THE RELATIONSHIP OF SOME BIOCHEMICAL AND PHYSIOLOGICAL FACTORS TO POSTMORTEM CHANGES IN PORCINE MUSCLE

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ABSTRACT

THE RELATIONSHIP OF SOME BIOCHEMICAL AND PHYSIOLOGICAL FACTORS TO POSTMORTEM CHANGES IN PORCINE MUSCLE

by Duane Elmer Koch

The results of this study were obtained from 146 market-weight pigs slaughtered in four groups. The distribution of red, white and intermediate muscle fibers, succinic dehydrogenase (SDH) activity, myoglobin, total lipid, phospholipid and glyceride ester (neutral lipid fraction) levels were determined on longissimus (LD) muscle samples obtained from the pigs in Group I at or shortly after exsanguination. The relationship of these parameters to 45 min postmortem pH values and to 24 hr postmortem subjective quality scores was observed on normal (slow-glycolyzing) and low quality (fast-glycolyzing) LD muscles from the three breeds (Chester White, Landrace, Poland China) of pigs included in Group I. Heart weights of the pigs in Groups I, III and IV were recorded and subsequently related to rates of postmortem pH decline and/or 24 hr transmission values of the LD muscle. Muscle (LD) or rectal temperatures were obtained on the pigs in Groups II, III and IV at the time of exsanguination and at 45 min postmortem and their relationship to 45 min pH, transmission values and subjective quality scores was observed. The effects of sample excision, at or shortly after exsanguination, upon postmortem pH, transmission values, glycogen, glucose-6-phosphate, lactic acid, ATP and creatine phosphate (CP) levels from the LD and rectus femoris (RF) muscles of the pigs included in Group IV were studied. In addition, glucose-1-phosphate, fructose-6-phosphate, glucose, ADP and AMP levels were compared among normal and low quality LD and RF muscles (Group IV) that had been incised at several

postmortem time periods. Transmission values and the 2 hr postmortem pH of the LD, RF, biceps femoris (BF) and supraspinatus (SS) muscles from the pigs in Group IV also were compared.

Normal LD muscles had more red and fewer white muscle fibers, higher SDH activities and greater total myoglobin contents than low quality LD muscles. The size of red and intermediate muscle fibers was larger among low quality than normal LD muscles. Normal LD muscles tended to have higher total lipid levels and greater neutral lipid ester contents than those of the low quality muscles. Landrace pigs tended to have more myoglobin and higher SDH activities than Poland Chinas or Chester Whites, while Poland China pigs tended to have more red and fewer white muscle fibers than the other two breeds.

Heart weights of the low quality pigs in Group I tended to be lighter than those from normal pigs. However, low and nonsignificant correlations were obtained between heart weights and either 45 min pH (Groups I and IV) or transmission values (Groups III and IV). From observations of several pigs with pericarditis, it appeared that heart function may be more important than heart weight as a contributory factor in the development of low quality porcine musculature.

Muscle (LD) temperatures at 45 min postmortem were found to be more highly correlated (negatively) with ultimate quality indices than muscle (Groups III and IV) or rectal (Group II) temperatures at the time of exsanguination. The in vivo temperatures at the time of exsanguination, as well as the effects of scalding, slaughter floor temperatures and time lapse before carcass chilling appeared to influence postmortem muscle temperatures.

Muscle (LD) incision (Group IV), at or shortly after exsanguination, stimulated contractile activity, significantly increased the rate of postmortem glycolysis and tended to decrease ultimate muscle qualitative properties. The LD muscles incised at the time of exsanguination had lower pH values, glycogen, ATP and CP levels and higher lactate contents at corresponding time periods, through 2 hr postmortem, than LD muscles not incised until 45 min after exsanguination. This effect of muscle incision was greater among normal than low quality LD muscles.

Exposure of the RF muscles (Group IV) to the atmosphere during sample excision and the concomitant chilling effects at early postmortem time periods (0 to 15 min) slowed down glycolytic rates and apparently nullified the influence of contractile activity associated with muscle incision. The RF muscles incised at or shortly after exsanguination exhibited significantly higher pH values, levels of glycogen and ATP and lower lactate contents at 2 hr postmortem than RF muscles not incised prior to this time. The effects of chilling associated with early postmortem incision of the RF muscles was greater among low quality than normal muscles. Postmortem levels of all the metabolites studied appeared to be related to the glycolytic rate in both the RF and LD muscles.

Low, but significant correlation coefficients were observed for transmission and 2 hr postmortem pH values of the RF, BF and SS muscles with those of the LD. These data indicate that the postmortem changes occurring in these muscles within a given porcine carcass tended to parallel each other.

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INTRODUCTION

While per capita meat consumption in the United States has been increasing, pork consumption has remained relatively constant during the past two decades and thus it is accounting for less of the total. Lack of consistent product uniformity in cutability (lean to fat and bone ratio) or palatability factors has been implicated in the static pork consumption pattern. However, the contribution of pork palatability factors to product acceptability has been poorly documented to date.

During the last 10 to 15 years, pale, soft and exudative (PSE) pork has received increasingly more attention. The incidence of the "lower quality" pork is quite variable and certain strains of pigs appear to be more predisposed to PSE development than others. Additionally environmental stressors and extremely high ambient temperatures or widely fluctuating daily temperatures seem to aggravate the incidence. Some, and possibly all, muscles of PSE pork carcasses are believed to be less palatable (decreased juiciness and tenderness) when cooked; to have greater shrink during processing; to exhibit more color variability in finished processed products; and to have lower emulsifying capacity due to decreased protein solubility than muscles resulting from "normal" carcasses. Research work to date has not conclusively established the factors responsible for the "normal" conversion of porcine muscle to pork, however, rapid postmortem pH drop while muscle temperature is relatively high ($\geq$ 35°C), results in excessive protein denaturation and this phenomenon appears to be the immediate cause of PSE muscle.

If PSE pork is a problem of economic importance, two approaches to its solution are apparent. The more immediate approach is that of reducing (or possibly eliminating) the incidence or severity of PSE muscle through selection toward resistant breeding stock, careful management during production, proper handling prior to and at the time of slaughter and rapid postmortem chilling of the carcasses. The other approach is to elucidate the ante- and/or postmortem factor(s) responsible for the variation in ultimate qualitative properties resulting during the conversion of porcine muscle to pork.

This study was conducted to provide preliminary information with respect to some of the variables involved in the conversion of porcine muscle to meat by:

1. Observing some aerobic and anaerobic characteristics (red, white and intermediate muscle fiber types, succinic dehydrogenase activity and myoglobin components) as well as lipid classes present in the longissimus muscle at the time of slaughter.
2. Determining what effect heart size and muscle temperature, at the time of slaughter, have on ultimate porcine muscle qualitative properties.
3. Establishing what effect sampling techniques have on postmortem glycolytic rate and ultimate muscle quality.
4. Determining if the qualitative properties of muscles from several different anatomical locations within PSE pork carcasses are similarly affected.

REVIEW OF LITERATURE

Bendall (1964, 1966a), Lawrie (1966a) and McLoughlin (1969) stated that variability in meat properties resulted from differences in post-mortem changes occurring within these muscles. They further stated that these differences are a reflection of the intended function of the particular muscle. Lawrie (1966a) indicated that muscles develop and differentiate for definite physiological purposes in response to various intrinsic and extrinsic stimuli (genetic, physiological and nutritional). Helander (1966) stated that muscle development (changes taking place in constitution, volume, and structure of skeletal muscle) is affected by age, degree of activity, hormonal state, metabolic state and pathological conditions.

The transverse elements of the sarcoplasmic reticulum conduct nerve impulses via Na/K depolarization from the sarcolemma to the triads of the longitudinal tubules (lace like network enveloping fibrils) of the sarcoplasmic reticulum (Copenhaver, 1964; Bendall, 1966a; Lawrie, 1966a). This depolarization wave releases Ca^{++} which in turn diffuse from the longitudinal tubules. The Ca^{++} releases ATP from its inert complex with Mg^{++} and activates myosin ATPase. The subsequent splitting of ATP provides the energy necessary for interaction of actin and myosin filaments resulting in muscle contraction. Immediately the Ca^{++} pump in the longitudinal tubules recaptures the Ca^{++} and contractile ATPase activity ceases. The ATP-Mg complex, which acts as a plasticizer in the resting state, again reforms.

Bendall (1966a) and Lawrie (1966a) reported that ATP is the immediate energy source in contraction. Bendall (1966a) stated that ATP is required to operate the Na/K pump in the sarcolemma and transverse tubules of the sarcoplasmic reticulum. Bendall (1966a) and Marsh (1966) reported that ATP is also required to operate the Ca^{++} pump in the longitudinal tubules of the sarcoplasmic reticulum. Bendall (1963) and Henneman and Olson (1965) observed the presence of a muscle mitochondrial ATPase.

White et al. (1964), Bendall (1966a) and Lawrie (1966a) reported that ATP and its immediate source of replenishment, creatine phosphate, can be maintained in slowly working muscle by oxidative phosphorylation. They further indicated that in rapidly contracting muscle, the oxygen supply becomes insufficient and ATP must be synthesized anaerobically via the glycolytic process.

Red and White Muscle Fibers

Needham (1926) broadly classified muscles as either red or white. Lawrie (1966a) indicated that this superficial differentiation reflected both histological and biochemical differences. The "redness" or "whiteness" of a muscle has been shown to be due to the varying proportion of red and white muscle fibers (Dubowitz and Pearse, 1961; Blanchaer et al., 1963; VanWijhe et al., 1963; Brooke, 1966). These authors reported that red fibers stained intensely when histochemically assayed for enzymes involved in aerobic metabolism; whereas, white fibers showed positive staining reactions to histochemical assays for the enzymes associated with anaerobic metabolism. With the same histochemical techniques, Ogata and Mori (1963), Dawson and Romanul (1964), Romanul (1964) and

Beatty et al. (1966) reported a variable number of fibers with intermediate staining reactions.

George and Berger (1966) reported that red fibers are smaller in diameter, have higher myoglobin levels and greater concentrations of mitochondria and oxidative enzymes than white fibers. On the other hand, white fibers have higher glycolytic enzyme activities, i.e. a greater capacity for anaerobic metabolism. Red fibers display a slow and sustained contractile activity as opposed to the fast but shorter duration contractile response of white fibers. Red fibers usually have higher lipid and lower glycogen contents than white fibers. Dawson and Romanul (1964) and Peachey (1968) indicated that the above classification is general and exceptions exist. George and Berger (1966) and Carrow et al. (1967) reported that red muscle fibers are supplied with a greater number of capillaries than white fibers.

Numerous studies have shown that muscle fiber characteristics are neurally controlled (Buller and Lewis, 1965; Henneman and Olson, 1965; Romanul and Van Der Meulen, 1967; Yellin, 1967; Guth, 1968; Karpati and Engel, 1968). Muscle fiber properties have been shown to be altered as a result of cross-innervating red and white muscles. Cross-innervated muscles reversed their speed of contraction and their enzymatic profiles (Close, 1965; Romanul and Van Der Meulen, 1967; Robbins et al., 1969). Exercise has also been shown to have an effect on muscle fiber type (Carrow et al., 1967; Holloszy, 1967; Edgerton et al., 1968; Peter et al., 1968). Carrow et al. (1967) also showed an increase of intramuscular capillary density with exercise.

While Briskey (1967) stated that a biochemical property of myosin is to catalyze ATP hydrolysis, Seidel and Gergely (1963), Sreter et al. (1966), Seidel et al. (1964) and Barany et al. (1965a) showed that myosin from red muscle has a lower ATPase activity than that from white muscle. Perry and Hartshorne (1963), Barany et al. (1965b) and Trayer and Perry (1966) reported that myosin ATPase activity increased progressively with muscle development and age. Buller et al. (1960) and Close (1964) reported that muscles of prenatal or neonatal mammals were all physiologically red. Thus Briskey (1967) indicated that the increase in myosin ATPase activity with postnatal development probably reflected the differentiation of red fibers to white fibers. Furthermore, Barany et al. (1965b) reported a direct relationship between speed of muscle contraction and myosin ATPase activity.

Postmortem Mammalian Muscle Changes

Lawrie (1966a) reported that the immediate result of blood removal at the time of slaughter is the depletion of oxygen supply and loss of all neural and hormonal control over metabolic processes. He further indicated that even though the muscle may not be actively contracting at this time, energy was required for maintenance of homeostasis. The latter author also stated that the rate and extent to which ATP becomes depleted readily affects the rate and extent of postmortem change, i.e. rigor mortis and glycolysis.

It is generally believed that all postmortem ATP breakdown is accomplished by sarcoplasmic ATPase activity (Bendall, 1960; Lawrie, 1966a). However, in certain instances, especially when rate of postmortem change is greatly accelerated and ATP is depleted at an abnormally high rate,

e.g. thaw rigor, cold shortening and rapid rigor mortis development as observed in some "degenerated" muscle, the myofibrillar ATPase system also becomes operative (Bendall, 1960; Marsh, 1966; Newbold, 1966).

Since exsanguination has inhibited most or all respiration, ATP must be replenished by anaerobic processes (Lawrie, 1966a). Bendall (1960), Newbold (1966) and Lawrie (1966a) reported that ATP is preferentially synthesized by transfer of high energy phosphate from creatine phosphate to ADP. They stated that when the supply of creatine phosphate becomes limited, anaerobic glycolysis becomes operative in order to maintain ATP levels. The rate and extent of glycolysis is dependent upon glycogen availability as well as the ATP and creatine phosphate levels at the time of exsanguination. When ATP is diminished more rapidly than resynthesized by the above reactions, myokinase catalyzes conversion of two moles of ADP to one mole each of ATP and AMP. Measurement of postmortem pH is frequently used as an indirect estimate of the extent of glycolysis.

Rigor mortis onset is dependent upon ATP disappearance from the muscle (Bendall, 1960; Lawrie, 1966a; Newbold, 1966; Davies, 1967). These same authors indicated that rigor mortis development is characterized by the loss of muscle extensibility caused by the transformation of the freely sliding actin and myosin filaments to the rigid actomyosin complex. Cassens (1966) and Davies (1967) stated that shortening may or may not occur during rigor. Bendall (1960), Lawrie (1966a) and Newbold (1966) reported that ATP complexed with Mg^{++} acts as a plasticizer for actin and myosin. Davies (1967) reported that ADP has a similar effect. Bendall (1960) observed that at lower pH values, lower ATP levels are needed before onset of rigor commences. Lawrie (1966a) reported that the rate and extent of postmortem changes are a reflection of muscle function

which in turn are influenced by species, breed, sex, age, anatomical location of the muscle, exercise and plane of nutrition. Lawrie (1966a, 1966b) also reported that metabolic stresses, e.g., activity, ambient temperature, humidity, atmospheric pressure, oxygen tension, feed, injury, pathological and psychological factors during or just preceding slaughter exert an influence on postmortem change. The rate at which postmortem changes occur increases with increasing temperature, especially in the range of 5° to 43°C (Bendall, 1960; Lawrie, 1966a; Newbold, 1966).

Bendall (1964), Briskey (1964), Lawrie (1966a), and McLoughlin (1969) reported that the rate and extent of postmortem changes affect the use of muscle as a food. A rapid glycolytic rate and an abnormally low ultimate pH usually result in a paler muscle color than that normally encountered (Cassens, 1966; Lawrie, 1966a); whereas, an exceptionally high ultimate pH gives rise to an unusually dark color (Lawrie, 1966a).

Water binding capacity is minimal at the isoelectric point which ranges from pH 5.1 to 5.5 for muscle proteins (Hamm, 1960). Bendall (1964) and Lawrie (1966a) reported that this point is at or near the normal ultimate pH of most muscles. Lawrie (1966a) found that even at high ultimate pH values there was a diminution of water binding capacity attributable to ATP disappearance and the consequential actomyosin formation. Lawrie (1966a), Bendall (1964) and Briskey (1964) indicated greater than normal loss of water holding capacity due to excessively low ultimate pH values or to very rapid postmortem pH declines, especially when these conditions were achieved at or above muscle temperatures of 35°C. They attributed this loss of water holding capacity to muscle protein denaturation, especially inplicating the sarcoplasmic fraction, and to loss of semipermeability of the sarcolemma.

Control of Glycolysis

Since rate of postmortem glycolysis probably plays a major role in ultimate meat quality, a brief discussion of glycolytic control will be presented. White et al. (1964) and Conn and Stumpf (1966) indicated that the rate of each enzyme-catalyzed reaction is related to the concentration of active enzyme; the availability of appropriate substrates, coenzymes and cofactors; the presence of activators or inhibitors; and the temperature and pH conditions. Scrutton and Utter (1968) stated that availability of substrate or regulation of catalytic activities of rate limiting enzymes, or both, are probably the major factors contributing to regulation of glycolytic flux.

Under normal conditions, two enzymes, i.e., phosphorylase and to a greater extent phosphofructokinase are almost always implicated as "the" rate limiting reactions of glycolysis (Lowry et al., 1964; Regen et al., 1964; Helmreich and Cori, 1965; Uyeda and Racker, 1965; Williamson, 1965; Wood, 1966; Scrutton and Utter, 1968). Randle (1964) indicated that glycogen degradation was controlled by phosphorylase; whereas, subsequent steps in the glycolytic pathway were controlled by the phosphofructokinase reaction. Wood (1966) reported that stimulation of phosphorylase activity did not necessarily increase phosphofructokinase reaction rates. Karpatkin et al. (1964) and Ozand and Narahara (1964) found that increased reaction rates of phosphorylase stimulated by epinephrine did not necessarily activate phosphofructokinase; whereas, muscle contraction elicited by electrical stimulation increased the rate of both reactions.

Atkinson (1966) and Wood (1966) reported that phosphorylase b can be converted to the active phosphorylase a by phosphorylase kinase in the presence of cyclic 3', 5'-AMP, Mg^{++}, ATP, and Ca^{++}. Atkinson (1966) and Scrutton and Utter (1968) noted that conversion of phosphorylase b to the a form was not absolutely necessary for muscle contraction or increased glycolytic rate. Wood (1966) reported that phosphorylase b is activated by AMP. Atkinson (1966) confirmed this observation and added that ATP and glucose-6-phosphate inhibited the conversion of phosphorylase b to the a form.

Phosphofructokinase is inhibited by ATP and citrate (Wood, 1966; and Scrutton and Utter, 1968); whereas, activation of phosphofructokinase is accomplished by AMP, ADP, Pi, fructose-6-phosphate and fructose-1,6-diphosphate (Mansour, 1965; Wood, 1966; and Scrutton and Utter, 1968).

Randle (1964) observed that anoxia and inhibition of oxidative phosphorylation increased glycolysis by increasing both phosphorylase and phosphofructokinase activity. He further indicated that facilitation of oxidative phosphorylation decreased glycolytic rate. In support of the above work, Lowry *et al.* (1964), Ramaiah *et al.* (1964) and Atkinson (1966) observed that an increased ATP to ADP, AMP, Pi ratio decreased glycolysis, while a decrease in this ratio increased glycolysis.

Postmortem Changes in Porcine Muscle

In the homeostatic state as well as immediately postmortem all porcine muscles are moderately dark in color, firm in texture and dry in appearance (Briskey, 1963, 1964; McLoughlin and Goldspink, 1963a). Anaerobic conditions develop rapidly following exsanguination and the rate and

extent of the resultant biochemical and physiological changes are largely responsible for the variation in ultimate meat characteristics (Briskey, 1963, 1964; Briskey et al., 1966). Forrest et al. (1963) noted that the ultimate gross morphology of porcine muscle ranged from excessively dark, firm and dry musculature to extremely pale, soft and exudative musculature. It has been shown that the pH pattern (Briskey, 1963, 1964) and/or the pH and temperature relationships in the muscle at the onset of rigor mortis are highly associated with the ultimate muscle classification (Sayre and Briskey, 1963; Briskey, 1964).

Briskey (1963, 1964) described the different pH patterns which are apparent in postmortem porcine muscle. If little postmortem glycolytic activity occurs and rigor mortis develops at high pH values or if glycolysis is extremely slow and rigor mortis occurs over a long time period, the muscle remains dark red in color, firm in texture and dry in appearance (Briskey et al., 1959a, b, c, 1962; Sayre and Briskey, 1963; Kastenschmidt et al., 1964). The ultimate pH of these muscles remained at or near 6.0 to 6.5. When postmortem glycolysis and rigor mortis occurs under normal conditions, i.e. at a moderate rate (4 to 6 hr for completion of rigor mortis and 6 to 12 hr to achieve an ultimate pH of 5.3 to 5.6), the muscles exhibit a grayish pink to red color (normal) and are moderately firm in structure and dry in appearance (Wismer-Pedersen and Briskey, 1961a; Briskey and Wismer-Pedersen, 1961a; Briskey et al., 1962; Sayre and Briskey, 1963). Fast onset of rigor mortis and an extremely rapid rate of glycolysis with the development of low pH values (< 5.6), at temperatures above 35°C, are associated with production of pale, soft

and exudative (PSE) muscles (Briskey et al., 1959b; Briskey and Wismer-Pedersen, 1961a; Briskey et al., 1962; Bendall and Wismer-Pedersen, 1962; Bendall et al., 1963; Briskey, 1963; Sayre and Briskey, 1963; Kastenschmidt et al., 1964).

Wismer-Pedersen (1959) and Briskey and Wismer-Pedersen (1961a, b) observed the development of PSE musculature with normal pH patterns. Likewise, Sayre et al. (1964) reported dark, soft and exudative porcine muscles resulting from rapid postmortem glycolytic rates.

Briskey (1963) and Sayre et al. (1963b) also noted a loss of normal intermuscular binding as a result of the rapid glycolytic rate. Bendall and Wismer-Pedersen (1962) and Cassens et al. (1963a, b) noted no histological abnormalities at the time of death for muscles which ultimately become PSE. Abnormally low ultimate pH values (Lawrie et al., 1958) and conditions characteristic of PSE musculature, such as "muscle degeneration" (Ludvigsen, 1953), "la myopathie exudative depigmentaire du porc" (Henry et al., 1955) and "white muscle disease" (Lawrie, 1960) have been reported from some European countries.

Rapid postmortem glycolytic rates are associated with rapid decreases of ATP and creatine phosphate (Briskey and Wismer-Pedersen, 1961a; Bendall and Wismer-Pedersen, 1962; Bendall et al., 1963; Briskey and Lister, 1968). Bendall et al. (1963) reported that rigor mortis onset occurs when ATP is at 30% of the initial level. Kastenschmidt et al. (1966, 1968) reported that muscles exhibiting fast glycolytic rates had lower ATP and creatine phosphate levels at the time of exsanguination than muscles which underwent slow glycolysis. They indicated that the fast

glycolyzing muscles may be in a highly anaerobic state at the time of exsanguination. Briskey and Lister (1968) stated that lactic acid content at the time of exsanguination was directly related to the rate of postmortem lactic acid accumulation. Although low ATP levels are associated with rapid glycolysis, it is not known whether low postmortem ATP levels result from rapid hydrolysis or inefficient resynthesis (Kastenschmidt et al., 1968).

Briskey (1964) and Cassens (1966) reported that a review of the literature showed no consistent histological observations which were associated with postmortem muscle properties. They stated that some of the changes observed may have resulted from different states of fiber contracture rather than from varying rates of postmortem pH decline. Nevertheless, Cassens et al. (1963a, b) noted greater sarcoplasmic disruption and decreased myofibrillar preservation as a result of rapid postmortem changes.

Sayre and Briskey (1963) and McLoughlin (1968) reported no differences in sarcoplasmic and myofibrillar protein extractabilities at the time of death, which were attributable to subsequent rates of postmortem reactions. Postmortem extractability of sarcoplasmic proteins was inversely related to rate of postmortem changes, especially glycolysis (Wismer-Pedersen and Briskey, 1961a; Bendall and Wismer-Pedersen, 1962; McLoughlin and Goldspink, 1963a; Scopes and Lawrie, 1963; McLoughlin, 1963, 1968; Sayre and Briskey, 1963; Briskey and Sayre, 1964; Topel et al., 1967). The decreased extractability was attributed to denaturation.

Decreased myofibrillar and sarcoplasmic protein extractability occurs when pH decline is rapid while postmortem muscle temperature is still high

[> 35°C] (Sayre and Briskey, 1963; McLoughlin and Goldspink,1963a, b; Briskey and Sayre, 1964; Bendall, 1964). Bendall and Wismer-Pedersen (1962) attributed the differences in myofibrillar protein extractability resulting from rapid postmortem pH fall to denatured sarcoplasmic protein precipitating onto the myofibrils rather than to myofibrillar protein alterations per se. However, Hart (1962a), Sayre and Briskey (1963) and McLoughlin (1963) noted a decrease in myofibrillar protein solubility above that attributed to sarcoplasmic protein precipitation.

In addition to the previously discussed effects of ultimate pH (Hamm, 1960) upon water holding capacity, decreased water retention is associated with rapid postmortem muscle changes as well as the degree of actomyosin formation (loss of binding sites) during rigor mortis development (Bendall and Wismer-Pedersen, 1962; Sayre et al., 1964; Bodwell et al., 1966).

Forrest et al. (1966) demonstrated that stimulation by electric shock shortly after slaughter resulted in stronger contractility for a longer duration among those muscles which subsequently underwent slow postmortem changes than for muscles which underwent rapid postmortem changes.

Briskey and Wismer-Pedersen (1961b) and Sayre et al. (1963b, c, d) showed that glycogen content per se had no effect on rate of postmortem change provided sufficient glycogen was present to attain normal ultimate pH values. Sayre et al. (1963a) and Kjolberg et al. (1963) reported that glycogen structure had little or no effect on rate of postmortem glycolysis. Bendall et al. (1963) and Sayre et al. (1963b, c) found no consistent association between muscle buffering capacity and rate of postmortem glycolysis.

Total protein, lipid and moisture contents of the muscle do not appear to be related to rate of postmortem change (Briskey et al., 1959c; Wismer-Pedersen, 1959; Lawrie, 1960; Wismer-Pedersen and Briskey, 1961a, b; Dahl, 1962). While Sink et al. (1967) found no relationship between chloroform-methanol or ether extractable lipids and rate of postmortem change, Krzywicki and Ratcliff (1967) found that phospholipid content was directly related to rate of postmortem pH decline.

Following a review of the literature, Briskey (1964) stated that rapid postmortem changes in muscle resulted in lower ultimate myoglobin contents. He emphasized that the total quantity as well as the chemical and physical state of myoglobin as affected by postmortem changes in porcine muscle required further study.

Briskey et al. (1959c) found no relationship between muscle potassium content and rate of postmortem changes. However, they noted higher sodium contents among muscles which underwent rapid glycolysis. Topel et al. (1967) found that neither muscle nor plasma sodium and potassium levels were associated with rate of pH decline. Cassens et al. (1963c, d) found no differences in zinc content of muscles exhibiting fast or slow glycolysis.

Sayre et al. (1963c) found that when muscle extracts were obtained 10 min postmortem phosphorylase was in the b form. These authors as well as Wismer-Pedersen (1959), Kjolberg et al. (1963) and Aberle and Merkel (1968b) could not relate total phosphorylase activity to postmortem glycolytic rate. Sayre et al. (1963b, d) found no apparent association between phosphofructokinase activity and rate of postmortem glycolysis. Kastenschmidt et al. (1966, 1968) studied the concentrations of glycolytic

intermediates in porcine muscle and they implicated phosphorylase, pyruvic kinase and especially phosphofructokinase as the rate limiting steps in postmortem glycolysis. Aberle and Merkel (1968a) reported no relationship between adenylic acid deaminase activity and rate of postmortem changes.

Factors Influencing the Rate of Postmortem Changes in Porcine Muscle

Predisposition.

Lawrie and Gatherum (1962) and Bendall et al. (1963) indicated that muscles from Large White pigs normally exhibit slower rates of postmortem glycolysis than those of the Danish Landrace breed. Clausen and Thomsen (1960) and Ludvigsen (1960) reported a higher incidence of PSE muscles from Pietrain than for Landrace pigs although both breeds have a rather high incidence. Bray (1968) in summarizing the work of Judge et al. (1959), Sayre et al. (1963d), and Allen et al. (1966) noted that in this country PSE pork is more prevalent among Poland China, Hampshire and Landrace pigs than in the Chester White or Duroc breeds. The latter author pointed out that ultimate qualitative properties might be more accurately ascribed as being due to strain rather than breed differences since the PSE condition occurs among all breeds, but some breeds or strains are more predisposed than others. Lasley (1968) and Christian (1968) stated that selection of pigs for increased lean yield may have resulted in unintentional selection toward inferior muscle quality. While the work of Omtvedt (1968) concurs with the latter observation, he reported that the genetic correlation between lean yield and qualitative

factors was low. He also stated that although additional investigations are needed, present heritability estimates for muscle qualitative factors are sufficiently high to be useful in the selection of breeding stock.

