THE RELATIONSHIP OF SOME ANATOMICAL AND PHYSIOLOGICAL CHARACTERISTICS OF SUS DOMESTICUS TO POSTMORTEM MUSCLE PROPERTIES

Thesis for the Degree of Ph. D.
MICHIGAN STATE UNIVERSITY
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1969

This is to certify that the

thesis entitled

THE RELATIONSHIP OF SOME ANATOMICAL AND PHYSIOLOGICAL CHARACTERISTICS OF SUS DOMESTICUS TO POSTMORTEM MUSCLE PROPERTIES

presented by

J. B. Weatherspoon

has been accepted towards fulfillment of the requirements for

Ph.D. degree in Food Science

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Date Dec. 19, 1969

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ABSTRACT

THE RELATIONSHIP OF SOME ANATOMICAL AND PHYSIOLOGICAL CHARACTERISTICS OF SUS DOMESTICUS TO POSTMORTEM MUSCLE PROPERTIES

By J. B. Weatherspoon

The cardiovascular system of the pig has been implicated in the etiology of pale, soft and exudative (PSE) musculature (Merkel, 1968). The immediate cause of PSE muscle is attributable to the rapid pH fall occurring postmortem while muscle temperature is still high (body temperature). This study was designed to measure some of the parameters associated with metabolic potential of muscle tissue prior to slaughter and immediately postmortem in two separate experiments. Three ham muscles (rectus femoris, semimembranosus and gluteus medius) of 79 market-weight pigs, representing five breeds (Poland China, Landrace, Chester White, Hampshire and Yorkshire), were included in this study. Two quality groups (normal and low quality) of 5 pigs each were selected within each breed, except for the Chester Whites since no low quality pigs were available for this breed.

Blood pressure, heart and respiratory rate, whole blood pH and hematocrit were measured on anesthetized pigs in Experiment I while heart rate and function were measured on conscious pigs in Experiment II. Total myoglobin and its derivatives (Experiment I) and anserine and carnosine content (both experiments) were determined on samples excised from the three ham muscles 30 minutes postmortem. The right ham of each pig was perfused with India ink to identify capillaries. Capillary and muscle fiber densities were determined.

The muscle fiber types were identified by succinic dehydrogenase (SDH) activity in Experiment I and SDH and phosphorylase activity in Experiment II and each was expressed as percent of total number and area.

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Skeletal muscle, myocardial and nerve tissues were examined histologically for evidence of degenerative changes and the degree of degeneration in the skeletal muscles was correlated with 45 min pH and 24 hr transmission values.

No significant correlations were found between any of the antemortem parameters and 45 min pH. Additionally, there were no significant ($P < .05$) differences between quality groups. Myoglobin data indicated that differences existed between muscles and breeds in their potential to utilize oxygen from myoglobin.

The number (percent of total fibers) of red fibers in the rectus femoris muscle was significantly ($P < .05$) correlated with 45 min pH and positive correlations were observed in all three muscles between these two characteristics. Low quality pigs had significantly ($P < .05$) larger mean muscle fiber sizes than normal pigs. Red fibers showed the greatest increase in size. Low quality pigs also had fewer capillaries (40-45%) in their muscles than normal pigs. The decreased capillary density (capillaries per square mm) in low quality muscles undoubtedly reduced the functional efficiency of the capillary bed and this situation was further aggravated by the increased fiber size. The net result of this dilution of the capillary bed was most pronounced in the red and intermediate fiber areas of the muscle, thus the aerobic potential was affected to the greatest extent. The reciprocal staining technique (Experiment II) was more effective in identifying the metabolic activity of each fiber type. The low quality Yorkshire pigs (Experiment II) had significantly less carnosine than the other quality groups. The Landrace pigs had significantly more carnosine than the other breeds in Experiment I and there were no differences between quality groups in Experiment II.

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Histological examination of the muscles in Experiment II showed some evidence of degeneration. The degenerative changes were observed more frequently and the observations more severe in the semimembranosus and gluteus medius muscles than in the rectus femoris. In addition, two pigs (Yorkshires) which showed moderate degenerative changes also showed some loss of myelin of the sciatic nerve, loss of phosphorylase reactivity (especially in the semimembranosus muscle) electrical axis deviation and the formation of long nuclear chains in the myocardium. The degree of muscle degeneration was more closely correlated with transmission values than with 45 min pH. Additionally, these data indicate that the ultimate muscle quality was more highly related to the physiological and anatomical parameters studied when both 45 min (postmortem) pH and 24 hr transmission values were used to categorize the pigs into quality groups.

The results of this study indicate that ultimate muscle qualitative properties were associated with decreased capillary density, increased fiber size per unit of muscle and with myopathic changes in the skeletal and cardiac muscle tissues. These myopathic observations were accompanied by other cardiac and neural anomolies.

THE RELATIONSHIP OF SOME ANATOMICAL AND PHYSIOLOGICAL CHARACTERISTICS OF *SUS DOMESTICUS* TO POSTMORTEM MUSCLE PROPERTIES

By

J. B. Weatherspoon

A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Department of Food Science

1969

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ACKNOWLEDGMENT

The author wishes to express his appreciation to his major professor, Dr. R. A. Merkel, for his guidance throughout this research study and in the preparation of this manuscript. The author also wishes to thank Drs. E. J. Benne, R. E. Carrow, A. M. Pearson and R. K. Ringer for serving as members of his guidance committee.

Special thanks are extended to the faculty, staff and fellow graduate students in the Food Science and Animal Husbandry Departments for their encouragement and assistance throughout this research study.

The author is also indebted to the American Meat Institute for financing a portion of the research.

The author wishes to express his gratitude to his parents for their encouragement to undertake graduate study.

Lastly, the author expresses his appreciation to his wife, Ann, and children, Jamel, Daryl and Lawrence for their encouragement and forebearance during the course of the graduate study.

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INTRODUCTION

During the past several decades consumer demands have placed increased emphasis on prefabricated and processed meat products. Such products necessitate uniformity of the quantitative and qualitative factors to insure consumer acceptance and a profitable return to the processor. During this same period producers have been selecting for faster growing, more efficient, heavier muscled animals, which will yield carcasses with a higher percentage of lean in relation to fat. In the case of swine, at least, the fast growing, meatier animals are seemingly more highly predisposed to developing pale, soft and exudative (PSE) musculature postmortem.

The immediate cause of PSE muscle is associated with the marked accumulation of lactic acid resulting from rapid glycolysis while muscle temperature is high (>35°C) or near body temperature. The ensuing protein denaturation affects the ultimate color and water binding properties of such muscle. While uniformity of color is an important aesthetic factor, the accompanying decreased water binding capacity affects processing properties and yields. Pale, soft and exudative muscle was first reported by Ludvigsen in 1953 and has been observed subsequently by many American and European researchers.

The increased glycolytic rate suggests an anaerobic environment immediately postmortem and, indeed, several investigators have shown that some biochemical and physiological parameters implicate involvement of the cardiovascular system in the development of PSE musculature. Sybesma (1965) reported an increase in meat quality following an injection of a peripherally vasodilatory catecholamine (isoxsuprine) 30 min antemortem.

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Among the functions of the circulatory system is that of carrying oxygen to the tissues for oxidative metabolism. This is performed by the arterial and capillary system and the latter also provides the important function of removal of waste metabolites. When concerning ourselves with meat we refer primarily to muscle, a tissue, which has a rather extensive blood supply. Postnatal muscle increases occur by changes in length and width (hypertrophy) of the muscle. Hypertrophy results from the increase in diameter of individual muscle fibers. Since well muscled pigs are more highly predisposed to PSE development, hypertrophy may aggravate the muscle fiber area to capillary ratio. Thus, this study was designed to observe capillarity density in relation to muscle fiber area between normal and low quality (PSE) pigs. In addition, some related physiological factors such as heart and respiration rates, blood pressure, heart function as well as histological observations of muscle and nerve fibers were also studied.

The objectives of this study were as follows:

1. To measure capillary density per unit of muscle area, the absolute capillary to fiber ratio and capillary density in relation to muscle fiber type.
2. To determine the ratio of red, white and intermediate muscle fibers in relation to postmortem muscle quality.
3. To determine if the carnosine and anserine content of muscle is related to glycolytic rate and/or some histological observations.
4. To determine if differences in some physiological parameters are related to the ultimate qualitative properties of muscle.

REVIEW OF LITERATURE

The gross morphology of postmortem porcine muscle has been shown to be affected by a number of factors, i.e., hormones (Ludvigsen, 1953, 1957; Henry, 1958; Sybesma, 1965; Topel and Merkel, 1967; Judge et al., 1968), other pharmacological agents (Ludvigsen, 1957), exercise and ration (Briskey et al., 1959, 1960), environment, breed and postmortem muscle temperature (Sayre et al., 1961, 1963, 1966) and glycolytic rate and postmortem muscle temperature (Wismer-Pedersen and Briskey, 1961b).

Lawrie (1966b) stated that genetic, nutritional and physiological stimuli influence the adaptation of muscle for specific physiological functions and he indicated that these functions affect their normal living metabolism as well as postmortem properties. Lawrie (1966a) also stated that psychological and metabolic stresses including activity, ambient temperature, relative humidity, atmospheric pressure, oxygen tension, feed, injury and path logical state during and immediately prior to slaughter greatly influence the postmortem changes in muscle. Sayre and Briskey (1963) showed that postmortem glycolytic rate has a marked effect on ultimate porcine muscle morphology. Bendall (1964), Briskey (1964), Lawrie (1966b) and Mc Loughlin (1969) reported that the extent of postmortem changes affects the use of muscle as a food.

The rate at which postmortem changes occur increases with increasing environmental temperature especially between 5°-43°C (Bendall, 1960; Lawrie, 1966a) Newbold, 1966). Koch (1969) found that muscle (longissimus) temperature at 45 min postmortem was more highly related to ultimate muscle quality than the temperature at the time of exsanguination. He also observed

that myotomy at the time of exsanguination stimulated contractile activity and significantly increased the postmortem glycolytic rate. In addition, Koch (1969) found that postmortem changes, as measured by pH and transmission values in the rectus femoris, biceps femoris, longissimus and supraspinatus muscles, paralleled each other within a given carcass.

Glycolytic Control

White et al. (1964), Bendall (1964) and Lawrie (1966b) reported that ATP and creatine phosphate levels are maintained in slow working muscles by oxidative phosphorylation, but in fast working muscles where oxygen supply is limited, ATP is synthesized anaerobically. It has been shown that ATP is preferentially formed by the transfer of the high energy phosphate of creatine phosphate to ADP but when the supply of creatine phosphate becomes limited, attempts to maintain ATP level occurs anaerobically via the glycolytic pathway (Bendall, 1960; Lawrie, 1966b; Newbold, 1966).

Lawrie (1966a) reported that the immediate consequence of blood removal at exsanguination is the depletion of oxygen supply and loss of neural and hormonal control. The inhibition of respiration by blood removal results in the anaerobic synthesis of ATP via glycolysis.

Several factors affect the rate of glycolysis such as the concentrations of ADP, ATP, creatine phosphate, glucose-6-phosphate citrate and inorganic phosphate (White et al., 1964; Lowry et al., 1964; Ramaiah et al., 1964; Wood, 1966; Atkinson, 1966; Scrutton and Utter, 1968). Among the enzymes of the glycolytic pathway, phosphorylase and phosphofructokinase have been shown to be rate limiting (Lowry et al., 1964; Randle, 1964; Helmreich and Cori, 1965; and Wood, 1966). Randle (1964) reported that anoxia and inhibition of oxidative phosphorylation increased glycolysis by increasing

both phosphorylase and phosphofructokinase activity. Bendall and Lawrie (1964) reported that poor blood circulation played an important role in oxygen deficiency and associated it with lactic acid accumulation following intensive muscle contraction during fighting. With poor blood circulation, aerobic production of ATP is reduced hence anaerobic glycolysis ensues and lactic acid accumulates.

Physiological Factors

Forrest et al. (1968) studied the changes in heart rate, blood pressure and respiration rate, in "stress-susceptible" and "stress-resistant" pigs exposed to a warm environment immediately preslaughter.

In a review of circulatory changes during hypoxia, Korner (1959) stated that the first detectable change during acute arterial hypoxia was an elevation of pulse rate. Chalmers et al. (1966) reported no change in blood flow to muscles during mild hypoxia (PO_2 35mm Hg) although cardiac output increased. They also stated that during moderate hypoxia (PO_2 30-35mm Hg) vasoconstriction occurred initially resulting from the strong chemoreceptor stimulation and that high quantities of adrenaline restored normal blood flow to muscle. Topel and Merkel (1967) and Judge et al. (1968) found lower levels of adenocorticoids in PSE pigs. Forrest et al. (1968) found decreased venous PO_2 and pH (< 6.8) and increased PCO_2 in "stress-susceptible" pigs. "Stress-resistant" pigs showed no significant change in these parameters during heat stress. The latter authors found that anesthetized pigs did not show the significant changes that were observed for the unanesthetized pigs. Korner (1959) stated that induced hypoxia increased cardiac output, heart rate, mean arterial pressure, and femoral arterial flow, while decreasing systemic and hindlimb vascular resistance.

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Booth et al. (1960) reported the systolic arterial blood pressure usually was higher under barbiturate narcosis than in conscious animals. Engelhardt (1966) reported that the blood pressure of 100 kg pigs is 140mm Hg. He further stated that swine react to fear and other disturbances with marked and sudden increases in heart rate and blood pressure. Platner et al. (1948), Lepeschkin (1951), and Dukes (1955) reported heart rates of pigs ranging from 60 to 200 beats/min. In all probability this range reflects variation in the excitability of swine.

Richter (1959) studied the effect of noise and strange environment on heart rate of pigs and reported rates of 99/min during handling by a familiar person in a familiar, quiet environment He also reported heart rates of 30-45 kg wild swine to be 104/min. during excitement. Lueckmann (1958) reported 10-30% lower heart rates in pigs when lying compared to standing resting pigs and 40-50% higher heart rates over the standing resting controls in excited pigs. Forrest et al. (1965) correlated heart and respiration rates of Poland China, Yorkshire X Duroc, Chester Whites and Hampshire pigs with gross morphology scores of the longissimus dorsi muscle. The PSE pigs had higher heart rates than normal pigs (156 compared to 130 beats per minute). Respiration rates were also slightly higher among PSE pigs than normal pigs (49 vs 43). The latter authors also reported variation between standing and lying positions and whether the animals were excited or not and they concluded that heart rates in pigs were extremely variable. These authors further concluded that the animal's ability to adjust to handling, through adjustments in heart and respiration rates may be an important factor influencing the postmortem properties of muscle. Artificially induced changes in heart and respiration rates gave about the same results as naturally occurring variations (Forrest et al., 1965).

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These authors indicated that a relationship existed between heart and respiration rates, blood pH and pressure with glycolytic rates and postmortem muscle properties. They also showed that the immediate environment influenced these parameters and the increased values were related to body demands for oxygen.

Although the postmortem changes of different muscles may parallel each other (Koch, 1969), their metabolic requirements are related to function (Lawrie, 1966a). The anaerobic and aerobic capacities of muscles are related to the relative proportions of fiber types.

Red and White Fibers

Three types of muscle fibers are generally recognized, red, intermediate and white with considerable variation in each class (Ogata, 1958). Red fibers are small in diameter and are basically aerobic in their metabolism, while white fibers are larger in diameter and have a more anaerobic type of metabolism (Beatty et al., 1963).

While red, white and intermediate fiber types are generally recognized, this terminology denoting fiber type is not consistent throughout the literature. Such terms as "fast" and "slow"; and type I and type II are also used. Type I, "slow" and red fibers are used synonymously. Likewise, "fast", type II and white fibers are also used synonymously. However, there is no conclusive evidence of the synonymity of the fiber types within each of these classifications (Brooke, 1966).

The diameters of intermediate muscle fibers vary between red and white fibers and have both aerobic and anaerobic metabolic capacity (George and Berger, 1966).

The majority of mammalian muscles are heterogeneous for muscle fiber types (Folkow and Halicka, 1968), but one fiber type may predominate in any given muscle. Fast contracting muscles have more white fibers than slow contracting muscles, the latter having predominantly red fibers (George and Berger, 1966). The proportioning of fiber types in a muscle is probably determined by function, however,this has not been conclusively elucidated.

Before muscle color was known to be influenced by the ratio of red to white fibers (Denny-Brown, 1929; Ogata, 1958; Dubowitz and Pearse, 1960a, 1960b; and Lawrie, 1966a), muscles were broadly classed as red and white (Ranvier, 1874; Knoll, 1891; Needham, 1926). Ogata (1958) and Dubowitz and Pearse (1960b) used histochemical techniques [succinic dehydrogenase (SDH) and phosphorylast activity] to demonstrate the three fiber types. The same techniques have been used to demonstrate three fiber types in a wide range of animals (Ogata and Mori, 1964; Padykula and Gauthier, 1963; Dawson and Romanul, 1964; Romanul, 1964; Cosmos et al., 1965; Cosmos, 1966; Gauthier and Padykula, 1966; Beatty et al., 1966; Beecher et al., 1965b; Bodwell et al., 1965; Cassens et al., 1968). Ogata (1965) has also demonstrated a difference in morphology of the motor end plate between fiber types.

Red fibers have more mitochondria, respiratory enzymes, and myoglobin than white fibers (Paul and Sperling, 1952; Lawrie, 1952, 1966a). Beecher et al., (1968) found a higher myoglobin content, a greater percentage of red fibers and more SDH activity in the dark portion of the semitendinosus muscle than the light portion of the same muscle. Earlier, Beecher et al., (1965a) showed that the light portion of the semitendinosus muscle

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had a higher glycolytic rate than the dark portion. However, these authors were unable to show a relationship for myoglobin content, percent red fibers or SDH activity with glycogen levels or pH values. Although non-significant, lactic acid levels were higher in the white portion of the muscle. Since red fibers contain more myoglobin and mitochrondia than white fibers, these facts suggest that their energy needs are provided by different systems (Dubowitz and Pearse, 1960b; Gauthier and Padykula, 1966). Beatty et al. (1963) reported the in vitro oxygen consumption of red muscle fibers was 30% higher than white fibers and that white fibers had a greater capacity to produce lactic acid.

The functional significance of fiber size and mitochondrial content remain uncertain, but various important relationships are apparent. Fibers of small diameter have a greater surface area for exchange of gases, ions and metabolites than equivalent total mass of large fibers (Gauthier and Padykula, 1966).

The number and diameter of muscle fibers have been shown to be related to meat properties (Schilling, 1966). Staun (1963) reported that number and diameter of muscle fibers differed with breed of pigs. He further stated that: (1) fiber diameter was influenced by age and weight; (2) number of fibers remained unchanged during the growth period; and (3) no significant difference in fiber number or diameter exists between boars, barrows or gilts. Significant correlations was found for number of fibers per square centimeter and fiber diameter with various expressions of muscling in pigs. Joubert (1956) reported that muscle fiber diameter was more closely associated with muscle weight than age, live weight or carcass weight. Exercise has been shown to have an effect on muscle fiber size, type, myoglobin

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Capillarity in Relation to Red and White Muscle

Krogh (1919) was among the first to study capillary density in relation to oxygen supply in muscle. He concluded that the number of capillaries per square millimeter of muscle was a function of their metabolism. Ranvier (1874) reported on the morphological arrangement of capillaries in skeletal muscle and stated that capillaries run longitudinally along the length of the fibers and occasionally transverse across them but not in a regular fashion. It was believed for many years that all muscle fibers were associated with an equal number of capillaries. Denny-Brown (1929), Martin et al. (1932), Smith and Ginouacchini (1956) and Romanul (1965) reported differences in capillary density between muscles in the same animal. Smith and Ginouacchini (1956) reported that the number of capillaries per fiber differed in red and white muscle with red muscle having the greater density. Schmidt-Nielsen and Pennycuik (1961) confirmed these findings in a study of capillary density among ten species.

Capillarity in Relation to Fiber Type

Nishiyami (1965) studied the relationship of fiber type to capillary density and found numbers of capillaries decreased from red to intermediate to white fibers. He concluded that since red fibers have greater oxidative enzyme capacity and more mitochondria, they require an abundant supply of oxygen and therefore are endowed with a greater capillary density. On the other hand, white fibers possess lower oxidative potential, fewer mitochondria, and since they require less oxygen they are supplied with fewer

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capillaries. Intermediate muscle fibers are intermediate in oxidative capacity, mitochondria and capillary density. Romanul (1965) stated that the oxidative capacity of a fiber is directly proportional to the number of capillaries surrounding it.

