg. F.)	Loin Eye Area (In. ²)	Longissimum Dorsi Wt. (1bs.)	Color Score (1-7)	Firmness Score (1-7)	Marbling Score (1-7)	Ether Extract Fat (2)	Hoisture (Z)	Fiber Diameter (Microns)	Degree Rigor (%)	Shear Value (1bs.)	NADH-TR Red Fibers (%)	NADH-TR White Fibers (%)	NADE-TR Intermed- iare Fibers (X)	l hr. pH	2 hr. pH	3 hr. pH	5 hr. pH	Initial pH (0 hr.)	Ultimate pH (24 hr.)
Low Initial									(2)	(2)	(1.5)			· · ·											
pH pH 5.35-5.99	219.17	76.02	63.44	42.74	29.92	106.6	5.58	4.93	Pale	Soft	V. Scant	1.73	74.56	72.45	55.31	22.83	12.66	64.59	22.75	5.54	5.43	5.44	5.36	5.60	5.31
Intermediate Initial			69 40	62.11	10.33	105 1		4 00	(4)	(4)	(3.37)						10.07	<i>(</i>) 10							
High Toitial	414.31	11-10	02.40	44.11	30.33	+03.3	5.55	4.92	(4.2)	(5)	(3 40)	1.88	/4.14	15.02	52.20	21.02	12.6/	04.12	22.01	2.91	3.76	2.74	2.38	0.15	3.31
pH pH 6.40-6.80	226	77.01	57.43	39.17	32,10	105	4.78	4.77	H. Pink	S. Firm	Slight	2.32	74.04	74.98	43.91	19.27	16.26	67.34	16.40	6.14	6.03	5.90	5.82	6.55	5.63

TABLE V

LISTING OF AVERAGES OF PARAMETERS MEASURED FOR ANIMALS IN THE THREE INITIAL pH RANGES

VITA Stanley Nathan Falk

Candidate for Degree of

Master of Science

Thesis: A CHARACTERIZATION OF MYODEGENERATION SYNDROME IN PORCINE MUSCLE

Major Field: Food Science

Biographical:

- Personal Data: Born in Little Rock, Arkansas, July 31, 1948, the son of Stanley E. and Jean C. Falk. Married Terrie Darlene Gregory, May 29, 1971.
- Education: Graduated from Hall High School, 1966. Received Bachelor of Science degree June, 1970, from the University of Arkansas at Little Rock with a major in Biology.
- Professional Experience: Undergraduate research and Teaching Assistant in Biology 1968-70 (UALR). Graduate Research and Teaching Assistant in Food Science, Oklahoma State University, 1970-71.
- Professional Organizations: Member of Institute of Food Technologists.