Briskey (1964) stated that adjacent porcine muscles frequently exhibit pronounced variation in color, gross morphology and general appearance. Lawrie and Pomeroy (1963) reported that considerable variability in sodium, potassium and myoglobin contents exists between muscles. Briskey et al. (1960b) indicated that muscles which exhibited relatively high ultimate pH values showed lower levels of initial glycogen, greater juice retention and higher myoglobin contents than muscles with lower pH values. Lawrie et al. (1963) indicated that variation in moisture content between muscles was directly related to ultimate pH values.

Lawrie et al. (1958) and Wismer-Pedersen (1959) noted that longissimus dorsi and semimembranosus muscles were susceptible to rapid postmortem changes. Briskey and Wismer-Pedersen (1961a) also implicated the susceptibility of the longissimus dorsi and biceps femoris muscles. Briskey et al. (1960b) found that the visual appearance of the gluteus medius was a good indicator of the water binding properties of the longissimus dorsi and biceps femoris muscles. Briskey (1964) stated that the number and location of muscles exhibiting the PSE condition varies considerably within carcasses. This observation may be related to differences in relative cooling rates, myoglobin content or oxygen storage capacity of the muscles. The latter author stated that light colored, inactive, tetanic muscles are more predisposed to development of rapid postmortem changes than dark colored, active, tonic muscles.

Lawrie et al. (1958) reported a wide range in ultimate pH values between various locations within the semimembranosus muscle. Lawrie (1960) observed lower ultimate pH values and concomitant greater degrees of exudation in the lumbar region of the longissimus dorsi muscle than in the thoracic region. Briskey (1964) reported greater severity of the PSE condition in the caudal and cranial regions of the longissimus dorsi muscle than in the mid section.

Beecher et al. (1965b) found no relationship between initial or ultimate glycogen and pH values with percent red fibers, myoglobin content or succinic dehydrogenase activities for muscles of varying degrees of "redness". However, they noted that ultimate lactic acid content tended to be higher in "white" muscles.

Beecher et al. (1965b) reported that rigor mortis developed earlier in the light portions of the biceps femoris and semitendinosus muscles than in the dark portions. Beecher et al. (1965a) further indicated that the light portion of the semitendinosus muscle exhibited faster glycolytic rates following exsanguination than the dark portion. Beecher et al. (1968) found that myoglobin content, percentage of red fibers and succinic dehydrogenase activity in the dark portion of the semitendinosus was approximately twice the corresponding levels in the white portion of the muscle. They also noted less moisture and sodium, but more lipid in the white portion, while calcium, potassium and magnesium contents were similar for the two sections of the muscle.

The influence of antemortem factors on postmortem changes

Thyroid (Ludvigsen, 1953) and adrenal (Ludvigsen, 1957) insufficiencies have been implicated in the susceptibility to rapid postmortem changes.

Ludvigsen (1960) postulated that in muscular pigs the increased muscle development was probably due to increased pituitary growth hormone production which would suppress thyrotropic and adrenocorticotropic activities. Wismer-Pedersen (1968) concurred with this postulation and further stated that the more muscular pigs are more predisposed to PSE development.

Ludvigsen (1953) reported that he increased the incidence of PSE pigs by feeding methylthiouracil 10 to 14 days prior to slaughter. Topel and Merkel (1966, 1967) followed the same experimental design and were unable to substantiate these findings.

Hedrick et al. (1963) and Ramsey et al. (1964) reported an improvement of porcine muscle color and increased ultimate pH by adrenaline injection 24 hr prior to slaughter; however, Aberle and Merkel (1968b) could not change ultimate qualitative characteristics by injecting epinephrine 10 min before exsanguination.

Topel et al. (1967) reported that decreased levels of plasma 17-hydroxycorticosteroids were associated with more rapid muscle postmortem pH declines. However, induced suppression of plasma 17-hydroxycorticosteroid levels showed no relationship to the rate of postmortem changes (Topel and Merkel, 1967; Aberle and Merkel, 1968b).

Judge et al. (1966, 1968a, b) noted that thyroid and adrenocortical insufficiencies were related to development of PSE muscle. Cassens et al. (1965) reported that adrenal glands displaying large lipid masses, which they interpreted as indicative of degenerative changes, were directly associated with rate of postmortem changes. Howe et al. (1969)

produced similar adrenocortical alterations by subjecting stress susceptible pigs to variable combinations of temperature and humidity environments.

Forrest et al. (1963) noted that the incidence of PSE paralleled the daily environmental temperature fluctuations and the incidence was highest when fluctuations were greatest. Sayre et al. (1961) decreased initial glycogen and ultimate lactic acid levels by subjecting pigs to an ice water bath prior to slaughter. Heat treatment (45°C) for 30 to 60 min prior to slaughter greatly accelerated postmortem glycolytic rates (Kastenschmidt et al., 1964, 1965). Sayre et al. (1963b) produced a similar response to heat treatment in Hampshire and Poland China pigs, but not among Chester Whites, even though the internal muscle temperature of all breeds was above 41°C. Kastenschmidt et al. (1964, 1965) greatly reduced the rate of postmortem pH decline by subjecting pigs to heat treatment followed immediately by cold treatment. Improvement in ultimate muscle properties was noted by this combination of preslaughter treatments even when internal muscle temperatures were not decreased (Kastenschmidt et al., 1965).

Ultimate muscle quality was adversely affected when growing pigs were subjected to alternating temperatures (Thomas et al., 1966; Addis et al., 1967a, b; Howe et al., 1968; Judge, 1968). This effect was most noticeable at moderate (40%) as opposed to low (17 to 30%) or high (85%) relative humidities. High relative humidity in combination with warm environmental temperatures had no detrimental effect on ultimate muscle quality. The latter authors indicated that it was possible to acclimate pigs to the heat stress. They ascribed this response to increased aerobic metabolic capacity.

Forrest et al. (1965) reported that greatly elevated heart and respiration rates, immediately prior to slaughter were associated with rapid rates of postmortem pH decline. They further noted that both heart and respiration rates tended to increase as muscle temperature increased. Forrest (1965) and Forrest et al. (1968) concluded that circulatory and respiratory difficulties leading to increased blood P_{CO_2} and decreased P_{O_2} and pH could be major contributors to production of PSE muscle, particularly when animals are subjected to warm temperatures immediately preslaughter. These authors observed that with the strains of pigs studied, the Chester Whites were able to maintain homeostatic conditions under heat stress to a greater extent than the Poland Chinas and thus the Chester Whites were more resistant to development of rapid postmortem muscle changes.

Engelhardt (1966) reported that domestic pigs have a smaller heart weight per unit of body weight when compared to wild pigs or other domestic animals. He indicated that this gave an unfavorable relationship between cardiac capability and body need.

Briskey and Lister (1968) reported that the development of anoxia associated with the death struggle and exsanguination process accounts for most of the muscle lactic acid content at slaughter.

Meyer et al. (1962) reported that hogs with high glucose tolerance levels tended to have increased muscle glycogen levels and faster rates of postmortem glycolysis. Wismer-Pedersen (1968) concluded that the occurrence of PSE musculature does not appear to be related with any known nutritional deficiency.

Briskey et al. (1959a, b, 1960a) reported that exhaustive exercise immediately preslaughter decreased initial glycogen levels and increased ultimate pH values. Lewis et al. (1959, 1961) improved ultimate muscle color as a result of periodic preslaughter electric shock. Sayre et al. (1963c) decreased initial glycogen levels and rate of postmortem changes by fasting for 70 hr prior to slaughter. Judge et al. (1967) increased rate and extent of postmortem glycolysis by physically restraining hogs prior to slaughter. Sayre et al. (1963c) increased the rate of postmortem glycolysis by short term exercise and excitement immediately prior to slaughter. Wismer-Pedersen (1959) stated that fright and shock rather than mere exercise were responsible for rapid postmortem pH fall. Wismer-Pedersen (1968) stated that exercising pigs (long term) during the feeding period might improve ultimate muscle characteristics but only if the exercise was relatively extensive.

Briskey (1963) and Lawrie (1966a) indicated that there appears to be little or no effect of stunning procedure upon rate or extent of postmortem changes as long as the medulla oblongata in the brain was not destroyed. Wismer-Pedersen and Rieman (1960) reported an increase in the incidence of low pH immediately after exsanguination when the medulla oblongata was cut. McLoughlin (1964) reported that stunning with a captive bolt pistol before exsanguination resulted in more rapid postmortem pH declines than when no stunning was performed. He also indicated that electrical stunning caused a more rapid pH decline in the muscle postmortem than the carbon dioxide immobilization method. Both methods produced more rapid pH falls postmortem than when stunning was not performed before exsanguination.

Briskey (1963) indicated that a slow rate of bleeding or retention of blood increased the rate of postmortem glycolysis. Wismer-Pedersen and Rieman (1960) reported increased rate of pH decline as the time between exsanguination and evisceration increased.

Sayre et al. (1966) reported that muscles of 5 to 10 kg pigs contained more glycogen and less myoglobin and total nitrogen than similar muscles from 250 to 300 kg pigs. They noted that muscles from more mature pigs tended to have faster postmortem glycolytic rates, with increased color loss and decreased protein solubility than muscles from younger pigs.

The influence of postmortem factors on muscle changes.

Briskey (1963) reported that removal of the skin from pork carcasses as opposed to conventional slaughter procedures (dehairing) facilitated faster chilling rates, which subsequently resulted in slower postmortem pH drops and darker colored musculature. Subjection of excised muscles or intact carcass sides to elevated temperatures (37°C) immediately postmortem, accelerated rate of pH drop (Wismer-Pedersen and Briskey, 1961a; Bendall and Wismer-Pedersen, 1962; Briskey, 1964; Beecher et al., 1965a; Bodwell et al., 1966). On the other hand, partial freezing of loins or hams in liquid nitrogen immediately postmortem was effective in improving ultimate meat qualitative properties (Borchert and Briskey, 1964). Additionally, Borchert and Briskey (1965) reported higher sarcoplasmic and myofibrillar protein extractabilities and improved emulsifying properties when the muscles were rapidly chilled or partially frozen (liquid nitrogen). Lewis et al. (1967) observed a decrease in muscle quality due to conventional freezing (-30°C) immediately after slaughter.

Direct electrical stimulation of excised muscles greatly accelerates postmortem glycolytic rate (Hallund and Bendall, 1965; Bendall, 1966b; Forrest and Briskey, 1967; McLoughlin, 1969). Forrest and Briskey (1967) reported accelerated postmortem changes after electrically stimulating the spinal cord of the split carcass immediately postevisceration. McLoughlin (1969) noted that contraction of muscle postmortem markedly increased glycolytic rate.

EXPERIMENTAL METHODS

Experimental Animals

Pigs, ranging in weight from 148 to 281 lb and originating from different management regimes, were slaughtered in four separate groups. Group I included 18 Poland China, 16 Landrace and 6 Chester White pigs, which were obtained from three Michigan hog producers. Group II included 40 pigs which were slaughtered for the 1968 Michigan Spring Barrow Show. These pigs represent 34 different Michigan hog producers. Group III included 44 pigs of various breeding, originating from either the Michigan Swine Testing Station or the Michigan State University swine herd. Group IV included 22 Yorkshire pigs which were obtained from the University swine herd.

Slaughter

The pigs in Groups II and III were slaughtered at the University Meat Laboratory in accordance with conventional procedures, i.e., they were electrically stunned, bled, scalded, dehaired, eviscerated and split into halves prior to washing and placing in a 3 to 4°C cooler. Animals in Groups I and IV were shackled and bled without stunning. Because of the muscle sampling procedure, these carcasses were not scalded but skinned, eviscerated and left unsplit, then washed and placed in 3 to 4°C coolers.

Sampling Procedure

Group I. Samples of the right longissimus (LD) muscle were excised from the 4th to 5th lumbar region at the time of exsanguination (0 hr). Subsequent LD samples were excised cranially from the initial sample site at 15 min, 45 min and 3 hr postmortem. A 24 hr postmortem sample was excised from the left LD in the 10th rib region. Histochemical samples were excised from the approximate geometric center of the right LD at 30 min postmortem. All muscle samples were frozen in liquid nitrogen immediately after excision.

Blood samples were collected at the time of exsanguination, allowed to clot, centrifuged and the resultant serum frozen and stored at -20°C. Within 5 min postexsanguination, a liver sample was excised and frozen in liquid nitrogen.

Group II. Rectal temperatures were recorded for the pigs in this group at the time of exsanguination. At 45 min postmortem, the right LD temperature of each pig was recorded and a sample was then excised from the right LD (10th rib region) for surface pH determination.

Group III. Muscle temperature was obtained at the time of exsanguination by inserting a thermometer directly into the right LD. LD temperature was also obtained on 20 of the carcasses in this group immediately after dehairing. At 45 min postmortem, LD temperature was recorded for all 44 carcasses in this group and a sample (right side) from the 11th rib region of the LD muscle was then excised for surface

pH measurement. Transmission values (measure of water extractable proteins) were determined on 24 hr muscle samples excised from the 9th rib region of the LD (right side).

Group IV. Samples from both the right and left LD and rectus femoris (RF) muscles were excised and frozen immediately in liquid nitrogen at the postmortem time periods shown in Table 1. The 0 hr LD samples were obtained at the time of exsanguination from the 2nd to 3rd lumbar region. All subsequent LD samples were excised by moving progressively cranial to the 0 hr sample. Samples were removed from the left LD directly opposite those of the right LD for corresponding postmortem time periods. The 0 hr RF samples were also obtained at the time of exsanguination. The anterior third of the RF was used for 0 hr or 15 min samples, the medial third for 45 min or 2 hr samples and the posterior third for the 24 hr samples.

Table 1. Postmortem time periods of sample excision for right and left sides of each muscle and the number of pigs included in each time period.

	Longissimus (LD)								
	Right					Left			
Postmortem time	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr
No. of pigs	12	12	12	12	12		12	12	12
No. of pigs	5	5	5	5	5	5	5	5	5
No. of pigs		5	5	5	5		5	5	5
	Rectus femoris (RF)								
No. of pigs	10		10		10		10		10
No. of pigs	5			5	5			5	5
No. of pigs		6		6	6			6	6

LD temperatures (right side) were recorded at the time of exsanguination and at 45 min postmortem (both sides). The RF temperatures from both sides of six pigs were recorded at 2 hr. Samples removed from the right LD and RF at the time of exsanguination and from both right and left sides at 24 hr postmortem were used for moisture determinations.

Samples of the biceps femoris (BF) and supraspinatus (SS) muscles from the right side were excised shortly after exsanguination (4 to 5 min) and at 1, 2 and 24 hr postmortem for pH determinations. Twenty-four hour (postmortem) samples from the LD and RF muscles from both right and left sides and right side BF and SS muscles were used to obtain transmission values.

Subjective Muscle Quality Appraisal

At 24 hr postmortem, LD muscles from all carcasses were subjectively evaluated using 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 normal or ideal for each of these characteristics, i.e., dry and firm, moderate or higher degrees of marbling, and grayish pink color. Scores of 0 were assigned to very soft exudative muscles which were devoid of visible marbling and very pale in color.

Heart Weights

The hearts from 31 pigs in Group I and all pigs (66) in Groups III and IV were removed, trimmed of excess fat and weighed to the nearest 0.1 g.

Powdering of Frozen Muscle and Liver Samples

The frozen muscle and liver samples (stored at -20°C following liquid nitrogen freezing) were powdered in a -20°C room as described by Borchert et al. (1965). Shattered pieces of the frozen samples were placed in a Waring blendor jar with chipped dry ice, pulverized for approximately 30 sec and then sifted. The coarse material which remained on the sieve was discarded. The powdered samples were placed in sterile polyethylene bags and 12 hr were allowed for CO_2 sublimation before sealing the containers or using the samples for subsequent analyses. The powdered samples were stored at -20°C until used for analysis.

Muscle pH

Approximately 5 g of frozen, powdered muscle were suspended in 25 ml 0.005 M sodium iodoacetate. The pH estimates were obtained from these suspensions with a Corning Model 12 expanded scale pH meter. For pH determination of fresh tissue, approximately 5 g of muscle were blended in 25 ml 0.005 M sodium iodoacetate in a small Waring blendor jar and pH of the suspension was recorded.

Muscle Moisture Determination

Approximately 2 g of finely ground muscle were dried in a 100°C oven for 18 hr. After cooling in a desiccator for approximately 1 hr, weight loss was recorded as moisture (AOAC, 1965).

Transmission Values

The transmission value procedure described by Hart (1962b) was used as an objective measure of muscle quality for the carcasses in Groups III and IV. Ten g of finely ground muscle sample (24 hr postmortem) were weighed in a centrifuge tube and cold distilled water added to bring to a total volume of 40 ml. The mixture was thoroughly stirred and held at 2 to 4°C for 20 hr after which time the mixture was restirred, centrifuged, and the supernatant filtered through Whatman No. 1 filter paper. One ml of clear filtrate was mixed in a test tube with 5 ml of prechilled (20°C) pH 4.6 buffer (9.35 parts of 0.2 M Na_2HPO_4 and 10.65 parts of 0.1 M citric acid). This mixture was incubated in a 20°C water bath for 30 min and after thorough mixing, percent transmission was read on a Bausch and Lomb Spectronic 20 colorimeter at 600 mμ against a blank (1 ml muscle filtrate and 5 ml distilled water).

Succinic Dehydrogenase Activity

The 0 hr LD samples from the carcasses in Group I were analyzed for succinic dehydrogenase (SDH) activity (Bonner, 1955). Approximately 4 g of powdered, frozen sample were extracted for 30 min with 15 ml 0.02 M phosphate buffer (pH 7.2). The samples were then centrifuged at 1500 x G

for 20 min and the supernatant (3°C) was adjusted to pH 5.7 with 1 N acetic acid. The resultant mixture was centrifuged at 1500 x G for 15 min and the precipitate suspended in 3 ml of 0.1 M phosphate buffer (pH 7.2).

A 0.3 ml aliquot of buffered suspension was added to a tube containing 1.9 ml of 0.15 M phosphate buffer (pH 7.2), 0.3 ml of 0.1 M KCN, 0.3 ml of 0.01 M K_3 Fe $(CN)_6$ and 0.2 ml of 0.2 M sodium succinate. Reduction of K_3 Fe $(CN)_6$ was read spectrophotometrically at 420 mμ with a Beckman DU Spectrophotometer. Absorbance was obtained initially and after 30 min of incubation at 35°C against a blank [identical to the sample tubes except distilled H_2O replaced K_3 Fe $(CN)_6$].

Red, White, and Intermediate Fibers

The fresh frozen LD samples for histochemical analysis of fiber type were sectioned (10 μ) on a Slee freeze microtome (-20°C). The sections were placed on coverslips and stained for SDH activity (Nachlas et al., 1957). The sections were incubated in a 0.05 M phosphate buffer (pH 7.6) at 37°C. This buffer also contained 0.05 mM of sodium succinate and 0.5 mg of Nitro BT [nitro-2,2:5,5'-tetraphenyl-3,3'-(3,3'-dimethoxy-4,4'-biphenylene) ditetrazolium chloride] per ml. After incubation (1 to 2 hr) the sections were washed in physiological saline (8.5 g NaCl, 0.2 g $CaCl_2$ and 0.1 g KCl in 1 l. of distilled water) and mixed in 10% formol saline for 10 min, rinsed in 15% ethanol for 5 min and mounted in glycerine jelly.

Prints (10 1/2" x 13 3/4") were made from the slides which were photographed from a magnification of 80X. Pictures were obtained from

three different areas of each muscle sample (LD) for the determination of red, white and intermediate fiber types. Relative numbers as well as relative areas of fiber types were determined. Area was measured with a compensating polar planimeter. Relative fiber size for each type was calculated from the number and total area of the respective fiber type.

Myoglobin

Total myoglobin concentration as well as estimates of the relative amounts of reduced myoglobin (Mb), metmyoglobin (MMb) and oxymyoglobin (O_2 Mb) were determined on the 0 hr LD samples from Group I carcasses according to the absorbancy ratio method of Broumand et al. (1958). Five g of frozen, powdered muscle were extracted in a stoppered Erlenmeyer flask with 20 ml cold distilled water by vigorous shaking for 1 1/2 min and the mixture was then filtered through Whatman No. 1 filter paper. The first 2 to 3 ml of filtrate were discarded and absorbance of the remaining filtrate was spectrophometrically determined at 473, 507, 573 and 597 mμ against a water blank. After obtaining the above readings, one drop of 0.5% KCN and one drop of 2% K_3 Fe $(CN)_6$ was added to the blank and each sample tube. Absorbance at 542 mμ was recorded as a measure of total myoglobin compounds converted to cyanometmyoglobin (CMMb).

The absorbancy ratio 507/573 mμ was used to estimate the % MMb while the ratio 473/597 mμ estimated the % Mb. These percentages were read directly from standardized curves presented with the original method. Percent O_2 Mb was determined by difference [% O_2 Mb = 100 - (% MMb + % Mb)]. The millimolar extinction coefficient of CMMb is 11.3 at 542 mμ.

Some Metabolites Involved in Glycolysis

Some of the metabolites involved in the glycolytic pathway were extracted from the frozen, powdered LD and RF samples obtained from the carcasses of Group IV and enzymatically determined by fluorometry in accordance with the procedures of Lowry et al. (1964) and Maitra and Estabrook (1964). The metabolites assayed included glycogen, glucose, glucose-1-phosphate (G-1-P), glucose-6-phosphate (G-6-P), fructose-6-phosphate (F-6-P), lactic acid, adenosine triphosphate (ATP), adenosine diphosphate (ADP), adenosine monophosphate (AMP) and creatine phosphate (CP).

Extraction

Frozen, powdered muscle samples were placed in test tubes containing 3 M $HClO_4$ (prechilled to 3°C) and after sample weight was obtained additional 3 M $HClO_4$ was added to give a $HClO_4$:muscle ratio of 2.86:1 (V/W). Following centrifugation (1500 x G for 15 min), 3.14 ml of 2 M $KHCO_3$/g of initial muscle sample weight were added to the decanted supernatants. After CO_2 evolution, 4.0 ml of 2 M Tris base/g of the initial muscle sample weight were added to bring the pH to 7.5 to 8.0. The supernatants containing 1 mg of muscle equivalent/0.01 ml were decanted from the $KClO_4$ precipitates and then stored at 3°C until analyses were completed (within 2 days).

Fluorometry

Analyses were conducted with 1 ml of solution which included reagents, enzyme(s) and neutralized sample extracts in 7 x 40 mm fused quartz

fluorometer tubes. The Aminco-Bowman Spectrophotofluorometer was employed to read the changes in concentration (fluorescence) of either the reduced nicotinamide dinucleotide phosphate (NADPH) or reduced nicotinamide dinucleotide (NADH). An excitation wavelength of 376 mμ and an emission wavelength of 464 mμ was used for all analyses. Standards and enzyme blanks were run with each analysis. All reactions were conducted at room temperature (25 to 28°C).

Reaction media, containing 0.01% bovine serum albumin, were prepared and stored at -20°C until needed. Where NADH was required, it was added shortly before each series of analyses. All enzymes (Sigma Chemical Co.) were diluted for use in these analyses with 0.02 M Tris buffer (pH 7.5) containing 0.02% bovine serum albumin to provide the required amounts in 0.01 ml.

G-6-P, ATP and CP

These compounds were measured on the same sample aliquot (0.02 ml of neutralized $HClO_4$ extract). Reactions were carried out in 0.1 M Tris buffer (pH 7.5). This buffer solution contained 1 mM of glucose, 0.3 mM of NADP and 5 mM of $MgCl_2$ per liter. Before adding 2 μg of yeast G-6-P dehydrogenase to each tube, the initial fluorescence reading was obtained. After the completion of NADPH increase (5 to 10 min), fluorescence was again recorded. Yeast hexokinase (2.5 μg) was then added and following an additional increase in NADPH (approximately 20 min) another reading was recorded. Ten μg of muscle phosphocreatine kinase and 0.03 μM of ADP (prepared within 1 hr of use) were added to each tube. Upon completion of this reaction (20 to 30 min), a final fluorescence reading for this series of reactions was recorded.

Glucose, G-1-P and F-6-P

Aliquots of the previously described muscle extract (0.02 ml) were added to 0.1 M Tris buffer (pH 7.5). This buffer solution contained 0.3 mM of ATP and NADP and 5 mM of $MgCl_2/1$. Initial fluorescence was obtained after completion of the reaction following the addition of 2 µg yeast G-6-P dehydrogenase. Additional fluorescence was recorded upon completion of the reaction initiated by the addition of 10 µg of muscle phosphoglucomutase to each tube (1 to 2 hr). Still another 1 to 2 hr elapsed following the addition of 10 µg of yeast phosphohexose isomerase to each tube before reading again. The last fluorescent reading for this series of compounds was obtained following the completion of the reaction catalyzed by the addition of 2.5 µg yeast hexokinase.

Glycogen

Approximately 25 ml of 1 N HCl were added to the insoluble residue which remained after $HClO_4$ extraction as previously described. This mixture was placed in a 100°C oven for 3 to 4 hr to convert muscle glycogen to glucose and after heating, additional 1 N HCl was added to obtain a HCl:muscle ratio of 20:1 (V/W). Aliquots (0.005 to 0.01 ml) of clear HCl hydrolysate (obtained by centrifugation) were added to the reaction media as previously described for glucose. Initial fluorescence was obtained 5 to 10 min after 2 µg of yeast G-6-P dehydrogenase had been added. Approximately 20 min after the addition of 2.5 µg of yeast hexokinase to the reaction media a final reading of NADPH fluorescence was recorded.

ADP and AMP

Reactions for ADP and AMP were conducted in a 50 mM potassium phosphate buffer (pH 7.0). This buffer also contained 3 μM NADH, 0.02 mM ATP, 0.02 mM phosphoenolpyruvate and 2 mM $MgCl_2$/1. A 0.02 ml aliquot of neutralized $HClO_4$ extract was used for these analyses. Fluorescence was obtained 10 min after the addition of heart lactic dehydrogenase (8 μg/tube). Additional fluorescence readings were recorded after the completion (10 min) of NADH decrease following the introduction of 1 μg of muscle pyruvate kinase. The final decrease in NADH was recorded about 10 min after the addition of 1 μg of muscle adenylic acid kinase to each tube.

Lactate

Lactic acid concentrations were fluorometrically determined according to an enzymatic procedure described by Hohorst (1963). Aliquots of neutralized $HClO_4$ extracts (0.005 to 0.01 ml) were added to a hydrazine-glycine buffer (7.2 g glycine, 5.2 g hydrazine sulphate, 0.2 g EDTA-NaH_2 .2 H_2O and sufficient 2 N NaOH to attain a pH of 9.5 in a total volume of 200 ml). This buffer contained 0.3 mM of NAD/1. Readings were recorded upon completion of the fluorescence increase (approximately 2 hr) after the addition of 10 μg heart lactic dehydrogenase.

Lipids

Extraction

Lipids were extracted by modification of the methods described by Ostrander and Dugan (1962) and Masaro *et al.* (1964) from the 0 hr LD, liver

and serum samples of the pigs in Group I. Sixty ml of chloroform-methanol (1:1 V/V) was added to either 20 ml of serum or 10 g of muscle or liver and stirred for 10 min. Twenty ml of 5% zinc acetate were added to the extraction mixture and stirred for an additional 1 min. The mixture was then filtered with the aid of suction through a Buchner funnel fitted with Whatman No. 1 filter paper. The precipitate was extracted an additional two times and filtered as described above. The combined filtrates were transferred to a separatory funnel and the chloroform layer removed. The water-methanol layer was washed twice with 75 ml chloroform. The chloroform fractions were combined and evaporated to dryness with a rotating flask evaporator. Traces of the solvent were removed by heating the residue in a 100°C oven for 5 min. Upon cooling in a dessicator, weight of the lipid material was obtained. This lipid material was redissolved in 10 ml chloroform and stored at -20°C for further analysis.

Phospholipids

Phospholipids of the LD samples were separated from neutral lipids by modification of the methods described by Bates (1958), Reiser et al. (1960) and Choudhury and Arnold (1960). Lipid material was dissolved in 50 ml of chloroform and mixed with 10 g of silicic acid [heated (110°C) for 2 hr]. The mixture was stirred for 10 min and filtered through a sintered glass funnel (medium porosity) by suction. Four-50 ml portions of chloroform were used to elute the neutral lipids. Phospholipids were then eluted with four-50 ml portions of methanol. Both fractions were evaporated to dryness, restored to a 10 ml volume with chloroform, and stored at -20°C for further analysis.