Beecher et al. (1965b) studied the relationship of red, intermediate and white fiber content to postmortem properties of porcine muscle. They reported an association existed between percent red fibers and myoglobin content, sarcomere length and SDH activity. Red fibers had more myoglobin, SDH activity and longer postrigor sarcomere lengths than white fibers.

The Effect of Capillarity on Rate of Metabolism

Capillary density in skeletal muscle has been shown to be directly proportionate to the metabolic rate (Krogh, 1919; Martin et al., 1932; Schmidt-Nielsen and Pennycuik, 1961; Romanul, 1965).

This relationship is not without exception, especially where meat producing animals are concerned (Schmidt-Nielsen and Pennycuik, 1961; Merkel, 1968). Schmidt-Nielsen and Pennycuik (1961) concluded that capillary density was influenced by several factors. Among these factors, capillary density is affected primarily by size of the muscle fibers which in turn varies considerably with the type of muscle. They further stated that circulation in muscle is most critical during maximum metabolic activity. Since the substrates for ATP production are present in muscle, the supply of oxygen is critical for the maintenance of hemeostasis. Hence, muscle circulatory potential and function is important for normal metabolic activity.

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Capillarity in Relation to Oxygen Supply

Krogh (1919) indicated that capillary density was most important for supplying oxygen to the tissue. He reported that capillarity in the muscles investigated was more than ample for adequate oxygen supply. The oxygen, reversibly bound to hemoglobin, is transported by the capillaries and by a process of diffusion passes through the capillary wall, the extracellular fluids and finally through the sarcolemma into the sarcoplasm (Comroe, 1966). Krogh (1919) stated that the number of capillaries per cross-sectional area of muscle varies directly with the gas exchanges and the distance oxygen must travel within the cell.

The total oxygen requirement of a muscle depends upon its size (Sybesma, 1965) which in turn is related to fiber diameter (Joubert, 1956). Large muscle fibers require more oxygen and produce more heat during contraction (Sybesma, 1965) than small fibers. An increase in the number of large fibers will reduce effective capillary density. A reduction in capillary density inversely affects gas exchange (Krogh, 1919; Schmidt-Nielsen and Pennycuik, 1961). The net result would be a decrease in oxygen supply to the tissue which necessitates ATP maintenance by anaerobic metabolism. Capillary density associated with an individual muscle fiber is not only correlated with the oxidative activity, but has a further meaning in terms of the energy metabolism of the cell (Romanul, 1965).

Factors Affecting Oxygen Supply

An adequate supply of oxygen to skeletal muscle tissues is dependent upon a number of factors; the more important ones being: (1) ambient oxygen

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pressure, (2) the effectiveness of respiratory exchange within the pulmonary system, (3) blood flow rate through the muscle, (4) capillary density and size, (5) concentration and oxygen binding capacity of hemoglobin, and (6) oxygen consumption of the muscle cell (Schmidt-Nielsen and Pennycuik, 1961; Thorling and Erslev, 1968). The dependency of red fibers on blood for oxygen supply is emphasized by its higher capillary density. White fibers on the other hand are less dependent on blood supply because of their greater anaerobic metabolism. The latter fibers require capillaries primarily for removal of waste metabolites. Merkel (1968) suggested that the ratio of fiber types and factors which affect oxygen supply to the muscle tissues, particularly just prior to and during exsanguination, probably influence postmortem muscle quality.

Some Effects of Domestication on Swine Cardiovascular System

Wachtel (1963) compared the cardiovascular system of domestic and European wild swine of similar body weight. Wild pigs had higher hemoglobin levels and 25-40% more oxygen carrying capacity than domestic pigs. They also utilized less oxygen than domestic pigs (34% vs 44%). Domestic pigs are evidently unable to increase blood volume or hemoglobin content during exercise (Lyhs and Wachtel, 1965). Hoernicke (1966) reported that wild pigs had smaller fiber diameters and greater fiber numbers than domestic pigs. Engelhardt (1966) also reported heavier heart weights in wild pigs than among domestic pigs. Michel (1963) reported that nuclear chains of 6-12 nuclei were found in the right ventricle myocardium of domestic pigs but they occur to a lesser degree in wild pigs and then chains of only 2-4

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nuclei were seen. He further stated that nuclear chaining is a part of the adaptation process and occurs only when muscle fibers are hypertrophied and overstrained. Engelhardt (1966) reported that domestic pigs possess less ventricular contraction force and shorter systolic time than other domestic farm animals.

Koch (1969) reported slightly heavier heart weights (20 gm) in normal pigs than among low quality pigs. He found no significant correlations between ultimate muscle quality or 45 minute pH values and heart weight. He suggested that impaired heart function rather than heart weight per se probably contributed to the lower quality muscle observed.

Several workers have indicated that porcine muscle has less myoglobin than other meat producing animals except the very young bovine (Lawrie, 1953a, 1953b, 1953c, 1966a; Bray et al., 1959; Forrest et al., 1964; Topel et al., 1966). Merkel (1968) stated that porcine skeletal muscle were less equipped for aerobic metabolism than other domesticated meat producing animals. This indicates that porcine skeletal muscles are more highly predisposed to glycolytic metabolism during normal activities than other meat producing animals. Pigs show a faster pH drop than the horse or ox immediately postmortem (Lawrie, 1966a).

Carnosine and Anserine

The integrity of muscle fibers is threatened by myolactosis, the latter accounting for the marked pH decline postmortem. The dipeptides, carnosine and anserine, have been reported to have a high buffering capacity in the physiological pH zone (Deutsch and Eggleton, 1938; Davey, 1960a).

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Hunter (1924), Eggleton and Eggleton (1933), Savron (1934), Tallan (1955) and Lukton and Olcott (1958) reported that white muscles contain more carnosine than red muscles but greater variability occurred in white muscles.

Savron (1934) also reported that the right and left "pod" of the same animal had equal amounts of carnosine. The wide occurrence of this peptide suggests an important physiological role (Zapp and Wilson, 1938a, 1938b). Wolff and Wilson (1932, 1935) suggested that the importance of anserine and carnosine may be their contribution to the total imidazole content.

McClintock and Hines (1925), Mason and Binkley (1931), Du Vigneaud and Hunt (1936) and Hunt and Du Vigneaud (1938) indicated that naturally occurring carnosine had a depressing effect on blood pressure. Yudelovich (1937) found 20-30% less lactic acid production in rat diaphragm muscle in the presence of carnosine. Meshkova and Zolatareuskaya (1937) reported carnosine had no effect upon the autolytic processes of muscle tissues.

The Effect of Carnosine and Anserine on Buffering Capacity

Bate-Smith (1938) studied the various buffering systems in muscle as related to the onset of rigor mortis. He stated that during the course of rigor, pH in mammalian muscle falls from 7.0-7.5 to 5.5-6.0. The latter pH values are the combined function of muscle buffering capacity and the amount of lactic acid produced postmortem. He further stated that buffering in rigor mortis differs from that in living tissues. In rigor all esterified phosphate is hydrolyzed to orthophosphate; and the bicarbonates are destroyed soon after death. Bate-Smith (1938) concluded that the main physiological role of anserine and carnosine was not that of buffering,

because of their individual and collective variation in concentration within muscle. He reported that these dipeptides could account for as much as 25% of the total buffering potential (at pH 7) of rigor muscle and up to 40% of the buffering in living tissue. The latter author further stated that carnosine accounted for 10.5% of the buffering in the psoas muscle of pigs.

Davey (1960b) reported that high concentrations of anserine and carnosine were associated with muscles which derived their energy from anaerobic metabolism. While it is not uncommon to find high concentrations of these dipeptides in muscles with high aerobic capacities in some species, he concluded that the aerobic capacity of a muscle is inversely related to the concentration of these dipeptides. Since these dipeptides occur in high concentration in muscles that derive their energy from anaerobic metabolism, their contribution to total buffering potential is all the more important in the conversion of muscle to meat. However, Deutsch and Eggleton (1938), Bate-Smith (1938), Davey (1960b), Bendall and Wismer-Pedersen (1962) reported that buffering potential was probably not the major physiological role of anserine and carnosine.

The Effect of Carnosine and Anserine on Glycolysis

Severin *et al.* (1948) reported that carnosine played a role in the formation of energy-rich phosphate compounds under anaerobic conditions. They reported that the primary effect of carnosine was on oxidoreduction enzymes when the experiment was performed in a phosphate buffer medium. Nagradova (1958, 1959) stated that neither carnosine, anserine nor histidine had a stimulatory effect on phosphoglyceric acid dehydrogenase (PGADH), but they relieved the inhibitory effect on PGADH by high concentrations

of inorganic phosphate. Severin and Yui (1958) stated that the action of these dipeptides could not be ascribed to their buffering properties, but was associated with preservation of the mitochondrial structure and to decreased uncoupling of oxidative phosphorylation in "aging". Davey (1960a) studied the effect of carnosine and anserine on glycolytic reactions in skeletal muscle and was unable to confirm the findings of Severin et al. (1948). Daveys' (1960a) results agreed with Nagradova (1958, 1959) who reported that activation could have been due to either: (1) increased pH and buffering capacity of the reaction mixture, or (2) to a removal of possible heavy-metal inhibitors as chelated complexes upon the addition of the dipeptides to the reaction mixture. Jencks and Hyatt (1959) reported that carnosine, in vitro, appeared to prevent oxidation of essential sulfhydryl groups which is catalyzed by heavy metals. They suggest that the in vivo effects of the dipeptides on glycolysis are due to their buffering potential. Goodall (1956) reported a relaxing effect of carnosine phosphates on glycerol-extracted muscle fibers at pH 6.0 but when the pH was raised to 7.0 they contracted. He suggested that carnosine phosphate may be a high energy bond donor to ADP. Davey (1960a) was unable to identify either carnosine or anserine phosphate in the muscles he studied.

The Effect of Carnosine on Nerves and Muscle Tissues

Severin et al. (1961) and Severin and Shestakov (1961) found that imidazole compounds had a stabilizing influence upon the formation of acetyl CoA during oxidation of pyruvic acid. This seemingly indirect effect on the biosynthesis of acetylcholine was confirmed by Lyu (1962).

Severin and Vul'son (1962) reported on the role of carnosine in neurotrophic relations. He reported that the addition of carnosine to Ringer's solution enabled the sartorius muscle of the frog to contract more strongly and for a longer period than the untreated control muscle. He concluded that carnosine acts on motor end plates in muscle and the mediator mechanism of neuromuscular transmission. He gave the following reasons for arriving at his conclusions: (1) carnosine does not appear in muscle until the embryonal stage at which the reflex arc, mediating motor responses to sensory stimuli begins to function. McManus and Benson (1967) reported large increases in the concentrations of these dipeptides between 24 hr and 21 days post ova; (2) denervation or deefferentation of the muscle leads to atrophy which is accompanied by early and characteristic decreases in the carnosine content of muscle; and (3) the characteristic localization of carnosine in muscles coincides with the distribution of nerve endings.

Shabanova (1953), Tallan (1955), and Shapira and Dreyfus (1957) reported decreased carnosine content in muscle atrophy. Schapira and Dreyfus (1957) observed that anserine content remained unchanged for long periods after degeneration. Slepanova and Grinio (1968) stated that all types of muscle atrophy were characterized by a decrease in carnosine level. A pronounced decrease in the free histidine content was observed in all cases of progressive muscular dystrophy.

Muscle Degeneration

Ludvigsen (1953) reported the existence of an alteration in muscle appearance of pigs which he termed "muscle degeneration" and he later (Ludvigsen, 1954, 1955) described macroscopically as a pale or greyish

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discoloration, resembling that of chicken or fish. Additionally the musculature is very watery, possesses a sour smell, and has an open structure. Microscopically, Ludvigsen (1954) reported a marked edema of the interstitial connective tissue and separation of the muscle fibers. He also observed swelling of the muscle fibers, loss of normal cross-striation but no destruction of the sarcolemma or nuclei.

Histological Changes Seen in Dystrophic Muscle

Julian and Asmundson (1963) described the microscopic changes seen in skeletal muscle of chickens afflicted with muscular dystrophy. Variation in fiber size was seen with some fibers being larger than normal and others are smaller than normal. Nuclear proliferation is seen early in the dystrophic process in both white and red muscle fibers. The mean number of subsarcolemmal nuclei of dystrophic and normal muscles do not overlap in red muscle. On the other hand, considerable overlap occurs in white muscle. Both single and multiple vacuoles are seen within muscle fibers. Fiber destruction is characteristic and is seen at almost any stage of the disease.

These findings have been reported for the species in which muscular dystrophy has been observed and in most types of the disease (Blaxter, 1957; Pearson, 1963; West, 1963; Harman et al., 1963; Dubowitz, 1963).

White muscle appears to be more predisposed to muscle degeneration than red muscle (Julian and Asmundson, 1963; Cosmos and Butler, 1966; Cosmos, 1969; Jasmin, 1966).

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Changes in Enzyme Activities in Dystrophic Muscle

Dreyfus and Schapiro (1962) stated that in muscular dystrophy glycogenolytic enzyme activities are always lower than normal. Dreyfus et al. (1954) and Ronzoni et al. (1958) reported lower phosphorylase and aldolase levels and Schapira et al. (1955) found lower phosphoglucomutase and all the glycogenolysis enzymes in dystrophic muscle. The activities of these enzymes fall to one-fourth of the normal level when expressed per unit of muscle weight, and to one-third, if expressed on a non-collagenous nitrogen basis. Ronzoni et al. (1958) found that hexokinase activity was unchanged but lactic acid dehydrogenase was increased in activity. In dystrophic chicken muscle, increased phosphorylase activity was noted early "ex ovo" (Cosmos and Butler, 1966). Intensity of the histochemical reaction for phosphorylase diminishes in white fibers of these dystrophic muscles and they frequently appear to be atrophied. Jasmin (1966) reported that muscle atrophy induced in rats by spinal cord transection caused a progressive loss of glycolytic enzymes as a result of primary involvement of white muscle fibers. These fibers underwent transformation into intermediate fiber types as indicated by changes in their size and staining reactivities. Cosmos (1969) stated that SDH activity increased in dystrophic tissues in certain muscle fibers only. These fibers increased in number and in intensity of staining reaction as the muscle matured. These fibers are not maintained, however, since they eventually decrease in number and are often absent in aged muscle. Golarz et al. (1961) reported both normal and subnormal oxidative enzyme activities in human patients. Fennell and West (1963) found hydrolase activity in dystrophic tissues, presumably from

an increase in lysosomal content in the muscle. Similar results have been reported by Tappel et al. (1962) for dystrophic mouse and chick muscle. Engel et al. (1966) stated that in denervation atrophy in cat muscle, the type I (red) fibers were relatively unchanged, while type II (white) fibers became severely dystrophic. Phosphorylase activity in the denervated muscle had not recovered after 10 weeks whereas tenotomized muscle showed recovery of phosphorylase activity after 4 weeks. Towers (1935, 1937) observed the same changes histologically in dystrophic tissue caused by either denervation or immobilization. She concluded that atrophy is not merely the result of disuse, but the result of the concomitant loss of some specific (trophic) nervous influence. West (1963) stated that peripheral nerves and motor end plates are generally unaffected in vitamin E deficiency muscular dystrophy. Mason (1960) indicated that when peripheral nerves and motor end plates are affected, the damage to them is secondary to the muscle injury. However, the nerves and motor end plates return to normal following the recovery of the animal from the deficiency state. Michel et al. (1969) reported that nutritional myopathy was observed microscopically, but not grossly, in pigs fed selenium-vitamin E deficient rations. The lesions observed by Michel et al. (1969) were the same as those reported by West (1963).

Cardiac Involvement in Muscle Degeneration

Storstein (1962) stated that in late stages of many types of human myopathies functional and structural changes in heart muscle are frequently observed. It has been estimated that the heart is affected in 50-85% of

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the advanced cases of muscle degeneration (Berenbaum and Horowitz, 1956; Storstein, 1962). Morphological examination of the heart of muscular dystrophy patients reveals myocardial lesions similar to those seen in skeletal muscle (Bajusz et al., 1966). A number of electrocardiographic (ECG) changes of nonspecific patterns are also seen. Cardiac enlargement not attributable to the usual causes is also seen. Bajusz et al. (1966) studied cardiac involvement in heredity muscular dystrophy of hamsters. He reported that in more than 90% of the animals, cardiac involvement advanced to the stage of congestive heart failure. It has been suggested that weakening of the myocardium by myopathic process is a predisposing factor, the actual failure being caused by the combined effect of various factors in which the respiratory muscles are involved (Kilburn et al., 1959). Sundermeyer et al. (1961) reported that oxidative phosphorylation uncoupling occurs in myopathic hamsters. Bajusz et al. (1966) reported that focal myocardial lesions produce changes in the cardiac excitation pathway which can be detected with the electrocardiogram with high frequency and high gain. Dreyfus and Schapira (1962) reported that an apparent acceleration of the peripheral circulation time occurs in sex linked Duchenne type muscular dystrophy. These findings implicate the involvement of the cardiovascular system in muscular degeneration. Dreyfus and Schapira (1962) stated that anoxia is likely to occur in progressive muscular dystrophy (PMD) to some degree. Démos and Ecoifier (1957) demonstrated circulatory disturbance in PMD patients, as shown by arteriography and measurements of the speed of circulation.

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EXPERIMENTAL METHODS

Experimental Animals

This study was conducted as two separate experiments. Experiment I included 19 Poland China, 16 Landrace and 8 Chester White pigs, and Experiment II consisted of 20 Hampshire and 16 Yorkshire pigs. The pigs were obtained from Michigan producers known to have a high incidence of the PSE condition. Ten pigs within each breed were classified as either high or low quality (five pigs per quality group) except for the Chester Whites, since no low quality pigs were available for this breed. Other workers have reported that Chester Whites are relatively resistant to the PSE condition.

Preslaughter Physiological Data

Blood Samples for Hematocrit and Whole Blood pH. Fifteen ml of blood were collected from the jugular vein in a 20 ml glass syringe attached to a number 16 needle via a two way Luer-Lok switch. The collection syringe and needle were rinsed in a heparinized solution before blood collection. Three ml of blood were placed in a 3 ml sample tube with one drop of heparin for the hematocrit determination. Hematocrits were determined by the micro-hematocrit technique described by McGovern et al. (1955). Blood samples were centrifuged at 10,000 rpm in an International "Hemacrit" centrifuge for 5 min and the percent red cell volume was read on an International "micro-capillary" reader. The remainder of the blood was carefully

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Whole blood pH was measured on a Corning model 12 expanded scale pH meter.

Anesthetization. A second syringe containing sodium pentabarbital (65 mg/ml) was attached to the two-way system previously described to anesthetize the pigs via injection into the jugular vein. Approximately 70% of the calculated dosage was administered rapidly and the remaining 30% was administered more slowly until the desired depth of anesthetization was reached. Each pig was placed on its back in a V-shaped trough and prepared for the determination of blood pressure.

Blood Pressure. The left hind limb was shaved, aseptically prepared and 5-10 ml of xylocaine (2.5 mg/ml) were injected into the inside lateral surface over the fibular tarsal bone. An incision approximately 2 in long was made at this site and the tibial artery was exposed. A plastic cannula (2 mm O.D.), filled with a 1% heparin solution was inserted into the tibial artery. The opposite end of the cannula was connected to a high pressure (Statham $P_{23}A$) transducer which conducted the impulse to a polygraph (Grass model 5P), operated at a chart speed of 5 mm/sec. The apparatus is shown in Appendix A. Mean arterial blood pressure was calculated from readings using the following formula:

$$\text{Mean Pressure} = \frac{\text{Sytolic pressure} + 2\ \text{diastolic pressure}}{3}$$

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Heart Rate. Heart rate was calculated from the polygram as beats per minute.

Respiration Rate. Respiration rate was measured by an actual visual count of the number of inspirations per minute. An average of three separate counts was recorded as respiration rate.

After the polygram was completed, the artery was ligated above the cannulation site and the cannula was then removed. The incision was sutured following an application of Combiotic powder. The pigs were slaughtered approximately 48 hr later.

Slaughter

The pigs were shackled by the right hind leg, hoisted off the floor and exsanguinated without prior stunning. The electrical stunning procedure was not used because occasionally it results in rupturing of blood vessels which would have interfered with the subsequent ink injection results. Immediately after exsanguination, the pigs were lowered onto a table and the entire posterior extremities were removed between the fifth and sixth lumbar vertebrae. The remaining portion of the carcass was skinned, eviscerated, left unsplit, washed, and retained on the slaughter floor until the 3 hr postmortem samples were removed. The carcasses were then placed in a 4°C cooler.

Injection Technique. The abdominal aorta was cannulated with a glass cannula. The common iliac artery to the left ham was ligated immediately

distal to the bifurcation of the abdominal aorta. Hemostats were applied to smaller arteries. The right ham was then flushed with Ringer's solution for 3 min or until the effluent from the abdominal vena cava became clear. The ham was then perfused with a 1:1 mixture of India ink and distilled water for 2 1/2 min at a constant pressure of 160-165 mm of Hg. After perfusion was completed, the right and left hams were separated by splitting the lumbar and sacral vertebrae. The perfused ham was defatted and the gluteus medius, semimembranosus and rectus femoris muscles were exposed but were left attached to their origins and insertions. The entire ham portion was then placed in 10% formalin for 48 hr. Samples were excised from the corresponding muscles for myoglobin, anserine and carnosine determinations as well as for histochemical and histological observations.

Sampling Procedure

Samples for histochemical observations (approximately 1 1/2 cm x 1 cm^2) were excised from the approximate geometric center of these same muscles and frozen in liquid nitrogen.

Experiment I. Approximately 30 min postmortem, 100 g of muscle were excised from the medial portion of the right gluteus medius, semimembranosus and rectus femoris, frozen immediately in liquid nitrogen and stored at -20°C for subsequent anserine and carnosine determination. An additional 50 g from the medial portion of each muscle were placed in a 100 g sample jar and held at 4°C for subsequent myoglobin determinations. The myoglobin determinations were completed within 12 hr of exsanguination. At 45 min

postmortem, a 50 g sample of the right longissimus muscle (2nd to 3rd lumbar region) was excised for pH determination. The excised sample was immediately frozen in liquid nitrogen and stored at -20°C. The muscle samples were powdered in a -20°C room as described by Borchert et al. (1965) and Koch (1969).

The perfused hams were removed from formalin after 48 hr. The three muscles were excised and the medial one-third of each muscle was removed and replaced in fresh 10% formalin for an additional 24 hr. Muscle sections (0.5 mm^3) were processed through a autotechnicon for 12 hr (see Appendix B for schedule of solution used in this process) and embedded in low melting paraffin. The paraffin blocks were stored at 4°C until sectioned.

Experiment II. The left hams were perfused as described in Experiment I. Following the removal of fat from the hams, they were hung in a 4°C cooler for 3 hr before excising the muscles. The samples for histochemical observations were removed and prepared as described for Experiment I. A sample was excised from the 3rd to 4th lumbar region of the right longissimus muscle at 45 min postmortem for surface pH measurement. Transmission values (measure of the extractability of water soluble proteins) were determined on 24 hr muscle samples excised from the 10th to 11th thoracic region of the left longissimus muscle.

The procedure for Experiment II was identical to that in Experiment I except that preslaughter observations included only electrocardiogram data. Electrocardiograms were run on unanesthetized pigs which were restrained in the normal standing position except the feet were raised off the floor by a ventral support. A lead was placed on the outer portion of

each fore leg just above the carpal bones and the inner portion of the hind legs at the dorsal tip of the fibular tarsal bone. The electrocardiograms were recorded by a Sanburn model 300 electrograph operated at standard speed (25 mm/sec).

The following measurements were made from the electrocardiograms: duration of P-wave, P-R segment, P-R interval, QRS complex (duration and magnitude), S-T segment, S-T interval, T-wave, Q-T interval, mean electrical axis, and heart rate. The durations were measured in seconds and the heart rate as beats per minute.

Mean Electrical Axis Determination

The direction of the electrical axis of the heart was calculated from the electrocardiograms according to the method of Burch and Winsor (1960). The direction of electrical axis of the heart was calculated from measurements of the QRS complex on leads II and III. The height of the R-wave is added algebraically to the sum of the depth of the Q + S waves (if no Q or S wave is present, its value equals 0) to obtain the absolute deflection of the QRS complex. The absolute deflection values from leads II and III are plotted on a circular graph designed for measuring electrical axis rotation. Those for lead I are plotted between 0-180°, lead II +60° to -120° and lead III +120° to -60°. Lines for the deflection values of leads II and III are drawn perpendicular to the II and III axis, respectively. At the point where these two lines intersect, a line is drawn from the center of the circle through the intersection to the perimeter of the circle. The degrees of rotation of the hearts electrical axis are read

from the perimeter of the circle between 0 and ± 180°. Positive values are located on the lower half of the circle 0 to + 180° and negative values are on the top half 0 to -180°. The values for normal human electrocardiograms were used for comparison. These values are as follows: 0 to +90° normal, +90° to +180° indicates right axis deviation and 0 to -180° indicates left axis deviation.

Muscle Quality Appraisal

Subjective Method

Experiment I. The longissimus muscles of all carcasses were subjectively evaluated at 24 hr postmortem according to a system similar to that described by Forrest et al. (1963). A score of 0-5 was recorded for each of the following three factors: structure and firmness, marbling and color. Highest values were assigned to the ideal for each of the three characteristics, i.e., dry and firm, moderate or higher degrees of marbling, and grayish pink color. Lowest values were assigned to soft, exudative muscles which were devoid of visible marbling and very pale in color. No subjective appraisal was made of the muscles in Experiment II.

Objective Methods - Muscle pH

Approximately 5 g of frozen, powdered muscle were suspended in 25 ml of 0.005 M sodium iodoacetate. The pH values were then obtained from these suspensions with a Corning model 12 expanded scale pH meter. Surface pH (Experiment II) was obtained directly on a section of the longissimus muscle.

Measurements were made at six locations on the surface of the muscle, averaged and recorded as the pH value.

Transmission Value

The transmission value as described by Hart (1962) was used as an objective measure of muscle quality for the carcasses in Experiment II. Ten g of finely ground muscle (24 hr postmortem) were weighed in a 50 ml graduated centrifuge tube and cold distilled water was added to bring the total volume to 40 ml. This mixture was stirred thoroughly and held at 4°C for 20 hr. The mixture was then restirred, centrifuged (3000 rpm for 20 min at 4°C) and the supernatant filtered through Whatman No. 1 filter paper. A one ml aliquot of clear filtrate was added to each of two test tubes. Five ml of cold (20°C) citrate buffer pH 4.6 (9.35 parts of 0.2 M Na_2HPO_4 and 10.65 parts of 0.1 M citric acid) were added to one of the tubes and 5 ml of cold (20°C) distilled water to the other tube. This mixture was incubated at 20°C for 30 min. Percent transmission was read on a Bausch and Lomb Spectronic 20 colorimeter at 600 mμ against the blank (1 ml muscle filtrate and 5 ml distilled H_2O).

Red, White and Intermediate Fibers

The blocks of fresh frozen samples of the rectus femoris, semimembranosus and gluteus medius muscles of the left ham prepared for histochemical analysis of fiber type were sectioned at 10-15 μ on a Slee-Pearse cryostat (-20°C). The sections were placed on coverslips and stained for SDH activity by the

procedure of Nachlas et al. (1957). The sections were incubated at 37°C for 1-2 hr in a medium containing 0.05 mM of sodium succinate and 0.5 mg of Nitro BT [nitro-2,2:5'-tetrapheny1-3,3'-(3,3'-dimethoxy-4,4'-biphenylene) ditetrazolium chloride] per ml at pH 7.6 in a phosphate buffer. After incubation the sections were washed in physiological saline (8.5 g NaCl, 0.2 g $CaCl_2$ and 0.1 g KCl per liter of distilled water) and fixed in 10% formol-saline for 10 min, dehydrated in 15% ethanol for 5 min and mounted in glycerine jelly.

Photomicrographs were taken from three different areas of each slide on 35 mm pantatomic X black and white film. Prints (26.5 x 35 cm on Kodak poly content paper) were made from a magnification of 21 X. Numbers of each fiber type were determined and expressed as percentage of total fibers. The size of the red fibers were determined by measurement with a compensating polar planimeter and used to calculate red fiber area which was expressed as percent of total area for the pigs in Experiment I.

In Experiment II red, white and intermediate fibers were identified by use of reciprocal stains of the metabolic activities of red and white fiber types according to Dubowitz and Pearse (1961). Serial sections to those incubated for SDH activity were incubated for phosphorylase activity according to the method of Takeuchi (1956). Incubation was carried out at 37°C for 3 hr [longer (up to 12 hr) with samples which were stored for longer periods] in a medium containing 50 mg of glucose-1-phosphate and 10 mg of muscle adenylic acid dissolved in 10 ml of distilled water and 5 ml of absolute alcohol. To this mixture was added a few drops of insulin

and 10 ml of acetate buffer at pH 5.7. After incubation the sections were fixed in absolute alcohol for 3-4 min then immersed in a dilute Gram's iodine iodide solution (2 g KI and 1 g I_2 in 100 ml of distilled water) for 5 min and mounted in iodine-glycerine jelly. Since slides prepared in this manner fade, they were kept under refrigeration and away from direct light. Phosphorylase activity is indicated by a deep brown to bluish-brown color. Fibers staining positive to SDH activity and negative to phosphorylase activity were designated red; fibers negative to SDH activity and positive to phosphorylase activity were designated white and fibers positive to both enzymes were designated as intermediate. Numbers as well as area of red, white and intermediate fibers were determined for each muscle. Area of each fiber type was measured with a compensating polar planimeter. Relative size of each fiber type was calculated from the number and total area of each fiber type.

Capillary Density

Experiment II

Capillary density was calculated from the pictures which were prepared for differentiating fiber type. The number of capillaries associated with each fiber type as well as with total area of each fiber type were determined.

In Experiment I capillary density was measured on sections from the paraffin embedded muscle samples. The sections were deparaffinized in xylene and stained with eosin (procedure described in Appendix B). Three

uniformly injected areas of each slide were photographed as described in the section on red and white fibers but at a magnification of 28 X. Capillaries per area of picture, fibers per area of picture and capillaries per fiber as well as per fiber type were determined from these pictures.

Myoglobin

Total myoglobin concentration as well as estimates of the relative amounts of reduced myoglobin (Mb), metmyoglobin (MMb) and oxymyoglobin (O_2Mb) were determined according to the absorbancy ratio method of Broumand et al. (1958) on samples from all muscles studied in Experiment I. Five g of fresh, chilled, finely-ground muscle were extracted with 20 ml of distilled water by shaking for 1 min in a stoppered Erlenmeyer flask. The extract was filtered through Whatman No. 1 filter paper and the first 2-3 ml of filtrate were discarded. The absorbancy of the remaining filtrate was determined spectrophometrically at wave lengths of 473, 507, 573 and 597 mμ in a Beckman DU spectrophometer against a water blank. One drop of 0.5% KCN and one drop of 2.0% $K_3Fe(CN)_6$ was added to the blank and each sample tube and mixed. The total myoglobin content was then measured as cyanometmyoglobin (CMMb) by recording the absorbance at 542 mμ.

The absorbancy ratio 507/573 mμ was used to estimate the % MMb and the absorbancy ratio of 473/597 mμ used to estimate the % Mb. These percentages were read directly from a standard curve presented with the original method to obtain absolute values. The millimolar extinction coefficient of CMMb is 11.3 at 542 mμ. This value was used to calculate the total concentration of CMMb. The % O_2Mb was calculated from the

formula [% O_2 b = 100 - (% MMb + % Mb)]. The results were expressed as millimicromoles (mμM) per gram of fresh tissue.

Anserine and Carnosine Determination

Preparation of Muscle Tissue

Anserine and carnosine determinations were made according to the diazo method of Parker (1966). Three g of fresh frozen sample were extracted (in duplicate) with 3 volumes of 1 N perchloric acid, blended for 1 min in a Virtis mixer and heated for 5 min at 100°C and filtered in Whatman No. 2 filter paper. The filtrate was neutralized with 1 N NaOH and diluted to 25 ml. The diluted filtrate was then frozen at -20°C and stored at this temperature until analyzed. Samples were thawed, centrifuged at 5000 rpm for 10 min and the supernate was decanted off and used for the analysis.

Carnosine determination

One ml of the extract was diluted with distilled water to a final peptide concentration of 0.05 to 0.15 mM. One ml of the final diluted sample was then pipetted in a test tube to which 1 ml of 0.04 M EDTA, 1 ml of 20% Na_2CO_3 and 2 ml of p-bromo analine were added. The reaction was allowed to proceed for 5 min and then stopped with the addition of 2 ml of 95% ethanol. Absorbance was read at 500 mμ against a reagent blank.

Total imidazole concentration

To 1 ml of the diluted sample as described in the carnosine determination, 0.8 ml of 4.0% $NaHCO_3$ and 0.2 ml of dinitrofluorobenzene (NDFB) were added and the mixture heated in a 55°C water bath for 30 min. Then 0.1 ml of 10 N NaOH and 1 ml of 20% HCl were added and the mixture was extracted with 2 ml of diethyl ether for 1 min.

Absorbance of the aqueous phase was measured against a reagent blank at 430 mμ. Anserine content was calculated by the following formula: total imidazole - carnosine = anserine. Anserine, carnosine and total imidazole are reported as μM/g of tissue. The preparation of the reagents used for the anserine and carnosine determinations is described in Appendix C.

Histological Examination

Histological examinations of selected tissues of the pigs in Experiment II were made to determine the extent of muscle degeneration, if any, and possible relationships to the PSE condition. Microscopic examinations were made of the three ham muscles, the left sciatic nerve and ventricular tissues (Yorkshire pigs only). The skeletal muscle tissues were fixed in 10% formalin and prepared for paraffin embedding by passing through the auto technicon overnight.

The fixing and staining procedures used for the cardiac muscle and nerve tissues are described in Appendix B.

The degree of histopathology was characterized according to the method of Homburger et al. (1966). The classification criteria are presented below.

Normal: no visible morphological changes

Suspicious: in longitudinally sectioned fibers, fewer than 4 internally rowed nuclei or less than 4 centrally placed nuclei per 100 in transversally sectioned fibers.

Mild: more than 4 centrally placed nuclei per 100 in transversely sectioned fibers. More than 4 internally rowed nuclei in longitudinally sectioned fibers.

Moderate: more than 8 centrally placed nuclei per 100 in transversely sectioned fibers. Internally rowing nuclei with longer rows.

Marked: Long chains of internally rowed nuclei, often with 2 or more rows of centrally placed nuclei per fiber in the majority of transversally sectioned fibers.

Other atypical observations such as perinuclear halos, "ring-binding" in the parenchyma, etc., were noted.

Microscopic observation was done with an A.O. type bioptical microscope.

Statistical Analysis

The statistical analysis followed were procedures described by Steel and Torrie (1960). Duncan's multiple range test was applied when analysis of variance data were significant ($P < .05$) to detect the significantly different means. Some simple correlation coefficients which were of interest were calculated.

RESULTS AND DISCUSSION

The Selection of Quality Groups

The pigs in Experiment I were categorized in quality groups based on their postmortem rate of pH decline of the longissimus muscle. Five pigs of each of three breeds (Poland China, Landrace and Chester White) with 45 min (postmortem) pH values greater than 6.0 were categorized as "normal". Additionally, five Landrace and five Poland China pigs with 45 min pH values less than 6.0 and with pale, soft and exudative (PSE) musclature (24 hr postmortem), as described by Briskey (1964), were categorized as "low quality". No low quality Chester White pigs were obtainable from the herd chosen as the source of this breed.

In Experiment II, the pigs were categorized in quality groups based on a combined evaluation of 45 min pH and 24 hr transmission values (protein extractability) of the longissimus muscle. As in Experiment I, five pigs from each of the Hampshire and Yorkshire breeds were categorized as "normal" and "low quality." Since transmission value does not always identify the low quality pigs (Ockerman and Cahill, 1968), 45 min (postmortem) pH values were considered more reliable for the categorization of quality groups. Therefore, pigs with the extreme in pH and transmission values were selected for comparison in the normal and low quality groups, respectively.

Mean pH and transmission values and subjective muscle quality scores of each quality group within each breed are presented in Table 1. As expected from the report of Briskey (1964), a significant ($P < .01$) difference

Table 1. Mean pH values, subjective quality scores and transmission values of the longissimus muscle by breed and quality group[1]

Characteristic measured	Experiment I					Experiment II			
	Breed and quality group					Breed and quality group			
	Poland China		Landrace		Chester White	Hampshire		Yorkshire	
	Normal	Low	Normal	Low	Normal	Normal	Low	Normal	Low
pH, 45 min	6.19^{b}	5.49^{a}	6.23^{b}	5.67^{a}	6.26^{b}	6.68^{ab}	6.27^{ab}	6.76^{b}	5.84^{a}
Subjective quality score[2]	10.0^{b}	5.6^{a}	11.0^{b}	6.8^{a}	13.0^{b}	X	X	X	X
Transmission value, 24 hr	X	X	X	X	X	8.80^{a}	49.8^{b}	12.5^{a}	61.7^{b}

[1]Means with the same superscripts are not significantly different ($P > .01$). Underlined values are significantly different at $P < .05$ level.

[2]Based on 15 point scale (0-5 for each of the three factors: marbling, color and structure).

X indicates no data were obtained.

for both 45 min pH and subjective quality scores was noted between quality groups in Experiment I. A significant ($P < .01$) correlation coefficient (0.63) was obtained between 45 min pH and subjective quality scores in Experiment I.

In Experiment II, 45 min pH values were significantly different between quality groups in both the Yorkshire and Hampshire breeds ($P < .01$ and $P < .05$, respectively). Transmission values were significantly ($P < .01$) different between quality groups in both breeds. Corresponding quality groups between breeds were not significantly different in either experiment. These data justify the method of categorization of the pigs used in this study.

Rate of pH fall was more rapid among low quality than normal pigs. Comparable (pH) curves within quality groups for each of the breeds used in Experiment I are presented in Figure 1.

The lower 45 min pH values in Experiment I compared to those in Experiment II probably reflect the difference in procedure used to measure pH (powdered sample homogenate in Experiment I and direct surface measurement in Experiment II). The correlation coefficient between 45 min pH and 24 hr transmission value (-.97) in the Yorkshire pigs was highly significant ($P < .01$). The correlation coefficient between these two parameters (-.44) was not significant in Hampshires. These data suggest that the factors influencing protein extractability may have been different in these two breeds. The correlation coefficient between 45 min pH and 24 hr transmission values (-.71) for all pigs in Experiment II was highly significant ($P < .01$).