Phospholipid phosphorus was determined as described by Beveridge and Johnson (1949). One ml of concentrated H_2SO_4 was added to dry phospholipid fractions in a microdigestion flask. The samples were placed on a burner (micro-Kjeldahl unit) and allowed to digest for six hours. When digestion was partially complete, 1 to 2 drops of 30% H_2O_2 were added and the flasks were shaken between the addition of each drop. Upon completion of the digestion, the samples were cooled and 9 ml of distilled water added. The samples were transferred to a 50 ml volumetric flask with 15 ml of distilled water. Twenty ml of freshly prepared molybdate-hydrazine sulfate reagent [25 ml of 2.5% (W/V) sodium molybdate dihydrate in 3 N H_2SO_4 and 10 ml 0.15% (W/V) hydrazine sulfate in 3 N H_2SO_4 brought to a total volume of 100 ml with distilled water] were added with a rapid delivery pipet. The samples were adjusted to volume with distilled water, mixed and heated in a boiling water bath for 10 min. Within 24 hr after cooling, color development was measured with a Spectronic 20 colorimeter at 830 mμ.

Neutral lipid ester determination

Ester concentration was measured on the neutral lipid fractions (Lands, 1958). Equal volumes of 4% NaOH and 4% hydroxylamine hydrochloride (W/V) in 95% ethanol were mixed and the resulting NaCl was removed by filtration. Two ml of the clear filtrate were added to a dry lipid sample in a screw cap culture tube. The tubes were shaken, heated (65°C) for 5 min and then cooled. Five ml of ferric perchlorate solution [4 ml of stock ferric perchlorate (Goddu *et al.*, 1955) and 2.5 ml of 70% $HClO_4$ diluted to 100 ml with cold absolute ethanol] were added as the tubes

cooled. Stock ferric perchlorate was prepared by dissolving five g of ferric perchlorate in 10 ml of 70% $HClO_4$ and 10 ml of H_2O. This mixture was diluted to 100 ml with anhydrous 2-butyl alcohol while cooling under a water tap.

Twenty min after the addition of the ferric perchlorate solution, color development was measured by recording absorbance with a Spectronic 20 colorimeter at 530 mμ. Triolein was used as a standard.

Statistical Analysis

The statistical procedures followed were those discussed by Steel and Torrie (1960). Duncan's new multiple range test was applied when analysis of variance data were significant in order to detect the significantly different means. Student's t values were obtained when only two means were compared. Where appropriate, simple correlation coefficients were obtained.

RESULTS AND DISCUSSION

Distribution of Muscle Fiber Types, Succinic Dehydrogenase Activity, Myoglobin and Lipid Levels

The pigs in Group I were categorized by breed according to the rate of postmortem pH decline in the LD muscle. Five pigs from each of three breeds (Poland China, Landrace and Chester White), which had 45 min postmortem pH values greater than 6.0 were called "normal". Five pigs from each of the Poland China and Landrace breeds were called "low quality" since they had 45 min pH values less than 6.0 and possessed ultimate LD muscle properties tending toward the pale, soft and exudative (PSE) condition described by Briskey (1964). The source of Chester White pigs used for this study were known to have a very low incidence of PSE and thus no attempt was made to obtain a low quality Chester White group.

The means of 45 min postmortem pH values and 24 hr subjective quality scores of the LD are presented in Table 2. As expected from the report of Briskey (1964), a significant ($P < .01$) difference for both 45 min pH values and subjective quality scores was noted between quality groups. These data are presented to justify the categorization of the pigs used in this study. A significant ($P < .01$) correlation coefficient of 0.63 was obtained between 45 min pH values and subjective quality scores of all the pigs in Group I (40).

The rate of postmortem pH fall was more rapid among the low quality pigs than for the normals (Figure 1). Comparable (pH fall) curves were observed within quality groups for the breeds included in this study.

Table 2. The mean pH values and subjective scores of the longissimus muscle by breed and quality group.[1]

	Breed				
	Poland China		Landrace		Chester White
Trait	Normal	Low quality	Normal	Low quality	Normal
45 min pH	6.19[a]	5.49[b]	6.23[a]	5.67[b]	6.26[a]
Subjective quality score[2]	10.0[a]	5.6[b]	11.0[a]	6.8[b]	13.0[a]

[1]Means with the same superscripts are not significantly different ($P > .01$).
[2]Score based on a 15 point scale with 5 possible points for each of the three muscle properties: marbling, color and structure (firmness and exudation).

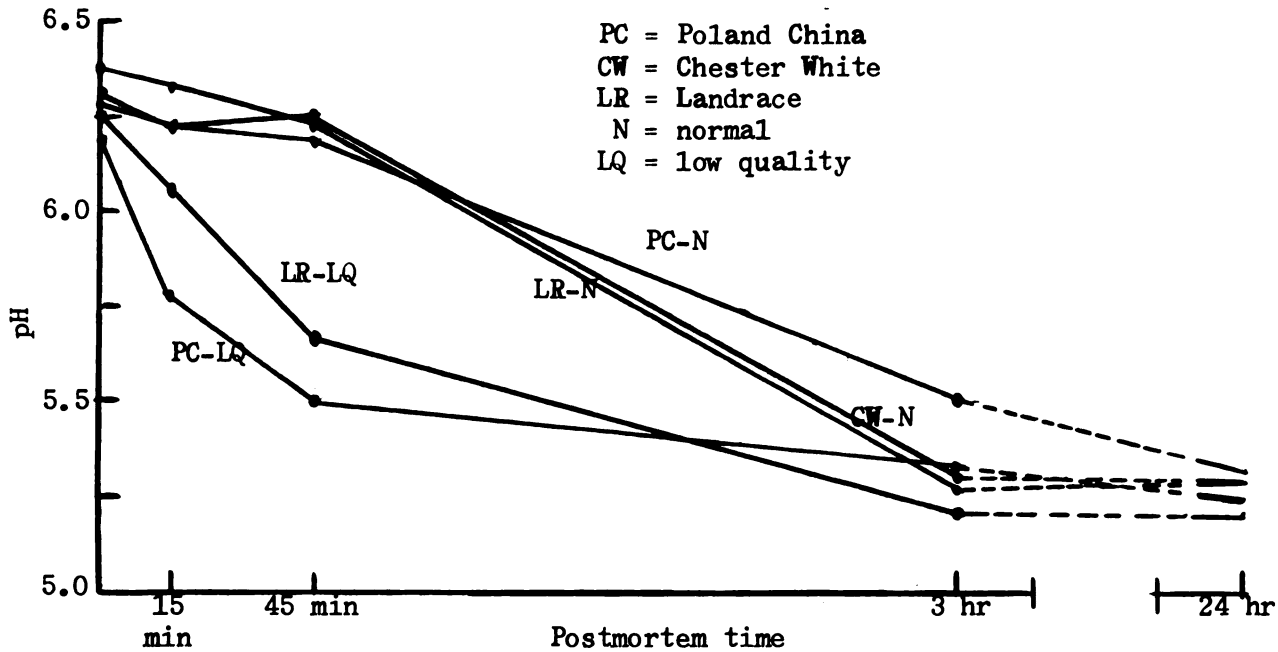

Figure 1. Postmortem pH curves of the longissimus muscle for the pigs in Group I.

While the initial (0 hr) pH values of the low quality pigs were slightly lower than normals, the rate and/or extent of decline was markedly different at 45 min postmortem. The rate of pH decline between quality groups became progressively narrower with postmortem time after 45 min and ultimate pH values (24 hr) were essentially the same. These data concur with the reports of Briskey and Wismer-Pedersen (1961a), Sayre and Briskey (1963) and Briskey (1964) and further substantiate the quality grouping used in this study.

Red, white and intermediate muscle fiber distribution.

White muscle fibers have a much greater anaerobic metabolic capacity and lower myoglobin and lipid levels than red fibers (George and Berger, 1966) and Beecher et al. (1965a, 1968) reported that differences in red and white fiber contents between the light and dark portions of the semitendinosus muscle were related to postmortem glycolytic rate. Thus it seems logical that differences in white or red fiber content between LD muscles could possibly be related to differences in ultimate quality characteristics. However, Briskey and Lister (1968) reported that a higher aerobic capacity (larger red fibers, higher levels of cytochrome oxidase and succinic dehydrogenase) existed for Poland China pigs than for Chester Whites and that the higher aerobic potential was related to development of PSE musculature. Thus this phase of the present study was undertaken to compare indices of aerobic metabolic capacity between normal and low quality LD muscle within and among several breeds of pigs.

In this study muscle (LD) fiber types were categorized as red (positive), white (negative) or intermediate on the basis of their staining

reaction for SDH activity. Intermediates were designated as those fibers which were not distinctly positive or negative but were intermediate in their staining reaction. The three fiber types were determined as a percentage of the total for both number and area. The relative fiber type size could then be calculated from these data.

When expressed as a percentage of total number, the normal Poland China pigs had more red fibers ($P < .05$) than either the low quality Poland China or Landrace pigs (Table 3). The low quality Landrace pigs had more white fibers than the normal groups and fewer intermediates than the other groups ($P < .05$).

On a percentage of total area basis (Table 3) both Poland China quality groups had significantly ($P < .05$) larger red fiber areas than all other groups. Both Poland China quality groups also had smaller white fiber areas than the normal Chester Whites or low quality Landrace pigs ($P < .05$). The low quality Poland Chinas had significantly ($P < .05$) larger intermediate fiber areas than the low quality Landrace pigs.

On a percentage of total area basis, Poland Chinas had more red and less white fibers than either Chester White or Landrace pigs; however, no significant ($P > .05$) differences exist between quality groups. On a percentage of total number basis, differences between breeds are apparent. Additionally, muscle from normal pigs contained more red and intermediate and less white fibers than that from low quality pigs ($P < .01$). From these data it is apparent that differences in relative areas or numbers of muscle fiber types vary considerably between breeds as well as quality groups.

Table 3. The distribution of red, white and intermediate fibers and succinic dehydrogenase activity in the longissimus muscle by breed and quality groups.[1]

Measurement and fiber type	Breed				
	Poland China		Landrace		Chester White
	Normal	Low quality	Normal	Low quality	Normal
Percent of total number					
Red	27.52[a]	22.67[b]	24.03[a,b]	20.34[b]	23.79[a,b]
White	63.13[b]	68.51[a,b]	65.86[b]	74.11[a]	67.22[b]
Intermediate	9.34[a]	8.83[a]	10.10[a]	5.54[b]	8.99[a]
Percent of total area					
Red	16.59[a]	16.79[a]	14.56[b]	14.09[b]	13.78[b]
White	76.51[b]	74.95[b]	78.22[a,b]	81.37[a]	80.39[a]
Intermediate	6.90[a,b]	8.25[a]	7.22[a,b]	4.53[b]	5.83[a,b]
Relative size[2]					
Red	10.02[a,b]	7.91[b]	10.97[a]	8.88[a,b]	10.31[a]
White	5.00	5.36	5.58	5.52	4.97
Intermediate	8.25[a,b]	6.50[b]	9.52[a]	7.87[a,b]	9.36[a]
Total fibers	6.05	5.87	6.63	6.07	5.95
Succinic dehydrogenase activity[3]	21.68[b]	18.49[b]	33.55[a]	17.91[b]	24.61[a,b]

[1]Means with the same superscripts do not differ significantly ($P > .05$).
[2]Number of fibers per square inch of picture.
[3]Millimicromoles succinate oxidized/min/g of muscle (0 hr).

These data explain why Briskey and Lister (1968) reported more red fibers among "stress-susceptible" pigs or those which ultimately developed PSE musculature. These authors used Chester Whites as "normal" or "stress-resistant" pigs, while Poland Chinas were used as the "stress-susceptible" pigs. The data in the present study supports the breed differences, but not the differences between quality groups.

Data for the relative size of each muscle fiber type are also presented in Table 3. Higher values correspond to relatively smaller fiber sizes. The relative size of the red and intermediate fiber types differ between breed-quality groups, whereas the white fiber size appears markedly similar. The "t" test analysis shows that normal muscles had smaller red ($P < .01$) and intermediate ($P < .05$) fibers than the low quality muscles. There do not appear to be any breed differences in relative size of the three fiber types.

A significant ($P < .05$) correlation coefficient ($r = 0.48$) was observed between percentage of red fiber numbers and 45 min pH; whereas, the percentage of red fiber area was negatively, but nonsignificantly ($P > .05$) correlated ($r = -.24$) with 45 min pH.

Succinic dehydrogenase activity.

The mean SDH activities between breed and quality groups are shown in Table 3. The Landrace pigs had significantly ($P < .05$) higher SDH activities than the normal Poland Chinas or the low quality pigs. Normal muscle possessed higher SDH activity than that from low quality muscle ($P < .05$). These data for SDH activities support that of the relative numbers and areas of fiber types previously discussed. LD muscles with higher percentages and/or smaller areas of red fibers tended to have higher SDH activity. Since the red and intermediate fibers from low quality muscle are larger than those from normal muscle, they are perhaps not as "red" or aerobic in nature as indicated by SDH activity.

The correlation between SDH activity and 45 min pH for all the pigs in Group I (40), although low ($r = 0.33$) was significant ($P < .05$). The

correlation coefficient for SDH activity and subjective quality scores ($r = 0.42$) was highly significant ($P < .01$).

Myoglobin.

Table 4 presents the mean levels of total Mb as well as relative amounts and absolute values of MMb, reduced Mb and O_2Mb. The normal Landrace pigs had significantly ($P < .05$) more total Mb than the other quality groups, while the low quality Poland Chinas had significantly ($P < .05$) less than all other groups. Breed differences were apparent since normal Landrace pigs had more total Mb than normal Chester Whites or both Poland China quality groups ($P < .05$). Low quality Landrace pigs had more total Mb than low quality Poland Chinas. Significant ($P < .05$) differences between quality groups were also apparent. Normal Landrace pigs had more total Mb than either the low quality Landrace or Poland Chinas and normal Chester White and Poland China pigs had more total Mb than low quality Poland Chinas.

There were no significant ($P > .05$) differences in percents MMb, reduced Mb or O_2Mb between any of the breeds or quality groups. Differences in levels of these Mb fractions reflect total Mb differences. Even though there appeared to be no consistent pattern among the percentages of these Mb fractions there was a trend toward the normal pigs having higher MMb and O_2Mb levels than the low quality groups.

Total Mb from 0 hr LD muscles (40 pigs) was significantly ($P < .01$) correlated with 45 min pH ($r = 0.41$).

From the data in Tables 3 and 4 it can be seen that higher proportions of red muscle fiber types were associated with increased SDH activities

Table 4. Myoglobin content of the longissimus muscle by breed and quality group.[1,2]

	Breed				
	Poland China		Landrace		Chester White
	Normal	Low quality	Normal	Low quality	Normal
Percent of total myoglobin					
Metmyoglobin	33.3	33.7	39.3	36.1	34.8
Reduced myoglobin	16.5	19.3	25.1	20.9	19.0
Oxymyoglobin	50.2	46.9	35.6	43.0	46.2
Millimicromoles /g of muscle					
Total myoglobin	80.2^{b}	55.4^{c}	114.8^{a}	89.4^{b}	79.4^{b}
Metmyoglobin	$26.3^{b,c}$	19.2^{c}	45.8^{a}	32.4^{b}	$28.0^{b,c}$
Reduced myoglobin	12.8^{b}	11.2^{b}	29.2^{a}	19.3^{b}	15.5^{b}
Oxymyoglobin	41.1^{a}	25.0^{b}	$40.1^{a,b}$	$37.7^{a,b}$	$36.0^{a,b}$

[1]Means with the same superscripts do not differ significantly ($P > .05$).
[2]0 hr samples.

and higher total Mb levels in normal muscle than in low quality muscle at or shortly after the time of exsanguination. While all of these observations were not significantly correlated with each other, individually they were significantly related to 45 min postmortem pH values. Thus it appears from these indices that a greater aerobic metabolic potential existed in normal muscle than in low quality muscle at the time of exsanguination. This may at least partially explain the postmortem differences observed between normal and low quality LD muscle. Also, this observation may explain why the 0 hr pH of the low quality muscles was lower ($P < .01$)

than that for normal muscles (Figure 1). Additionally, Kastenschmidt et al. (1966, 1968) indicated that "fast-glycolyzing" muscles may already be in a highly anaerobic state at the time of exsanguination. However, it remains to be established to what degree these differences are inherent or attributable to management practices during growth and development.

Lipids.

Table 5 summarizes the serum, liver and LD lipid analysis of the pigs by breed and ultimate quality group. The lower quality Landrace pigs had higher serum lipid levels than the normal Landrace or both Poland China quality groups ($P > .05$). Liver lipid content did not vary significantly ($P > .05$) between any of the breed or quality groups.

While muscle lipid levels did not vary significantly ($P > .05$) between groups, there was a trend toward normal muscle among the Poland China and Landrace breeds to have higher concentrations of lipids than the low quality groups. Percent of muscle lipid was significantly ($P < .05$) correlated with 45 min pH ($r = 0.39$).

There were no significant ($P > .05$) differences in phospholipid content when expressed on either a total muscle basis or muscle lipid basis (Table 5). Phospholipid content on a total muscle was negatively, but nonsignificantly ($P > .05$) correlated with 45 min pH ($r = -.23$). These data do not support the highly significant relationship between muscle phospholipid content and 45 min pH observed by Krzywicki and Ratcliff (1967). However, their phospholipid values represented only the myofibrillar and reticular fractions of muscle, whereas the values reported in this study were determined on the whole muscle.

Table 5. Serum, liver and longissimus muscle lipids by breed and quality group.

	Breed				
	Poland China		Landrace		Chester White
	Normal	Low quality	Normal	Low quality	Normal
Lipid source[1,2]					
Serum	0.305[b]	0.314[b]	0.308[b]	0.374[a]	0.330[a,b]
Liver	5.38	5.36	5.29	5.58	5.63
Longissimus	4.20	3.20	4.77	4.18	3.98
Lipid component					
Phospholipids (μ eq P/g LD lipid)	164	242	171	164	146
(μ eq P/g LD)	6.16	7.13	7.69	6.56	5.94
Glyceride esters[2,3] (μ eq /g LD lipid)	763[b,c]	702[c]	871[a,b]	820[a,b,c]	915[a]
(μ eq/g LD)	33.5[a,b]	22.0[b]	40.7[a]	34.4[a,b]	36.6[a]

[1]% of lipid material on a fresh weight basis.
[2]Means with the same superscripts do not differ significantly ($P > .05$).
[3]Determined on the neutral lipid fraction only.

Glyceride ester contents expressed on a total muscle and muscle lipid basis are presented in Table 5. On a muscle lipid basis, Chester White pigs had higher glyceride ester contents than both Poland China quality groups ($P < .05$). Normal Landrace muscles had greater glyceride ester contents (muscle lipid basis) than the low quality Poland Chinas ($P < .05$). Low quality Poland China samples contained less glyceride esters than those for either normal Chester White or Landrace pigs on a whole muscle basis ($P < .05$). On a whole muscle basis glyceride ester levels were significantly ($P < .01$) correlated with 45 min pH values ($r = 0.55$).

These data indicate that if muscle lipid content is a factor in determining the rate or extent of postmortem changes this effect would result from the neutral lipid fraction. Fritz et al. (1958), Issekutz et al. (1964), Spitzer and Gold (1964) and Masaro (1967) reported that lipid oxidation may provide an important energy source for muscle activity. While the phospholipids of muscle are structural-functional elements, they are not mobilized for energy purposes; whereas triglycerides serve as the ultimate lipid energy reservoir (Masaro, 1967). Beatty et al. (1963) stated that red muscle fibers, commensurate with the greater aerobic metabolic capacity, would more readily oxidize fat for energy than white fibers. Thus the trend toward increased glyceride ester and total lipid contents in normal muscle may be associated with or are a result of the greater aerobic potential among these muscles.

Heart Weights

A deficiency of cardiovascular capacity (anatomical and physiological) has been implicated in the etiology of PSE musculature (Forrest et al., 1965, 1968; Merkel, 1968). Additionally, Engelhardt (1966) reported that wild pigs have heavier heart weights, and thus more favorable relationships between cardiac capability and body need, than domestic pigs. The above reports prompted a collection of heart weights in this study.

In order to minimize live weight differences, all heart weights were converted linearly to that of a 200 lb live weight pig. Mean heart weights for the normal and low quality Poland China pigs were 274.9 g and 270.7 g, respectively; 282.2 g and 236.4 g for the normal and low quality Landrace, respectively, and 286.1 g for the normal Chester Whites in Group I of

this study. Since only three heart weights were obtained for the normal Poland Chinas, this group was not included in the statistical analysis. Analysis of the above data indicate that low quality Landrace pigs had significantly ($P < .05$) lighter heart weights than normal Landrace or Chester Whites. However, heart weights were nonsignificantly ($P > .05$) correlated with either 45 min pH values of LD muscles from Groups I and IV or transmission values from Groups III and IV ($r = 0.07$ and $-.07$, respectively).

These results indicate that heart weight per se was not associated with the rate of postmortem pH decline or ultimate muscle quality. Two pigs in Group III, which had moderate to severe cases of pericarditis (detected upon postmortem examination) resulted in carcasses with severe PSE musculature. The heart weights of these two pigs (248 g and 235 g, respectively) when adjusted to a 200 lb live weight basis were comparable to the mean (253 g) of the low quality Landrace and Poland Chinas in Group I. These lower weights are contrasted to the 281 g mean heart weight of the normal pigs in Group I. However, it is likely that the impaired heart function rather than heart weight per se could have contributed to the low quality muscle of these carcasses.

Muscle Temperature

The adverse effects of high temperature-low pH relationships on ultimate muscle quality have been well documented (Briskey and Wismer-Pedersen, 1961a; Bendall and Wismer-Pedersen, 1962; Briskey, 1964). The incidence of low quality muscle development was reported to be markedly

increased when body temperature at the time of exsanguination is near or exceeds 42°C (Merkel, 1968). Hoernicke (1966) observed insufficient temperature regulation among some pigs. Thus, a phase of the present study involved the relationship of muscle temperature at the time of exsanguination and during early postmortem periods to some muscle quality parameters.

Temperature of the LD muscle 45 min postmortem was significantly ($P < .01$), but negatively correlated with (Groups II, III and IV) subjective quality scores ($r = -.32$) and with (Groups II and III) 45 min pH ($r = -.52$). Muscle temperature 45 min postmortem was also significantly ($P < .01$) correlated with (Groups III and IV) transmission values ($r = 0.56$). The higher LD temperatures 45 min postmortem either played a role in development of low quality muscle or they possibly resulted from the exothermic reactions associated with increased glycolysis.

Temperatures at the time of exsanguination showed lower relationships to rate of pH decline and ultimate quality properties than those at 45 min postmortem. Rectal temperature (Group II) was nonsignificantly ($P > .05$) correlated with 45 min pH ($r = -.29$). LD temperature at exsanguination (Group III) was nonsignificantly ($P > .05$) correlated with 45 min pH ($r = -.29$). A low, but significant ($P < .05$) correlation ($r = 0.27$) was observed between LD temperatures at the time of exsanguination and transmission values (Groups III and IV). Thus it appears from this study that muscle temperature at the time of exsanguination was not as important as that at 45 min postmortem in influencing ultimate muscle qualitative properties.

LD temperatures at 45 min postmortem (Table 6) were significantly ($P < .01$) higher than 0 hr rectal temperatures (Group II) or 0 hr LD temperatures (Group III). The greater temperature differential between 0 hr and 45 min postmortem in Group II possibly resulted from the fact that 0 hr temperatures were obtained rectally rather than directly from the LD muscle. However, pigs in Group II were slaughtered as a group (similar environmental temperature conditions); whereas, the pigs in Group III were slaughtered (in lots ranging from 2 to 10) over a three month period. The relatively "hot and humid" conditions associated with extensive use of hot water and steam on the slaughter floor while the large number of pigs in Group II were slaughtered may be at least partially responsible for the elevated 45 min temperatures observed in this group. The prolonged exposure (1 to 1 1/2 hr) of the pigs in Group II to "hot and humid" slaughter conditions before chilling is unusual since the time required for dehairing, evisceration and cleaning before chilling varies to some extent with the number of pigs and normally far fewer are slaughtered at any one time.

The scalding operation which involved soaking the entire pig in hot water (60°C) for 5 to 10 min prior to dehairing could possibly have elevated the 45 min muscle temperature. From limited observations (20 pigs) in Group III, the temperature after scalding (15 min postmortem), while not significant ($P > .05$), tended to be higher than the initial temperature. The pigs in Group IV were not scalded or the carcasses skinned and eviscerated until approximately 2 hr postmortem, and the 45 min temperatures were essentially the same as the initial temperatures

(Table 6). Thus subjection to scalding and/or "hot and humid" slaughter floor conditions may have contributed to increased muscle temperatures at 45 min postmortem.

Table 6. Some rectal and longissimus muscle temperatures at several postmortem time periods.[1]

	Time periods		
No. of pigs	0 hr	15 min	45 min
40[2]	39.3[b]	--	40.6[a]
44[3]	39.9[b]	--	40.4[a]
20[3]	40.3	40.7	40.5
19[4]	39.6	--	39.6

[1]Means with superscripts are significantly different ($P < .01$).
[2]Group II pigs, 0 hr temperature was rectal rather than LD.
[3]Group III pigs, LD temperatures.
[4]Group IV pigs, LD temperatures.

Two Yorkshires, which were intended to be slaughtered among the pigs in Group III, died prior to slaughter while either in transit to or shortly after arrival at the Meat Laboratory. One of these pigs had an LD temperature of 44.7°C approximately 1 hr postmortem with a pH value of 5.45; the other had a LD temperature of 44.0°C and a pH of 5.70 within 30 min postmortem. Two additional Yorkshire pigs had LD temperatures of 41.9°C and 42.2°C, respectively, at the time of exsanguination, while 45 min temperature was 41.7°C for both pigs. The LD pH at 45 min postmortem was 5.65 and 5.70 for these pigs, respectively. These latter two pigs were the same ones previously indicated to have had pericarditis and which ultimately possessed severe PSE musculature. Cardiovascular

impairment may very likely have contributed to the elevated temperature in the latter two pigs.

While observations in this study indicate that temperature 45 min postmortem appeared to exert a significant influence on ultimate porcine muscle properties, the factor(s) which contributed to elevated muscle temperatures (45 min postmortem) have not been positively identified.

The Effect of 0 hr Sample Excision on Postmortem Muscle Changes

Observations made while excising muscle samples from the pigs in Group I indicated that many of the postmortem changes which occurred were affected by the sampling technique. The 3 hr LD sample (excised from the same muscle which was incised for the earlier postmortem samples) frequently exhibited a trend toward PSE development, while the 24 hr sample from the opposite LD (not previously incised) appeared more "normal". Thus the pigs in Group IV were slaughtered to determine what, if any, effect(s) sampling procedure had on glycolytic rate and ultimate muscle qualitative characteristics.

Longissimus muscle.

Table 7 summarizes the postmortem pH patterns and illustrates the experimental design. A combination of three different initial sampling times were used to compare the right (LD-R) and left (LD-L) longissimus muscles as follows: 0 hr LD-R with 45 min LD-L (line 1, Table 7), 0 hr LD-R with 15 min LD-L (line 2, Table 7) and 15 min LD-R with 45 min LD-L (line

Table 7. The effect of 0 hr sample excision on postmortem pH decline of the longissimus muscle.[1]

No. of pigs	Right longissimus					Left longissimus			
	Postmortem time period					Postmortem time period			
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr
12	6.18^{b}	6.15^{b}	6.01^{b}	5.62^{c}	5.20^{d}	--	6.46^{a}	6.02^{b}	5.23^{d}
5	$6.20^{a,b,c}$	$6.12^{b,c}$	$6.04^{c,d}$	5.69^{e}	5.18^{f}	6.42^{a}	$6.34^{a,b}$	$5.81^{d,e}$	5.19^{f}
5	--	6.57^{a}	6.45^{a}	5.80^{b}	5.27^{c}	--	6.50^{a}	6.00^{b}	5.29^{c}

[1]Means with the same superscripts do not differ significantly ($P < .05$).

3, Table 7). Hereafter these three initial sampling times from the LD-R and LD-L will be referred to as 0-45, 0-15 and 15-45, respectively.

It is readily apparent from the data in Table 7 that 0 hr sample excision had a significant effect on the rate of postmortem pH decline. In the 0-45 group, the LD-L 45 min pH was significantly ($P < .05$) greater than LD-R 0 hr, 15 min or 45 min pH. The LD-L pH at 2 hr postmortem was comparable to the LD-R 0 hr pH. A significant ($P < .05$) difference between the two sides at 2 hr was readily apparent. In the 0-15 group, LD-L 45 min pH was significantly ($P < .05$) greater than LD-R 45 min pH. When comparing pH values among the three combinations of initial sampling times, it is apparent that LD muscles not incised at 0 hr maintained a high pH for at least 2 hr postmortem. No marked differences in 24 hr pH values were observed for any LD muscle indicating that only rate and not extent of postmortem glycolysis was affected.