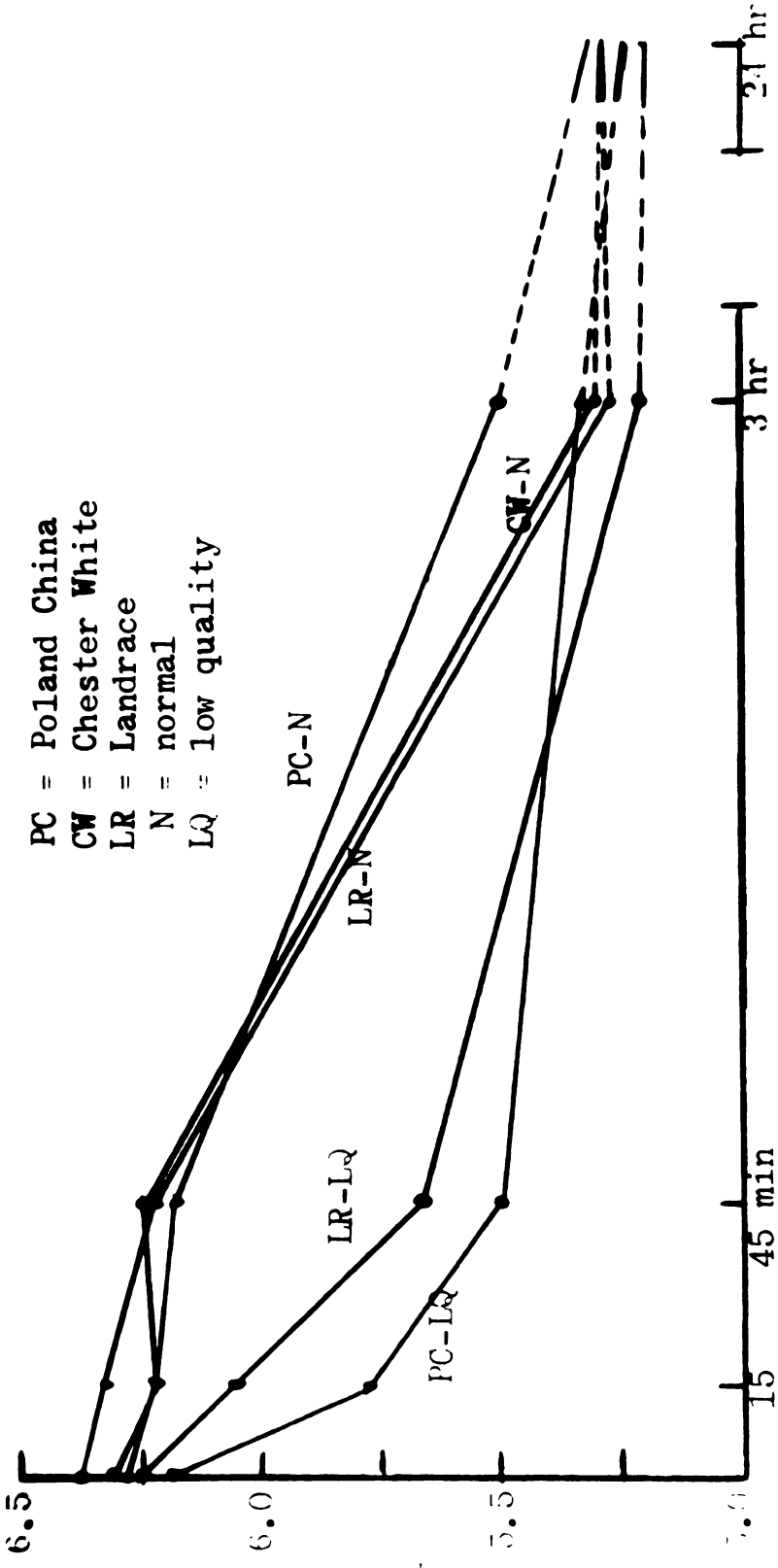

Figure 1. Postmortem pH curves of the longissimus muscles of the pigs in Experiment I by breed and quality group

Preslaughter Physiological Values

A summary of the preslaughter physiological data from Experiment I is presented in Table 2.

Respiration rate - Respiration rates were not significantly different between quality groups. The low quality Poland China pigs had a higher mean respiration rate than all other pigs. Respiration rates were not significantly correlated with 45 min pH (-.14).

Table 2. Mean values of some preslaughter physiological parameters by breed and quality group.

	Experiment I				
	Breed and quality group[1]				
	Poland China		Landrace		Chester White
Parameter	Normal	Low	Normal	Low	Normal
Live body weight, kg	80.2	87.1	92.5	99.8	93.4
Respiration rate, inspiration/min	18.1	22.8	19.4	19.3	18.3
Blood pressure, mm Hg	153.0	175.8	158.8	144.0	157.0
Heart rate, beats/min	125.4	137.6	163.2	127.6	156.0
Hematocrit, % red cell volume	35.5	40.0	39.0	39.6	38.4
Whole blood pH	7.34[2]	7.33	7.38	7.37	7.28

[1]Five pigs per quality group within each breed.
[2]Only one pig was measured.

Blood pressure - Mean blood pressure was not statistically significant ($P > .05$) between quality groups. The low quality Poland China pigs had the highest mean blood pressure of all quality groups. On the other hand, the low quality Landrace had a lower mean blood pressure than normal Landrace pigs. The mean blood pressure of the normal quality pigs in all breeds were comparable. Because of the high values of the low quality Poland China pigs, they had a higher, although not statistically significant, mean blood pressure than either Chester White or Landrace pigs.

The correlation between 45 min pH and blood pressure was low (-.06).

Heart rate - Heart rates were not significantly ($P > .05$) different between quality groups. Heart rates roughly paralleled blood pressure values within and between quality groups. Heart rates of Poland China and Landrace pigs were lower than Chester Whites, with Poland China pigs having the lowest values. A low nonsignificant correlation (0.15) was found between 45 min pH and heart rate.

In Experiment II heart rates were determined from electrocardiograms (ECG) on conscious pigs. The mean values were generally lower but comparable to values obtained in Experiment I. These data are not in agreement with reported results in that heart rates taken from electrocardiograms of conscious pigs were frequently higher than those on anesthetized pigs (Hausmann, 1934; Luisada et al., 1944; Platner et al., 1948; and Grauwiler, 1965). The mean heart and respiration rates observed in Experiment I were slightly lower but comparable to those reported by Forrest et al. (1968) on anesthetized pigs. These same authors reported that conscious pigs

had higher heart and respiration rates than anesthetized pigs. The difference in restraining method used in these two experiments may possibly account for these results. Low quality pigs in both breeds (Experiment II) had higher mean heart rates than those with normal quality. Mean heart rate for all pigs in Experiment I was 142.0 in contrast to 130.7 in Experiment II. The mean heart rates for Experiment II are presented in Table 3.

Electrocardiograph data - These data were obtained on the pigs in Experiment II are summarized in Table 3. Low quality Yorkshire pigs had

Table 3. Mean electrocardiogram data for the pigs in Experiment II by breed and quality group.[1]

Parameter	Breed and quality group				Mean of all pigs
	Hampshire		Yorkshire		
	Normal	Low	Normal	Low	
Heart rate, beats/min	121.6	128.4	122.3	145.4	130.7
P-wave, sec	0.062[b]	0.057[a,b]	0.051[a,b]	0.047[a]	0.054
P-R segment, sec	0.045	0.043	0.038	0.042	0.042
P-R interval, sec	0.107	0.100	0.089	0.089	0.096
QRS complex, sec	0.046	0.045	0.048	0.048	0.047
S-T segment, sec	0.114	0.111	0.111	0.108	0.111
S-T interval, sec	0.181	0.182	0.170	0.176	0.180
T-wave, sec	0.067	0.071	0.058	0.068	0.066
Q-T interval, sec	0.236	0.230	0.232	0.243	0.235

[1]Means with the same or no superscripts are not significantly different ($P > .05$).

a significantly ($P < .05$) shorter p-wave than normal Hampshires. Additionally, Yorkshire pigs had a shorter mean p-wave than Hampshires. None of the other intervals were significantly different between quality groups or breeds. The duration of the T interval and waves are in agreement with the values reported by Luisada et al. (1944), Miller et al. (1957) and Miller et al. (1966). The mean systole/diastole quotient (0.4) was not significantly different ($P > .05$) between quality groups for either Hampshire or Yorkshire pigs. These values are lower than those reported by Engelhardt (1966) for the domestic pig (0.7-0.8).

The direction of the electrical axis was calculated from the electrocardiogram of each pig in Experiment II and the results are presented in Table 4. The pigs in all quality groups showed electrical heart axis deviation but the deviations were more frequent among low quality pigs. The deviation data of all pigs in Experiment II are presented in Appendix D.

Table 4. Electrical axis rotation data of each pig in Experiment II.

	Breed and quality group			
	Hampshire		Yorkshire	
	Normal	Low	Normal	Low
	Degrees	Degrees	Degrees	Degrees
	+40	+98	+20	-68
	- 1	+47	+15	-122
	+73	+93	-30	+17
	- 6	+52	+36	+76
	+97	+45	+114	+137
Group mean	+40.6	+67	+35	+ 8

Hampshire pigs tended to show more right axis (+) deviation while Yorkshires showed more left axis deviation. These trends suggest breed differences in the position of the electrical axis of the heart. The electrical axis deviation data were not significantly correlated with 45 min pH. The direction of the electrical axis in the Hampshire pigs (20) was inversely related to 45 min pH (-.27) while the same correlation for Yorkshires was positive (0.42). These data along with the low systole/diastole quotient, and shorter P-wave in low quality pigs compared to normals suggest that these differences in cardiac function may be associated with the development of PSE musclature. This would support the suggestion of Koch (1969) and the implication by Forrest et al. (1968) and Merkel (1968) that cardiac function may be a factor in the development of PSE musclature postmortem.

Whole blood pH and hematocrit values - Whole blood pH was not significantly different between quality groups or breeds. Mean values are within the normal reported range (Ruch and Patton, 1966). None of the pigs showed whole blood pH values as low (6.8) as the stress susceptible pigs reported by Forrest et al. (1968). Their pigs were stressed (warm environment) for 20-60 min, whereas, attempts were made to minimize stresses prior to and during slaughter in the present study. Hematocrit values are also within the normal range of reported values (Miller et al., 1967; Bidner, 1969). Normal Poland Chinas had lower hematocrits than all other pigs. Hematocrit was nonsignificantly correlated with 45 min pH (-.15).

None of the preslaughter physiological parameters measured were significantly correlated with 45 min pH values, however, the results may have been affected by anesthetization (Experiment I) or masked by the body's compensatory response during the time interval between measurement and slaughter (48 hr). The 48 hr period between the recording of preslaughter physiological data and the time of slaughter was allowed for the pigs to recover from surgical and other accompanying stresses which, in turn, might have affected the ultimate qualitative properties of the carcass.

Postmortem Data

Myoglobin

Mean levels of total myoglobin (Mb) as well as absolute amounts of metmyoglobin (MMb), reduced Mb and oxymyoglobin (O_2Mb) of the three ham muscles of the pigs in Experiment I are presented in Table 5.

Rectus femoris muscle. The rectus femoris muscle of Chester White pigs had significantly ($P < .05$) more total Mb than normal Poland China or low quality Landrace pigs, but they were not significantly different from low quality Poland China or normal Landrace pigs. In the Poland China breed, normal pigs had less total Mb than those with low quality, while normal Landrace had more total Mb than those with low quality. No significant ($P > .05$) difference in MMb content was found between the quality groups, although the values tended to parallel those of total Mb. Reduced Mb values were highest among Chester White pigs, being significantly ($P < .05$) higher than normal Poland China and low quality Landrace

Table 5. Mean myoglobin data of the rectus femoris, semimembranosus and gluteus medius muscles by breed and quality group.[1,2]

Muscle and myoglobin fraction[2]	Breed and quality group				
	Poland China		Landrace		Chester White
	Normal	Low	Normal	Low	Normal
Rectus femoris					
Total myoglobin	110.1^{a}	195.2^{ab}	214.1^{ab}	139.3^{a}	260.5^{b}
Metmyoglobin	46.3	88.9	89.5	50.4	117.4
Reduced myoglobin	35.8^{a}	94.9^{ab}	91.9^{ab}	38.7^{a}	125.4^{b}
Oxymyoglobin	28.0^{ab}	11.4^{a}	32.7^{ab}	50.2^{b}	17.7^{a}
Semimembranosus					
Total myoglobin	68.9^{a}	84.4^{a}	143.6^{b}	108.2^{ab}	151.7^{b}
Metmyoglobin	23.5^{a}	35.2^{ab}	61.4^{b}	39.1^{ab}	62.7^{b}
Reduced myoglobin	18.4	34.0	62.2	32.6	65.0
Oxymyoglobin	27.0	15.1	20.0	36.5	24.1
Gluteus medius					
Total myoglobin	55.1^{a}	107.9^{ab}	148.9^{b}	113.3^{ab}	152.5^{b}
Metmyoglobin	18.1^{a}	50.1^{ab}	66.5^{ab}	43.3^{a}	70.7^{b}
Reduced myoglobin	16.2	52.9	62.5	40.4	70.1
Oxymyoglobin	20.8^{bc}	4.9^{a}	19.9^{bc}	29.6^{c}	11.6^{ab}

[1]Means with the same or no superscripts do not differ significantly ($P > .05$).
[2]Millimicromoles/g of fresh muscle.

pigs. Oxymyoglobin content was significantly higher ($P < .05$) in low quality Landrace than either low quality Poland China or normal Chester White pigs.

Semimembranosus muscle. Although total Mb content of the semimembranosus muscles was lower than those of the rectus femoris, they paralleled the values of the latter muscle. Normal Chester White and Landrace pigs had significantly ($P < .05$) more total Mb than either of the Poland China quality groups but the values were not significantly different from the low quality Landrace pigs. Metmyoglobin content was significantly ($P < .05$) different between quality groups and the values paralleled those of total Mb. There were no significant ($P > .05$) differences in either reduced Mb or O_2Mb levels between quality groups in the semimembranosus muscle.

Gluteus medius muscle. The myoglobin data of the gluteus medius followed the same pattern as the rectus femoris and semimembranosus muscles. Normal Chester White and Landrace pigs had significantly ($P < .05$) more total Mb than normal Poland Chinas. Total Mb content of low quality Poland China and Landrace pigs were not significantly ($P > .05$) different from the other quality groups and both low quality groups had more total Mb than high quality Poland Chinas. These observations were similar in all three muscles. Koch (1969) used the same pigs as those included in this study and found more total Mb in the longissimus muscles of the normal Poland China and Landrace pigs than in those with low quality.

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Breed differences. Landrace pigs had significantly ($P < .05$) more O_2Mb in the rectus femoris muscle than either Poland Chinas or Chester Whites. Chester Whites had significantly ($P < .05$) more total Mb and MMb in the semimembranosus muscle than either Poland China or Landrace pigs. The gluteus medius muscle showed more variability between breeds than the other two muscles. Poland China pigs had significantly ($P < .05$) less total Mb and MMb than the other two breeds while Landrace pigs had significantly ($P < .05$) more O_2Mb than Poland China and Chester Whites. The low quality Poland China pigs had more total Mb, MMb and reduced Mb and less O_2Mb than the normal pigs in all three muscles. Corresponding values for the Landrace pigs were directly opposite those of Poland Chinas.

Muscle differences. As expected, mean total Mb as well as most of the other Mb fractions of all quality groups were higher in the rectus femoris than in the semimembranosus or gluteus medius muscles. The relative amounts of MMb, reduced Mb and O_2Mb (expressed as percent of the total Mb) are presented in Table 6. These data tend to suggest that breed and muscle differences exist in the capacity to utilize the oxygen from O_2Mb. Low quality Poland China pigs had consistently less O_2Mb than the other quality groups while low quality Landrace had consistently more O_2Mb than the other quality groups in all three muscles. All muscles had similar amounts of MMb and reduced Mb within each quality group. The mean O_2Mb values of all quality groups decreased in absolute content from rectus femoris to semimembranosus to gluetus medius muscles. However, all quality groups and breeds showed a slight increase in O_2Mb when expressed as % of

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Table 6. Mean myoglobin data of the rectus femoris, semimembranosus and gluteus medius muscles (expressed as percentage of the total).

Muscle and myoglobin fraction	Breed and quality group				
	Poland China		Landrace		Chester White
	Normal	Low	Normal	Low	Normal
Rectus femoris					
Metmyoglobin	37.2	44.8	39.2	34.8	45.4
Reduced myoglobin	25.2	45.8	37.4	25.8	46.8
Oxymyoglobin	37.6	9.4	23.4	39.4	8.0
Semimembranosus					
Metmyoglobin	31.6	40.6	42.6	33.0	41.0
Reduced myoglobin	20.8	37.0	42.6	25.2	41.0
Oxymyoglobin	47.6	22.4	14.8	41.8	18.0
Gluteus medius					
Metmyoglobin	33.4	45.8	44.0	33.2	46.2
Reduced myoglobin	25.6	48.2	42.6	29.0	45.4
Oxymyoglobin	41.0	6.0	13.4	37.8	8.4

total Mb in the semimembranosus. These results suggest that differences exist in oxygen utilization and/or requirements between muscles.

The trend of the relative amounts (% of total Mb) of the various myoglobin fractions in the three muscles are coincident with the finding of Koch (1969) for the longissimus muscles of these same pigs. However, these data do not concur with his suggestion that normal muscles have greater

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aerobic metabolic potential, at least in the Poland China breed, but rather they suggest that muscle and breed differences exist in the efficiency of aerobic metabolic potential. Difference in time of sampling (Koch sampled at the time of exsanguination compared to 30 min postmortem in the present study) and sample preparation (powdered sample vs freshly ground sample) probably account for the quantitative difference found in total myoglobin between the two studies.

The mean total myoglobin content observed in postmortem muscle paralleled the antemortem physiological parameters. Respiration and heart rate, blood pressure and hematocrit showed the same pattern among the Poland China and Landrace quality groups as that observed for total Mb postmortem.

Total Mb in the semimembranosus muscle was significantly ($P < .05$) correlated with 45 min pH (0.43) while corresponding correlations for the gluteus medius and rectus femoris were nonsignificant (0.30 and 0.10, respectively).

Distribution of Red, White and Intermediate Muscle Fibers

In Experiment I distribution of fiber types were expressed as percent of the total number of fibers. Fiber types were identified histochemically on the basis of their relative staining intensity for SDH activity. The mean percent of each fiber type of each muscle within breeds and quality group is presented in Table 7. No significant differences were found in the percent of each fiber type between either quality groups or breeds. Low quality Poland China pigs had lower percents of red fibers and higher percents of white fibers than the normal Poland Chinas in all three muscles.

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Table 7. Distribution of fiber types in three porcine muscles by breed and quality group (Experiment I).[1,2]

Muscle and fiber type	Breed and quality group				
	Poland China		Landrace		Chester White
	Normal	Low	Normal	Low	Normal
Rectus femoris					
Red	42.9	35.8	40.9	51.9	41.2
White	42.0	47.4	43.9	30.7	40.3
Intermediate	15.1	16.8	15.2	17.4	18.5
Semimembranosus					
Red	40.5	34.3	35.3	34.8	40.6
White	43.4	50.3	49.3	53.7	45.9
Intermediate	16.1	15.2	15.4	11.4	13.4
Gluteus medius					
Red	34.8	29.7	35.9	29.3	34.2
White	49.5	55.6	50.4	60.6	54.2
Intermediate	15.9	14.8	13.6	10.0	11.6

[1]Identified by succinic dehydrogenase activity.
[2]Numbers of each fiber type are expressed as % of total fiber number.

Low quality Landrace had higher percents of red fibers in the rectus femoris muscle and lower percentages of white fibers than normal Landrace pigs. Normal Landrace had higher percents of red fibers and lower percentages of white fibers in the semimembranosus and gluteus medius muscles than low quality pigs. Landrace pigs tended to have less intermediate fibers than

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Poland Chinas in both quality groups and they also had 5% fewer red fibers in the normal quality semimembranosus muscle than either the Poland China or Chester Whites. Chester White pigs tended to be intermediate to normal Poland China and Landrace in percentages of the three fiber types. These data suggest that the low quality muscles had a greater potential for anaerobic metabolism than those of normal quality. As expected the rectus femoris had significantly more red fibers than the semimembranosus ($P < .05$) and gluteus medius ($P < .01$) muscles. No significant ($P > .05$) differences were found in the percentages of fiber types between the semimembranosus and gluteus medius muscles.

The area of each fiber type was calculated and expressed as percentages of total fiber area. The mean values of the pigs in Experiment I are presented in Table 8. These data indicate that numbers of each fiber type within a muscle are not consistent with the total area of each fiber type. While no statistical analysis was calculated on these data, the red fibers showed more change between quality groups than the other fiber types. Low quality Landrace had greater red fiber areas than normal Landrace pigs in the rectus femoris and semimembranosus muscles. While the individual red fiber areas of the gluteus medius muscle in low quality pigs were larger than normal pigs, total area of red fibers was lower. Poland Chinas had larger red fiber areas in the rectus femoris and gluteus medius muscles in the low quality pigs than in normals. These data show that differences in fiber size exist between muscles.

When fiber types were compared on the basis of percent of total numbers, the normal pigs, with the exception of the rectus femoris muscle in Landrace,

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Table 8. Relative area of each fiber type in three porcine muscles by breed and quality group.[1,2]

Muscle and fiber type	Breed and quality group				
	Poland China		Landrace		Chester White
	Normal	Low	Normal	Low	Normal
Rectus femoris					
Red	30.7	33.7	33.9	47.6	27.2
White	50.5	50.7	51.4	33.6	52.5
Intermediate	18.8	15.6	14.7	18.8	20.3
Semimembranosus					
Red	32.3	21.9	26.1	29.5	33.9
White	54.9	61.8	59.0	60.1	54.0
Intermediate	12.8	16.3	14.9	10.4	12.1
Gluteus medius					
Red	22.7	24.7	26.1	24.8	25.6
White	62.7	58.3	62.7	66.0	65.3
Intermediate	14.6	17.0	11.2	9.2	9.1

[1]Data calculated from mean number percent and mean fiber area for each fiber type.
[2]Area of each fiber type expressed as % of total area.

had more aerobic potential than low quality pigs. On the basis of area percent low quality pigs had the greater aerobic potential. Koch (1969) reported a similar pattern in fiber type distribution of the longissimus muscle of these same pigs. These data indicate that the histological

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observations per se were not entirely coincident with the postmortem muscle qualitative properties. Thus, chemical determinations, such as enzyme activities, probably would provide a clearer understanding of total metabolic potential of each muscle.