Table 8 presents the means for transmission values and subjective quality scores as affected by 0 hr sampling. While none of the differences were significant ($P > .05$), there was a trend toward lower ultimate quality as a result of 0 hr sampling in the 0-45 group. One of the carcasses in the 0-45 sampling group had 2 hr pH values of 5.31 and 6.31, transmission values of 95.0 and 18.5 and subjective quality scores of 5 and 10 for the LD-R and LD-L, respectively. Two other carcasses from the same sampling group had transmission values of 85.2 and 38.8, and 56.0 and 19.2 for LD-R and LD-L, respectively. Thus it is apparent that ultimate muscle properties of some carcasses were markedly affected by 0 hr sampling of the LD muscle.

Table 8. Effect of 0 hr sample excision on qualitative properties of the longissimus muscle.

LD-R, LD-L initial sampling time	Transmission values[1]		Subjective scores[2]	
	LD-R	LD-L	LD-R	LD-L
0-45	49.7	30.9	8.4	9.9
0-15	21.4	24.3	11.2	11.0
15-45	22.7	20.7	10.4	11.6

[1]Lower values correspond to more normal musculature.
[2]Higher scores correspond to more normal musculature.

To support the pH data shown in Table 7, glycogen, lactate, G-6-P, ATP and CP levels for the corresponding sampling groups are presented in Tables 9 through 13. Glycogen levels of the LD-L at 45 min postmortem were significantly ($P < .05$) greater than those at 0 hr, 15 min or 45 min for the LD-R muscle (Table 9). Glycogen content of the LD-L at 2 hr was significantly ($P < .05$) greater than that of the LD-L muscle at 2 hr postmortem. There were no significant differences within the 0-15 and 15-45 sampling groups between the LD-R and LD-L muscles at the same postmortem time periods. The LD-L muscles tended to maintain higher glycogen levels than the LD-R in the 0-15 group, especially at the early postmortem time periods. From a comparison of the glycogen levels of the LD-L and LD-R muscle samples in the 15-45 group at all postmortem time periods with levels in the other two sampling groups, it is readily apparent that glycogen content remained higher when no 0 hr sampling was performed. There appeared to be no marked differences in glycogen content among any of the muscle samples at 24 hr, thus indicating that only the rate and not the extent of glycolysis was affected by sampling schedule.

Table 9. The effect of 0 hr sample excision on postmortem glycogen levels of the longissimus muscle.[1,2]

No. of pigs	Right longissimus					Left longissimus			
	Postmortem time period					Postmortem time period			
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr
12	38.1^{b}	40.7^{b}	37.4^{b}	18.5^{c}	2.1^{d}		55.2^{a}	33.6^{b}	3.5^{d}
5	42.9^{a}	$39.4^{a,b}$	40.5^{a}	23.3^{c}	2.9^{d}	51.7^{a}	48.5^{a}	$26.2^{b,c}$	4.7^{d}
5		55.2^{a}	55.1^{a}	28.3^{b}	2.2^{c}		57.0^{a}	34.6^{b}	2.4^{c}

[1]Values expressed as micromoles of glucose equivalents/g of muscle.

[2]Means with the same superscripts do not differ significantly ($P > .05$).

The lactate values are presented in Table 10. Lactate levels in the LD-L 45 min muscle samples of the 0-45 group were significantly ($P < .05$) lower than in either 0 hr, 15 min or 45 min LD-R samples. A significant ($P < .05$) difference between sides was observed at 2 hr. There were no significant ($P > .05$) differences in lactate content between the LD-R and LD-L muscles within the same postmortem time periods in the 0-15 or 15-45 sampling groups. Lactate levels of the 15-45 group were consistently lower than the levels of both the 0-45 and 0-15 groups through at least 2 hr postmortem. No marked differences in lactate levels were evident between 24 hr means. These data support the observations for pH and glycogen levels.

Table 11 summarizes the effects of 0 hr sample excision on G-6-P levels. It can readily be seen that the levels were relatively high at 0 hr, then reached a low between 15 min and 2 hr and increased again at 24 hr. This pattern agrees with that reported by Kastenschmidt et al. (1968). However, these authors reported slightly higher values than those observed in this study. As observed with the other glycolytic metabolites, the 24 hr G-6-P values for all muscle samples were similar. In the 0-45 group, the LD-L muscles 45 min and 2 hr postmortem levels were significantly ($P < .05$) lower than LD-R 45 min and 2 hr levels, respectively. When comparing sides (LD-R vs LD-L) of all the sampling groups, it again is apparent that 0 hr sample excision resulted in elevated G-6-P levels at least through 2 hr postmortem.

Table 10. The effect of 0 hr sample excision on postmortem lactate levels of the longissimus muscle.[1,2]

No. of pigs	Right longissimus					Left longissimus			
	Postmortem time period					Postmortem time period			
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr
12	42.3^{d}	$48.9^{c,d}$	55.4^{c}	74.2^{b}	91.3^{a}		30.3^{e}	$53.1^{c,d}$	93.1^{a}
5	$40.8^{c,d}$	$45.6^{b,c,d}$	$53.0^{b,c,d}$	$69.3^{a,b}$	88.0^{a}	32.3^{d}	35.0^{d}	$65.9^{a,b,c}$	89.2^{a}
5		22.1^{c}	31.3^{c}	57.0^{b}	89.8^{a}		25.7^{c}	49.0^{b}	96.9^{a}

[1]Values expressed as micromoles/g muscle.

[2]Means with the same superscripts do not differ significantly ($P > .05$).

Table 11. The effect of 0 hr sample excision on postmortem glucose-6-phosphate levels of the longissimus muscle.[1,2]

No. of pigs	Right longissimus					Left longissimus			
	Postmortem time period					Postmortem time period			
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr
12	$5.64^{b,c}$	$3.76^{d.e}$	$2.66^{e,f}$	$5.06^{c,d}$	$6.78^{a,b}$		1.29^{g}	$2.19^{f,g}$	7.18^{a}
5	$5.95^{a,b,c}$	$4.45^{b,c,d}$	2.66^{d}	$4.23^{c,d}$	$6.69^{a,b}$	2.97^{d}	2.18^{d}	$4.03^{c,d}$	7.02^{a}
5		$1.56^{c,d}$	0.62^{d}	3.71^{b}	7.29^{a}		0.53^{d}	$2.50^{b,c}$	7.48^{a}

[1] Values expressed as micromoles/g muscle.

[2] Means with the same superscripts do not differ significantly ($P > .05$).

Table 12 shows the effects of 0 hr sample excision on ATP levels of the longissimus muscle. In the 0-45 sampling group, the LD-L 45 min ATP levels were definitely higher than those of the 0 hr, 15 min or 45 min LD-R muscle samples ($P < .05$). Levels of ATP in the 2 hr LD-L muscles were significantly ($P < .05$) higher than those in 2 hr LD-R. While no significant differences existed within the same postmortem time period in the 0-15 sampling group, there was an obvious trend for ATP levels in LD-L to remain higher than those at corresponding postmortem times in the LD-R muscles. When comparing sides (LD-R vs LD-L) of all three sampling groups, it is readily apparent that 0 hr sampling was responsible for lower ATP levels. This effect upon ATP level was probably attributable to the vigorous muscle contraction attendant with LD sample excision at or shortly after the time of exsanguination. The lower ATP levels very likely resulted from the activation of myofibrillar ATPase required to

Table 12. The effect of 0 hr sample excision on postmortem ATP levels of the longissimus muscle.[1,2]

	Right longissimus				Left longissimus		
	Postmortem time period				Postmortem time period		
No. of pigs	0 hr	15 min	45 min	2 hr	15 min	45 min	2 hr
12	2.10^{b}	2.00^{b}	1.58^{b}	0.43^{c}		3.72^{a}	1.52^{b}
5	$2.16^{a,b}$	$2.03^{a,b}$	$1.65^{a,b}$	0.92^{b}	3.26^{a}	3.46^{a}	1.16^{b}
5		3.62^{a}	3.28^{a}	0.92^{b}		3.48^{a}	1.63^{b}

[1]Values expressed as micromoles/g muscle.
[2]Means with the same superscripts do not differ significantly ($P > .05$).

operate the contractile mechanism. The lower ATP levels, in all probability, could be responsible for the increased glycolytic rate observed as a result of 0 hr sample excision. The effect might be expected from the reported controlling influence that ATP levels have on glycolysis, especially on the phosphofructokinase reaction (Wood, 1966; Scrutton and Utter, 1968). Electrical stimulation of the contractile mechanism also has been reported to activate the phosphorylase and phosphofructokinase enzymes (Karpatkin et al., 1964; Ozand and Narahara, 1964).

Table 13 summarizes the CP levels observed at various postmortem time periods as a result of 0 hr sample excision. In the 0-45 sampling group, LD-L 45 min CP levels were significantly ($P < .05$) higher than those at 0 hr, 15 min or 45 min in the LD-R muscles. In the 0-15 group, LD-L 15 min CP levels were considerably higher ($P > .05$) than those at 0 hr or 15 min in the LD-R muscles. The CP levels at 2 hr postmortem were quite similar in all sampling groups from both right and left LD muscles.

Table 13. The effect of 0 hr sample excision on postmortem creatine phosphate levels of the longissimus muscle.[1,2]

	Right longissimus				Left longissimus		
	Postmortem time period				Postmortem time period		
No. of pigs	0 hr	15 min	45 min	2 hr	15 min	45 min	2 hr
12	0.49^{b}	0.36^{b}	0.24^{b}	0.19^{b}		1.85^{a}	0.30^{b}
5	0.30^{b}	0.10^{b}	0.11^{b}	0.13^{b}	1.81^{a}	0.59^{b}	0.20^{b}
5		2.20^{a}	$0.92^{a,b}$	0.04^{b}		$1.56^{a,b}$	0.09^{b}

[1]Values expressed as micromoles/g muscle.
[2]Means with the same superscripts do not differ significantly ($P > .05$).

When comparing all three sampling groups, it is apparent that the 0 hr sample excision drastically reduced CP levels. Thus higher CP levels among the muscles not incised at 0 hr provided for a ready source of ATP. This observation could possibly help account for the slower rate of glycolysis observed among those pigs not sampled at 0 hr. The CP levels observed in this study were lower than those reported by Kastenschmidt et al. (1968), but no explanation was apparent except for possible breed differences.

To further examine the effects of 0 hr sample excision on rate and extent of postmortem glycolysis of the LD muscle, three pigs from the two sampling groups, 0-45 and 0-15, were designated as "normal" and three pigs from these same sampling groups were designated as "low quality" based upon rate of postmortem pH declines and 24 hr transmission values and subjective quality scores. Normal LD muscles had significantly ($P < .05$) slower pH declines, lower transmission values and higher subjective quality scores than the low quality LD muscles (Table 14, Figure 2). In addition to the glycolytic metabolites previously discussed levels of G-1-P, F-6-P, glucose, ADP and AMP were also determined on samples from both (right and left) LD muscles of these pigs. These data are presented graphically (Figures 3 and 4) to illustrate the results previously discussed and to show the differences in response to the effects of 0 hr sample excision between normal and low quality LD muscles.

Figure 2 shows that there was a difference in the postmortem pH pattern of LD muscle between the normal and low quality pigs. The 2 hr pH values of normal LD muscles were significantly ($P < .01$) higher than those of the low quality pigs (Table 14). At 45 min postmortem, only the

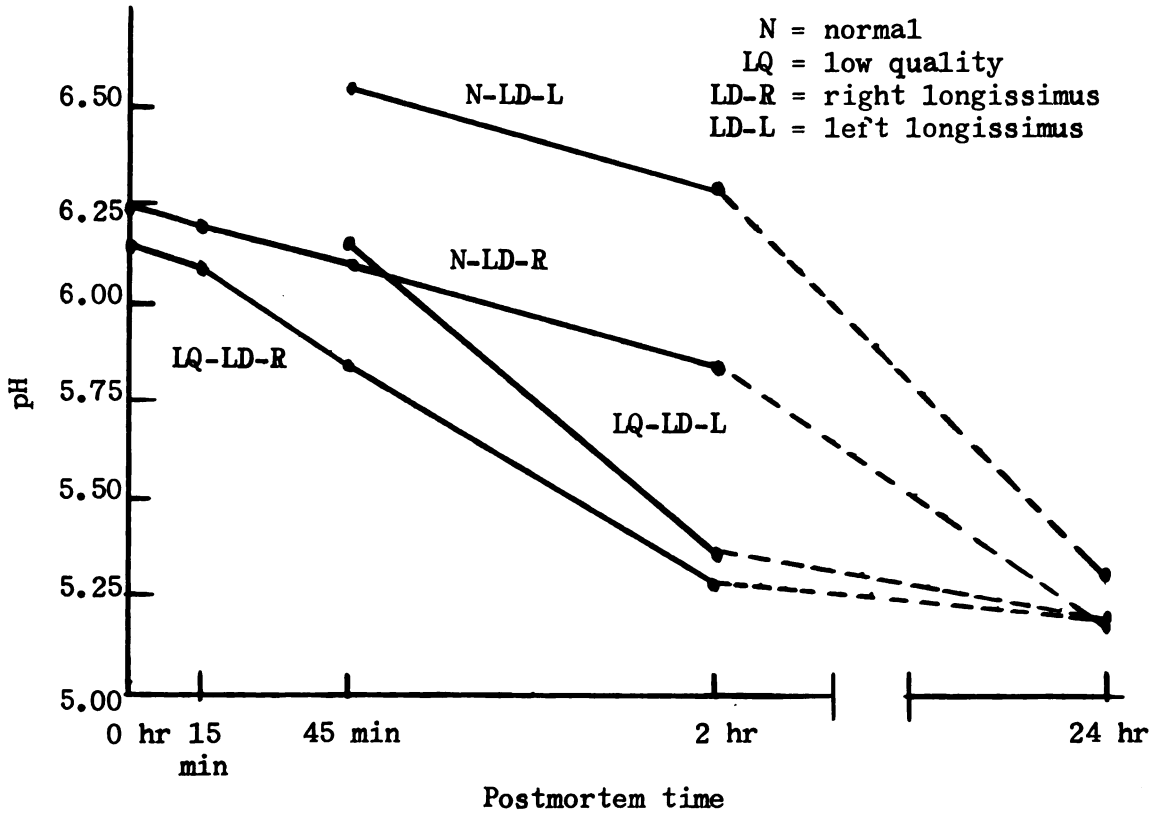

Figure 2. Postmortem pH pattern of normal and low quality longissimus muscle as affected by 0 hr sample excision.

Table 14. The effect of 0 hr sample excision on certain qualitative assessments for normal and low quality longissimus muscle.

Quality assessment	Normal quality		Low quality	
	Right longissimus	Left longissimus	Right longissimus	Left longissimus
Transmission values[1,4]	12.7[b]	11.3[b]	69.9[a]	66.8[a]
Subjective quality scores[2,3]	11.3[a]	12.3[a]	6.0[b]	6.3[b]
pH, 45 min[3]	6.10[b]	6.56[a]	5.84[c]	6.16[b]
pH, 2 hr[4]	5.85[b]	6.29[a]	5.29[c]	5.37[c]

[1]Lower values correspond to more normal musculature..
[2]Higher values correspond to more normal musculature.
[3]Means with the same superscripts are not significantly different ($P > .05$).
[4]Means with the same superscripts are not significantly different ($P > .01$).

LD-L pH values were significantly ($P < .05$) different between normal and low quality pigs. The pH values of the LD-L muscles 45 min postmortem were significantly ($P < .05$) greater than those of the LD-R muscles within quality groups. At 2 hr postmortem only the LD-L pH values of the normal muscles were significantly ($P < .01$) higher than those of the LD-R. These data indicate that 0 hr sample excision had a greater effect on normal muscles than on the low quality LD muscles.

Postmortem changes in muscle glycogen and lactate quantities (figure 3) corresponded to the magnitude of the pH declines, i.e. glycogen diminution essentially paralleled the pH drop, while lactate accumulated in inverse proportion to these levels. Glycogen values were consistently lower and lactate levels uniformily higher in the low quality muscles than those of the normal pigs at least until 2 hr postmortem. The 0 hr sample excision had a markedly greater effect on both of these metabolites in normal muscles than in those of lower quality. At 2 hr postmortem, glycogen and lactate levels were similar between the low quality LD-R and LD-L samples; whereas normal LD-R had considerably less glycogen and more lactate than those of the LD-L muscles. The glycogen and lactate curves (Figure 3) support the pH curves (Figure 2) in that low quality muscle exhibited a faster rate of postmortem glycolysis than normal muscle, while 0 hr sample excision more readily affected postmortem glycolysis in normal LD than LD muscle in the low quality carcasses.

The postmortem hexosemonophosphate (G-1-P, G-6-P and F-6-P) curves are presented in Figure 3. No explanation is apparent as to why the F-6-P curves did not parallel those of G-6-P as observed by Kastenschmidt et al.

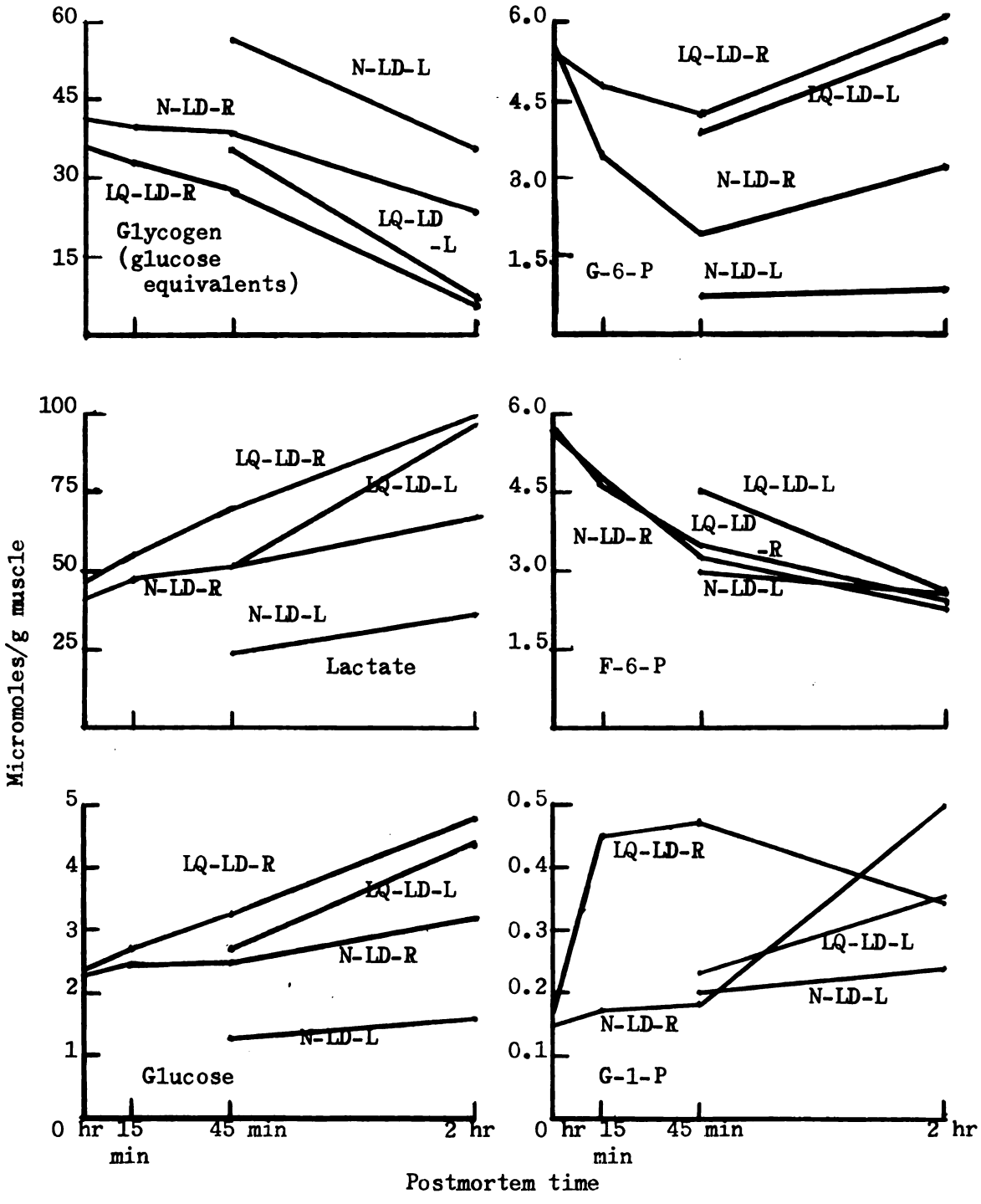

Figure 3. The effect of 0 hr sample excision on postmortem levels of glycogen, lactic acid, glucose, glucose-1-phosphate, glucose -6-phosphate and fructose-6-phosphate between normal (N) and low quality (LQ) longissimus muscle (LD-R = right longissimus; LD-L = left longissimus).

(1968). The latter authors reported considerably lower levels of F-6-P in porcine LD muscle than those obtained in this study. The only differences observed for the F-6-P curves in the present study was that the low quality group LD-L 45 min postmortem muscles contained higher levels (F-6-P) than the other muscle samples at 45 min postmortem.

Except for the 0 hr samples, G-6-P levels were consistently higher among low quality muscles than normal LD muscle, especially at 45 min and 2 hr postmortem. In general, the G-6-P levels were relatively high at 0 hr and minimal between 15 min and 2 hr postmortem; however, in all cases the 2 hr samples had higher G-6-P levels than those at 45 min postmortem. The G-6-P levels of the low quality LD muscles were essentially the same or slightly higher at 2 hr than at 0 hr; whereas, the G-6-P levels of the normal LD muscles were considerably lower 2 hr postmortem than at 0 hr. The LD-R muscles, which were incised at 0 hr, exhibited higher G-6-P levels at 45 min and 2 hr postmortem than the LD-L muscles. This effect of 0 hr sample excision on G-6-P levels was particularly obvious among the normal muscles.

The 0 hr G-1-P levels (Figure 3) were similar between normal and low quality LD muscles. The G-1-P levels of low quality LD-R muscles increased markedly until 15 min postmortem, remained relatively constant between 15 min and 45 min and then gradually decreased until the 2 hr postmortem sampling period. The G-1-P levels of the normal LD-R muscles increased gradually from 0 hr until 45 min and then increased more rapidly until 2 hr postmortem. The LD-L muscles of both quality groups gradually increased

in G-1-P level between 45 min and 2 hr; however, the low quality muscles had consistently higher values at both postmortem time periods. At 2 hr the normal LD-R muscles had higher G-1-P levels than the low quality LD-R muscle samples.

There were no differences in 0 hr glucose levels between the normal and low quality LD-R muscles (Figure 3). Glucose levels and the postmortem patterns agree favorably with the work reported by Kastenschmidt et al. (1968). Glucose levels increased more rapidly with time postmortem among the low quality muscles than in normal muscle and they were higher in the low quality muscles than in the normal LD muscles at all of the postmortem time periods studied. The LD-R samples (0 hr incision) had more glucose than LD-L muscles at corresponding postmortem time periods. This difference was most obvious among normal muscles than for the low quality muscles. Accumulation of glucose postmortem can result from α-amylase activity upon glycogen as reported by Lawrie (1966a).

Levels of ATP (Figure 4) were consistently higher among normal LD muscles especially the LD-L muscles than the low quality muscle samples. Except for the normal LD-L muscles, ATP levels gradually decreased from 0 hr until 2 hr postmortem. The normal LD-L muscles exhibited a considerably higher ATP level at 45 min than all other groups. Even though a marked decrease in ATP levels occurred in these normal LD-L muscles between 45 min and 2 hr postmortem, the (ATP) levels at 2 hr were comparable to those of the normal LD-R muscles at 0 hr. The LD-L muscles contained consistently more ATP than the LD-R muscle, within quality groups, especially among normal muscle samples.

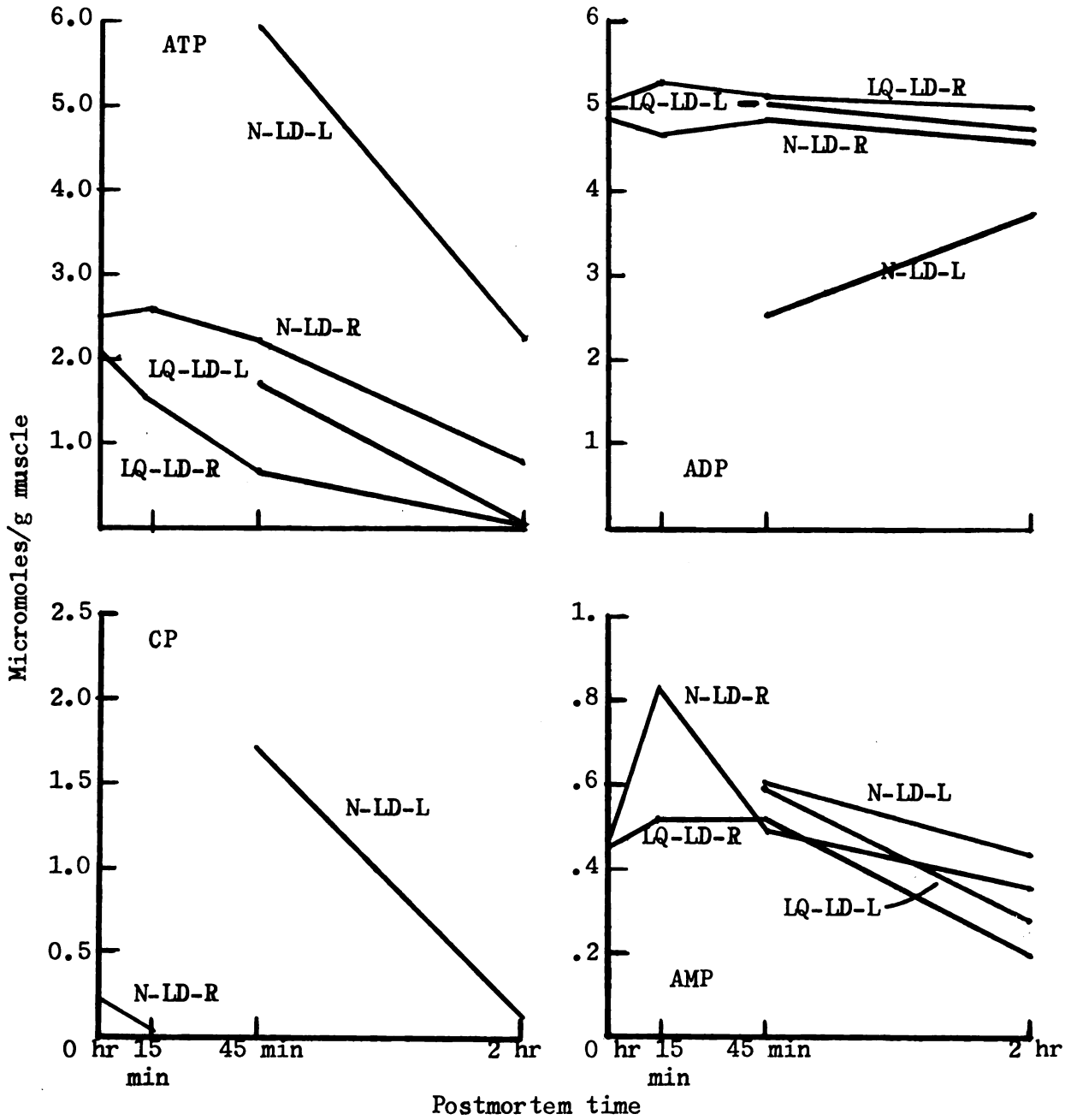

Figure 4. The effect of 0 hr sample excision on postmortem levels of ATP, ADP, AMP and creatine phosphate between normal (N) and low quality (LQ) longissimus muscle (LD-R = right longissimus, LD-L = left longissimus).

The levels of CP (Figure 4) were unexplainably low among all of the muscles included in this phase of the experiment, and in fact, CP levels were not detectable in any of the low quality muscle samples. The normal LD-R muscles had very low levels of CP at 0 hr, but no detectable quantities after 15 min postmortem. The normal LD-L samples had the highest CP levels at 45 min postmortem, and though its levels decreased, CP was still detectable at 2 hr.

The ADP levels (Figure 4) were unexplainably higher than those reported by Kastenschmidt et al. (1968). The normal LD-L muscles had appreciably less ADP than all the other muscle samples. While ADP levels remained relatively constant among the normal LD-R and low quality LD-R and LD-L muscle samples through 2 hr postmortem, the ADP levels of the normal LD-L muscles increased between 45 min and 2 hr.

The AMP levels (Figure 4) were similar among all the LD-L muscle samples studied, except that normal LD-R muscles had an obviously greater AMP content than low quality LD-R muscles at 15 min postmortem. While the LD-L muscles had higher AMP levels than the LD-R muscles at 45 min and 2 hr, the AMP levels gradually decreased in the muscles of all quality groups during this postmortem time interval.