The distribution of red, white and intermediate fibers was also studied in Experiment II, however reciprocal stains of serial sections for SDH and phosphorylase activity was used to identify fiber types. Reciprocal stains were used in this experiment to more nearly identify the actual metabolic potential of each muscle studied. These results are presented in Table 9. No significant differences ($P > .05$) in fiber types (expressed as percent of either total number or area) were found between quality group or breeds, however, it was readily apparent that a larger number and area of fibers were categorized as intermediate than in Experiment I. The reciprocal staining technique used on the serial sections indicated that a large proportion of the fibers reacted positively for both SDH and phosphorylase activity and thus they were categorized as intermediate fibers. As can be seen from Figure 2, many fibers that were strongly positive for SDH activity also were strongly positive for phosphorylase. Thus, in Experiment I (only SDH activity assessed) more fibers were categorized as red than should have been based on their total metabolic potential as determined in Experiment II. The normal pigs had a higher percent of both number and area of red fibers in the rectus femoris and semimembranosus muscles than low quality pigs. The gluteus medius muscle of Hampshires had lower percents of number and area of red fibers in normal than low quality pigs. The opposite

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Table 9. Distribution of fiber types in three porcine muscles by breed and quality group (Experiment II).

Muscle and fiber type	% of total number[1]				% of total area[2]			
	Hampshire		Yorkshire		Hampshire		Yorkshire	
	Normal	Low	Normal	Low	Normal	Low	Normal	Low
Rectus femoris								
Red	16.4	11.8	9.8	8.3	11.1	9.7	7.1	5.9
White	29.7	35.9	44.8	47.9	32.6	39.3	50.6	55.1
Intermediate	53.9	52.3	45.3	44.9	56.3	51.1	42.3	39.1
Semimembranosus								
Red	24.9	12.1	19.6	15.4	21.1	9.4	15.5	11.9
White	47.8	37.8	42.5	40.1	51.0	39.5	49.5	48.4
Intermediate	27.4	50.2	37.9	44.2	28.0	51.2	35.0	39.8
Gluteus medius								
Red	16.1	21.2	15.5	7.0	13.0	18.1	13.4	5.1
White	52.8	48.7	42.9	53.7	52.7	50.4	43.0	57.2
Intermediate	31.1	30.1	41.6	39.3	34.3	31.5	43.6	37.7

[1]Each fiber type expressed as a % of total fiber number.
[2]Each fiber type expressed as a % of total fiber area.

pattern was observed in the gluteus medius muscle of Yorkshire pigs. The percents of total number and area of white fibers were inversely related to those of the red fibers. Area percent and number of intermediate fibers were higher in the rectus femoris and gluteus medius muscles of normal pigs but they were highest in the semimembranosus muscles of low quality pigs.

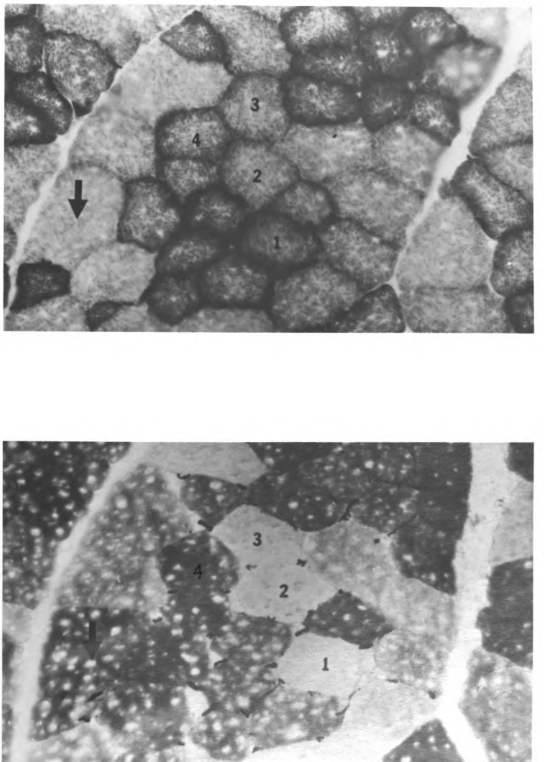

Figure 2. Section showing the reciprocal staining reactions to succinic dehydrogenase (SDH) and phosphorylase (P) top and bottom, respectively. Fibers 1, 2 and 3 stained positive for SDH and negative for P and were classified as red; fiber 4 stained positive for SDH and P and was labeled intermediate. The fiber identified by the arrow stained negative for SDH and positive for P and was classified as white.

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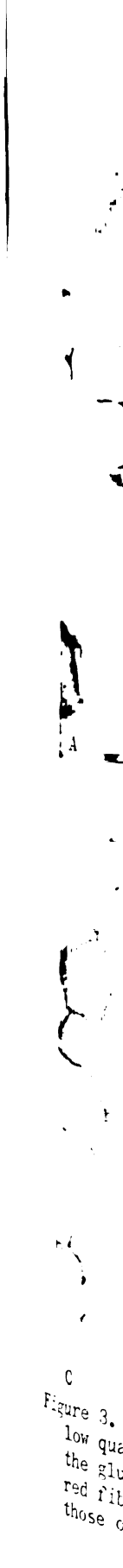
A higher percentage of red fibers was observed in the semimembranosus than in the rectus femoris muscle of normal Hampshires and both quality groups of Yorkshire pigs. Low quality Hampshire pigs had approximately the same percentages (number and area) of all fiber types in the rectus and semimembranosus muscles and they also had a higher number and area (percent) of red fibers in the gluteus medius.

Considerable variation in fiber types was observed within and between muscles and quality groups. While differences in relative numbers of each fiber type between muscles has been well documented, the variation observed between quality groups in this study can not be explained, since numerous factors are known to affect fiber type. However, greater differences in fiber type were observed between quality groups than between breeds in this study. Observations to be discussed later may help to explain the fiber type data.

Relative Size of Fiber Types

Figure 3 shows the difference in fiber sizes between quality groups and between muscles. Figures 3-A and 3-B show the rectus femoris and gluteus medius muscles, respectively, from a normal pig and Figures 3-C and 3-D show the same muscles from a low quality pig. The photomicrographs show increased size of red fibers between muscles (Figures 3-A and 3-B) and quality groups (3-A and 3-C, and 3-B and 3-D).

In the low quality pigs greater differences in size of the relative fiber types were observed in the semimembranosus muscle of both Hampshire and Yorkshire pigs than in the rectus femoris or gluteus medius muscles.



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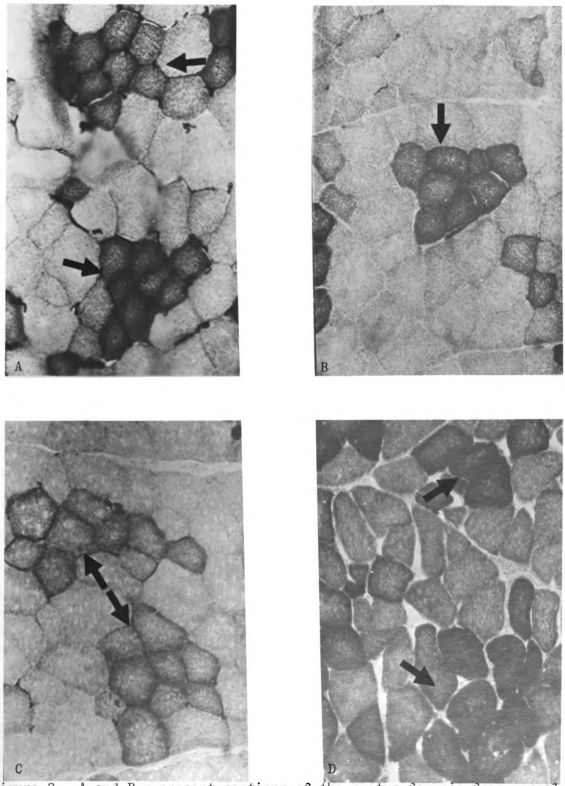

Figure 3. A and B represent sections of the rectus femoris from normal and low quality muscle, respectively. Sections C and D represent sections of the gluteus medius from normal and low quality muscle, respectively. The red fibers (indicated by arrows) of low quality muscles are larger than those of corresponding normal muscles.

The greatest increase in fiber size was in the red fibers in each muscle. This observation supports other data from Experiments I and II which indicate a larger red fiber area in the muscles of the pigs in the low quality groups. Fiber sizes, however, were not significantly ($P > .05$) correlated (see Appendix E) with 45 min pH in any of the muscles studied in Experiment II. The correlations between individual white fiber size and pH were positive in all muscles except the gluteus medius (0.23, 0.30 and -.12 for the rectus femoris, semimembranosus and gluteus medius muscles, respectively). Differences in fiber sizes between quality groups were more apparent among Yorkshire pigs. They also had lower pH values at 45 min. Percent of total white fiber area was also negatively correlated with 45 min pH in the gluteus medius muscles (-.13). Percents of total area of red and white fiber areas were positively correlated to 45 min pH values in the rectus femoris and semimembranosus muscles (0.33, 0.30 and 0.13 and 0.30, respectively). Percent intermediate fiber area was negatively related to 45 min pH in all three muscles (-.21, -.44, -.13 in the rectus femoris, semimembranosus and gluteus medius, respectively). Intermediate fibers in the semimembranosus muscle were more highly correlated with 45 min pH than any of the other fiber types.

The low positive correlations between the relative size of individual fiber types and 45 min pH suggest that fiber types *per se* are not affecting the development of low qualitative muscle properties. However, these data indicate that morphology of the fibers, especially size was associated with ultimate muscle quality. Cooper *et al.* (1969) reported larger fiber sizes in stress-susceptible than in stress-resistant pigs.

Capillary and Fiber Density

Capillary density is important to the maintenance of muscle homeostatic function because capillaries transport nutrients needed for metabolism to the cell and remove waste products from the cell. Sybesma (1965) stated that when large muscles contract there is an inefficient use of oxygen as well as greater heat production than in smaller muscles. This suggests that heavily muscled pigs require a denser capillary bed to supply the exaggerated oxygen demands of the larger muscles as well as for the removal of the greater heat produced during contraction. In the preceding discussion, it was observed that the muscle fibers, especially red fibers, tended to be larger among low quality pigs than in normals.

The right ham was perfused with India ink to identify and determine capillary bed density and the relationship, if any, of capillary density to postmortem properties. These capillary density results together with total muscle fiber densities for both experiments are presented in Table 10. The capillaries and fibers per square inch of picture were statistically analyzed and the mean values of each experiment were converted to capillaries or fibers per square millimeter of muscle. These values are presented in Table 10. In Experiment I, all the normal quality pigs were comparable in both capillary and fiber densities per unit of muscle. No significant ($P > .05$) differences in capillary density were observed between quality groups in the rectus femoris and semimembranosus muscles. However, the low quality pigs (Experiment I) consistently had lower capillary densities in these muscles than normal muscles. The number of fibers

Table 10. Capillary and fiber density for three porcine muscles by breed and quality group.[1,2]

Muscle and density item	Experiment I					Experiment II			
	Breed and quality group					Breed and quality group			
	Poland China		Landrace		Chester White	Hampshire		Yorkshire	
	Normal	Low	Normal	Low	Normal	Normal	Low	Normal	Low
Rectus femoris									
Capillaries	480	308	478	368	505	401^{b}	517^{b}	408^{b}	266^{a}
Fibers	505^{ab}	323^{a}	506^{ab}	323^{a}	615^{b}	378	424	424	293
Semimembranosus									
Capillaries	338	218	392	258	358	329^{a}	316^{a}	563^{b}	276^{a}
Fibers	390^{ab}	265^{a}	358^{ab}	265^{a}	505^{b}	293^{a}	273^{a}	454^{b}	243^{a}
Gluteus medius									
Capillaries	300^{b}	163^{a}	$\underline{480}^{b}$	283^{b}	285^{b}	332	372	408	247
Fibers	$\underline{450}^{b}$	293^{a}	$\underline{480}^{b}$	240^{a}	$\underline{518}^{b}$	250	303	355	230

[1]Mean values with the same or no superscript do not differ significantly ($P > .05$). Underlined means differ from other values at the $P < .01$ level.
[2]Capillary and fiber density is expressed per mm^2 of muscle.

per square millimeter of muscle were significantly different ($P < .05$) in these two muscles. Chester White pigs had the smallest fibers of all breeds in all muscles. Normal Poland China and Landrace pigs had significantly smaller ($P < .05$ and $P < .01$, respectively) fibers than the corresponding low quality pigs in the gluteus medius muscle, but not in the rectus femoris or semimembranosus. Normal Landrace pigs had significantly ($P < .05$) larger fibers in the semimembranosus muscle than Chester Whites.

Normal Landrace had significantly ($P < .01$) higher capillary density in the gluteus medius muscle than all the other pigs. Chester White, Poland China and low quality Landrace pigs had significantly ($P < .05$) more capillarity than low quality Poland China pigs in the gluteus medius muscle. In Experiment I, no significant breed differences for capillary density were observed in the rectus femoris and semimembranosus muscles. Landrace pigs showed significantly ($P < .01$) more capillary density in the gluteus medius muscle and had a significantly ($P < .05$) higher capillary to fiber ratio than the other breeds. In general, there were no differences in capillary to fiber ratios between quality group.

The results of Experiment II essentially followed the same pattern as in Experiment I, particularly among the Yorkshire pigs. In the Hampshire pigs, only the semimembranosus muscles followed the same pattern as the other breeds and quality groups. The differences between quality groups in the Hampshires was not as marked as in the Yorkshire breed. However, the differences in the postmortem muscle properties between quality groups were also less marked among Hampshires than in the other breeds. The

normal pigs in Experiment II had lower capillary and fiber densities than those in Experiment I. While low quality pigs in Experiment II generally had lower capillary and fiber densities than those in Experiment I, they more nearly approximated the low quality values in Experiment I.

Fiber density was more closely correlated with 45 min pH than capillary density. In Experiment I, the correlation coefficients between capillary density and 45 min pH for the rectus femoris, semimembranosus and gluteus medius muscles were 0.43, 0.53 and 0.45, respectively. These correlation coefficients for the rectus femoris and gluteus medius were significant at the $P < .05$ level while that for the semimembranosus muscle was significant at the $P < .01$ level. Fiber densities, on the other hand, were significantly correlated ($P < .01$) with 45 min pH in all three muscles (0.63, 0.59 and 0.73 for the rectus femoris, semimembranosus and gluteus medius muscles, respectively).

The correlation coefficients for these same parameters in Experiment II were not significantly correlated with 45 min pH. Fiber and capillary density were nonsignificantly correlated with 45 min pH (0.21 and 0.15; 0.32 and 0.31; 0.21 and 0.20 for fiber and capillary densities of the rectus femoris, semimembranosus and gluteus medius muscles, respectively). It is concluded from these data that the increased fiber size in low quality pigs exerted a greater influence on the rate of postmortem pH fall than capillary density. These data also suggest that the dilution of the capillary bed is a direct result of increased fiber size. Red fibers showed the greatest increase in size among the low quality pigs.

Anserine and Carnosine Determination

The development of PSE musclature has been attributed to the rapid pH fall postmortem while muscle temperature is still at or near body temperature. One of the factors affecting postmortem pH fall is the buffering capacity of the muscle. Thus, muscle anserine and carnosine contents were determined since Deutsch and Eggleton (1938) reported that the pK's of anserine and carnosine make them ideally suited for physiological buffering.

The anserine, carnosine and total imidazole content of the three ham muscles was determined by the diazo method (Parker, 1966) and the results are presented in Table 11 for Experiments I and II. No significant difference was observed between quality groups within breeds for any of the muscles studied. However, breed differences were readily apparent. In the rectus femoris muscle, Landrace had significantly ($P < .05$) more carnosine and total imidazole than Chester White and Poland China pigs. No significant differences were observed in the semimembranosus muscles between breeds. Chester White pigs had significantly ($P < .05$) less carnosine and more ($P < .05$) anserine in the gluteus medius muscle than the other breeds.

Even though the content of anserine and carnosine in the low quality group were equal to or greater than levels in normal pigs, faster pH declines were observed among low quality pigs (Figure 1). It appears that these dipeptides had little, if any, effect on the rate of pH fall. It should be emphasized, however, that the concentration of these dipeptides and pH fall were measured in different muscles.

Table 11. Total imidazole, carnosine and anserine content of three porcine muscles by breed and quality group.[1]

Muscle and chemical component	Experiment I[2]					Experiment II[3]			
	Breed and quality group					Breed and quality group			
	Poland China		Landrace		Chester White	Hampshire		Yorkshire	
	Normal	Low	Normal	Low	Normal	Normal	Low	Normal	Low
Rectus femoris									
Total imidazole	6.66^{a}	7.01^{a}	9.58^{b}	8.82^{b}	6.59^{a}	7.84^{b}	8.82^{b}	7.91^{b}	6.46^{a}
Carnosine	3.89	5.28	3.40	3.54	4.44	6.94	6.87	6.32	5.69
Anserine	2.77^{a}	1.73^{a}	6.18^{b}	5.28^{b}	2.15^{b}	.90	1.95	1.59	.77
Semimembranosus									
Total imidazole**	8.54	9.02	10.00	8.47	7.98	8.47^{b}	8.40^{b}	8.26^{b}	6.80^{a}
Carnosine**	5.14	6.25	7.36	7.15	5.62	8.26^{b}	8.33^{b}	7.98^{b}	6.29^{a}
Anserine	3.40	2.77	2.64	1.32	2.36	.21	.07	.28	.51
Gluteus medius									
Total imidazole	7.01$_{ab}$	7.84$_{b}$	7.50$_{b}$	7.71$_{b}$	7.72$_{a}$	9.44^{b}	9.44^{b}	8.47^{ab}	7.49^{a}
Carnosine**	5.83^{ab}	6.32^{b}	6.46^{b}	7.08^{b}	5.14^{a}	8.82^{b}	8.68^{b}	7.64^{a}	7.08^{a}
Anserine	1.18^{a}	1.52^{a}	1.04^{a}	.63^{a}	2.58^{b}	.62	.76	.93	.41

[1]Values expressed as μM/g of fresh muscle.
[2]Means with the same or no superscripts do not differ significantly (P > .05).
[3]Means with the same or no superscripts do not differ significantly (P > .01).
_____ underlined means differ at P < .01 level in Experiment I.
_____ underlined means differ at P < .05 level in Experiment II.

As expected, the rectus femoris (a red muscle) had significantly ($P < .01$) more anserine and less carnosine than the semimembranosus or gluteus medius muscles. Carnosine content of the gluteus medius was more closely correlated with 45 min pH than that in any of the other muscles. The correlation coefficient (-.49) between carnosine and 45 min pH in this muscle was highly significant ($P < .01$). The correlation between carnosine content and 45 min pH in the gluteus medius muscle of Landrace pigs (-.62) was also significant ($P < .05$). Total imidazole and anserine contents were positively, but nonsignificantly, correlated with 45 min pH. The higher correlations between pH values of the longissimus muscle and carnosine content of the gluteus medius, as compared to the other two ham muscles, may be attributable to their anatomical and morphological similarities.

In Experiment II, Hampshire pigs showed no difference between quality groups in total imidazole, carnosine or anserine content among any of the muscles. Normal Yorkshire pigs had significantly ($P < .05$) less carnosine in the gluteus medius muscle than normal Hampshires. Low quality Yorkshire pigs had significantly ($P < .01$) less total imidazole in the rectus femoris and semimembranosus muscles than the other quality groups. They also had significantly ($P < .01$) less total imidazole in the gluteus medius muscle than Hampshire pigs. Carnosine content of the semimembranosus muscle of the low quality Yorkshire pigs was significantly ($P < .01$) lower than that for all other quality groups.

Hampshire pigs tended to have higher total imidazole, anserine and carnosine contents than Yorkshires. These data are consistent with the results of Experiment I since significant ($P < .05$) breed differences were apparent as already discussed.

Low, nonsignificant correlation coefficients were obtained between carnosine content and 45 min pH as well as transmission values in all three muscles of Hampshire pigs. Yorkshire pigs on the other hand, showed significant ($P < .05$) positive correlations between 45 min pH and carnosine content of the rectus femoris and semimembranosus muscles (0.68 and 0.69, respectively). A substantial (0.50), but nonsignificant, correlation was obtained between the same two parameters in the gluteus medius muscle. Transmission values were negatively correlated with carnosine content of the semimembranosus muscle (-.69, $P < .05$). These data indicate that carnosine content was associated with postmortem muscle pH but the mechanism of its involvement needs further study. Since the pKa value of carnosine is 6.9, it may be reasonable to assume that its greatest influence would occur immediately prior to, or just after exsanguination. Shabonova (1954) and Tallan (1955) reported lower carnosine levels in degenerative muscle than in normal muscles. Sybesma (1965) concluded that transmission values were more closely associated with muscle degenerative changes than with postmortem pH patterns. Since the low quality Yorkshire pigs in this study had less carnosine than normal pigs, muscles of all the pigs in Experiment II were examined histologically for possible myopathic changes.

Histopathological Observations of Muscle and Nerve Fibers

Skeletal muscle fibers. Histological observation for possible degenerative changes were made on longitudinal and cross sections of skeletal, neural and cardiac (Yorkshires only) tissues of the pigs in Experiment II.

The cardiac and skeletal muscle tissues were embedded in paraffin and stained with hematoxylin and eosin (H and E).

Longitudinal and cross sections of a portion of the sciatic nerve were also embedded in paraffin and stained with Cresyl-echt violet and luxol fast blue to observe possible neural morphological alterations.

Histological observations of the skeletal muscles of the normal Hampshire pigs ranged from suspicious to moderate degenerative changes according to the classification system of Homburger et al. (1966) among the three ham muscles. The changes most frequently observed included nuclear chaining, centrally placed nuclei (> 5/100) and nuclear proliferation. To a lesser extent variations in fiber size due to splitting, vacualization and perinuclear haloes were observed. In most cases degeneration was based primarily on centrally placed nuclei, nuclear rowing, nuclear proliferation and fragmentation. Numerical values ranging from 1 to 5 were assigned to the classification of degrees of degeneration as described in the experimental section. Normal muscles were assigned the value of 5 and the most marked degenerative muscles a score of 1. The semimembranosus and gluteus medius muscles were more degenerative than the rectus femoris, but the rectus femoris muscles of low quality Yorkshires were also affected.

Several normal pigs in both breeds showed enough degenerative changes to be classified as suspicious. Mean values of degrees of degenerative changes between breeds and quality group are presented in Table 12. The gluteus medius muscle was the most suspicious in the normal Yorkshire pigs while the semimembranosus muscle of normal Hampshires was most suspicious.

Table 12. Mean subjective scores of muscle degeneration in three porcine muscles by breed and quality group.[1]

Muscle	Breed and quality group			
	Hampshire		Yorkshire	
	Normal	Low	Normal	Low
Rectus femoris	4.5	4.4	4.6	3.8
Semimembranosus	4.2	4.5	4.8	3.2
Gluteus medius	4.5	3.0	4.4	3.2

[1]Degenerative classifications were assigned the following values: normal = 5, suspicious = 4, mild = 3, moderate = 2 and marked = 1.

The low quality Hampshire pigs showed more pronounced degenerative changes in the gluteus medius muscle, although moderate degeneration was observed in the semimembranosus muscle of one low quality Hampshire pig. Yorkshire pigs, on the other hand, showed degenerative changes in all three muscles of the low quality group.

Three of the low quality Yorkshires showed mild to moderate degrees of degeneration in the semimembranosus and gluteus medius muscles. No significant differences ($P > .05$) were found between quality groups for degenerative scores. Thus the values of all pigs were combined for the correlation analysis between subjective degenerative scores and objective quality assessments within each muscle. Significant correlation coefficients were found between muscle degeneration scores and 45 min pH values and with 24 hr transmission values. Forty-five min pH values were significantly ($P < .05$) correlated with degeneration scores in the rectus femoris (0.46) but not in the semimembranosus (0.38) or gluteus medius (0.31)

muscles. These relationships are surprising since the most marked degenerative changes were observed in the gluteus medius muscle. On the other hand, 24 hr transmission values were significantly correlated with degeneration scores in two of the three muscles studied. The correlation coefficient between the latter two parameters (-.38) in the rectus femoris was nonsignificant, whereas, the same correlations in the semimembranosus and gluteus medius were (-.62 and -.61, respectively) significant at the 0.01 level. These data are in agreement with the report of Hart (1962) in that transmission values were more highly correlated with muscle quality associated with degeneration changes than with 45 min pH values.

Since carnosine content has been reported to be lower in degenerative muscles (Tallan, 1955), correlations were calculated between carnosine content and degenerative scores. The correlations indicate that carnosine content was not significantly related to degenerative score in any of the muscles. These results suggest that degenerative changes probably did not reach the clinical stage.

Giant muscle fibers (large round fiber which usually stains intermediate to SDH and negative to phosphorylase) were observed in most of the low quality pigs and when found they occurred in all three muscles. No account was made of their frequency in this study but they appeared to be more numerous in muscles from the low quality pigs than in normals. Cooper et al. (1969) reported more of these fibers in stress-susceptible than in stress-resistant pigs. Giant fibers are shown in Figure 4.

Nutritional dystrophies in domestic animals closely resembles certain human progressive myopathies morphologically but they constitute a separate disease entity (Homburger et al., 1966). Vitamin E deficient rations have

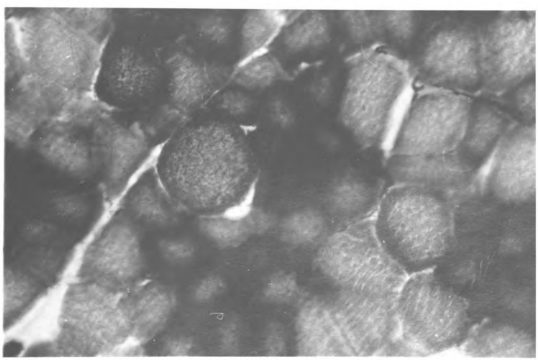

Figure 4. Giant (large rounded fibers) fibers were observed in the muscle sections of some pigs. They appeared to be more numerous among low quality pigs.

been reported to result in muscle degenerative changes among some commercial swine herds in Michigan (Michel, 1969). Since vitamin E deficiency results in pathological and morphological observations resembling the changes found in this study, the histochemical slides were reviewed for possible alterations to support the histological data obtained. Figures 5 and 6 show some of the degenerative changes observed in the pigs of Experiment II. The normal histochemical pattern, with the reciprocal stains of SDH and phosphorylase activity, is shown in Figure 2. Deviation from the pattern shown in Figure 2 was observed in three of the low quality Yorkshire pigs but none of the Hampshires. This deviation showed that muscle fibers which stained strongly positive to SDH activity also stained strongly positive for phosphorylase activity and fibers negative to SDH activity were also negative to phosphorylase activity (Figure 7). Muscles with normal staining patterns were incubated in the same media as those showing the deviations in staining reactions, thus the possibility of differences in incubating media can be ruled out. Engel et al. (1966) reported that phosphorylase activity (histochemical) was lost in the early stages of denervated and tenotomized cat muscle. These authors also found that TPNH or DPNH dehydrogenase reaction showed positive reciprocal stain activity to menadione-mediated α-glycerophosphate dehydrogenase in normal fibers. They further observed that a high degree of activity may occur in the same cell in abnormal fibers and that neither phosphorylase nor oxidative enzyme reactions are reliable for detecting the original fiber type of abnormal fibers. The histological evidence of muscle degeneration

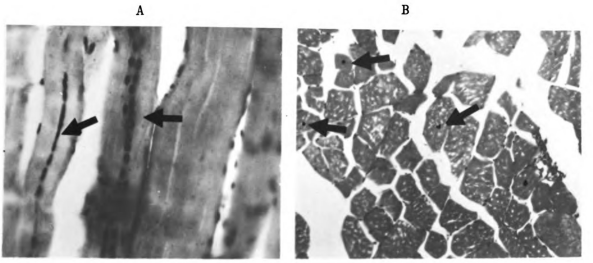

Figure 5. Section A illustrates nuclear chaining (arrows) and absence of normal ultra structure. Section B shows centrally placed nuclei (arrows).

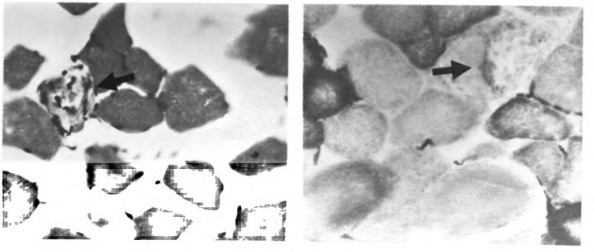

Figure 6. Section A shows a degenerative muscle fiber (arrow) prepared with H & E stain. Section B likewise shows muscle fiber degenerative changes (arrow) but stained for SDH activity.

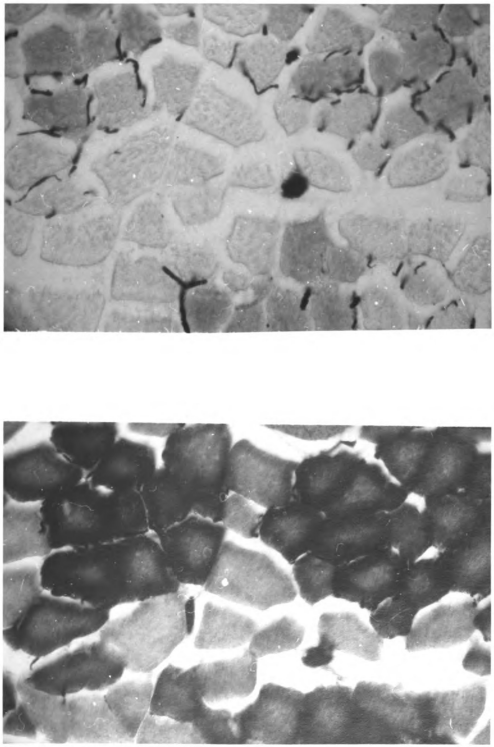

Figure 7. The lower section shows normal staining reaction for succinic dehydrogenase activity while the upper section illustrates abnormal staining reaction for phosphorylase activity when reciprocal stains were compared. The normal reciprocal staining reactions are shown in Figure 2.

has been described in a previous section for these same pigs. Since some histological evidence of degeneration has been observed in Experiment II, the fiber type pattern as shown by SDH and phosphorylase activity may not represent the true pattern.

Nerve fibers. In order to examine the possibility of neural involvement in the altered fiber type patterns, histological observation of the sciatic nerves of these pigs were made and they showed indications of loss of myelin. Figure 8 shows photomicrographs of normal and abnormal nerve fibers. This loss of myelin (Figure 8 B) may possibly interfere with the normal passage of neural impulses. However, it is not possible to discern from these data if the demyelinated nerve fibers innervated the specific muscle sections that were examined in this study. Additionally it can not be determined from the data whether the muscle observations resulted from the nerve fiber abnormalties or vice versa. The relationship of these histological and histochemical observations to the development of PSE musclature need further elucidation.

Cardiac muscle fibers. Ventricular muscle tissues were also examined histologically to determine if degenerative changes had occurred in the myocardium. Indications of overexertion of cardiac muscle such as the formation of nuclear chains were observed. No evidence of degenerative heart muscles was found in any of the pigs. Nuclear chains were observed in several pigs but they were not specifically confined to any one quality group. Figure 9 shows a series of nuclear chains that were observed in

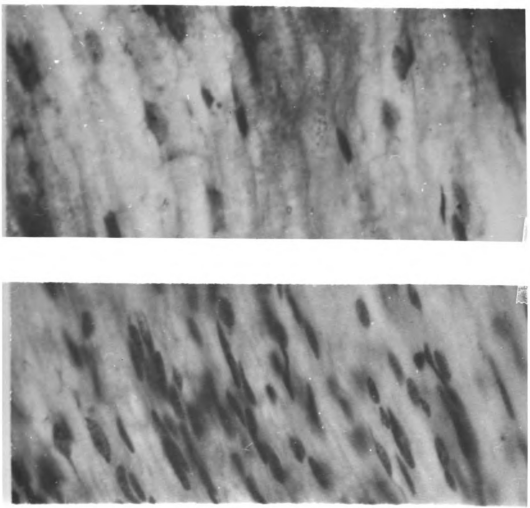

Figure 8. The upper section shows a normal sciatic nerve fiber, while the lower section represents abnormal histology of the sciatic nerve.

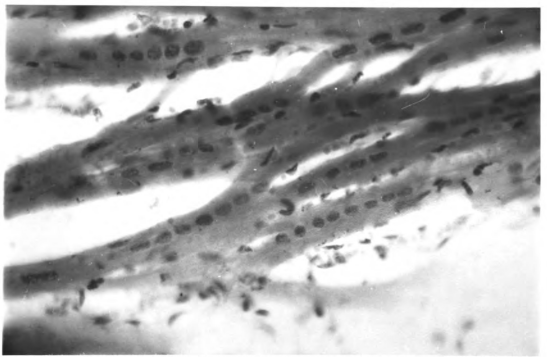

Figure 9. This section illustrates nuclear rowing in porcine cardiac muscle.

one of the Yorkshire pigs in this study. Nuclear chains consisting of 3 to 4 nuclei occurred more frequently than longer chain lengths of 6-10 nuclei. Electrical axis deviation and long nuclear chains were found in two Yorkshire pigs both of which were low quality. These histological observations together with the electrical axis deviation data lend support to the suggestion of Koch (1969) that abnormal cardiac function may affect the ultimate qualitative properties of the porcine muscle.

SUMMARY

The results of this study were obtained from two separate experiments designed to study the relationship of some physiological and anatomical parameters to the ultimate qualitative properties of porcine muscle. Forty-three Poland China, Landrace and Chester White pigs were included in Experiment I. Thirty-six pigs (Hampshire and Yorkshire) were included in Experiment II. Two quality groups (normal and low quality) of 5 pigs each were selected within each breed, except Chester Whites (only normal quality pigs), on the basis of 45 min pH and 24 hr subjective quality scores in Experiment I, and in Experiment II, 45 min pH and 24 hr transmission values.

Physiological data including heart rates, blood pressure and cardiac function were determined from polygraphs (Experiment I) and electrocardiograms (Experiment II). Respiration rates (actual count) and whole blood pH were also determined. Muscle fiber types were identified by SDH activity in Experiment I and by the reciprocal relationship of SDH and phosphorylase activity in Experiment II. Both area and number of each fiber type in the rectus femoris, semimembranosus and gluteus medius muscles were determined.

The right ham of each pig was perfused with an India ink solution and the capillary and fiber densities determined from paraffin embedded (Experiment I) or fresh frozen (Experiment II) sections. Absolute amounts of myoglobin and its derivatives were determined in Experiment I, and anserine and carnosine content of the three ham muscles was determined in both experiments. Histological examination of skeletal and myocardial tissues

as well as nerve fibers were made for possible evidence of degenerative changes.

The two quality groups were significantly different ($P < .01$) in the parameters used for categorization into groups in all breeds except the Hampshires (significant at the $P < .05$ level). No significant differences were found between quality groups for any of the antemortem physiological data obtained in Experiment I. Mean blood pressures of the normal quality pigs were comparable, while the low quality Poland Chinas had higher mean blood pressure than all other quality groups. On the other hand, the low quality Landrace had lower mean blood pressures than normal Landrace pigs. Blood pressures of Chester Whites were intermediate to those of normal Poland China and Landrace pigs. The mean values for all the physiological data were within reported ranges for swine.

None of the physiological data were significantly correlated with objective muscle quality values.

The muscles of low quality Poland China pigs had more total myoglobin (Mb) than normals, while normal Landrace had more total mb than those with low quality. The total Mb content of Chester Whites was comparable to that of the normal Landrace and low quality Poland China pigs. Reduced myoglobin and metmyoglobin values paralleled those of total myoglobin between quality groups while oxymyoglobin showed the reverse pattern of total myoglobin. These data indicate that the capacity to use oxygen from myoglobin may differ with breed.

Normal pigs had more red muscle fibers in the rectus femoris muscle than low quality pigs of all breeds except the Landrace in Experiment I.

Percentage of red fibers (expressed as percent of total fibers) decreased from the rectus femoris to semimembranosus to gluteus medius while white fibers increased correspondingly. Regardless of fiber type, larger areas were observed in the low quality pigs than normals. In general, red fiber area was larger among low quality pigs than in those with normal quality muscle. Fiber sizes were significantly correlated ($P < .01$) with 45 min pH in the three ham muscles.

The number of capillaries per unit of muscle was lower in the low quality pigs than in normals. Normal pigs had significantly ($P < .01$) more capillarity in the gluteus medius muscle than those with low quality. There was no significant ($P > .05$) difference in the number of capillaries associated with fiber types between quality groups. Dilution of the capillary bed was observed between quality groups in all breeds but all breeds were not affected to the same extent. Thus low quality pigs not only had quantitatively less capillarity (capillaries per sq mm of muscle), but the efficiency of the capillary bed was further aggravated by fiber hypertrophy in these muscles.

Anserine, carnosine and total imidazole content was not directly associated with the rate of pH fall. However, carnosine content was significantly correlated with transmission values. The low quality Yorkshires had significantly less ($P < .05$) carnosine than the other quality groups (Experiment II). Carnosine content was more highly correlated with transmission values among the Yorkshire pigs than in Hampshires.

Histopathological observation of muscles (cardiac and skeletal) of the pigs in Experiment II revealed some muscle degeneration in both quality groups. Yorkshire pigs showed more degeneration than Hampshires,

and the semimembranosus and gluteus medius muscles were affected to a greater extent than the rectus femoris muscle.

Histochemical observation of these muscles showed severe degeneration in some muscles with a concomitant loss of phosphorylase activity. Loss of myelin was observed in two of the pigs which showed loss of phosphorylase activity. Additionally, these same two pigs showed electrical axis deviation of the heart and nuclear chaining of the myocardium. Myopathic lesions, heart axis deviation and decreased carnosine levels suggest that the difference in ultimate muscle quality observed in the pigs in Experiment II may have been complicated by other factors. Vitamin E and/or selenium deficiencies show histological changes similar to those observed in this study. Michel et al. (1969) reported that field cases show a deficiency of these nutrients in some commercial swine herds in Michigan. The possibility of a relationship between these nutrients and muscle quality needs further study.

Transmission value was significantly ($P < .01$) correlated with muscle degenerative scores in the semimembranosus and gluteus medius muscles. The correlation between 45 min pH and degenerative score was significant at the 0.05 level (0.46)in the rectus femoris muscle only. These data indicate that both of these estimates (45 min pH and transmission value) should be used to evaluate muscle quality objectively.

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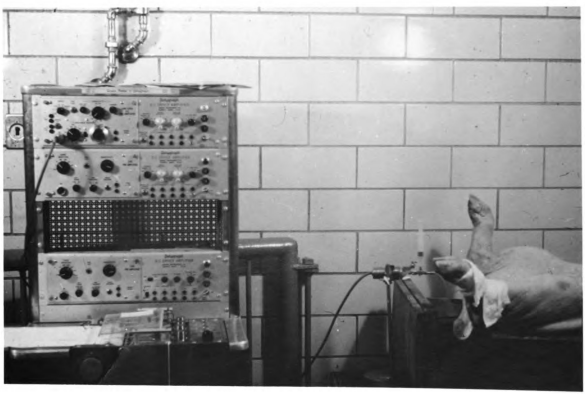
APPENDIX



Appendix A. Photograph of blood pressure apparatus.