Table 15 summarizes the levels (24 hr postmortem) of all the glycolytic metabolites studied, except those of ATP and CP, since the latter two compounds were detected in very minute amounts and then only in a few of the samples. Levels of the glycolytic metabolites present at 24 hr postmortem should provide an indication of the extent of glycolysis. Although no marked differences existed between quality groups or between sides

Table 15. The levels of some glycolytic metabolites in normal and low quality longissimus muscle at 24 hr postmortem.[1]

	Quality group			
	Normal		Low quality	
Metabolite	Right longissimus	Left longissimus	Right longissimus	Left longissimus
Glycogen[2]	3.2	5.6	1.9	2.1
Lactate	93.1	90.9	98.5	93.9
G-1-P	0.12	0.19	0.29	0.25
G-6-P	6.01	6.89	5.75	5.92
F-6-P	2.36	2.53	2.37	2.30
Glucose	5.80	4.68	6.31	6.30
ADP	5.54	4.10	5.86	5.72
AMP	0.28	0.26	0.20	0.24

[1]Levels expressed as micromoles/g muscle.
[2]Glycogen levels expressed as micromoles glucose equivalents/g muscle.

(LD-R vs LD-L) for these metabolites, several small differences were observed. The low quality LD muscles had higher levels of lactate, G-1-P, glucose and ADP and lower levels of glycogen, G-6-P and AMP at 24 hr postmortem than normal muscles. Within quality groups, the LD-R muscles had consistently higher levels of lactate and ADP and lower levels of glycogen and G-6-P than the LD-L muscle samples. The normal LD-R muscles also exhibited higher levels of glucose than the normal LD-L samples. From the data for glycogen and lactate levels, it appeared that the low quality muscles underwent (slightly) more extensive glycolysis than normal muscles, and that the LD-R muscle samples exhibited (slightly) more extensive glycolysis than LD-L samples.

In summarizing the effects of 0 hr sample excision on the LD muscles, it was obvious that resection of LD muscle at the time of exsanguination definitely resulted in a more rapid rate of postmortem glycolysis and that the ultimate muscle properties were altered. Normal muscles or those exhibiting a relatively slow rate of postmortem glycolysis were more markedly affected by 0 hr sample excision than the low quality muscles or those which showed a relatively fast rate of postmortem glycolysis. Excising the 0 hr muscle sample resulted in stimulation of contractile activity within the entire incised LD muscle; however the excised sample per se exhibited especially marked contractile activity before it was frozen in liquid nitrogen. ATP and CP are utilized by the contractile mechanism (Bendall, 1966; Lawrie, 1966a) and "slow-glycolyzing" muscle was reported to contain more ATP and CP than "fast-glycolyzing" muscle at the time of exsanguination (Briskey and Lister, 1968; Kastenschmidt et al., 1968). Thus the stimulation of contractile activity by the 0 hr sample excision could have conceivably reduced the ATP and CP levels to those normally observed in "fast-glycolyzing" muscle at the time of exsanguination.

From an examination of Figures 2-4, it appears that the rate of glycolysis was inversely proportional to ATP levels. The considerably higher ATP levels, present in normal LD muscles not sampled at 0 hr, were most likely responsible for the reduced rates of glycolysis. The increased G-6-P levels after 45 min postmortem would be expected if inhibition of the phosphofructokinase enzyme occurred (Wilson et al., 1967). Phosphofructokinase activity is reportedly pH sensitive and to be inhibited by

pH values of 6.0 or lower (Mansour, 1965). In the present study these low pH values were attained at or shortly after 45 min postmortem in all the LD muscles except for the normal LD-L samples.

Rectus femoris muscle.

The RF muscle is considered a "red" muscle as opposed to the LD which is classified as a "white" muscle. As such, the RF is implicated (Briskey et al., 1960b; Beecher et al., 1965b) as being more resistant to the development of low quality musculature than the LD. Thus, the RF muscle was included in this study to compare its response to 0 hr sample excision with that of the LD.

The experimental design for sample excision from the RF muscle was similar to that previously described for the LD. A combination of three different initial sampling times were used to compare the right (RF-R) and left (RF-L) rectus femoris muscles as follows: 0 hr RF-R with 45 min RF-L (line 1, Table 16), 0 hr RF-R with 2 hr RF-L (line 2, Table 16) and 15 min RF-R with 2 hr RF-L (line 3, Table 16). Hereafter these three initial sampling times from the RF-R and RF-L will be referred to as 0-45, 0-2 and 15-2.

The effects of 0 hr sample excision on postmortem pH decline of the rectus femoris muscle are presented in Table 16. The 0-45 group showed no significant ($P > .05$) differences in 0 hr or 45 min pH values between the two sides (RF-R vs RF-L). The 24 hr pH values for this sampling group were lower ($P < .01$) than the earlier postmortem values, but were nearly identical between the two sides. In both the 0-2 and 15-2 groups the 2 hr pH values for the RF-L were significantly lower than 2 hr

Table 16. The effect of 0 hr sample excision on postmortem pH decline of the rectus femoris muscle.[1]

No. of pigs	Right rectus femoris					Left rectus femoris		
	Postmortem time period					Postmortem time period		
	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
10	6.44^{a}		6.39^{a}		5.44^{b}	6.41^{a}		5.43^{b}
5	6.55^{a}			6.32^{a}	5.48^{c}		5.84^{b}	5.39^{c}
6		6.62^{a}		6.32^{b}	5.56^{d}		5.90^{c}	5.47^{d}

[1]Means with the same superscripts do not differ significantly ($P > .01$).

pH for the RF-R muscles. While the 2 hr pH value of the 0-2 sampling group was not significantly ($P > .05$) different from the 0 hr pH among the RF-R muscles, the 2 hr pH values of the 15-2 group were significantly ($P < .01$) lower than the 15 min pH of the RF-R muscle samples. While the 24 hr pH values of the latter two sampling groups (0-2 and 15-2) were significantly ($P > .01$) lower than the earlier postmortem pH values in these groups, the 24 hr pH values of the RF-R samples tended to be slightly higher than those of the RF-L samples. These data indicate that the RF muscles were affected opposite the LD muscles, i.e. 0 hr sample excision appeared to inhibit rather than stimulate postmortem glycolysis. These differences in results between the two muscles were not expected since the RF muscles appeared to contract just as violently or even more so than those of the LD following 0 hr sample excision.

Subsequent to the detection of the 2 hr pH differences between the RF muscles in the 0-2 sampling group, muscle (RF) temperatures were obtained at 2 hr postmortem in the 15-2 sampling group. The temperatures

at 2 hr postmortem were 34.2°C and 38.4°C for RF-R and RF-L samples, respectively. This difference was statistically significant ($P < .01$). The removal of skin and subcutaneous fat to facilitate excision of the RF-R muscle samples apparently allowed the remaining portion of the incised RF-R muscles to dissipate heat, while the RF-L which were not exposed until 2 hr postmortem tended to maintain in vivo temperatures. Despite the differences in postmortem pH declines no significant ($P > .05$) effect on RF transmission values were noted. The RF-R and RF-L transmission values for the 0-45, 0-2 and 15-2 sampling groups were 15.6 and 18.4, 11.3 and 9.9, and 11.2 and 12.4, respectively.

The RF values of glycogen (Table 17) and lactate (Table 18) appeared to substantiate the pH patterns. In the 0-45 sampling group neither glycogen nor lactate levels were significantly ($P > .05$) different among 0 hr RF-R or 45 min RF-R and RF-L muscle samples. Significantly ($P < .05$) less glycogen and more lactate was found at 2 hr postmortem in the RF-L muscles as opposed to the RF-R muscle samples in both the 0-2 and 15-2 sampling groups. Significantly ($P < .05$) more lactate accumulated at 2 hr than at 15 min postmortem in the RF-R muscles of the 15-2 sampling group. Comparable values for the 0-2 sampling group between 0 hr and 2 hr were nonsignificant ($P > .05$). No marked differences in 24 hr glycogen or lactate levels were apparent between sides (RF-R vs RF-L) in any of the sampling groups. Thus, it appears that the rate rather than the extent of postmortem glycolysis was affected by 0 hr sample excision.

Table 17. The effect of 0 hr sample excision on postmortem glycogen levels of the rectus femoris muscle.[1,2]

No. of pigs	Right rectus femoris					Left rectus femoris		
	Postmortem time period					Postmortem time period		
	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
10	30.5^{a}		33.7^{a}		6.6^{b}	35.8^{a}		5.1^{b}
5	36.4^{a}			30.8^{a}	4.9^{c}		15.2^{b}	3.8^{c}
6		39.2^{a}		32.9^{a}	5.1^{c}		17.8^{b}	3.5^{c}

[1]Levels are expressed as micromoles glucose equivalents/g muscle.
[2]Means with the same superscripts do not differ significantly ($P > .05$).

Table 18. The effect of 0 hr sample excision on postmortem lactate levels of the rectus femoris muscle.[1,2]

No. of pigs	Right rectus femoris					Left rectus femoris		
	Postmortem time period					Postmortem time period		
	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
10	27.2^{b}		34.2^{b}		73.2^{a}	32.2^{b}		76.8^{a}
5	19.8^{b}			34.6^{b}	68.8^{a}		69.3^{a}	77.6^{a}
6		18.5^{c}		34.6^{b}	71.2^{a}		67.2^{a}	74.2^{a}

[1]Levels are expressed as micromoles/g muscle.
[2]Means with the same superscripts do not differ significantly ($P > .05$).

Table 19 summarizes the G-6-P levels in the RF-R and RF-L muscle samples. The G-6-P levels appeared to be relatively high initially, dropped to low levels between 15 min and 2 hr postmortem and then reached higher levels at 24 hr than those found initially. No significant ($P > .05$) differences at 45 min (0-45 sampling group) or 2 hr (0-2 and 15-2 sampling

groups) for G-6-P levels were noted between RF-R or RF-L samples. However, at 2 hr postmortem in both the 0-2 and 15-2 sampling groups the RF-L muscles had more than twice as much G-6-P as the RF-R muscle samples. While the differences in G-6-P levels at 24 hr postmortem were only significant ($P < .05$) in the 15-2 sampling group, the levels tended to be higher among RF-R than RF-L muscle samples in the other two sampling groups.

Table 19. The effect of 0 hr sample excision on postmortem glucose-6-phosphate levels of the rectus femoris muscle.[1,2]

	Right rectus femoris					Left rectus femoris		
	Postmortem time period					Postmortem time period		
No. of pigs	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
10	4.07^{b}		0.91^{c}		7.02^{a}	0.91^{c}		$6.04^{a,b}$
5	3.12^{b}			1.12^{b}	7.02^{a}		2.94^{b}	5.70^{a}
6		1.74^{c}		1.12^{c}	8.24^{a}		2.66^{c}	5.78^{b}

[1]Levels are expressed as micromoles/g muscle.
[2]Means with the same superscript do not differ significantly ($P > .05$).

The ATP and CP levels of the RF muscles are presented in Tables 20 and 21, respectively. No significant ($P > .05$) differences in ATP or CP levels at either 0 hr or 45 min postmortem were evident between RF-R and RF-L muscle samples for the 0-45 sampling group. These results as well as the other glycolytic metabolite data for the RF muscles indicate that stimulation of the contractile machinery by sample excision did not cause a reduction of ATP levels and thus did not enhance the glycolytic rate

like that observed for the LD muscle. If myofibrillar ATPase was stimulated to any extent, then a very efficient maintenance of ATP levels occurred or the chilling effect associated with the RF muscle excision may have offset any stimulatory effects of the 0 hr muscle incision.

Table 20. The effect of 0 hr sample excision on postmortem ATP levels of the rectus femoris muscle.[1,2]

	Right rectus femoris					Left rectus femoris		
	Postmortem time period					Postmortem time period		
No. of pigs	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
10	2.73[a]		2.50[a]		0.12[b]	2.64[a]		0.08[b]
5	3.72[a]			2.51[b]	0.20[c]		0.72[c]	0.13[c]
6		3.60[a]		2.49[b]	0.19[c,d]		0.76[c]	0.10[d]

[1]Levels are expressed as micromoles/g muscle.
[2]Means with the same superscript do not differ significantly ($P > .05$).

Table 21. The effect of 0 hr sample excision on postmortem creatine phosphate levels of the rectus femoris muscle.[1,2]

	Right rectus femoris					Left rectus femoris		
	Postmortem time period					Postmortem time period		
No. of pigs	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
10	0.80[a]		0.78[a]		0.07[b]	0.45[a,b]		0.06[b]
5	2.32[a]			0.39[b]	0.09[b]		0.19[b]	0.11[b]
6		2.13[a]		0.18[b]	0.14[b]		0.14[b]	0.09[b]

[1]Levels are expressed as micromoles/g muscle.
[2]Means with the same superscript do not differ significantly ($P > .05$).

The ATP levels at 2 hr postmortem were significantly ($P < .05$) higher among the RF-R muscles than in the RF-L muscle samples in the 0-2 and 15-2 sampling groups. The ATP levels in the RF-R samples were significantly ($P < .05$) lower at 2 hr postmortem than at 0 hr or 15 min for both groups. The CP levels paralleled ATP concentrations. In contrast to the LD muscle previously discussed, low, but detectable levels of ATP and CP were found in most 24 hr RF muscle samples.

A comparison of the glycolytic metabolites discussed above, together with levels of G-1-P, F-6-P, glucose, ADP and AMP was made for the RF muscle between normal and low quality groups similar to that presented for the LD muscle. In both the normal and low quality groups the same three carcasses included in the discussion of the LD muscles plus one additional carcass were used in each group. The 0 hr values represented the same three carcasses used for the LD muscle; 45 min values included two of the three carcasses; 2 hr values included the remaining carcass of these three plus the additional carcass (indicated above); and the 24 hr values included all four carcasses in each group. This approach was followed because 45 min and 2 hr RF samples were not obtained from the same carcasses with the sampling procedure used for this phase of the study. Thus, it should be kept in mind during the subsequent discussion that direct comparisons of glycolytic metabolites in the RF muscles between postmortem time periods are limited by this sampling procedure. However, direct comparisons between muscles (RF-R vs RF-L) and quality (normal vs low quality) groups can be made for individual postmortem time periods.

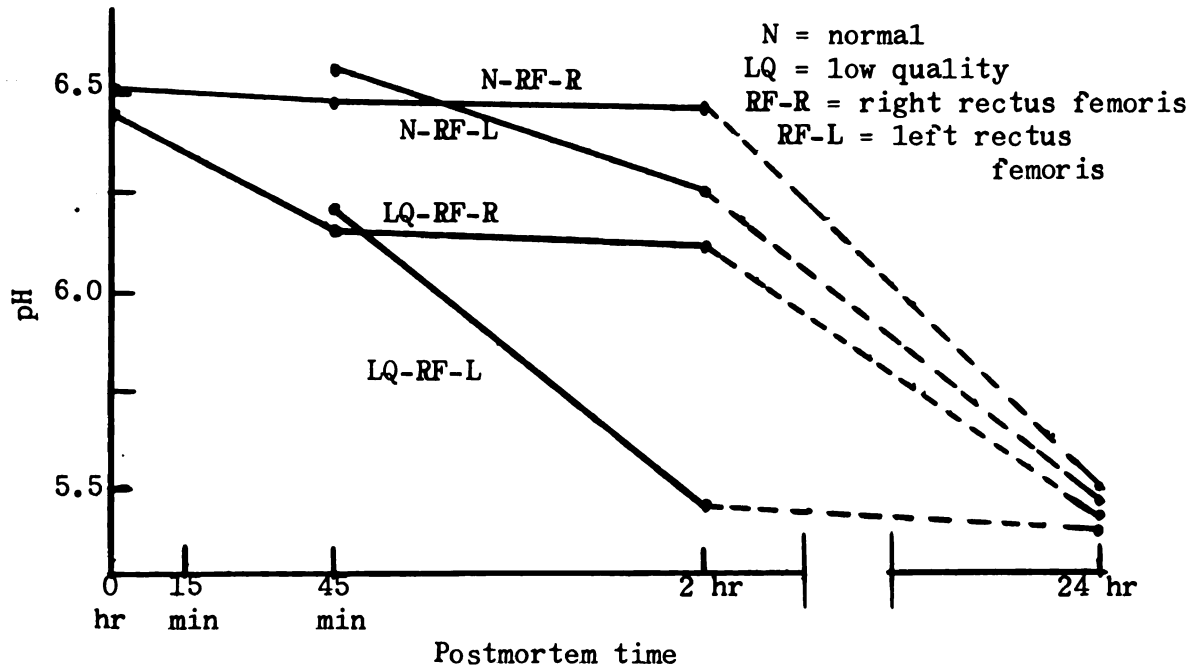

Figure 5. Postmortem pH pattern of normal and low quality rectus femoris muscle as affected by 0 hr sample excision.

The pH patterns for normal and low quality RF-R and RF-L muscles are presented in Figure 5. There appeared to be a more rapid pH decline in the low quality RF muscles than among normal muscles. This observation was especially obvious in the RF-L muscle samples. Thus it appears that the temperature effects (cooling) caused by 0 hr sample excision influenced the rate of postmortem pH decline more among low quality RF muscles than in normal muscles.

Glycogen and lactate levels (Figure 6) substantiated the results obtained for the pH patterns. The normal muscle samples retained more glycogen and accumulated less lactate than the low quality samples through the 2 hr postmortem time period. These observations were more obvious for RF-L than for RF-R muscles. Normal RF muscles contained more glycogen and less lactate than the low quality muscle samples at both 45 min and 2 hr postmortem. Differences in glycogen and lactate levels between sides

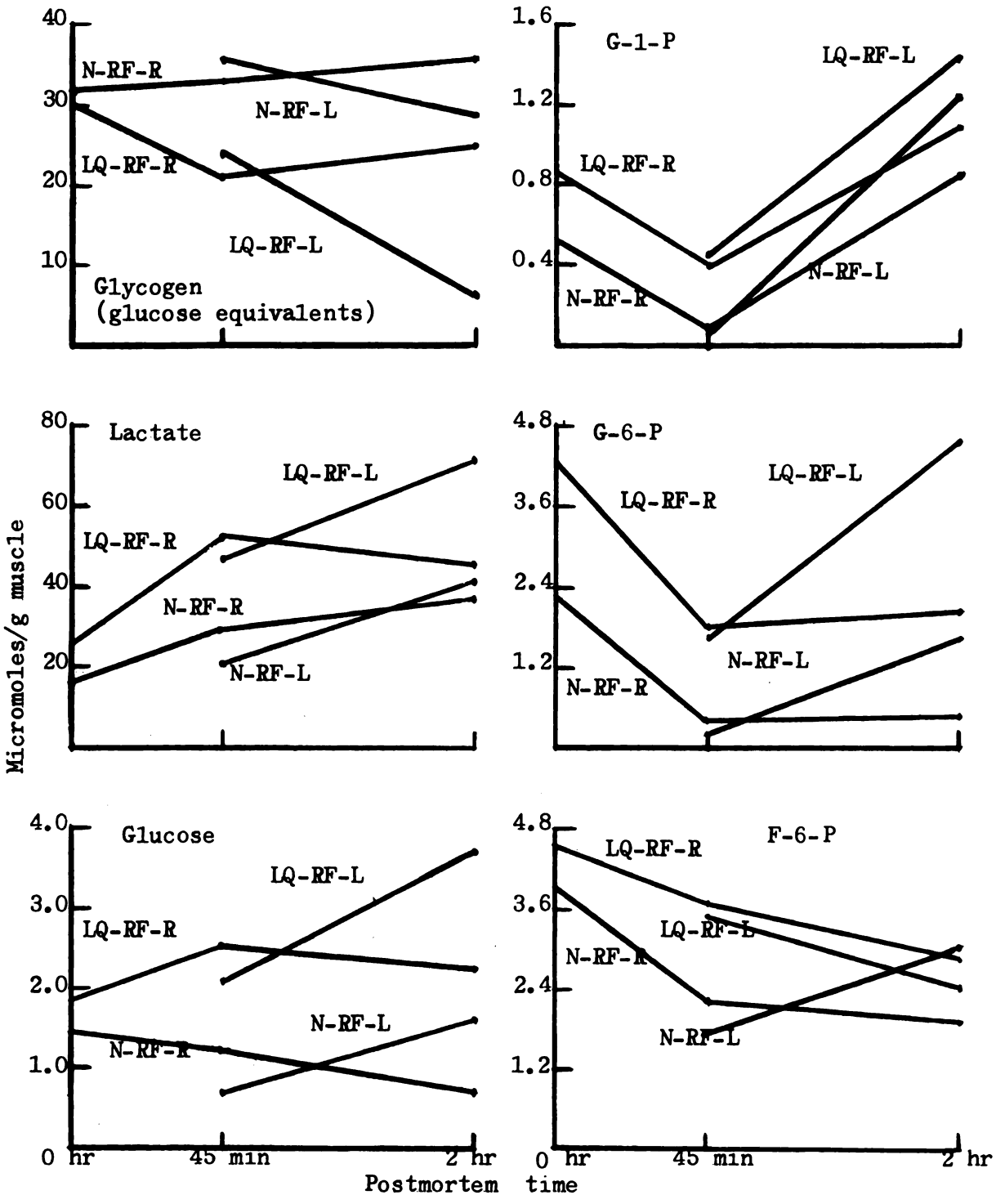

Figure 6. The effect of 0 hr sample excision on postmortem levels of glycogen, lactic acid, glucose, glucose-1-phosphate, glucose-6-phosphate and fructose-6-phosphate between normal (N) and low quality (LQ) rectus femoris muscle (RF-R = right rectus femoris; RF-L = left rectus femoris).

(RF-R vs RF-L) at 2 hr postmortem were greater in low quality than in normal RF muscles.

The G-1-P levels (Figure 6) decreased from 0 hr to 45 min postmortem and then increased markedly from 45 min to 2 hr among all muscle samples. Normal samples had lower G-1-P levels than low quality muscles at each postmortem time period.

The G-6-P levels (Figure 6) decreased from 0 hr to 45 min postmortem in all muscles and remained relatively constant between 45 min and 2 hr in the RF-R muscles. The RF-L muscles showed marked increases in G-6-P levels from 45 min to 2 hr postmortem. At 2 hr postmortem, the levels of G-6-P were higher in the RF-L muscle samples than in RF-R muscles and these higher levels were greater among low quality muscles than the normals.

While the normal RF-L muscles exhibited an increase in F-6-P levels (Figure 6) from 45 min to 2 hr postmortem, the F-6-P levels of all the other samples gradually decreased from 0 hr to 2 hr. Additionally, the low quality muscles exhibited slightly higher F-6-P levels than the normal RF-R samples.

Glucose levels (Figure 6) remained relatively constant in the RF-R muscles from 0 hr to 2 hr, but they increased between 45 min and 2 hr in the RF-L muscles. This increase was slightly greater among the low quality muscles than in the normal RF muscle samples. Glucose levels of low quality muscles were consistently higher than those of normal RF muscles.

The ATP levels (Figure 7) of the normal RF samples were consistently higher than those of the low quality muscle samples. While normal RF-R muscles showed little change in ATP levels from 0 hr to 2 hr postmortem, the ATP levels of the low quality RF-R samples decreased gradually during

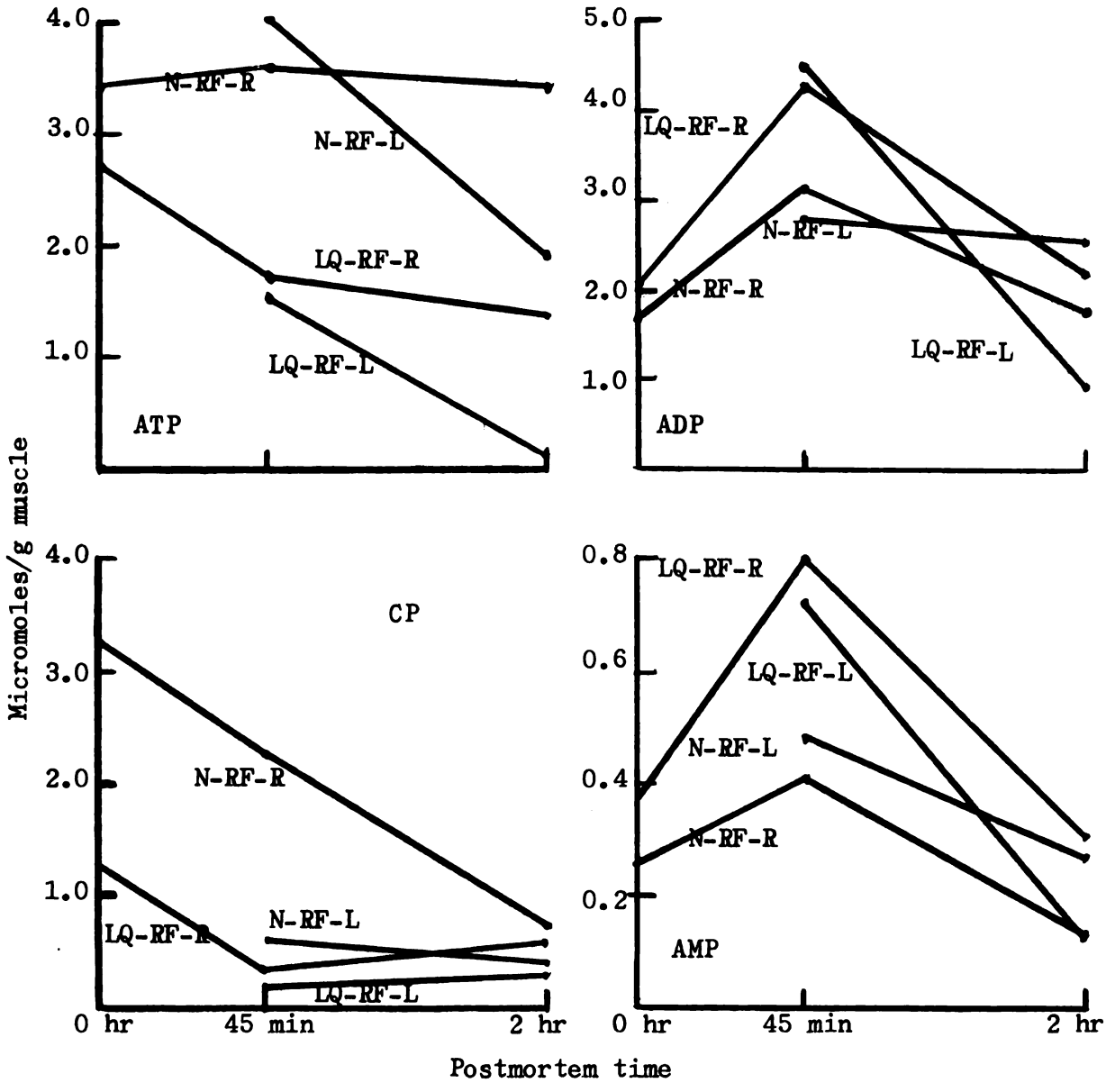

Figure 7. The effect of 0 hr sample excision on postmortem levels of ATP, ADP, AMP and creatine phosphate between normal (N) and low quality (LQ) rectus femoris muscle (RF-R = right rectus femoris; RF-L = left rectus femoris).

this same postmortem time period. The RF-L muscles showed a definite decrease in ATP levels from 45 min to 2 hr postmortem. The ATP levels of low quality RF-L muscles were almost completely diminished at 2 hr postmortem.

The CP levels (Figure 7) of the normal RF-R samples remained higher than those of the other muscle samples and they decreased steadily from 0 hr to 2 hr postmortem. The CP levels of the low quality RF-R muscles gradually decreased from 0 hr to 45 min and remained relatively constant until 2 hr postmortem. The CP levels of the normal and low quality RF-L samples were low and remained essentially the same between 45 min and 2 hr postmortem.

The ADP levels (Figure 7) increased from 0 hr to 45 min and then decreased from 45 min to 2 hr postmortem in all samples. Low quality RF-R muscles consistently had higher ADP levels than the normal RF-R samples. The low quality RF-L muscles had higher ADP levels at 45 min postmortem and lower levels at 2 hr than all other muscle samples; whereas the normal RF-L samples had lower levels at 45 min and higher levels at 2 hr than all other muscle samples.

The AMP levels (Figure 7) increased from 0 hr to 45 min postmortem and then decreased in all muscle samples from 45 min to 2 hr postmortem. Low quality RF-R muscle samples consistently had higher AMP levels than the normal RF-R muscles. The low quality RF-L muscles had higher AMP values at 45 min and slightly lower levels at 2 hr postmortem than the normal RF-L muscle samples. While the postmortem curves of AMP and ADP were similar, levels of both of these nucleotides showed a more marked change between 45 min and 2 hr postmortem in low quality than in normal RF-L muscles.