Appendix B. Histological procedures:

I. Hematoxylin and eosin:

A. Fixative:

10% Formalin solution

37.40% Formaldehyde	100 ml
Distilled water	900 ml

B. Autotechnicon - dehydrating, clearing and infiltration:

1.	70% Ethyl alcohol	1 hr
2.	70% Ethyl alcohol	1 hr
3.	80% Ethyl alcohol	1 hr
4.	95% Ethyl alcohol	1 hr
5.	95% Ethyl alcohol	1 hr.
6.	Absolute ethyl alcohol	1 hr
7.	Absolute ethyl alcohol	1 hr
8.	Xylene	1 hr
9.	Xylene	1 hr
10.	Paraffin (Paraplast)	1 hr
11.	Paraffin (Paraplast)	2 hr
12.	Embed in paraffin (Paraplast) and cool rapidly	

C. Dehydration - clearing and infiltration (without Autotechnicon):

1.	Fixative - 10% Formalin	
2.	Wash tap water	overnight
3.	50% Ethyl alcohol	4 hr
4.	70% Ethyl alcohol	4 hr
5.	80% Ethyl alcohol	2 hr
6.	95% Ethyl alcohol (3 changes)	1 hr each
7.	Absolute ethyl alcohol (2 changes)	1 hr each
8.	Absolute alcohol and terpinol (1:1, v/v)	overnight at 2°C
9.	Paraffin (4 changes)	1 hr each at 60°C
10.	Embed as described above	

D. Stock solutions and staining procedures:

Solutions:

Hematoxylin and eosin

Eosin (stock solution)

Eosin	1 g
95% Ethyl alcohol	100 ml

Dilute 1:1 with distilled water before use.

Appendix B. (continued)

Harris hematoxylin (stock solution)
Hematoxylin 2.5 g
Aluminum ammonium sulfate 50.0 g
Mercuric oxide 1.25 g
Absolute alcohol 25.0 ml
Distilled water 500.0 ml
Dilute 1:1 with distilled water and filter before use.

Staining:

1.	Xylene	3 min
2.	Xylene	3 min
3.	Absolute alcohol	2 min
4.	Absolute alcohol	2 min
5.	95% Ethyl alcohol	2 min
6.	80% Ethyl alcohol	2 min
7.	70% Ethyl alcohol	2 min
8.	50% Ethyl alcohol	2 min
9.	Distilled water	rinse until clear
10.	Harris hematoxylin	5 min
11.	Tap water	rinse until clear
12.	Acid alcohol (0.25% HCl)	5 dips
13.	Ammonia in tap water	dip until blue
14.	Distilled water	rinse
15.	Eosin	1 min
16.	95% Ethyl alcohol	wash (2-5 dips)
17.	Absolute alcohol	wash (5-10 dips)
18.	Xylene	2 min
19.	Xylene	5-10 min
20.	Remove excess moisture from freshly stained slide and mount in permamount and coverslip.	

E. Staining procedure for eosin (Experiment I):

Follow procedure for hematoxylin and eosin except for steps 9-13.

F. Solutions and staining procedures of nerve fibers:

Fixative 10% formalin

Solutions:

1. Luxol Fast Blue solution
Luxol fast blue 0.1 g
95% Ethyl alcohol 100.0 ml
Add 5 ml of 10% acetic acid to each 1000 ml of solution. Solution is stable.

Appendix B. (continued)

2. Cresyl-Echt Violet solution
   Cresyl-echt violet acetate 0.1 g
   Distilled water 100.0 ml
   Just before using, add five drops of 10% acetic acid to every 30 ml of solution and filter.

3. Lithium Carbonate solution
   Lithium carbonate 0.05 g
   Distilled water 100.0 ml

Staining procedure:

1. Deparaffinized through xylene and absolute alcohol and then through several changes (minimum of 3) of 95% alcohol.
2. Stain overnight (16 to 24 hr) in Luxol fast blue solution at 37°C.
3. Wash in 95% alcohol to remove excess stain.
4. Wash in distilled water.
5. Begin differentiation by quick immersion in 0.05% lithium carbonate.
6. Continue differentiation in 70% alcohol until gray and white matters can be distinguished.
7. Wash in distilled water.
8. Finish differentiation by rinsing briefly in 0.05% lithium carbonate and then put through several changes (minimum of 3) of 70% alcohol until the white matter contrasts (greenish blue) sharply with the gray matter (colorless).
9. Wash thoroughly in distilled water.
10. Stain for 6 min in warm cresyl violet solution.
11. Differentiate in several changes of 95% alcohol.
12. Dehydrate in absolute alcohol, clear in xylene and mount.

Appendix C. Solutions used in anserine and carnosine determination:

A. Diazotized p-Bromoaniline:

1. Bromoaniline - dissolve 2.25 g of bromoaniline in 22.5 ml of 37% HCl in a 250 ml volumetric flask then dilute to 250 ml with distilled water. Make fresh daily.

2. Sodium nitrite - dissolve 4.64 g of $NaNO_2$ in distilled water and dilute to 100 ml. Store at 4°C.

3. Preparation of diazotized p-bromoaniline - add 5 ml of bromoaniline solution (1) to 5 ml of sodium nitrite solution (2) and allow to react for 5 min (in ice bath). After 5 min add 20 ml of $NaNO_2$ solution (2) and allow to react for 15 min. Take 9 ml of this mixture and dilute to 50 ml with cold distilled water. This solution should be made from stock solutions (1 and 2) every 4 hr. Solutions must be kept cold.

B. 2,4-Dinitro-1-fluorobenzene solution (NDFB):

Dissolve 0.4 ml NDFB into 50 ml of 95% ethanol. Solution is stable for 10 days.

Appendix D. Data used to categorize the pigs into quality groups.

Experiment I[a,b]			Experiment II[a,b]		
Pig No.	45 min pH	Subjective quality score	Pig No.	45 min pH	24 hr transmission value, %
PC-1	6.06	13	H-1	6.50	37.2
PC-2	5.80	7	H-2	6.60	18.6
PC-3	6.55	11	H-3	5.78	37.0
PC-4	6.14	5	H-4	6.70	7.0
PC-5	5.72	5	H-5	6.55	7.0
PC-6	6.06	8	H-6	6.65	6.0
PC-7	5.67	9	H-7	6.40	32.0
PC-8	6.10	11	H-8	6.02	70.0
PC-9	5.63	7	H-9	6.57	60.0
PC-10	6.05	7	H-10	6.58	54.0
PC-11	6.05	10	H-11*	6.80	18.0
PC-12	5.65	5	H-12	6.55	35.0
PC-13	6.22	11	H-13	6.40	22.0
PC-14	5.61	4	H-14	6.58	29.0
PC-15	5.42	5	H-15	6.66	12.0
PC-16	5.27	8	H-16	6.40	39.0
PC-17	5.33	5	H-17	6.86	12.0
PC-18	5.50	11	H-18	6.64	17.0
PC-19	5.80	6	H-19	6.40	13.0
LR-1	5.48	6	H-20	6.51	12.0
LR-2	6.26	9	Y-1	6.80	8.0
LR-3	5.70	7	Y-2	6.80	10.5
LR-4	6.08	6	Y-3	6.50	34.0
LR-5	5.97	11	Y-4	6.50	22.0
LR-6	5.20	5	Y-5	5.50	67.0
LR-7	6.16	6	Y-6	6.70	11.0
LR-8	6.26	10	Y-7	6.80	18.0
LR-9	6.18	10	Y-8	6.40	13.0
LR-10	6.18	11	Y-9	5.50	79.5
LR-11	5.98	5	Y-10*	6.06	10.0
LR-12	6.27	12	Y-11	5.56	74.0
LR-13	6.20	13	Y-12	6.35	46.0
LR-14	6.02	9	Y-13	6.38	20.0
LR-15	6.12	9	Y-14	6.68	15.0
LR-16	6.10	8	Y-15	6.58	14.0
CW-1	6.07	12	Y-16	6.23	42.0
CW-2	6.45	13			
CW-3	6.16	7			
CW-4	6.26	14			
CW-5	6.38	13			
CW-6	6.13	13			

[a]Pigs in Experiment I which were categorized as normal: PC-3, 8, 10, 11, 13, LR-2, 8, 10, 12, 13 and CW-1, 2, 4, 5 and 6. Experiment II: H-4, 5, 6, 15 and 17 and Y-1, 2, 6, 7 and 14.

[b]Pigs in Experiment I which were categorized as low quality: PC-14, 15, 16, 17, 19 and LR-1, 3, 5, 6 and 11. Experiment II: H-3, 8, 9, 10, 16 and Y-5, 9, 11, 12 and 14.

*Postmortem inspection indicated pericarditis.

Appendix E. Simple correlation coefficients.

Quality evaluations vs 45 min pH	
Subjective quality score Experiment I only	0.63**
24 hr transmission value (Yorkshires only)	-.97**
24 hr transmission value (Hampshires only)	-.44
24 hr transmission value (all pigs in Experiment II)	-.71**
Physiological data vs 45 min pH	
Respiration rate	-.14
Blood pressure	-.06
Heart rate	0.15
Electrical axis rotation (Hampshire pigs)	-.27
Electrical axis rotation (Yorkshire pigs)	0.42
Hematocrits (all pigs in Experiment I)	-.15

Muscle data vs 45 min pH

	Muscles		
	Rectus femoris	Semimembranosus	Gluteus medius
Total myoglobin	0.10	0.43*	0.30
Area/white fiber	0.23	0.30	-.12
Percent white fiber area	0.13	0.30	-.13
Percent red fiber area	0.33	0.30	0.35
Percent intermediate fiber area	-.21	-.44	-.12
Capillarity density			
Experiment I (all pigs)	0.43*	0.53**	0.45*
Experiment II	0.15	0.32	0.20
Fiber densities			
Experiment I (all pigs)	0.63**	0.59**	0.73**
Experiment II	0.21	0.31	0.21
Red fiber per unit of red fibers	0.29	0.37	0.26
White fiber per unit of white fibers	0.23	0.31	0.20
Intermediate fiber per unit of intermediate fibers	0.17	0.41	-.12
Carnosine content (all pigs)			-.49**
Carnosine content (Landrace pigs only)			-.62**
Carnosine content (Yorkshire pigs only)	0.68*	0.69*	0.50
Muscle degeneration scores	0.46*	0.38	0.31

*Significant at $P < .05$ level
**Significant at $P < .01$ level.

Appendix E. (continued)

	Muscles		
	Rectus femoris	Semimembranosus	Gluteus medius
Muscle data vs quality evaluations			
Muscle degeneration scores with 24 hr transmission values	-.38	-.62**	-.61**
Carnosine content with subjective quality scores (Experiment I)	-.15	-.13	-.44

*Significant at $P < .05$ level.
**Significant at $P < .01$ level.

Appendix F. Physiological data

Pig No.	Heart rate	Respiration rate	% Hematocrit	Blood pressure	Whole blood pH
PC 1	X	X	34.0	X	X
PC 2	X	X	39.0	X	X
PC 3	87	11.1	27.3	113.0	X
PC 4*	--	16.0	33.0	-	X
PC 5	95	29.0	34.0	160.0	X
PC 6	103	20.0	36.8	173.0	X
PC 7	120	21.8	22.8	150.0	X
PC 8	156	25.8	40.0	133.0	X
PC 9	144	18.5	18.9	174.0	X
PC 10	96	18.0	24.5	100.0	X
PC 11	168	13.0	43.0	191.0	X
PC 12	168	20.0	45.0	196.0	7.26
PC 13	120	22.5	42.5	227.5	7.34
PC 14	132	12.5	42.0	149.3	7.38
PC 15	136	27.0	42.0	180.0	7.30
PC 16	132	28.5	42.0	172.5	7.34
PC 17	132	25.0	37.2	179.7	7.24
PC 18	120	19.0	41.3	159.5	7.56
PC 19	156	21.0	37.3	197.5	7.39
LR 1	156	14.0	41.5	113.0	7.32
LR 2	180	25.5	41.5	157.0	7.35
LR 3	108	13.0	40.1	152.0	7.33
LR 4	156	25.5	42.0	165.0	7.37
LR 5	138	13.5	45.0	196.0	7.26
LR 6	128	17.5	28.0	130.0	7.56
LR 7	131	16.5	27.0	190.0	--
LR 8	175	18.0	39.8	175.0	7.52
LR 9	132	15.5	39.5	135.0	7.45
LR 10	168	18.0	37.8	141.0	7.40
LR 11	108	38.5	42.8	129.0	7.37
LR 12	168	18.5	37.0	163.0	7.47
LR 13	168	17.0	40.0	158.0	7.18
LR 14	168	19.5	37.0	133.0	7.42
LR 15	156	16.5	36.3	133.0	7.42
LR 16	125	43.0	37.0	176.0	7.48
CW 1*	168	30.0	41.3	171.0	7.42
CW 2*	156	12.5	36.0	161.0	8.04
CW 3	180	12.0	44.0	178.0	7.33
CW 4	144	12.5	25.0	136.0	7.33
CW 5	120	22.5	39.5	134.0	7.40
CW 6	180	10.0	47.3	180.0	7.21
CW 7	144	35.0	36.0	153.0	7.38
CW 8	132	22.0	40.0	164.0	7.15
PC $\bar{X}$	129.1	20.5	35.8	165.8	7.35 (8)
LR $\bar{X}$	145.1	20.7	38.5	152.9	7.39 (15)
CW $\bar{X}$	153.0	19.6	38.6	156.4	7.41 (8)

*Died

Appendix F. (continued)

Pig No.	Heart rate	P-wave	P-R seg.	P-R int.	Qrs. comp.	S-T seg.	S-T int.	T-wave	Q-T int.
	Electrocardial intervals (time)(seconds)								
H-3	139.3	0.060	0.051	0.111	0.052	0.102	0.182	0.082	0.245
H-4	174.9	0.068	0.021	0.091	0.051	0.106	0.162	0.064	0.204
H-5	94.2	0.060	0.078	0.138	0.038	0.144	0.217	0.073	0.258
H-6	132.1	0.060	0.040	0.100	0.046	0.087	0.149	0.062	0.211
H-8	151.3	0.060	0.040	0.099	0.050	0.086	0.165	0.079	0.221
H-9	110.8	0.055	0.039	0.094	0.051	0.127	0.196	0.069	0.226
H-10	110.1	0.053	0.040	0.093	0.036	0.118	0.173	0.055	0.221
H-15	102.2	0.060	0.047	0.107	0.050	0.101	0.186	0.080	0.256
H-16	130.7	0.056	0.045	0.101	0.040	0.124	0.193	0.069	0.235
H-17	99.4	0.060	0.041	0.101	0.044	0.132	0.189	0.057	0.251
Y-1	152.9	0.045	0.040	0.085	0.049	0.092	0.146	0.054	0.230
Y-2	120.9	0.060	0.031	0.082	0.050	0.114	0.173	0.059	0.238
Y-5	126.5	0.048	0.050	0.098	0.046	0.130	0.200	0.070	0.252
Y-6	128.2	0.060	0.036	0.096	0.053	0.089	0.161	0.072	0.213
Y-7	109.0	0.052	0.045	0.099	0.048	0.128	0.185	0.053	0.238
Y-9	131.0	0.040	0.039	0.079	0.057	0.095	0.178	0.083	0.252
Y-11	151.9	0.048	0.032	0.080	0.042	0.083	0.129	0.046	0.197
Y-12	106.4	0.049	0.042	0.091	0.040	0.128	0.239	0.061	0.273
Y-14	100.7	0.040	0.040	0.080	0.040	0.132	0.186	0.054	0.240
Y-16	111.1	0.048	0.049	0.097	0.056	0.103	0.183	0.080	0.241

Electrical axis rotation

Pig No.	Deflection of R-wave mm Lead II	Lead III	Degrees rotation
H-1	2.0	3.0	108
H-2	6.5	-1.0	22
H-3	1.5	0	45
H-4	3.0	-5.0	- 6
H-5	4.0	4.5	97
H-6	6.5	-1.0	40
H-7	9.5	3.2	48
H-8	2.0	2.5	98
H-9	3.0	1.0	47
H-10	4.5	4.8	93
H-11	5.0	-2.0	10
H-12	8.5	4.0	54
H-13	1.0	2.0	120
H-14	8.0	4.5	62
H-15	1.5	-2.0	- 1
H-16	1.3	0.5	52
H-17	11.0	7.5	73
H-18	6.5	7.0	96
H-19	4.5	4.0	84

Appendix F. (continued)

Pig No.	Electrical axis rotation: Deflection of R-wave mm Lead II	Lead III	Degrees rotation
H-20	6.0	2.5	55
Y-1	6.0	-1.0	20
Y-2	3.0	-2.0	15
Y-3	0	-10.0	-30
Y-4	-0.8	-0.5	-89
Y-5	-6.5	-10.0	-68
Y-6	0	-6.5	-30
Y-7	6.0	0.5	36
Y-8	5.0	-9.0	- 5
Y-9	-7.5	-3.5	-122
Y-10	-2.5	-2.0	-104
Y-11	3.5	-1.0	17
Y-12	5.0	4.0	76
Y-13	7.0	4.5	67
Y-14	2.5	4.0	114
Y-15	3.8	1.0	45
Y-16	0.5	4.1	137

Normal Hampshires 4, 5, 6, 15 and 17.
Normal Yorkshires 1, 2, 6, 7 and 14.
Low quality Hampshires 3, 8, 9, 10 and 16.
Low quality Yorkshires 5, 9, 11, 12 and 16.

Appendix G. Red, white and intermediate fiber types expressed as percent of total number.

Experiment I

Pig No.	Muscles								
	Rectus femoris			Semimembranosus			Gluteus medius		
	Red	White	Int.	Red	White	Int.	Red	White	Int.
PC 3	51.71	35.80	12.46	52.35	33.10	14.52	47.67	34.46	17.86
PC 8	42.66	37.64	20.97	22.40	57.71	19.86	31.82	49.95	19.39
PC 10	38.64	48.81	12.55	58.30	28.83	12.85	48.14	40.57	11.27
PC 11	60.85	24.92	14.21	50.18	38.48	11.33	23.85	66.58	9.55
PC 13	21.08	63.08	15.81	19.24	58.61	22.12	22.87	55.84	21.26
PC 14	32.25	51.44	16.30	20.72	64.68	14.91	23.11	61.52	15.36
PC 15	31.98	52.00	16.01	32.25	50.57	16.98	36.73	46.24	17.03
PC 16	39.61	40.86	19.52	40.23	47.99	11.77	27.24	60.53	12.21
PC 17	38.11	46.94	14.93	35.48	48.44	15.40	23.58	64.88	11.52
PC 19	37.13	45.70	17.14	42.74	39.01	16.68	37.86	44.78	17.66
LR 2	37.86	48.27	13.96	42.70	40.41	16.87	39.14	43.34	17.52
LR 8	31.35	52.10	16.53	37.03	47.61	15.34	35.26	48.57	16.15
LR 10	54.02	31.25	18.38	24.09	59.67	16.32	33.28	53.36	13.34
LR 12	47.58	31.07	21.33	37.74	44.72	17.52	35.95	51.24	12.79
LR 13	33.71	56.78	9.48	34.79	54.15	11.04	36.09	55.72	8.18
LR 1	58.63	19.87	21.47	44.49	42.03	13.46	14.53	78.28	7.13
LR 3	45.62	34.54	19.83	28.01	62.67	9.33	29.11	63.19	7.60
LR 5	40.68	54.22	5.00	32.45	63.75	3.78	30.00	62.82	7.15
LR 6	50.62	26.80	22.56	30.46	55.36	14.43	30.50	55.66	13.81
LR 11	63.81	18.10	18.06	38.75	44.68	16.23	42.58	43.31	14.09
CW 1	37.58	39.16	23.23	35.28	46.32	18.38	34.08	49.82	16.09
CW 2	52.90	33.57	14.78	55.49	34.00	10.48	43.15	45.88	10.94
CW 4	43.45	30.09	25.42	37.30	48.79	13.90	34.26	53.40	12.33
CW 5	31.91	50.39	17.69	29.60	55.76	12.63	23.22	68.34	8.33
CW 6	40.45	48.29	11.24	44.97	43.94	11.07	36.08	53.69	10.21

Normal: PC 3, 8, 10, 11 and 13; LR 2, 8, 10, 12 and 13; CW 1, 2, 4, 5 and 6.
Low quality: PC 14, 15, 16, 17 and 19; LR 1, 3, 5, 6 and 11.

Appendix G. (continued)

Experiment II

	Number percent									Area percent								
	Muscles									Muscles								
	Rectus femoris			Semimembranosus			Gluteus medius			Rectus femoris			Semimembranosus			Gluteus medius		
Pig No.	Red	White	Int.	Red	White	Int.	Red	White	Int.	Red	White	Int.	Red	White	Int.	Red	White	Int.
H-3	-	-	100.0	-	-	100.0	X	X	X	-	-	100.0	-	-	100.0	X	X	X
H-4	-	-	100.0	56.3	31.7	12.1	13.2	57.9	29.0	-	-	100.0	53.9	32.8	13.4	12.6	53.4	34.0
H-5	14.3	17.1	68.6	11.0	65.4	23.6	12.5	54.2	33.4	8.6	20.0	71.4	8.6	69.9	22.1	10.6	51.9	38.6
H-6	24.6	33.4	42.0	16.5	57.4	26.2	16.4	36.5	47.2	14.5	39.7	45.8	12.7	59.8	28.6	13.0	42.3	44.7
H-8	16.4	23.6	60.0	15.2	55.1	29.7	X	X	X	13.2	25.3	61.5	13.4	54.3	32.4	X	X	X
H-9	11.8	39.2	49.0	17.7	46.6	35.8	19.0	43.0	38.0	9.5	51.5	39.0	11.2	32.3	56.6	17.2	44.0	38.8
H-10	12.3	59.6	28.2	-	47.7	52.4	17.5	52.6	29.8	10.5	59.2	30.4	-	47.6	52.5	11.3	58.6	30.1
H-15	25.4	48.1	26.6	16.3	40.5	43.3	25.2	52.9	22.0	20.8	51.3	28.0	8.8	53.6	37.7	18.5	51.9	29.6
H-16	18.4	57.1	24.5	27.5	39.4	33.1	27.0	50.4	22.6	15.1	60.5	24.5	22.2	39.1	38.7	25.9	48.6	25.5
H-17	16.4	29.7	53.9	24.3	44.1	31.7	13.4	62.8	23.9	11.1	32.6	56.3	21.4	39.9	38.7	10.2	64.1	25.8
Y-1	17.3	20.5	62.2	12.9	34.1	53.1	38.6	0.6	60.9	14.7	27.3	58.1	9.8	41.8	48.4	35.6	0.9	64.0
Y-2	6.4	66.4	27.3	21.6	41.9	36.6	-	70.5	29.6	4.3	71.9	23.8	19.0	44.9	36.3	-	64.8	35.2
Y-5	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
Y-6	0.4	49.4	49.2	X	X	X	-	45.7	54.4	0.6	52.5	46.9	X	X	X	-	43.9	56.1
Y-7	-	43.2	56.8	X	X	X	X	X	X	-	50.2	49.8	X	X	X	X	X	X
Y-9	-	47.0	53.0	7.3	34.2	56.5	1.3	46.8	51.9	-	60.8	39.2	6.9	36.0	57.1	1.6	49.9	48.5
Y-11	1.3	30.4	68.4	20.9	42.5	36.7	0.7	59.2	40.2	1.6	41.4	57.0	14.0	49.7	36.4	1.6	53.6	44.8
Y-12	6.8	56.9	36.4	11.1	33.3	55.6	-	53.6	46.4	8.0	57.7	34.4	11.8	47.8	40.4	-	60.4	39.6
Y-14	24.8	44.3	31.0	24.4	51.6	24.0	23.5	55.1	21.5	16.0	51.0	33.0	17.7	62.0	20.4	18.0	62.3	19.8
Y-16	21.6	51.3	27.7	22.0	50.0	28.0	26.0	55.2	18.8	14.2	60.5	25.4	14.7	60.0	25.3	17.3	64.9	17.9

Counts not made.
Absence of fiber type.
Normal: Hampshire 4, 5, 6, 15 and 17; Yorkshire 1, 2, 6, 7 and 14.
Low quality: Hampshire 3, 8, 9, 10 and 15; Yorkshire 5, 9, 11, 12 and 16.

Appendix H. Capillary and fiber density data.

Pig No.	Muscle: Rectus femoris Cap/fiber	Rectus femoris Cap/in.^{2}	Rectus femoris Fibers/in.^{2}	Semimembranosus Cap/fiber	Semimembranosus Cap/in.^{2}	Semimembranosus Fibers/in.^{2}	Gluteus medius Cap/fiber	Gluteus medius Cap/in.^{2}	Gluteus medius Fibers/in.^{2}
Experiment I									
PC-3	0.65	1.25	1.91	0.72	1.74	2.46	0.66	1.50	2.40
PC-8	0.63	0.84	1.65	0.90	0.78	0.86	0.58	0.71	1.24
PC-10	1.22	4.03	3.31	1.00	1.57	1.62	0.70	1.56	2.24
PC-11	1.06	2.29	2.21	1.01	1.37	1.36	0.90	1.39	1.54
PC-13	0.80	1.17	1.46	0.81	1.27	1.52	0.55	0.87	1.59
PC-14	0.92	1.51	1.65	0.99	0.94	0.96	0.36	0.55	1.51
PC-15	1.02	1.26	1.25	0.85	0.85	0.98	0.33	0.42	1.20
PC-16	1.43	1.33	0.96	0.80	1.01	1.27	0.70	0.68	0.94
PC-17	0.63	0.80	1.30	0.64	0.67	1.04	0.91	0.93	1.02
PC-19	1.00	1.23	1.29	0.82	0.87	1.07	0.56	0.64	1.18
LR-1	0.89	1.27	1.43	1.28	1.55	1.21	0.62	1.70	1.03
LR-2	0.94	1.52	1.62	1.25	1.90	1.50	1.55	1.97	1.32
LR-3	2.08	2.51	1.21	0.63	0.56	0.93	0.95	1.10	1.18
LR-5	0.99	0.98	0.99	0.97	0.93	0.95	0.87	0.68	0.78
LR-6	1.32	1.08	0.82	0.91	0.89	0.80	0.71	1.07	0.72
LR-8	1.11	1.79	1.62	0.89	0.50	0.55	0.84	1.43	1.60
LR-10	1.28	2.28	1.79	1.15	1.95	1.69	1.06	2.48	2.32
LR-11	0.77	1.50	2.03	0.88	1.23	1.42	1.05	1.13	1.08
LR-12	0.61	1.76	2.88	0.99	1.77	1.81	0.73	1.79	2.45
LR-13	0.99	2.20	2.34	1.07	1.73	1.62	1.02	1.95	1.94
CW-1	1.71	3.50	2.05	0.74	1.42	1.92	0.52	1.16	2.33
CW-2	0.77	2.68	3.45	0.75	1.44	1.94	0.63	1.49	2.41
CW-4	0.81	1.81	2.23	0.73	0.99	1.36	0.69	1.13	1.64
CW-5	0.43	0.74	1.73	0.71	1.82	2.53	0.41	0.93	2.11
CW-6	0.35	1.35	2.88	0.63	1.47	2.34	0.54	1.00	1.87
Experiment II									
H-3	1.38	1.57	1.12	1.35	1.28	0.95	--	--	--
H-4	0.99	0.84	0.88	0.81	1.24	1.59	1.68	1.71	0.98
H-5	1.17	1.10	0.84	1.29	0.84	0.64	0.98	0.68	0.70
H-6	0.91	1.54	1.16	1.61	0.69	0.68	0.98	0.47	0.48
H-8	1.24	1.59	1.29	1.02	0.84	0.82	--	--	--
H-9	1.03	1.81	1.74	1.05	1.00	0.96	1.18	0.77	0.65
H-10	1.03	1.07	1.03	1.16	0.31	0.27	1.21	1.25	1.03
H-15	1.07	1.77	1.57	1.36	1.30	1.00	1.23	1.41	1.07
H-16	1.23	1.56	1.28	1.21	1.37	1.15	1.11	1.20	1.09
H-17	1.44	0.85	0.53	1.69	0.95	0.56	1.39	0.78	0.57
Y-1	1.11	1.49	1.35	1.32	1.18	0.89	1.23	0.95	0.77
Y-2	0.60	0.55	0.91	1.30	1.88	1.46	0.65	0.65	1.00
Y-5	--	--	--	--	--	--	--	--	--
Y-6	0.85	1.09	1.27	--	--	--	1.79	1.37	0.77
Y-7	0.78	1.12	1.42	--	--	--	--	--	--
Y-9	0.92	0.53	0.58	1.30	0.66	0.51	1.62	0.53	0.33
Y-11	0.94	0.66	0.71	1.40	0.52	0.38	0.84	0.65	0.80
Y-12	0.83	0.48	0.56	1.07	0.48	0.44	1.07	0.50	0.47
Y-14	1.16	1.62	1.22	1.15	2.07	1.80	1.12	1.98	1.77
Y-16	0.93	1.75	1.89	1.04	1.54	1.48	1.11	1.34	1.22

Appendix H. (continued)

Pig No.	Muscle								
	Rectus femoris			Semimembranosus			Gluteus medius		
	Red	White	Int.	Red	White	Int.	Red	White	Int.
Capillaries associated with each fiber type (Experiment II)									
H-3	-	-	2.05	-	-	2.56	-	-	-
H-4	-	-	1.68	3.07	0.60	1.57	6.04	2.15	5.07
H-5	2.80	1.25	1.90	5.27	1.88	3.72	3.34	1.58	1.58
H-6	3.76	1.49	2.31	3.58	1.34	2.61	3.43	1.75	2.00
H-8	3.44	0.95	2.30	3.85	1.21	2.43	-	-	-
H-9	4.25	1.35	2.58	3.14	1.04	2.69	3.60	1.44	2.03
H-10	4.24	1.62	2.58	-	1.40	2.16	4.80	1.30	2.88
H-15	3.91	1.07	2.69	3.72	1.89	3.53	4.55	1.40	3.47
H-16	4.70	1.32	2.75	3.81	1.55	2.40	3.89	1.40	2.41
H-17	4.62	1.99	3.62	5.35	2.37	3.77	4.99	1.63	4.29
Y-1	3.79	1.52	2.53	4.33	1.44	2.63	2.97	1.00	1.72
Y-2	3.71	0.63	1.80	4.37	1.24	3.34	-	0.71	2.55
Y-5	X	X	X	X	X	X	X	X	X
Y-6	2.00	0.99	2.25	X	X	X	-	1.72	4.64
Y-7	-	1.00	2.04	X	X	X	X	X	X
Y-9	-	1.47	2.08	3.13	1.43	2.48	4.00	1.65	3.44
Y-11	3.50	1.57	2.07	3.60	1.72	2.21	3.00	1.00	2.21
Y-12	3.34	1.43	2.21	5.00	1.50	1.97	-	1.63	2.08
Y-14	3.34	1.65	2.87	4.49	1.84	2.80	4.46	1.57	3.15
Y-16	4.46	1.37	2.55	3.96	1.47	3.26	3.40	1.41	3.21

X No count made.
- Absence of fiber type.

Appendix I. Myoglobin data.[a]

Pig No.	Muscle: Rectus femoris Total myoglobin	MMb	Reduced Mb	O_2Mb	Semimembranosus Total myoglobin	MMb	Reduced Mb	O_2Mb	Gluteus medius Total myoglobin	MMb	Reduced Mb	O_2Mb
PC-3	113.5	52.2	29.5	31.8	42.2	16.0	--	26.2	40.4	20.6	2.8	17.0
PC-8	140.1	78.5	50.5	11.2	29.6	8.0	7.4	14.2	55.1	18.1	16.2	20.8
PC-10	68.2	17.7	8.2	42.3	63.5	18.4	11.4	33.7	70.1	23.8	40.7	5.6
PC-11	54.2	8.1	--	46.1	56.8	5.1	0	51.7	64.4	19.3	19.3	25.8
PC-13	174.4	75.0	90.7	8.7	152.2	70.0	73.1	9.1	45.3	8.6	1.8	34.9
PC-14	121.2	50.9	52.1	18.2	96.0	40.3	48.0	7.7	76.1	40.3	33.5	2.3
PC-15	85.9	37.8	29.2	18.9	86.6	32.0	31.2	23.4	80.0	33.6	38.4	8.0
PC-16	188.7	83.0	88.7	17.0	78.2	38.3	29.7	10.2	66.4	25.9	31.2	9.3
PC-17	290.6	136.6	154.0	--	47.6	14.3	5.7	27.6	145.8	70.0	75.8	--
PC-19	289.8	136.2	150.7	2.9	113.4	51.0	55.6	6.8	171.4	80.6	85.7	5.1
LR-1	107.8	35.6	30.2	42.0	64.8	14.3	5.8	44.7	61.1	15.3	15.3	30.5
LR-2	165.2	57.8	56.2	51.2	178.7	76.8	82.2	19.7	145.8	59.8	59.8	26.2
LR-3	96.4	21.2	5.8	69.4	83.3	19.2	10.8	53.3	64.5	14.2	9.7	40.6
LR-5	173.7	67.7	60.8	45.2	156.9	64.3	54.9	37.7	177.6	72.8	76.4	28.4
LR-6	148.3	59.3	35.6	53.4	70.0	24.5	16.8	28.7	59.4	18.4	10.7	30.3
LR-8	103.7	31.1	6.2	66.4	116.0	45.2	34.8	36.0	101.7	38.6	47.8	15.3
LR-10	398.7	191.4	203.3	4.0	139.5	62.8	58.6	18.1	170.4	81.8	81.8	6.8
LR-11	170.3	68.1	61.3	40.9	166.1	73.1	74.7	18.3	203.9	95.8	89.7	18.4
LR-12	222.9	93.6	109.2	20.1	137.8	57.9	56.5	23.4	175.6	86.0	49.2	40.4
LR-13	179.8	73.7	84.5	21.6	146.1	64.3	78.9	2.9	151.2	66.5	74.1	10.6
CW-1	318.6	137.0	165.7	15.9	100.6	42.3	40.2	18.1	137.8	62.0	51.0	24.8
CW-2	167.8	68.8	65.4	33.6	106.4	40.4	30.9	35.1	144.4	66.4	69.3	8.7
CW-4	308.8	132.8	151.3	24.7	235.6	101.3	117.8	16.5	211.8	101.7	110.1	--
CW-5	201.5	110.8	76.6	14.1	166.0	66.4	71.4	28.2	140.8	66.2	60.5	14.1
CW-6	305.8	137.6	168.2	--	150.0	63.0	64.5	22.5	127.5	57.4	59.9	10.2

Normal: PC 3, 8, 10, 11 and 13; LR 2, 8, 10, 12 and 13; CW 1, 2, 4, 5 and 6.
Low quality: PC 14, 15, 16, 17 and 18; LR 1, 3, 5, 6 and 11.
[a]Data are expressed as μM/g of fresh tissue.

Appendix J. Total imidazole, carnosine and anserine data.[a]

	Muscle								
	Rectus femoris			Semimembranosus			Gluteus medius		
Pig No.	Total imidazole	Carnosine	Anserine	Total imidazole	Carnosine	Anserine	Total imidazole	Carnosine	Anserine
PC-3	6.25	5.00	1.25	8.19	6.53	1.66	7.57	5.83	1.74
PC-8	7.98	5.83	2.15	9.51	5.35	4.16	7.64	6.25	1.39
PC-10	4.86	2.22	2.64	7.36	3.68	3.68	7.15	5.62	1.53
PC-11	4.86	3.33	1.53	10.27	5.28	4.99	6.80	5.76	1.04
PC-13	9.37	3.12	6.25	7.36	4.79	2.57	5.76	5.62	0.14
PC-14	7.76	6.04	1.72	10.82	6.80	4.02	8.89	7.50	1.39
PC-15	7.15	3.89	3.26	10.27	6.80	3.47	7.43	5.55	1.88
PC-16	6.80	4.01	2.79	7.63	4.51	3.12	7.15	5.35	1.80
PC-17	7.01	6.53	0.48	8.19	8.19	0.00	8.33	8.19	0.15
PC-19	6.46	5.76	0.70	8.33	4.79	3.54	7.29	5.14	2.15
LR-1	10.20	3.54	6.66	6.11	6.11	0.00	8.33	7.98	0.35
LR-2	9.30	3.47	5.83	8.54	7.91	0.63	7.15	7.15	0.00
LR-3	7.15	2.29	4.86	9.44	7.22	2.22	9.37	8.46	0.91
LR-5	7.84	3.89	3.95	7.57	5.07	2.50	5.90	5.41	0.49
LR-6	8.19	5.14	3.05	9.72	6.46	3.26	8.75	7.91	0.84
LR-8	6.24	3.68	2.56	6.66	4.23	2.43	6.25	5.83	0.42
LR-10	10.10	4.93	5.17	10.69	8.19	2.50	7.91	6.46	1.45
LR-11	10.69	2.99	7.70	9.72	8.12	1.60	6.25	5.69	0.56
LR-12	11.25	1.80	9.45	10.20	7.64	2.56	7.98	7.08	0.90
LR-13	10.97	3.19	7.78	13.88	8.89	4.99	8.26	5.97	2.29
CW-1	6.46	4.30	2.16	8.33	5.21	3.12	7.98	5.00	2.98
CW-2	7.36	3.47	3.89	7.64	6.46	1.18	7.29	5.35	1.94
CW-4	6.18	4.72	1.46	8.05	5.35	2.70	7.64	5.41	2.23
CW-5	4.93	4.72	0.21	7.98	5.69	2.29	7.64	4.72	2.92
CW-6	8.12	5.00	3.12	7.98	5.55	2.43	8.33	5.35	2.98

[a]Data are expressed as μM/g of fresh tissue.

Appendix J. (continued)

Pig No.	Muscle: Rectus femoris — Total imidazole	Rectus femoris — Carnosine	Rectus femoris — Anserine	Semimembranosus — Total imidazole	Semimembranosus — Carnosine	Semimembranosus — Anserine	Gluteus medius — Total imidazole	Gluteus medius — Carnosine	Gluteus medius — Anserine
H-3	7.36	6.18	1.18	7.84	7.76	0.08	8.82	8.68	0.14
H-4	6.94	6.18	0.76	8.47	8.33	0.14	10.48	9.58	0.90
H-5	9.02	8.33	0.69	8.33	8.26	0.07	9.37	8.82	0.55
H-6	7.43	7.01	0.42	8.54	8.19	0.35	8.96	8.89	0.07
H-8	9.86	6.39	3.47	8.47	8.40	0.07	9.44	8.61	0.83
H-9	9.37	8.54	0.83	8.19	8.12	0.07	9.72	8.25	0.97
H-10	10.07	7.08	2.99	8.33	8.33	0.00	9.02	8.33	0.69
H-15	7.64	5.55	2.09	8.33	7.64	0.69	8.86	7.76	1.10
H-16	7.50	6.04	1.46	9.02	8.19	0.83	10.34	8.89	1.45
H-17	8.33	7.70	0.63	8.75	8.75	0.00	9.65	9.02	0.63
Y-1	8.54	6.73	1.81	7.64	7.01	0.63	8.19	7.08	1.11
Y-2	8.33	6.18	2.15	9.86	9.51	0.35	9.02	8.68	0.34
Y-5	4.86	4.51	0.35	6.25	5.41	0.84	6.32	5.41	0.91
Y-6	7.64	5.41	2.23	9.51	8.68	0.83	8.89	8.33	0.56
Y-7	7.08	6.94	0.14	7.84	7.84	0.00	8.19	7.29	0.90
Y-9	6.39	5.14	1.25	6.11	6.04	0.07	7.71	7.29	0.42
Y-11	6.11	6.11	0.00	7.22	6.66	0.56	7.29	7.22	0.07
Y-12	7.22	6.39	0.83	6.80	6.39	0.41	7.76	7.15	0.61
Y-14	7.98	6.32	1.66	6.59	6.46	0.13	7.91	6.94	0.97
Y-16	7.64	6.39	1.25	7.78	7.50	0.28	8.47	8.26	0.21

[a]Data are expressed as μM/g of fresh tissue.

Appendix K. Observation of muscle degeneration (Experiment II).

	Muscle degeneration scores		
	Muscle		
Pig No.	Rectus femoris	Semimembranosus	Gluteus medius
H-3	4	5	4
H-4	X	5	5
H-5	4	3	3
H-6	4	3	X
H-8	4	3	4+
H-9	5	2	2+
H-10	4	4	2+
H-15	5	5	5
H-16	5-	4	3
H-17	5	5-	5-
Y-1	4	5	5
Y-2	4	5	4
Y-5	4	4	4
Y-6	5-	4	5
Y-7	5-	5-	4
Y-9	4	3	3
Y-11	3	2+	2
Y-12	3	2	2
Y-14	5-	5	4
Y-16	5-	5-	5-

Normal: Hampshire 4, 5, 6, 15 and 17; Yorkshire 1, 2, 6, 7 and 14.
Low quality: Hampshire 3, 8, 9, 10 and 16; Yorkshire 5, 9, 11, 12 and 16.
X not examined.