From Figure 5 it can be seen that normal muscle samples had slightly higher 24 hr pH values than low quality RF muscles and that the RF-L 24

hr pH values tended to be lower than corresponding RF-R values. Table 22 summarizes the 24 hr levels of the metabolites studied. Normal muscles had slightly more residual glycogen, especially among the RF-L samples, and less lactate than the low quality muscle samples. Additionally, the RF-L muscles accumulated more lactate than the RF-R muscles especially among the low quality samples. The 24 hr G-1-P and G-6-P levels were higher among the normal than the low quality muscle samples. The RF-R muscles tended to have higher levels of G-1-P and G-6-P than the RF-L muscles. Glucose levels were higher among low quality muscle samples than in normal muscles and the RF-L muscles contained slightly more glucose than the RF-R samples. The ATP and CP levels were slightly higher in normal than in low quality muscle samples at 24 hr postmortem; whereas, low quality samples had higher levels of ADP and AMP than normal muscles.

From the data presented in Tables 16-22 and Figures 5-7, it is apparent that pH decline and glycogen degradation paralleled the postmortem ATP diminution. Although lactate levels were inversely related to the above metabolites, the accumulation of lactate was also proportional to pH fall and the reduction of glycogen and ATP. The ATP levels apparently played a role in the control of glycolysis among the RF muscles similar to that previously discussed for the LD muscles. The higher ADP and AMP levels in the low quality muscles, at least until 45 min postmortem, in all probability enhanced the glycolytic rate among these muscles.

With the introduction of the temperature variable in the RF muscles, it was not possible to determine if sample excision, at or shortly after

Table 22. The levels of some glycolytic metabolites in normal and low quality rectus femoris muscle at 24 hr postmortem.[1]

	Quality group			
	Normal		Low quality	
Metabolite	Right rectus femoris	Left rectus femoris	Right rectus femoris	Left rectus femoris
Glycogen[2]	3.3	3.4	3.1	2.2
Lactate	68.8	69.7	71.1	75.0
G-1-P	1.22	1.09	0.94	0.80
G-6-P	5.29	4.40	4.82	3.83
F-6-P	2.30	1.95	2.48	2.00
Glucose	3.18	3.74	4.36	4.60
ATP	0.17	0.09	0.07	0.03
CP	0.19	0.22	0.12	0.10
ADP	0.66	0.47	1.60	1.54
AMP	0.12	0.16	0.16	0.22

[1]Levels expressed as micromoles/g muscle.
[2]Glycogen levels expressed as micromoles glucose equivalents/g muscle.

exsanguination, affected the RF muscle similarly to that previously described for the LD muscle. Levels of glycolytic metabolites and the pH values at 45 min tend to indicate that the RF-R exhibited slightly faster rates of glycolysis than the RF-L at this postmortem time period. However, much of the difference was, in all probability, nullified because of the lower muscle temperatures already existing in the RF-R than in the RF-L muscles at 45 min postmortem.

A Comparison of Postmortem Differences between Several Porcine Muscles within the Same Carcass

Briskey (1964) suggested that some muscles are more resistant to the development of PSE conditions than others. He attributed this muscle difference to variation in cooling rates or oxygen-retaining capacities between muscles. From observations of the carcasses in Groups I, II and III of this study, it appeared that most, if not all, of the muscles of a low quality carcass were affected when rapid glycolysis occurred in the LD muscle. Thus, in addition to the LD and RF, two other muscles, i.e., the biceps femoris (BF) and supraspinatus (SS) were sampled (0, 1, 2 and 24 hr postmortem) from the carcasses in Group IV to study some qualitative properties. The six most "normal" and the six "lowest quality" carcasses of the pigs in Group IV were compared. The basis for categorization of these 12 carcasses into the two quality groups included the combination of transmission values, rate of postmortem pH declines and subjective quality scores of the LD muscle only.

Transmission values and 2 hr pH values of each muscle were compared between normal and low quality carcasses (Table 23). It was decided to use 2 hr pH values rather than the pH at some other postmortem time period, because the transmission values of both LD muscles of the pigs in Group IV were more highly correlated with 2 hr pH than with 45 min pH ($r = -.69$ and $r = -.49$, respectively). To justify this categorization of the carcasses into normal and low quality groups, normal LD muscles had significantly ($P < .01$) lower transmission values and higher 2 hr pH values than low quality LD muscles. There was a definite trend for all of the muscles

Table 23. Transmission values and 2 hr postmortem pH values of several muscles from normal and low quality carcasses.

Muscle	Transmission value		pH (2 hr postmortem)	
	Normal	Low quality	Normal	Low quality
Right longissimus[1]	13.6	66.2	5.86	5.42
Left longissimus[1]	12.9	56.5	6.15	5.56
Right rectus femoris	10.4	15.4		
Left rectus femoris[2]	9.7	20.7		
Biceps femoris[3]	8.1	14.7	6.45	6.04
Supraspinatus	13.6	20.3	6.31	6.22

[1]Means were significantly different ($P < .01$) for both transmission and 2 hr pH values.
[2]Means were significantly different ($P < .05$) for transmission value only.
[3]Means were significantly different ($P < .05$) and ($P < .01$) for transmission values and 2 hr pH values, respectively.

studied (BF, SS, RF-R and RF-L) to have lower transmission values and higher 2 hr pH values among normal carcasses than those of low quality carcasses. Normal BF muscles had significantly ($P < .05$) lower transmission values and higher ($P < .01$) 2 hr pH values than the low quality BF muscles. The normal RF-L muscles had significantly ($P < .05$) lower transmission values than the low quality RF-L muscles. Because of the sampling procedure used, there was an insufficient number of 2 hr pH values to allow for an evaluation of the RF muscle. However, from the postmortem pH patterns shown in Figure 5, it is apparent that the low quality RF muscles had more rapid pH declines than normal RF muscles.

Simple correlation coefficients for transmission and 2 hr pH values of the LD-L muscles with those of the LD-R, RF-L, RF-R, BF and SS muscles

from all of the carcasses (22) of Group IV were calculated (Table 24). Although low, correlations of 2 hr LD-L pH with 2 hr pH values of the LD-R, BF and SS were significant. Correlations between transmission values of the LD-L and those of the LD-R, RF-R and BF were also significant ($P < .01$), but low. Thus, it appears that none of the muscles studied (BF, RF and SS) was entirely resistant to rapid rates of postmortem glycolysis or development of low quality muscle characteristics when the LD of a given carcass was affected. The BF and SS muscle incision and exposure (cooling) shortly after exsanguination most likely affected these muscles similarly to the sampling effects found in the LD and RF muscles.

Table 24. Simple correlation coefficients for transmission values and 2 hr postmortem pH values between some muscles.[1,2]

Transmission values		2 hr postmortem pH values	
Muscle	Left longissimus	Muscle	Left longissimus
Right longissimus	0.64	Right longissimus	0.65
Left rectus femoris	0.35		
Right rectus femoris	0.67		
Biceps femoris	0.67	Biceps femoris	0.58
Supraspinatus	0.40	Supraspinatus	0.47

[1]Correlation coefficients > 0.423 are significant ($P < .05$).
[2]Correlation coefficients > 0.537 are significant ($P < .01$).

SUMMARY

The results of this study were obtained from 146 market-weight pigs slaughtered in four different groups. The distribution of red, white and intermediate muscle fibers, succinic dehydrogenase (SDH) activity, myoglobin, total lipid, phospholipid and glyceride ester (neutral lipid fraction) levels were determined on longissimus (LD) muscle samples obtained from the pigs in Group I at or shortly after exsanguination. The relationship of these parameters to rates of pH decline and subjective quality scores of the ultimate muscle properties was observed on normal and low quality LD muscles from the three breeds of pigs included in Group I. Heart weights of the pigs in Group I, III and IV were recorded and their relationship to rate of postmortem pH decline and/or 24 hr transmission values observed. Muscle or rectal temperatures were obtained on the pigs in Groups II, III and IV at the time of exsanguination and at 45 min postmortem and their relationships to 45 min pH, transmission values and subjective quality scores was also observed. The effects of sample excision, at or shortly after exsanguination, upon pH and transmission values and glycogen, glucose-6-phosphate, lactate, ATP and creatine phosphate (CP) levels from the LD and rectus femoris (RF) muscles were studied for the pigs included in Group IV. In addition to the above observations for Group IV pigs, glucose-1-phosphate, fructose-6-phosphate, glucose, ADP and AMP levels were compared among normal and low quality LD and RF muscle samples excised at several postmortem time periods. Transmission values and 2 hr postmortem pH values of the LD, RF, biceps femoris (BF) and supraspinatus (SS) muscles from the pigs in Group IV were compared.

Normal LD muscles had more red and fewer white muscle fibers, higher SDH activities and greater total myoglobin contents than low quality LD muscles. The fiber size of red and intermediate muscle fiber types was larger in low quality than in normal LD muscle. Normal LD muscles tended to have higher total lipid levels and greater glyceride ester contents of the neutral lipid fraction than those of the low quality LD muscles. Landrace pigs tended to have more myoglobin and higher SDH activities than Poland Chinas or Chester Whites, while Poland China pigs tended to have more red fibers than the other two breeds.

Heart weights of the low quality pigs in Group I tended to be lighter than those of the normal pigs. However, no significant correlations were obtained between heart weights and either 45 min pH (Groups I and IV) or transmission values (Groups III and IV). Observations from several pigs which had pericarditis indicated that heart function may be more important than heart weight in contributing toward development of low quality porcine musculature.

Muscle (LD) temperature at 45 min postmortem was found to be more highly related (negatively) to ultimate muscle quality indices than muscle (Groups III and IV) or rectal (Group II) temperature at the time of exsanguination. While no identification of the factor(s) contributing to the differences in LD temperatures was made in this study, in all likelihood, the in vivo temperatures at the time of exsanguination, as well as scalding, slaughter floor temperatures, and time lapse before carcass chilling contributed to postmortem muscle temperatures.

Muscle (LD) incision of the pigs in Group IV, at or shortly after the time of exsanguination, stimulated contractile activity, significantly

increased the rate of postmortem glycolysis and tended to decrease ultimate muscle qualitative characteristics. The LD muscles incised at the time of exsanguination had lower pH values, glycogen, ATP and CP levels and higher lactate contents at corresponding time periods through 2 hr postmortem, than LD muscles not incised until 45 min after exsanguination. This effect of muscle incision was greater among the normal than low quality LD muscles.

Excision of RF muscle samples involved removal of skin and subcutaneous tissues thus exposing the RF muscles to the atmosphere. The chilling effect resulting from exposure of the RF muscles tended to slow down glycolytic rates and apparently even nullified the effects of contractile activity resulting from muscle incision at an early postmortem time (0 to 15 min). The RF muscles not incised until 2 hr postmortem exhibited significantly lower pH values and levels of glycogen and ATP and higher lactate contents than RF muscles incised at or shortly after the time of exsanguination. The effect of chilling associated with early postmortem incision of the RF muscles was greater among the low quality than normal muscle samples. Postmortem levels of all the metabolites studied appeared to be related to the glycolytic rate in both the RF and LD muscles.

Transmission and 2 hr postmortem pH values of the RF, BF and SS muscles showed low, but significant correlations with those of the LD muscles. The data indicated that the postmortem changes in these muscles within a given porcine carcass tended to parallel each other.

Since the results of this study indicate that muscle sample excision, at or shortly after exsanguination, markedly altered subsequent postmortem characteristics, these effects must be recognized and taken into account when the postmortem changes in porcine muscle are to be studied. Additionally, consideration must be given to the ante- and/or postmortem environmental conditions as well as breed effects upon muscle qualitative properties for studies of this nature.

BIBLIOGRAPHY

Aberle, E. D. and R. A. Merkel. 1968a. 5'-adenylic acid deaminase in porcine muscle. J. Food Sci. 33:27.

Aberle, E. D. and R. A. Merkel. 1968b. Physical and biochemical properties of porcine muscle as affected by exogenous epinephrine and prednisolone. J. Food Sci. 33:43.

Addis, P. B., H. R. Johnson, C. J. Heidenreich, H. W. Jones and M. D. Judge. 1967a. Effect of humidity level in a warm growing environment on porcine carcass composition and quality. J. Animal Sci. 26:705.

Addis, P. B., H. R. Johnson, N. W. Thomas and M. D. Judge. 1967b. Effect of temperature acclimation on porcine physiological responses to heat stress and associated properties of muscle. J. Animal Sci. 26:466.

Allen, E., J. C. Forrest, A. B. Chapman, N. First, R. W. Bray and E. J. Briskey. 1966. Phenotypic and genetic associations between porcine muscle properties. J. Animal Sci. 25:962.

AOAC. 1965. Official Methods of Analysis. 10th ed. p. 346. Assoc. of Agri. Chemists, Washington, D. C.

Atkinson, D. E. 1966. Regulation of enzyme activity. Ann. Rev. Biochem. 35:85.

Barany, M., A. F. Turci, K. Barany, A. Volpe and T. Reckard. 1965a. Myosin of newborn rabbits. Arch. Biochem. Biophys. 111:727.

Barany, M., K. Barany, T. Reckard and A. Volpe. 1965b. Myosin of fast and slow muscles of the rabbit. Arch. Biochem. Biophys. 109:185.

Bates, M. W. 1958. Turnover rates of fatty acids of plasma triglyceride, cholesterol ester and phospholipid in the postabsorptive dog. Am. J. Physiol. 194:446.

Beatty, C. H., R. D. Peterson and R. M. Bocek. 1963. Metabolism of red and white muscle fiber groups. Am. J. Physiol. 204:939.

Beatty, C. H., G. M. Basinger, C. C. Dully and R. M. Bocek. 1966. Comparison of red and white voluntary skeletal muscles of several species of primates. J. Histochem. Cytochem. 14:590.

Beecher, G. R., E. J. Briskey and W. G. Hoekstra. 1965a. A comparison of glycolysis and associated changes in light and dark portions of the porcine semitendinosus. J. Food Sci. 30:477.

Beecher, G. R., R. G. Cassens, W. G. Hoekstra and E. J. Briskey. 1965b. Red and white fiber content and associated post-mortem properties of seven porcine muscles. J. Food Sci. 30:969.

Beecher, G. R., L. L. Kastenschmidt, R. G. Cassens, W. G. Hoekstra and E. J. Briskey. 1968. A comparison of the light and dark portions of a striated muscle. J. Food Sci. 33:84.

Bendall, J. R. 1960. Post-mortem changes in muscle. In Structure and Function of Muscle. Ed., G. H. Bourne. Vol. III, p. 227. Academic Press, New York.

Bendall, J. R. and J. Wismer-Pedersen. 1962. Some properties of the fibrillar proteins of normal and watery pork muscle. J. Food Sci. 27:144.

Bendall, J. R. 1963. Physiology and chemistry of muscle. p. 33. Proc. Meat Tenderness Symposium. Campbell Soup Co., Camden, N. J.

Bendall, J. R., O. Hallund and J. Wismer-Pedersen. 1963. Post-mortem changes in the muscles of Landrace pigs. J. Food Sci. 28:156.

Bendall, J. R. 1964. Meat proteins. In Proteins and Their Reactions. Eds., H. W. Schultz and A. F. Anglemier. p. 225. AVI Publishing Co., Inc., Westport, Conn.

Bendall, J. R. 1966a. Muscle as a contractile machine. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 7. University of Wisconsin Press, Madison.

Bendall, J. R. 1966b. The effect of pre-treatment of pigs with curare on the post-mortem rate of pH fall and onset of rigor mortis in the musculature. J. Sci. Food Agric. 17:333.

Beveridge, J. M. R. and S. E. Johnson. 1949. The determination of phospholipid phosphorus. Canadian J. Res. 27E:159.

Blanchaer, M. C., M. Van Wijhe and D. Mozersky. 1963. The oxidation of lactate and α-glycerophosphate by red and white skeletal muscle. I. Quantitative studies. J. Histochem. Cytochem. 11:500.

Bodwell, C. E., A. M. Pearson, J. Wismer-Pedersen and L. J. Bratzler. 1966. Post-mortem changes in muscle. II. Chemical and physical changes in pork. J. Food Sci. 31:1.

Bonner, W. D. 1955. Succinic dehydrogenase. In Methods in Enzymology. Eds., S. P. Colowick and N. O. Kaplan. Vol. I. p.722. Academic Press, Inc., New York.

Borchert, L. L. and E. J. Briskey. 1964. Prevention of pale, soft, exudative porcine muscle through partial freezing with liquid nitrogen post-mortem. J. Food Sci. 29:203.

Borchert, L. L. and E. J. Briskey. 1965. Protein solubility and associated properties of porcine muscle as influenced by partial freezing with liquid nitrogen. J. Food Sci. 30:138.

Bray, R. W. 1968. Variation of quality and quantity factors within and between breeds. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. P. 136. Iowa State Univ. Press, Ames, Iowa.

Briskey, E. J., R. W. Bray, W. G. Hoekstra, R. H. Grummer and P. H. Phillips. 1959a. The effect of various levels of exercise in altering the chemical and physical characteristics of certain pork ham muscles. J. Animal Sci. 18:153.

Briskey, E. J., R. W. Bray, W. G. Hoekstra, P. H. Phillips and R. H. Grummer. 1959b. The chemical and physical characteristics of various pork ham muscles. J. Animal Sci. 18:146.

Briskey, E. J., R. W. Bray, W. G. Hoekstra, P. H. Phillips and R. H. Grummer. 1959c. The effect of exhaustive exercise and high sucrose regimen on certain chemical and physical pork ham muscle characteristics. J. Animal Sci. 18:173.

Briskey, E. J., R. W. Bray, W. G. Hoekstra, P. H. Phillips and R. H. Grummer. 1960a. Effect of high protein, high fat, and high sucrose rations on the water-binding and associated properties of pork muscle. J. Animal Sci. 19:404.

Briskey, E. J., W. G. Hoekstra, R. W. Bray and R. H. Grummer, 1960b. A comparison of certain physical and chemical characteristics of eight pork muscles. J. Animal Sci. 19:214.

Briskey, E. J. and J. Wismer-Pedersen. 1961a. Biochemistry of pork muscle structure. I. Rate of anaerobic glycolysis and temperature change versus the apparent structure of muscle tissue. J. Food Sci. 26:297.

Briskey, E. J. and J. Wismer-Pedersen. 1961b. Biochemistry of pork muscle structure. II. Preliminary observations of biopsy samples versus ultimate muscle structure. J. Food Sci. 26:306.

Briskey, E. J., R. N. Sayre and R. G. Cassens. 1962. Development and application of an apparatus for continuous measurement of muscle extensibility and elasticity before and during rigor mortis. J. Food Sci. 27:560.

Briskey, E. J. 1963. Influence of ante- and post-mortem handling practices on properties of muscle which are related to tenderness. p. 195. Proc. Meat Tenderness Symposium. Campbell Soup Co., Camden, N. J.

Briskey, E. J. 1964. Etiological status and associated studies of pale, soft, exudative porcine musculature. Adv. Food Res. 13:89.

Briskey, E. J. and R. N. Sayre. 1964. Muscle protein extractability as influenced by conditions of post-mortem glycolysis. Proc. Soc. Exptl. Biol. Med. 115:823.

Briskey, E. J., L. L. Kastenschmidt, J. C. Forrest, G. R. Beecher, M. D. Judge, R. G. Cassens and W. G. Hoekstra. 1966. Biochemical aspects of post-mortem changes in porcine muscle. J. Agr. Food Chem. 14:201.

Briskey, E. J. 1967. Myofibrillar proteins of skeletal muscle. Proc. of the 19th Res. Conf. Am. Meat Inst. Found. p. 1.

Briskey, E. J. and D. Lister. 1968. Influence of stress syndrome on chemical and physical characteristics of muscle post-mortem. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 177. Iowa State Univ. Press, Ames, Iowa.

Brooke, M. H. 1966. The histological reaction of muscle to disease. In The Physiology and Biochemistry of Muscle as a Food. Eds. E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 113. University of Wisconsin Press, Madison.

Broumand, H., C. O. Ball and E. J. Stier. 1958. Factors affecting the quality of prepackaged meat. II. E. Determining the proportions of heme derivatives in fresh meat. Food Technol. 12:65.

Buller, A. J., J. C. Eccles and R. M. Eccles. 1960. Differentiation of fast and slow muscles in the cat hind limb. J. Physiol. 150:399.

Buller, A. J. and D. M. Lewis. 1965. Further observations on mammalian cross-innervated skeletal muscle. J. Physiol. 178:343.

Carrow, R. E., R. E. Brown and W. D. Van Huss. 1967. Fiber size and capillary to fiber ratios in skeletal muscle of exercised rats. Anat. Rec. 159:33.

Cassens, R. G., E. J. Briskey and W. G. Hoekstra. 1963a. Electron microscopy of post-mortem changes in porcine muscle. Nature 197:1119.

Cassens, R. G., E. J. Briskey and W. G. Hoekstra. 1963b. Electron microscopy of post-mortem changes in porcine muscle. J. Food Sci. 28:680.

Cassens, R. G., E. J. Briskey and W. G. Hoekstra. 1963c. Relation of pork muscle quality factors to zinc content and other properties. Food Technol. 17:4.

Cassens, R. G., E. J. Briskey and W. G. Hoekstra. 1963d. Variation in zinc content and other properties of various porcine muscles. J. Sci. Food Agr. 14:427.

Cassens, R. G., M. D. Judge, J. D. Sink and E. J. Briskey. 1965. Porcine adrenocortical lipids in relation to striated muscle characteristics. Proc. Soc. Exptl. Biol. Med. 120:854.

Cassens, R. G. 1966. General aspects of post-mortem changes. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 181. University of Wisconsin Press, Madison.

Choudhury, R. B. R. and L. K. Arnold. 1960. The determination of the neutral oil content of crude vegetable oils. J. Am. Oil Chem. Soc. 37:87.

Christian, L. L. 1968. Limits for rapidity of genetic improvement for fat, muscle, and quantitative traits. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 154. Iowa State Univ. Press, Ames, Iowa.

Clausen, H. and R. N. Thomsen. 1960. Report on investigations with pigs. Natl. Research Inst. on Animal Husbandry, Copenhagen. Rept. No. 317.

Close, R. 1964. Dynamic properties of fast and slow skeletal muscles of the rat during development. J. Physiol. 173:74.

Close, R. 1965. Effects of cross-union of motor nerves to fast and slow skeletal muscles. Nature 206:831.

Conn, E. E. and P. K. Stumpf. 1966. Outlines of Biochemistry. 2nd ed. p. 402. John Wiley and Sons, Inc., New York.

Copenhaver, W. M. 1964. Bailey's Textbook of Histology. 15th ed. p. 160. Williams and Wilkins Co., Baltimore.

Dahl, O. 1962. Amino acid composition of normal and degenerated pig muscle. J. Food Sci. 27:5.

Davies, R. E. 1967. Recent theories on the mechanism of muscle contraction and rigor mortis. Proc. of the 19th Res. Conf. Am. Meat Inst. Found. p. 39.

Dawson, D. M. and F. C. A. Romanul. 1964. Enzymes in muscle. II. Histochemical and quantitative studies. Arch. Neurol. 11:369.

Dubowitz, V. and A. G. E. Pearse. 1961. Enzymatic activity of normal and dystrophic human muscle: A histochemical study. J. Pathol. Bacteriol. 81:365.

Cassens, R. G., E. J. Briskey and W. G. Hoekstra. 1963d. Variation in zinc content and other properties of various porcine muscles. J. Sci. Food Agr. 14:427.

Cassens, R. G., M. D. Judge, J. D. Sink and E. J. Briskey. 1965. Porcine adrenocortical lipids in relation to striated muscle characteristics. Proc. Soc. Exptl. Biol. Med. 120:854.

Cassens, R. G. 1966. General aspects of post-mortem changes. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 181. University of Wisconsin Press, Madison.

Choudhury, R. B. R. and L. K. Arnold. 1960. The determination of the neutral oil content of crude vegetable oils. J. Am. Oil Chem. Soc. 37:87.

Christian, L. L. 1968. Limits for rapidity of genetic improvement for fat, muscle, and quantitative traits. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 154. Iowa State Univ. Press, Ames, Iowa.

Clausen, H. and R. N. Thomsen. 1960. Report on investigations with pigs. Natl. Research Inst. on Animal Husbandry, Copenhagen. Rept. No. 317.

Close, R. 1964. Dynamic properties of fast and slow skeletal muscles of the rat during development. J. Physiol. 173:74.

Close, R. 1965. Effects of cross-union of motor nerves to fast and slow skeletal muscles. Nature 206:831.

Conn, E. E. and P. K. Stumpf. 1966. Outlines of Biochemistry. 2nd ed. p. 402. John Wiley and Sons, Inc., New York.

Copenhaver, W. M. 1964. Bailey's Textbook of Histology. 15th ed. p. 160. Williams and Wilkins Co., Baltimore.

Dahl, O. 1962. Amino acid composition of normal and degenerated pig muscle. J. Food Sci. 27:5.

Davies, R. E. 1967. Recent theories on the mechanism of muscle contraction and rigor mortis. Proc. of the 19th Res. Conf. Am. Meat Inst. Found. p. 39.

Dawson, D. M. and F. C. A. Romanul. 1964. Enzymes in muscle. II. Histochemical and quantitative studies. Arch. Neurol. 11:369.

Dubowitz, V. and A. G. E. Pearse. 1961. Enzymatic activity of normal and dystrophic human muscle: A histochemical study. J. Pathol. Bacteriol. 81:365.

Edgerton, V. R., L. Gerchman and R. Carrow. 1969. Histochemical changes in rat skeletal muscle after exercise. Exptl. Neurol. (In press).

Engelhardt, W. V. 1966. Swine cardiovascular physiology - a review. In Swine in Biomedical Research. Eds., L. K. Bustad and R. O. McClellan. p. 307. Pacific Northwest Laboratory, Richland, Wash.

Forrest, J. C., R. F. Gundlach and E. J. Briskey. 1963. A preliminary survey of the variations in certain pork ham muscle characteristics. Proc. of the 15th Res. Conf. Am. Meat Inst. Found. p. 81.

Forrest, J. C. 1965. Porcine physiology as related to post-mortem muscle properties. Proc. Recip. Meat Conf. 18:270.

Forrest, J. C., L. L. Kastenschmidt, G. R. Beecher, R. H. Grummer, W. G. Hoekstra and E. J. Briskey. 1965. Porcine muscle properties. B. Relation to naturally occurring and artificially induced variation in heart and respiration rates. J. Food Sci. 30:492.

Forrest, J. C., M. D. Judge, J. D. Sink, W. G. Hoekstra and E. J. Briskey. 1966. Prediction of the time course of rigor mortis through response of muscle tissue to electrical stimulation. J. Food Sci. 31:13.

Forrest, J. C. and E. J. Briskey. 1967. Response of striated muscle to electrical stimulation. J. Food Sci. 32:483.

Forrest, J. C., J. A. Will, G. R. Schmidt, M. D. Judge and E. J. Briskey. 1968. Homeostasis in animals (Sus domesticus) during exposure to a warm environment. J. Applied Physiol. 24:33.

Fritz, I. B., D. G. Davis, R. H. Holtrap and H. Dundee. 1958. Fatty acid oxidation by skeletal muscle during rest and activity. Am. J. Physiol. 194:379.

George, J. C. and A. J. Berger. 1966. Avian Myology. p. 25. Academic Press, New York.

Goddu, R. F., N. F. LeBlanc and C. M. Wright. 1955. Spectrophotometric determination of esters and anhydrides by hydroxamic acid reaction. Anal. Chem. 27:1251.

Guth, L. 1968. "Trophic" influences of nerve on muscle. Physiol. Rev. 48:645.

Hallund, O. and J. R. Bendall. 1965. The long-term effect of electrical stimulation on the post-mortem fall of pH in the muscles of Landrace pigs. J. Food Sci. 30:296.

Hamm, R. 1960. Biochemistry of meat hydration. Adv. Food Res. 10:355.

Hart, P. C. 1962a. Physico-chemical characteristics of degenerated meat in pigs. Tijdschr. Diergeneesk 87:3.

Hart, P. C. 1962b. The transmission value, a method for meat quality evaluation. Research Institute for Animal Husbandry "Schoonoord". Zeist. The Netherlands. (mimeograph)

Hedrick, H. B., M. E. Bailey, F. C. Parrish and D. A. Naumann. 1963. Effect of adrenaline stress on pork quality. J. Animal Sci. 22:827 (abstr.).

Helander, E. 1966. General considerations of muscle development. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 19. University of Wisconsin Press, Madison.

Helmreich, E. and C. F. Cori. 1965. Regulation of glycolysis in muscle. Adv. Enz. Regulation. 3:91.

Henneman, E. and C. B. Olson. 1965. Relations between structure and function in the design of skeletal muscles. J. Neurophysiol. 28:581.

Henry, M., J. Billon and G. Haouza. 1955. Contribution à l 'etude de l acidose des viandes der porc, dites exsudatives. Rev. Pathol. Gén Comparée. 55:857.

Hoernicke, H. 1966. Review of the Berlin symposium on swine circulatory system. In Swine in Biomedical Research. Eds. L. K. Bustad and R. O. McClellan. p. 419. Pacific Northwest Laboratory, Richland, Wash.

Hohorst, H. J. 1963. L-(+)-lactate. Determination with lactic dehydrogenase and DPN. In Methods of Enzymatic Analysis. Ed., H. V. Bergmeyer. p. 266. Academic Press Inc., New York.

Holloszy, J. O. 1967. Biochemical adaptations in muscle. Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. J. Biol. Chem. 242:2278.

Howe, J. M., N. W. Thomas, P. B. Addis and M. D. Judge. 1968. Temperature acclimation and its effects on porcine muscle properties in two humidity environments. J. Food Sci. 33:235.

Howe, J. M., P. B. Addis, R. D. Howard and M. D. Judge. 1969. Environment-induced adrenocortical lipid in "stress-susceptible" pigs. J. Animal Sci. 28:70.

Issekutz, B. Jr., H. I. Miller, P. Paul and K. Rodahl. 1964. Source of fat oxidation in exercising dogs. Am. J. Physiol. 207:583.

Judge, M. D., V. R. Cahill, L. E. Kunkle and W. H. Bruner. 1959. Pork quality. I. Influences of some factors on pork muscle characteristics. J. Animal Sci. 18:448.

Hart, P. C. 1962b. The transmission value, a method for meat quality evaluation. Research Institute for Animal Husbandry "Schoonoord". Zeist. The Netherlands. (mimeograph)

Hedrick, H. B., M. E. Bailey, F. C. Parrish and D. A. Naumann. 1963. Effect of adrenaline stress on pork quality. J. Animal Sci. 22:827 (abstr.).

Helander, E. 1966. General considerations of muscle development. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 19. University of Wisconsin Press, Madison.

Helmreich, E. and C. F. Cori. 1965. Regulation of glycolysis in muscle. Adv. Enz. Regulation. 3:91.

Henneman, E. and C. B. Olson. 1965. Relations between structure and function in the design of skeletal muscles. J. Neurophysiol. 28:581.

Henry, M., J. Billon and G. Haouza. 1955. Contribution à l 'etude de l acidose des viandes der porc, dites exsudatives. Rev. Pathol. Gén Comparee. 55:857.

Hoernicke, H. 1966. Review of the Berlin symposium on swine circulatory system. In Swine in Biomedical Research. Eds. L. K. Bustad and R. O. McClellan. p. 419. Pacific Northwest Laboratory, Richland, Wash.

Hohorst, H. J. 1963. L-(+)-lactate. Determination with lactic dehydrogenase and DPN. In Methods of Enzymatic Analysis. Ed., H. V. Bergmeyer. p. 266. Academic Press Inc., New York.

Holloszy, J. O. 1967. Biochemical adaptations in muscle. Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. J. Biol. Chem. 242:2278.

Howe, J. M., N. W. Thomas, P. B. Addis and M. D. Judge. 1968. Temperature acclimation and its effects on porcine muscle properties in two humidity environments. J. Food Sci. 33:235.

Howe, J. M., P. B. Addis, R. D. Howard and M. D. Judge. 1969. Environment-induced adrenocortical lipid in "stress-susceptible" pigs. J. Animal Sci. 28:70.

Issekutz, B. Jr., H. I. Miller, P. Paul and K. Rodahl. 1964. Source of fat oxidation in exercising dogs. Am. J. Physiol. 207:583.

Judge, M. D., V. R. Cahill, L. E. Kunkle and W. H. Bruner. 1959. Pork quality. I. Influences of some factors on pork muscle characteristics. J. Animal Sci. 18:448.

Judge, M. D., E. J. Briskey and R. K. Meyer. 1966. Endocrine related post-mortem changes in porcine muscle. Nature 212:287.

Judge, M. D., R. G. Cassens and E. J. Briskey. 1967. Muscle properties of physically restrained stressor-susceptible and stressor-resistant porcine animals. J. Food Sci. 32:565.

Judge, M. D. 1968. Influence of controlled environmental temperature and humidity. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 187. Iowa State Univ. Press, Ames, Iowa.

Judge, M. D., E. J. Briskey, R. G. Cassens, J. C. Forrest and R. K. Meyer. 1968a. Adrenal and thyroid function in stress-susceptible pigs (Sus domesticus). Am. J. Physiol. 214:146.

Judge, M. D., J. C. Forrest, J. D. Sink and E. J. Briskey. 1968b. Endocrine related stress responses and muscle properties of swine. J. Animal Sci. 27:1247.

Karpati, G. and W. K. Engel. 1968. "Type grouping" in skeletal muscles after experimental reinnervation. Neurol. 18:447.

Karpatkin, S., E. Helmreich and C. F. Cori. 1964. Regulation of glycolysis in muscle. II. Effect of stimulation and epinephrine in isolated frog sartorius muscle. J. Biol. Chem. 239:3139.

Kastenschmidt, L. L., E. J. Briskey and W. G. Hoekstra. 1964. Prevention of pale, soft, exudative porcine muscle through regulation of antemortem environmental temperature. J. Food Sci. 29:210.

Kastenschmidt, L. L., G. R. Beecher, J. C. Forrest, W. G. Hoekstra and E. J. Briskey. 1965. Porcine muscle properties. A. Alteration of glycolysis by artificially induced changes in ambient temperature. J. Food Sci. 30:565.

Kastenschmidt, L. L., E. J. Briskey and W. G. Hoekstra. 1966. Metabolic intermediates in skeletal muscles with fast and slow rates of post-mortem glycolysis. Nature 212:288.

Kastenschmidt, L. L., W. G. Hoekstra and E. J. Briskey. 1968. Glycolytic intermediates and co-factors in "fast-" and "slow-glycolyzing" muscles of the pig. J. Food Sci. 33:151.

Kjolberg, O., D. J. Manners and R. A. Lawrie. 1963. The molecular structure of some pig muscle glycogens. Biochem. J. 87:351.

Krzywicki, K. and P. W. Ratcliff. 1967. The phospholipids of pork muscle and their relation to the post-mortem rate of glycolysis. J. Sci. Food Agr. 18:252.

Lands, W. E. M. 1958. Metabolism of glycerolipides: A comparison of lecithin and triglyceride synthesis. J. Biol. Chem. 231:883.

Lasley, J. F. 1968. Relationship of breeding and reproduction to carcass quality and quantity characteristics. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 145. Iowa State Univ. Press, Ames, Iowa.

Lawrie, R. A., D. P. Gatherum and H. P. Hale. 1958. Abnormally low ultimate pH in pig muscle. Nature 182:807.

Lawrie, R. A. 1960. Post-mortem glycolysis in normal and exudative longissimus dorsi muscles of the pig in relation to so-called white muscle disease. J. Comp. Pathol. Therap. 70:273.

Lawrie, R. A. and D. P. Gatherum. 1962. Studies on the muscles of meat animals. II. Differences in the ultimate pH and pigmentation of longissimus dorsi muscles from two breeds of pigs. J. Agric. Sci. 58:97.

Lawrie, R. A. and R. W. Pomeroy. 1963. Sodium and potassium in pig muscle. J. Agric. Sci. 61:409.

Lawrie, R. A., R. W. Pomeroy and A. Cuthbertson. 1963. Studies on the muscles of meat animals. III. Comparative composition of various muscles in pigs of three weight groups. J. Agric. Sci. 60:195.

Lawrie, R. A. 1966a. Meat Science. Pergamon Press, New York.

Lawrie, R. A. 1966b. Metabolic stresses which affect muscle. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 137. University of Wisconsin Press, Madison.

Lewis, P. K., C. J. Brown and M. C. Heck. 1959. The effect of periodic electric shock prior to slaughter on the eating quality of fresh and cured pork. J. Animal Sci. 18:1477. (abstr.).

Lewis, P. K., M. C. Heck and C. J. Brown. 1961. Effect of stress from electrical stimulation and sugar on the chemical composition of swine carcasses. J. Animal Sci. 20:727.

Lewis, P. K., C. J. Brown and M. C. Heck. 1967. Effect of ante-mortem stress and freezing immediately after slaughter on certain organoleptic and chemical characteristics of pork. J. Animal Sci. 27:1276.

Lowry, O. H., J. V. Passonneau, F. X. Hasselberger and D. W. Schultz. 1964. Effect of ischemia on known substrates and cofactors of the glycolytic pathway in brain. J. Biol. Chem. 239:18.

Ludvigsen, J. 1953. "Muscular degeneration" in hogs (preliminary report). Proc. XV. Intern. Veterin. Cong. Vol. I, part 1, p. 602. Boktryckeri, Stockholm.

Ludvigsen, J. 1957. On the hormonal regulation of vasomotor reactions during exercise with special reference to the action of adrenal cortical steroids. Acta Endocrinologica 26:406.

Ludvigsen, J. 1960. Maladaptation syndromes in pigs. Proc. 2nd Intern. Animal Nutritional Conf. Madrid, Spain.

Maitra, P. K. and R. W. Estabrook. 1964. A fluorometric method for the enzymic determination of glycolytic intermediates. Anal. Biochem. 7:472.

Mansour, T. E. 1965. Studies on heart phosphofructokinase. Active and inactive form of the enzyme. J. Biol. Chem. 240:2165.

Marsh, B. B. 1966. Relaxing factor in muscle. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 225. University of Wisconsin Press, Madison.

Masaro, E. J., L. B. Rowell and R. M. McDonald. 1964. Skeletal muscle lipids. I. Analytical method and composition of monkey gastrocnemius and soleus muscles. Biochim. Biophys. Acta 84:493.

Masaro, E. J. 1967. Skeletal muscle lipids. III. Analysis of the functioning of skeletal muscle lipids during fasting. J. Biol. Chem. 242:1111.

McLoughlin, J. V. 1963. Studies on pig muscle. II. The effect of rapid post-mortem pH fall on the extraction of the sarcoplasmic and myofibrillar proteins of post-rigor muscle. Irish J. Agric. Res. 2:115.

McLoughlin, J. V. and G. Goldspink. 1963a. Post-mortem changes in the colour of pig longissimus dorsi muscle. Nature 198:584.

McLoughlin, J. V. and G. Goldspink. 1963b. Studies on pig muscle. I. Exudative pig muscle. Irish J. Agric. Res. 2:27.

McLoughlin, J. V. 1964. Observations on pale exudative musculature. Proc. Recip. Meat Conf. 17:171.

McLoughlin, J. V. 1968. Sarcoplasmic and myofibrillar protein in skeletal muscle of two breeds of pig. J. Food Sci. 33:383.

McLoughlin, J. V. 1969. Relationship between muscle biochemistry and properties of fresh and processed meats. Food Manufacture 44:36.

Merkel, R. A. 1968. Implication of the circulatory system in skeletal muscle to meat quality. Proc. Recip. Meat Conf. 21:204.

Meyer, J. A., E. J. Briskey, W. G. Hoekstra and R. W. Bray. 1962. Glucose tolerance in swine as related to post-mortem muscle characteristics. J. Animal Sci. 21:543.

Nachlas, M. M., K. Tsou, E. DeSouza, C. Cheng and A. M. Seligman. 1957. Cytochemical demonstration of succinic dehydrogenase by the use of a new p-nitrophenyl substituted ditetrazole. J. Histochem. Cytochem. 5:420.

Needham, D. M. 1926. Red and white muscle. Physiol. Rev. 6:1.

Newbold, R. P. 1966. Changes associated with rigor mortis. In The Physiology and Biochemistry of Muscle as a Food. Eds., E. J. Briskey, R. G. Cassens and J. C. Trautman. p. 213. University of Wisconsin Press, Madison.

Ogata, T. and M. Mori. 1963. A histochemical study of hydrolytic enzymes in muscle fibers of various animals. J. Histochem. Cytochem. 11:645.

Omtvedt, I. T. 1968. Some heritability characteristics and their importance in a selection program. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 128. Iowa State Univ. Press, Ames, Iowa.

Ostrander, J. and L. R. Dugan, Jr. 1962. Some differences in composition of covering fat, intermuscular fat and intramuscular fat of meat animals. J. Am. Oil Chem. Soc. 39:178.

Ozand, P. and H. T. Narahara. 1964. Regulation of glycolysis in muscle. III. Influence of insulin, epinephrine, and contraction on phosphofructokinase activity in frog skeletal muscle. J. Biol. Chem. 239: 3146.

Peachey, L. D. 1968. Muscle. Annual Review of Physiology. 30:401.

Penny, I. F., C. A. Voyle and R. A. Lawrie. 1964. Some properties of freeze-dried pork muscles of high or low ultimate pH. J. Sci. Food Agric. 15:559.

Perry, S. V. and D. J. Hartshorne. 1963. The proteins of developing muscle. In The Effect of Use and Disuse on Neuromuscular Functions. Eds., E. Gutman and P. Hnik. p. 491. Elsevier Publishing Co., New York.

Peter, J. B., R. N. Jeffress and D. R. Lamb. 1968. Exercise: Effects on hexokinase activity in red and white skeletal muscle. Science 160:200.

Ramaiah, A., J. A. Hathaway and D. E. Atkinson. 1964. Adenylate as a metabolite regulator. Effect on yeast phosphofructokinase kinetics. J. Biol. Chem. 239:3619.

Randle, P. J. 1964. Fuel and power in the control of carbohydrate metabolism in mammalian muscle. In Homeostasis and Feedback Mechanisms. Symposia of the Society for Experimental Biology No. XVIII. p. 129. Academic Press Inc., New York.

Regen, D. M., W. W. Davis, H. E. Morgan and C. R. Park. 1964. The regulation of hexokinase and phosphofructokinase activity in heart muscle. Effects of alloxan diabetes, growth hormone, cortisol, and anoxia. J. Biol. Chem. 239:43.

Reiser, R., M. C. Williams and M. F. Sorrels. 1960. Tissue lipide relationships from their composition and acetate incorporation. Arch. Biochem. Biophys. 86:42.

Robbins, N., G. Karpati and W. K. Engel. 1969. Histochemical and contractile properties in the cross-innervated guinea pig soleus muscle. Arch. Neurol. 20:318.

Romanul, F. C. A. 1964. Enzymes in muscle. I. Histochemical studies of enzymes in individual muscle fibers. Arch. Neurol. 11:355.

Romanul, F. C. A. and J. P. Van Der Meulen. 1967. Slow and fast muscles after cross innervation. Enzymatic and physiological changes. Arch. Neurol. 17:387.

Sayre, R. N., E. J. Briskey, W. G. Hoekstra and R. W. Bray. 1961. Effect of preslaughter change to a cold environment on characteristics of pork muscle. J. Animal Sci. 20:487.

Sayre, R. N. and E. J. Briskey. 1963. Protein solubility as influenced by physiological conditions in the muscle. J. Food Sci. 28:675.

Sayre, R. N., E. J. Briskey and W. G. Hoekstra. 1963a. Porcine muscle glycogen structure and its association with other muscle properties. Proc. Soc. Exptl. Biol. Med. 112:164.

Sayre, R. N., E. J. Briskey and W. G. Hoekstra. 1963b. Alteration of post-mortem changes in porcine muscle by pre-slaughter heat treatment and diet modification. J. Food Sci. 28:292.

Sayre, R. N., E. J. Briskey and W. G. Hoekstra. 1963c. Effect of excitement, fasting and sucrose feeding on porcine muscle phosphorylase and post-mortem glycolysis. J. Food Sci. 28:472.

Sayre, R. N., E. J. Briskey and W. G. Hoekstra. 1963d. Comparison of muscle characteristics and post-mortem glycolysis in three breeds of swine. J. Animal Sci. 22:1012.

Sayre, R. N., B. Kiernat and E. J. Briskey. 1964. Processing characteristics of porcine muscle related to pH and temperature during rigor mortis development and to gross morphology 24 hr post-mortem. J. Food Sci. 29:175.

Sayre, R. N., J. Para and E. J. Briskey. 1966. Protein alterations and associated changes in porcine muscle as influenced by maturity, genetic background and post-mortem muscle temperature. J. Food Sci. 31:819.

Scopes, R. K. and R. A. Lawrie. 1963. Post-mortem lability of skeletal muscle proteins. Nature 197:1202.

Scrutton, M. C. and M. F. Utter. 1968. The regulation of glycolysis and gluconeogenesis in animal tissues. Ann. Rev. Biochem. 37:249.

Seidel, J. C. and J. Gergely. 1963. Studies on myofibrillar adenosine triphosphatase with calcium-free adenosine triphosphate. I. The effect of ethylenediaminetetraacetate, calcium, magnesium, and adenosine triphosphate. J. Biol. Chem. 238:3648.

Seidel, J. C., F. A. Sreter, M. M. Thompson and J. Gergely. 1964. Comparative studies of myofibrils, myosin and actomyosin from red and white rabbit skeletal muscle. Biochem. Biophys. Res. Commun. 17:662.

Sink, J. D., R. W. Bray, W. G. Hoekstra and E. J. Briskey. 1967. Lipid composition of normal and pale, soft, exudative porcine muscle. J. Food Sci. 32:258.

Spitzer, J. J. and M. Gold. 1964. Free fatty acid metabolism by skeletal muscle. Am. J. Physiol. 206:159.

Sreter, F. A., J. C. Seidel and J. Gergely. 1966. Studies on myosin from red and white skeletal muscles of the rabbit. J. Biol. Chem. 241:5722.

Steel, R. G. D. and J. H. Torrie. 1960. Principles and Procedures of Statistics. McGraw-Hill Book Co., Inc., New York.

Thomas, N. W., P. B. Addis, H. R. Johnson, R. D. Howard and M. D. Judge. 1966. Effects of environmental temperature and humidity during growth on muscle properties of two porcine breeds. J. Food Sci. 31:309.

Topel, D. G. and R. A. Merkel. 1966. Effect of exogenous goitrogens upon some physical and biochemical properties of porcine muscle and adrenal gland. J. Animal Sci. 25:1154.

Topel, D. G. and R. A. Merkel. 1967. Effect of exogenous prednisolone and methyl-prednisolone upon plasma 17-hydroxycorticosteroid levels and some porcine muscle characteristics. J. Animal Sci. 26:1017.

Topel, D. G., R. A. Merkel and J. Wismer-Pedersen. 1967. Relationship of plasma 17-hydroxycorticosteroid levels to some physical and biochemical properties of porcine muscle. J. Animal Sci. 26:311.

Trayer, I. P. and Perry, S. V. 1966. The myosin of developing skeletal muscle. Biochem. Zeit. 345:87.

Uyeda, K. and E. Racker. 1965. Regulatory mechanisms in carbohydrate metabolism. VII. Hexokinase and phosphofructokinase. J. Biol. Chem. 240:4682.

Van Wijhe, M., M. C. Blanchaer and W. R. Jacyk. 1963. The oxidation of lactate and α-glycerophosphate by red and white skeletal muscle. II. Histochemical studies. J. Histochem. Cytochem. 11:505.

White, A., P. Handler and E. L. Smith. 1964. Principles of Biochemistry. 3rd ed. McGraw-Hill Book Co., New York.

Williamson, J. R. 1965. Glycolytic control mechanisms. I. Inhibition of glycolysis by acetate and pyruvate in the isolated, perfused rat heart. J. Biol. Chem. 240:2308.

Wilson, J. E., B. Sacktor and C. G. Tiekert. 1967. In situ regulation of glycolysis in tetanized cat skeletal muscle. Arch. Biochem. Biophys. 120:542.

Wismer-Pedersen, J. 1959. Quality of pork in relation to rate of pH change post mortem. Food Res. 24:711.

Wismer-Pedersen, J. and H. Rieman. 1960. Pre-slaughter treatment of pigs as it influences meat quality and stability. Proc. 12th Res. Conf., Am. Meat Inst. Found. p. 89.

Wismer-Pedersen, J. and E. J. Briskey. 1961a. Rate of anaerobic glycolysis versus structure in pork muscle. Nature 189:318.

Wismer-Pedersen, J. and E. J. Briskey. 1961b. Relationship of post-mortem acidity and temperature. Food Technol. 15:232.

Wismer-Pedersen, J. 1968. Modern production practices and their influence on stress conditions. In The Pork Industry: Problems and Progress. Ed., D. G. Topel. p. 163. Iowa State Univ. Press, Ames, Iowa.

Wood, W. A. 1966. Carbohydrate metabolism. Am. Rev. Biochem. 35:521.

Yellin, H. 1967. Neural regulation of enzymes in muscle fibers of red and white muscle. Exptl. Neurol. 19:92.

APPENDIX

Appendix A. Heart weight, LD pH and subjective quality scores (Group I[a,b]).

Animal	Slaughter wt (lbs)	Heart wt (g)	LD post-mortem pH 0 hr	15 min	45 min	3 hr	24 hr	Quality M	C	F
1-PC-B	195	-	6.44	6.18	6.06	5.55	5.58	3	5	5
2-PC-G	180	-	6.18	5.92	5.80	5.64	5.56	1	3	3
3-PC-G	190	-	6.44	6.54	6.55	5.92	5.26	3	4	4
4-PC-G*	194	-	6.35	6.28	6.14	5.24	5.22	1	2	2
5-PC-G	172	-	6.02	5.52	5.72	5.26	5.28	1	2	2
6-PC-G	180	-	6.10	6.12	6.06	5.29	5.25	2	3	3
7-PC-G	148	-	6.15	5.94	5.67	5.48	5.30	1	4	4
8-PC-G	161	-	6.28	6.18	6.10	5.55	5.56	2	4	5
9-PC-G	168	-	6.19	5.88	5.63	5.54	5.37	1	3	3
10-PC-G	163	249.7	6.26	6.14	6.05	5.40	5.28	1	3	3
11-PC-G	163	535.7	6.36	6.11	6.05	5.31	5.27	2	4	4
12-PC-G	210	291.3	6.06	5.76	5.65	5.28	5.23	1	2	2
13-PC-G	209	249.6	6.21	6.15	6.22	5.37	5.25	3	4	4
14-PC-G	191	237.3	6.14	5.80	5.61	5.34	5.30	1	2	1
15-PC-G	204	255.1	6.19	5.58	5.42	5.40	5.33	1	2	2
16-PC-G	165	222.1	6.20	5.68	5.27	5.34	5.24	2	3	3
17-PC-G	198	311.2	6.02	5.66	5.33	5.26	5.20	1	2	2
18-PC-G	205	327.8	6.35	5.90	5.50	5.40	5.29	2	5	4
19-PC-G	201	272.9	6.33	6.14	5.80	5.30	5.20	1	2	3
20-LR-G	216	248.1	6.30	5.98	5.48	5.14	5.19	1	3	2
21-LR-G	211	269.4	6.48	6.43	6.26	5.20	5.23	2	4	3
22-LR-G	220	248.2	6.22	6.05	5.70	5.32	5.27	1	3	3
23-LR-G	226	282.7	6.40	6.33	6.08	5.14	5.15	1	3	2
24-LR-G	226	258.9	6.17	6.12	5.97	5.23	5.23	3	5	3
25-LR-G	240	276.3	6.20	5.84	5.20	5.16	5.18	1	2	2
26-LR-B	208	258.2	6.34	6.30	6.16	5.22	5.19	1	3	2
27-LR-B	230	312.7	6.36	6.31	6.26	5.21	5.21	3	4	3
28-LR-G	183	251.8	6.22	6.18	6.18	5.27	5.26	3	4	3
29-LR-G	196	264.5	6.36	6.28	6.18	5.30	5.31	3	5	3
30-LR-B	196	262.1	6.34	6.27	5.98	5.22	5.20	1	2	2
31-LR-B	191	299.5	6.36	6.29	6.27	5.33	5.38	3	5	4
32-LR-G	190	285.0	6.30	6.32	6.20	5.36	5.36	4	5	4
33-LR-B	192	231.6	6.28	6.24	6.02	5.24	5.25	2	4	3
34-LR-G	201	258.5	6.36	6.26	6.12	5.28	5.22	2	4	3
35-LR-G	192	278.9	6.32	6.26	6.10	5.28	5.23	1	4	3
36-CW-G	204	305.5	6.14	6.09	6.07	5.22	5.26	3	5	4
37-CW-B	225	263.4	6.34	6.36	6.45	5.28	5.30	3	5	5
38-CW-B	213	265.1	6.14	6.22	6.16	5.24	5.25	3	2	2
39-CW-G	198	304.6	6.31	6.30	6.26	5.32	5.26	4	5	5
40-CW-G	192	322.6	6.28	6.32	6.38	5.58	5.36	4	4	5
41-CW-G	210	265.6	6.31	6.24	6.13	5.22	5.26	4	5	4

*Killed by overdose of Na pent. anesthesia.

[a]Hogs included in text as "normal" (N): 3, 8, 10, 11, 13, 21, 27, 29, 31, 32, 36, 37, 39, 40, 41.

[b]Hogs included in text as "lower quality" (LQ): 14, 15, 16, 17, 19, 20, 22, 24, 25, 30.

Appendix A. Heart weight, LD pH and subjective quality scores (Group I[a,b]).

Animal	Slaughter wt (lbs)	Heart wt (g)	LD post-mortem pH 0 hr	15 min	45 min	3 hr	24 hr	Quality M	C	F
1-PC-B	195	-	6.44	6.18	6.06	5.55	5.58	3	5	5
2-PC-G	180	-	6.18	5.92	5.80	5.64	5.56	1	3	3
3-PC-G	190	-	6.44	6.54	6.55	5.92	5.26	3	4	4
4-PC-G*	194	-	6.35	6.28	6.14	5.24	5.22	1	2	2
5-PC-G	172	-	6.02	5.52	5.72	5.26	5.28	1	2	2
6-PC-G	180	-	6.10	6.12	6.06	5.29	5.25	2	3	3
7-PC-G	148	-	6.15	5.94	5.67	5.48	5.30	1	4	4
8-PC-G	161	-	6.28	6.18	6.10	5.55	5.56	2	4	5
9-PC-G	168	-	6.19	5.88	5.63	5.54	5.37	1	3	3
10-PC-G	163	249.7	6.26	6.14	6.05	5.40	5.28	1	3	3
11-PC-G	163	535.7	6.36	6.11	6.05	5.31	5.27	2	4	4
12-PC-G	210	291.3	6.06	5.76	5.65	5.28	5.23	1	2	2
13-PC-G	209	249.6	6.21	6.15	6.22	5.37	5.25	3	4	4
14-PC-G	191	237.3	6.14	5.80	5.61	5.34	5.30	1	2	1
15-PC-G	204	255.1	6.19	5.58	5.42	5.40	5.33	1	2	2
16-PC-G	165	222.1	6.20	5.68	5.27	5.34	5.24	2	3	3
17-PC-G	198	311.2	6.02	5.66	5.33	5.26	5.20	1	2	2
18-PC-G	205	327.8	6.35	5.90	5.50	5.40	5.29	2	5	4
19-PC-G	201	272.9	6.33	6.14	5.80	5.30	5.20	1	2	3
20-LR-G	216	248.1	6.30	5.98	5.48	5.14	5.19	1	3	2
21-LR-G	211	269.4	6.48	6.43	6.26	5.20	5.23	2	4	3
22-LR-G	220	248.2	6.22	6.05	5.70	5.32	5.27	1	3	3
23-LR-G	226	282.7	6.40	6.33	6.08	5.14	5.15	1	3	2
24-LR-G	226	258.9	6.17	6.12	5.97	5.23	5.23	3	5	3
25-LR-G	240	276.3	6.20	5.84	5.20	5.16	5.18	1	2	2
26-LR-B	208	258.2	6.34	6.30	6.16	5.22	5.19	1	3	2
27-LR-B	230	312.7	6.36	6.31	6.26	5.21	5.21	3	4	3
28-LR-G	183	251.8	6.22	6.18	6.18	5.27	5.26	3	4	3
29-LR-G	196	264.5	6.36	6.28	6.18	5.30	5.31	3	5	3
30-LR-B	196	262.1	6.34	6.27	5.98	5.22	5.20	1	2	2
31-LR-B	191	299.5	6.36	6.29	6.27	5.33	5.38	3	5	4
32-LR-G	190	285.0	6.30	6.32	6.20	5.36	5.36	4	5	4
33-LR-B	192	231.6	6.28	6.24	6.02	5.24	5.25	2	4	3
34-LR-G	201	258.5	6.36	6.26	6.12	5.28	5.22	2	4	3
35-LR-G	192	278.9	6.32	6.26	6.10	5.28	5.23	1	4	3
36-CW-G	204	305.5	6.14	6.09	6.07	5.22	5.26	3	5	4
37-CW-B	225	263.4	6.34	6.36	6.45	5.28	5.30	3	5	5
38-CW-B	213	265.1	6.14	6.22	6.16	5.24	5.25	3	2	2
39-CW-G	198	304.6	6.31	6.30	6.26	5.32	5.26	4	5	5
40-CW-G	192	322.6	6.28	6.32	6.38	5.58	5.36	4	4	5
41-CW-G	210	265.6	6.31	6.24	6.13	5.22	5.26	4	5	4

*Killed by overdose of Na pent. anesthesia.

[a]Hogs included in text as "normal" (N): 3, 8, 10, 11, 13, 21, 27, 29, 31, 32, 36, 37, 39, 40, 41.

[b]Hogs included in text as "lower quality" (LQ): 14, 15, 16, 17, 19, 20, 22, 24, 25, 30.

Appendix B. Muscle fiber types and succinic dehydrogenase activities (Group I).

Animal	SDH activity[a]	Muscle fiber types: Number % R	W	I	Area % R	W	I	Relative size[b] R	W	I	Total
1-PC-B	36.5	--	--	--	--	--	-	--	-	--	---
2-PC-G	24.8	--	--	--	--	--	-	--	-	--	---
3-PC-G	25.9	27.3	63.6	9.1	15.6	78.0	6.4	12.0	5.6	9.7	6.8
4-PC-G*	29.2	--	--	--	--	--	-	--	-	--	---
5-PC-G	13.2	--	--	--	--	--	-	--	-	--	---
6-PC-G	22.7	--	--	--	--	--	-	--	-	--	---
7-PC-G	27.2	--	--	--	--	--	-	--	-	--	---
8-PC-G	15.8	29.2	60.1	10.7	16.8	75.4	7.8	9.1	4.2	7.3	5.3
9-PC-G	19.8	--	--	--	--	--	-	--	-	--	---
10-PC-G	19.3	26.5	65.4	8.1	16.3	77.7	6.0	9.7	5.0	8.0	6.0
11-PC-G	33.7	26.4	63.0	10.6	17.2	74.1	8.7	10.3	5.7	8.1	6.7
12-PC-G	11.1	--	--	--	--	--	-	--	-	--	---
13-PC-G	13.7	28.2	63.6	8.2	17.2	77.3	5.5	9.0	4.6	8.2	5.6
14-PC-G	14.5	26.8	63.6	9.6	18.6	73.2	8.2	9.3	5.6	7.5	6.4
15-PC-G	12.2	18.6	73.0	8.4	15.1	77.3	7.6	7.0	5.4	6.2	5.7
16-PC-G	20.0	19.9	73.5	6.6	15.2	79.2	5.6	6.7	4.8	6.1	5.1
17-PC-G	14.8	20.6	72.7	6.7	17.3	76.7	6.0	7.2	5.8	6.8	6.1
18-PC-G	45.0	20.9	72.5	6.6	20.9	72.7	6.4	7.3	7.4	7.6	7.4
19-PC-G	31.0	27.4	59.7	12.9	17.8	68.3	13.9	9.3	5.3	5.8	6.0
20-LR-G	24.4	23.0	72.6	4.4	15.1	80.9	4.0	7.9	4.7	5.8	5.2
21-LR-G	25.2	24.2	66.4	9.4	15.7	78.2	6.2	10.0	5.6	10.0	6.5
22-LR-G	12.0	16.1	77.9	6.0	13.8	80.3	5.9	5.8	4.8	5.1	5.0
23-LR-G	25.7	--	--	--	--	--	-	--	-	--	---
24-LR-G	17.9	18.8	75.1	6.1	12.2	83.0	4.8	9.2	5.4	7.6	6.0
25-LR-G	17.7	21.6	75.1	3.3	14.1	83.8	2.1	10.6	6.2	11.0	6.9
26-LR-B	28.8	--	--	--	--	--	-	--	-	--	---
27-LR-B	25.4	20.2	70.0	9.8	12.6	80.3	7.1	10.8	5.9	9.3	6.8
28-LR-G	22.9	--	--	--	--	--	-	--	-	--	---
29-LR-G	34.1	25.7	63.3	11.0	15.3	75.7	9.0	8.0	4.0	5.8	4.8
30-LR-B	17.5	22.2	69.9	7.9	15.3	78.8	5.9	10.8	6.5	9.8	7.3
31-LR-B	45.0	26.1	63.8	10.1	14.7	78.7	6.6	13.6	6.2	11.7	7.6
32-LR-G	38.0	24.0	65.8	10.2	14.6	78.3	7.1	12.4	6.3	10.8	7.5
33-LR-B	26.9	--	--	--	--	--	-	--	-	--	---
34-LR-G	35.0	--	--	--	--	--	-	--	-	--	---
35-LR-G	49.2	--	--	--	--	--	-	--	-	--	---
36-CW-G	28.3	22.5	67.4	10.1	14.7	78.7	6.6	8.2	4.5	8.1	5.3
37-CW-B	26.2	27.2	65.4	7.4	13.8	82.2	3.9	11.4	4.6	11.1	5.8
38-CW-B	27.1	--	--	--	--	--	-	--	-	--	---
39-CW-G	28.5	25.2	64.8	9.9	14.0	79.9	6.1	11.2	5.1	10.1	6.2
40-CW-G	16.9	21.4	70.8	7.8	12.8	81.9	5.3	10.8	5.6	9.6	6.4
41-CW-G	23.1	22.6	67.7	9.7	13.6	79.2	7.2	10.0	5.1	8.0	6.0

*Killed by overdose of Na pent. anesthesia.

[a] μ moles succinate oxidized/min/g frozen, powdered muscle at 37°C.

[b] Fibers/sq. in. of picture analyzed.

Appendix C. Myoglobin (Group I).

	Myoglobin (0 hr LD)						
	Total	MMb		Mb		O_2Mb	
Animal	Mb[a]	%	Amount[a]	%	Amount[a]	%	Amount[a]
1-PC-B	63.4	31.5	20.0	20.5	13.0	48.0	30.4
2-PC-G	48.1	40.0	19.2	16.5	7.9	43.5	20.9
3-PC-G	75.6	41.5	31.4	24.2	18.4	34.2	25.9
4-PC-G*	52.2	30.8	16.1	14.2	7.4	55.0	28.7
5-PC-G	53.5	31.8	17.0	16.2	8.7	52.0	27.8
6-PC-G	60.2	39.2	23.6	31.5	19.0	29.2	17.6
7-PC-G	88.9	35.7	31.7	21.3	19.0	43.0	38.2
8-PC-G	76.4	37.2	28.4	22.0	16.8	40.8	31.2
9-PC-G	61.6	29.5	18.2	11.0	6.8	59.5	36.7
10-PC-G	59.7	31.5	18.8	16.8	10.0	51.8	30.9
11-PC-G	64.1	28.8	18.4	8.8	5.6	62.5	40.1
12-PC-G	55.3	32.2	17.8	13.0	7.2	54.8	30.3
13-PC-G	125.3	27.7	34.7	10.7	13.4	61.7	77.3
14-PC-G	61.2	34.0	20.8	17.5	10.7	48.5	29.7
15-PC-G	74.4	40.3	30.0	26.8	20.0	32.8	24.4
16-PC-G	34.3	29.0	9.9	13.5	4.6	57.5	19.7
17-PC-G	40.2	31.9	12.8	20.6	8.3	47.5	19.1
18-PC-G	99.4	27.5	27.3	13.2	13.1	59.3	59.0
19-PC-G	67.0	33.5	22.4	18.2	12.2	48.2	32.3
20-LR-G	102.3	36.8	37.6	24.2	24.8	39.0	39.9
21-LR-G	106.0	35.5	37.6	19.2	20.4	45.2	48.0
22-LR-G	93.0	37.2	34.6	26.5	24.6	36.2	33.7
23-LR-G	106.3	37.0	39.3	24.0	25.5	39.0	41.4
24-LR-G	84.8	32.2	27.4	14.0	11.9	53.8	45.6
25-LR-G	66.4	34.8	23.1	14.2	9.5	51.0	33.8
26-LR-B	75.7	33.5	25.4	16.5	12.5	50.0	37.9
27-LR-B	143.8	43.2	62.2	29.8	42.8	27.0	38.8
28-LR-G	89.9	44.2	39.8	27.2	24.5	28.5	25.6
29-LR-G	114.1	41.8	47.6	28.5	32.5	29.8	33.9
30-LR-B	100.7	39.3	39.6	25.7	25.8	35.0	35.2
31-LR-B	103.4	36.8	38.1	23.5	24.3	39.7	41.0
32-LR-G	106.6	39.2	41.8	24.5	26.1	36.2	38.6
33-LR-B	89.5	41.8	37.4	25.8	23.0	32.5	29.1
34-LR-G	77.9	37.5	29.2	20.5	16.0	42.0	32.7
35-LR-G	78.2	39.0	30.5	20.5	16.0	40.5	31.7
36-CW-G	73.8	28.5	21.0	12.0	8.8	59.5	43.9
37-CW-B	77.8	37.7	29.3	20.3	15.8	42.0	32.7
38-CW-B	57.8	28.8	16.6	12.2	7.1	59.0	34.1
39-CW-G	75.7	27.0	20.4	11.5	8.7	61.5	46.6
40-CW-G	73.9	38.5	28.5	22.3	16.5	39.2	29.0
41-CW-G	95.9	42.3	40.6	28.8	27.6	28.8	27.6

*Killed by overdose of Na pent. anesthesia.
[a]mμ moles/g frozen, powdered muscle.

Appendix D. Lipids (Group I).

Animal	Lipid (% fresh wt)			Phospholipid (0 hr LD)		Glyceride esters (0 hr LD)	
	Serum	Liver	LD (0 hr)	μ eq P/ g LD	μ eq P/ g lipid	μ eq/ g LD	μ eq/ g lipid
1-PC-B	-	-	-	-	-	-	-
2-PC-G	-	-	-	-	-	-	-
3-PC-G	0.31	4.92	5.04	5.40	107.1	42.9	851.2
4-PC-G*	-	-	-	-	-	-	-
5-PC-G	-	-	-	-	-	-	-
6-PC-G	-	-	-	-	-	-	-
7-PC-G	-	-	-	-	-	-	-
8-PC-G	0.27	5.16	3.96	7.92	201.0	31.5	795.4
9-PC-G	-	-	-	-	-	-	-
10-PC-G	0.32	5.41	2.62	5.48	211.4	15.6	595.4
11-PC-G	0.29	5.70	3.39	7.71	227.3	22.1	651.9
12-PC-G	-	-	-	-	-	-	-
13-PC-G	0.34	5.69	6.01	4.30	71.5	55.4	921.8
14-PC-G	0.35	5.79	2.22	4.16	162.0	19.1	860.4
15-PC-G	0.36	4.87	5.25	7.32	139.4	31.6	601.9
16-PC-G	0.26	6.01	2.14	8.18	383.2	13.5	630.8
17-PC-G	0.30	4.53	2.61	8.48	325.8	17.0	651.3
18-PC-G	-	-	-	-	-	-	-
19-PC-G	0.31	5.62	3.78	7.49	198.1	29.0	767.2
20-LR-G	0.34	4.97	3.26	6.25	192.8	23.3	714.7
21-LR-G	0.29	5.15	3.83	6.28	164.0	37.5	979.1
22-LR-G	0.33	6.38	5.52	7.14	143.6	42.7	773.6
23-LR-G	-	-	-	-	-	-	-
24-LR-G	0.40	4.93	4.30	4.52	105.4	40.5	941.9
25-LR-G	0.38	5.97	3.98	7.68	192.9	33.2	834.2
26-LR-B	-	-	-	-	-	-	-
27-LR-B	0.26	5.20	6.92	7.18	103.8	52.0	751.4
28-LR-G	-	-	-	-	-	-	-
29-LR-G	0.32	4.81	4.20	8.34	198.8	35.6	847.6
30-LR-B	0.43	5.63	3.85	7.20	186.6	32.1	833.8
31-LR-B	0.34	4.81	3.65	8.80	241.4	34.4	942.5
32-LR-G	0.33	6.48	5.27	7.85	149.1	44.0	834.9
33-LR-B	-	-	-	-	-	-	-
34-LR-G	-	-	-	-	-	-	-
35-LR-G	-	-	-	-	-	-	-
36-CW-G	0.31	5.61	3.56	4.88	137.3	31.5	884.8
37-CW-B	0.37	5.89	4.11	8.40	204.5	33.1	805.4
38-CW-B	-	-	-	-	-	-	-
39-CW-G	0.26	5.66	5.25	5.28	100.6	53.1	1011.42
40-CW-G	0.34	5.67	3.75	3.25	88.0	32.2	858.7
41-CW-G	0.36	5.34	3.25	7.88	202.3	32.9	1012.3

*Killed by overdose of Na pent. anesthesia.

Appendix E. Group II.

Barrow	Carcass wt (lb)	Length (in.)	Backfat thickness (in.)	Ham %	Loin eye area ($in.^2$)	Quality M	C	F	Temperature (°F) Rectal (0 hr)	LD (45 min)	LD pH (45 min)
1-Y	135	29.9	1.27	22.7	4.70	4	4	5	102.5	104.1	6.55
2-H	158	31.0	1.27	21.3	5.55	3	4	4	102.4	105.0	6.50
3-H	137	29.2	1.00	22.5	4.22	4	4	4	101.8	104.0	6.24
4-XB	174	32.1	1.17	21.1	4.73	2	3	3	102.4	105.3	6.30
5-Y	154	31.5	1.63	20.0	4.03	4	4	4	103.4	106.5	6.01
6-H	146	31.5	1.23	22.3	4.37	1	2	3	104.6	106.8	5.54
7-CW	126	28.3	1.33	21.8	3.67	3	3	5	103.3	102.3	6.12
8-SP	159	28.8	1.63	18.6	4.46	4	5	5	104.0	106.6	6.33
9-PC	141	28.5	1.63	19.0	3.37	1	2	4	103.0	104.2	5.70
10-XB	132	28.2	1.23	23.8	5.07	2	3	2	103.1	105.5	6.10
11-XB	158	30.5	1.53	19.0	4.47	2	5	5	104.0	104.8	6.36
12-D	133	29.0	1.17	21.5	3.63	2	2	2	103.6	105.7	5.63
13-H	159	31.0	1.33	21.6	4.61	3	5	5	102.1	104.6	6.42
14-XB	148	28.9	1.47	21.4	5.21	3	4	4	102.3	103.6	6.66
15-LR	138	29.8	1.27	20.1	3.89	5	5	5	104.8	106.8	6.14
16-H	144	30.0	1.17	23.3	5.58	4	4	5	101.8	105.3	6.36
17-H	151	30.5	1.23	21.7	3.84	5	5	4	102.6	104.6	6.31
18-B	133	29.7	1.20	21.4	3.79	2	3	4	102.5	105.2	6.20
19-XB	154	29.8	1.33	23.0	4.42	1	5	4	101.8	103.5	6.81
20-H	156	28.3	1.53	21.5	5.43	1	2	1	103.4	106.5	6.00

Appendix E. Group II (continued).

Barrow	Carcass wt (lb)	Length (in.)	Backfat thickness (in.)	Ham %	Loin eye area ($in.^2$)	Quality M	C	F	Temperature (°F) Rectal (0 hr)	LD (45 min)	LD pH (45 min)
21-H	158	32.0	1.20	23.7	4.07	1	3	3	102.6	104.5	5.95
22-XB	142	28.9	1.23	22.2	4.20	4	4	3	102.0	103.8	6.12
23-CW	146	29.3	1.43	17.9	4.13	5	4	4	102.0	104.4	6.28
24-H	135	28.9	1.20	21.6	4.96	3	4	3	102.6	104.3	6.62
25-D	140	28.8	1.33	18.1	3.35	5	4	5	103.2	104.6	6.47
26-H	148	30.3	1.17	22.2	4.50	2	3	3	102.8	105.5	5.92
27-XB	138	28.5	1.33	21.2	4.77	4	4	5	103.0	105.4	6.15
28-Y	142	29.3	1.20	22.6	5.35	2	3	3	103.0	106.3	5.97
29-XB	139	29.9	1.27	20.7	4.91	5	5	5	102.4	104.9	6.40
30-H	138	31.3	1.13	21.7	3.44	3	4	5	102.0	103.6	6.64
31-Y	140	30.3	1.13	20.8	4.95	3	4	4	102.2	103.6	5.80
32-CW	162	30.3	1.33	20.9	4.29	3	5	5	103.2	104.1	6.49
33-H	136	29.0	1.23	20.9	4.92	1	3	3	101.9	104.8	5.92
34-H	148	29.2	1.37	20.7	4.81	2	3	4	105.6	106.8	6.32
35-PC	167	30.6	1.67	18.6	4.42	1	3	3	102.0	105.4	5.68
36-XB	136	29.6	1.37	20.1	3.33	4	4	3	102.6	105.1	6.04
37-D	163	29.3	1.53	21.1	4.48	5	5	5	101.5	104.8	6.20
38-PC	170	30.0	1.43	19.8	4.85	1	3	4	103.0	106.6	5.70
39-H	162	31.2	1.43	21.8	4.74	3	4	5	101.3	104.3	6.21
40-CW	149	29.0	1.13	22.4	4.01	5	5	5	102.4	105.0	6.18

Appendix F. Group III.

Animal	Slaughter wt (lb)	Heart wt (g)	LD temperature (°F) 0 hr	Out of scald	45 min	LD pH 45 min	Transmission value	Quality M	C	F	Backfat thickness (in.)	Loin eye area ($in.^2$)	Ham and loin %
1-H-G	182	276.1	105.0	105.7	106.8	5.95	--	2	4	2	1.07	6.53	42.5
2-D-B	191	240.9	104.0	104.2	104.1	6.25	--	5	4	5	1.33	4.44	40.8
3-Y-B	200	255.3	104.0	104.8	104.0	5.90	76.2	3	4	2	1.20	4.29	40.2
4-Y-G	190	231.3	104.7	105.4	104.5	6.10	40.8	3	5	4	1.03	4.38	40.9
5-Y-G	189	217.0	105.8	106.9	106.8	5.80	59.5	2	4	2	1.03	4.04	39.6
6-H-G	192	299.3	103.8	103.6	102.0	6.40	42.0	3	5	5	0.95	5.14	42.2
7-Y-B	190	263.5	105.2	105.7	105.2	5.65	83.5	2	2	1	1.15	4.12	42.2
8-Y-B*	208	258.5	107.5	108.3	107.0	5.65	99.5	1	1	1	1.36	4.64	39.1
9-Y-B*	196	230.0	108.0	109.2	107.1	5.70	100.0	1	1	1	1.38	3.89	38.8
10-Y-B	206	250.0	105.7	106.1	104.6	6.55	55.5	2	4	4	1.65	3.49	38.7
11-Y-G	202	261.0	104.7	104.6	104.8	6.45	47.0	4	5	5	1.40	4.94	40.5
12-PC-G	206	268.8	104.8	106.4	106.7	5.80	93.5	1	2	2	1.20	5.99	39.7
13-Y-G	200	219.9	103.6	104.6	104.8	5.95	82.8	2	3	3	1.43	4.46	39.4
14-Y-B	202	261.5	103.3	104.5	104.4	6.15	72.5	3	4	3	1.67	4.50	39.6
15-Y-G	200	249.8	103.5	104.4	103.2	5.65	93.5	2	2	2	1.33	4.61	40.9
16-H-G	220	335.6	103.2	103.9	104.0	6.55	72.5	3	5	5	1.17	5.16	40.5
17-H-B	222	308.9	104.0	104.5	103.8	6.45	79.0	4	4	5	1.17	4.27	39.5
18-Y-B	219	284.3	103.4	104.2	103.8	6.60	67.0	5	5	5	1.47	4.09	36.5
19-H-G	208	285.1	104.6	105.0	105.7	6.20	84.5	3	4	4	1.13	4.26	40.8
20-Y-G	218	308.3	104.2	104.8	105.5	6.20	83.5	5	5	4	1.33	3.85	37.7

*Postmortem inspection indicated pericarditis.

Appendix F. Group III (continued).

Animal	Slaughter wt (lb)	Heart wt (g)	LD temperature (°F) 0 hr	45 min	LD pH 45 min	Transmission value	Quality M	C	F	Backfat thickness (in.)	Loin eye area ($in.^2$)	Ham and loin %
21-H-B	216	260.5	101.2	102.8	6.35	56.2	3	5	4	1.23	4.27	40.0
22-CW-G	195	261.5	105.5	104.2	6.50	30.2	4	5	5	1.18	4.06	40.1
23-Y-B	214	289.6	102.2	102.7	6.00	65.2	4	4	5	1.10	4.69	41.5
24-Y-B	240	269.6	103.0	103.2	6.60	36.8	5	5	5	1.47	3.96	37.4
25-Y-B	240	313.3	104.0	104.8	6.50	13.8	5	5	5	1.53	5.10	38.6
26-Y-B	234	296.3	104.0	103.8	6.55	27.5	5	4	5	1.28	5.97	40.1
27-H-B	238	295.1	102.1	104.4	6.30	59.8	2	4	3	1.20	5.55	39.6
28-H-G	196	347.6	104.1	105.4	6.35	24.8	3	5	4	0.93	4.52	42.1
29-Y-G	208	253.9	103.4	102.3	6.00	43.5	2	3	4	1.30	4.16	40.0
30-Y-G	182	275.9	103.6	104.5	5.70	72.0	2	4	4	0.83	4.67	46.2
31-Y-B	215	245.0	103.0	104.4	5.90	40.0	3	5	5	1.57	3.50	36.6
32-Y-B	198	220.4	103.4	104.8	5.90	46.5	5	3	4	1.53	3.11	35.2
33-H-G	182	289.3	100.2	105.2	6.40	62.5	3	4	4	1.00	4.25	43.3
34-H-G	234	245.6	103.6	104.0	6.10	93.0	1	4	2	1.37	4.30	40.5
35-CW-G	192	274.9	104.2	105.5	6.30	23.5	4	4	4	1.13	3.92	40.8
36-Y-G	192	257.3	104.7	105.6	5.95	17.0	3	5	5	1.07	X	41.2
37-Y-B	236	264.9	103.8	105.7	5.60	99.0	2	2	2	1.57	X	38.7
38-Y-B	208	227.7	103.0	104.6	5.75	66.5	3	4	4	1.37	X	38.9
39-Y-G	224	261.8	104.0	104.9	6.05	89.5	4	5	5	1.23	X	42.7
40-Y-G	228	247.7	103.1	104.0	6.15	39.5	4	4	4	1.43	X	38.8
41-Y-G	226	280.8	104.0	105.2	5.60	97.0	2	2	2	1.23	X	40.7
42-Y-G	210	218.6	102.8	104.6	6.05	51.5	5	5	4	1.20	X	38.4
43-H-B	228	247.9	102.9	105.1	6.20	96.5	2	3	3	1.47	X	37.9
44-H-G	228	258.5	102.8	104.8	6.25	73.5	3	5	4	1.23	X	37.9

Appendix G. pH and quality scores of longissimus muscle and heart weight (Group IV).

Animal	Slaughter wt (lbs)	Heart wt (g)	pH									Quality scores					
			LD-R postmortem time					LD-L postmortem time				LD-R			LD-L		
			0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	M	C	F	M	C	F
1-G	193	243.9	6.07	5.91	5.85	5.80	5.19	X	6.42	6.14	5.18	2	4	3	3	5	4
2-G	222	246.5	6.32	6.25	6.19	5.92	5.25	X	6.54	6.30	5.38	2	4	5	3	5	5
3-B	266	270.8	6.13	6.15	5.93	5.32	5.30	X	6.03	5.33	5.31	1	2	2	1	2	2
4-B	188	213.9	6.27	6.25	6.13	5.85	5.13	X	6.61	6.25	5.12	2	4	3	2	4	4
5-G	226	303.4	6.11	6.22	6.10	5.52	5.12	X	6.37	5.73	5.17	2	3	3	2	3	3
6-G	211	231.3	6.23	6.32	6.18	5.64	5.13	X	6.42	6.00	5.11	2	3	3	2	3	3
7-G	209	266.4	6.12	5.98	5.93	5.66	5.19	X	6.50	6.16	5.20	2	4	4	3	5	4
8-G	249	270.3	6.21	6.22	6.11	5.65	6.15	X	6.51	6.16	5.19	2	4	3	2	4	4
9-G	261	332.7	6.15	6.10	5.94	5.66	5.28	X	6.51	6.13	5.30	2	4	4	2	4	4
10-B*	198	255.6	6.19	6.13	6.14	5.81	5.19	X	6.59	6.32	5.26	3	3	3	4	4	4
11-G	237	277.8	6.22	6.16	5.68	5.31	5.20	X	6.29	5.44	5.21	2	3	3	2	3	4
12-G	196	254.1	6.20	6.17	6.16	5.82	5.12	6.47	6.35	5.87	5.13	4	4	5	4	4	5
13-G	204	244.1	6.29	6.23	6.16	5.96	5.12	6.64	6.64	6.43	5.25	5	4	4	5	4	5
14-G	240	284.3	6.19	6.10	5.84	5.42	5.21	6.31	6.30	5.58	5.24	3	4	4	3	4	4
15-G	240	251.8	6.18	6.15	6.13	6.00	5.25	6.31	6.27	5.83	5.17	4	5	5	4	4	4
16-G	281	279.2	6.12	5.96	5.92	5.25	5.20	6.36	6.16	5.33	5.18	1	2	2	1	2	2
17-G	180	221.4	X	6.48	6.31	5.66	5.20	X	6.32	5.67	5.18	3	3	4	4	3	4
18-B	216	293.7	X	6.56	6.45	5.82	5.25	X	6.69	6.34	5.31	3	4	4	4	5	5
19-G	198	243.1	X	6.59	6.53	5.90	5.32	X	6.67	6.35	5.37	2	4	4	4	5	5
20-B	200	279.2	6.17	6.10	5.90	5.31	5.30	X	6.68	6.31	5.28	1	2	2	2	4	4
21-B	200	272.3	X	6.55	6.37	5.50	5.30	X	6.32	5.55	5.32	3	4	4	3	4	4
22-G	174	237.0	X	6.66	6.59	6.13	5.26	X	6.49	6.02	5.27	2	4	4	1	3	4

*Postmortem inspection indicated slight pericarditis.
X Samples not obtained.

Appendix H. pH values of rectus femoris, biceps femoris and supraspinatus (Group IV).

	pH															
	Rectus femoris (RF)								Biceps femoris (BF) postmortem time				Supraspinatus (SS) postmortem time			
	RF-R postmortem time					RF-L postmortem time										
Animal	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr	0 hr	1 hr	2 hr	24 hr	0 hr	1 hr	2 hr	24 hr
1-G	6.35	6.51	6.45	6.17	X	X	6.04	X	6.57	6.58	6.32	X	6.64	6.45	6.33	X
2-G	6.49	6.57	6.53	6.30	5.48	6.58	6.12	5.51	6.50	6.58	6.40	5.39	6.68	6.61	6.45	5.77
3-B	6.35	6.15	6.22	5.63	5.30	6.23	5.38	5.32	6.47	6.50	5.54	5.34	6.32	6.00	5.77	5.67
4-B	6.45	X	6.54	X	5.27	6.50	X	5.41	6.75	6.68	6.36	5.38	6.55	6.39	6.28	5.43
5-G	6.36	X	6.30	X	5.45	6.21	X	5.33	6.46	6.55	6.13	5.44	6.51	6.37	6.34	5.58
6-G	6.46	X	6.45	X	5.38	6.34	X	5.30	6.43	6.43	6.24	5.39	6.50	6.50	6.35	5.58
7-G	6.39	X	6.44	X	5.48	6.43	X	5.47	6.48	6.60	6.43	5.45	6.58	6.62	6.31	5.63
8-G	6.56	X	6.43	X	5.35	6.55	X	5.40	6.52	6.74	6.64	5.36	6.69	6.58	6.49	5.64
9-G	6.38	X	6.43	X	5.50	6.56	X	5.49	6.45	6.61	6.23	5.37	6.49	6.49	6.42	5.66
10-B*	6.45	X	6.48	X	5.72	6.53	X	5.59	6.27	6.29	6.00	5.48	6.46	6.42	6.11	5.70
11-G	6.46	X	6.09	X	5.45	6.19	X	5.48	6.50	6.41	5.93	5.39	6.62	6.39	6.28	5.62
12-G	6.61	X	X	6.43	5.56	X	5.93	5.38	6.53	6.53	6.31	5.45	6.44	6.35	6.24	5.68
13-G	6.67	X	X	6.51	5.43	X	6.30	5.38	6.69	6.75	6.63	5.44	6.66	6.60	6.35	5.62
14-G	6.38	X	X	6.14	5.48	X	5.58	5.42	6.42	6.54	6.36	5.47	6.48	6.44	6.36	5.65
15-G	6.53	X	X	6.41	5.53	X	6.02	5.44	6.59	6.73	6.65	5.37	6.62	6.68	6.05	5.60
16-G	6.54	X	X	6.11	5.40	X	5.37	5.34	6.42	6.61	6.15	5.33	6.65	6.70	6.30	5.64
17-G	X	6.57	X	6.35	5.52	X	5.60	5.42	6.64	X	6.36	5.36	6.43	X	6.09	5.59
18-B	X	6.77	X	6.43	5.63	X	6.21	5.55	6.84	X	6.47	5.46	6.65	X	6.35	5.65
19-G	X	6.56	X	6.40	5.52	X	6.14	5.54	6.68	X	6.50	5.36	6.64	X	6.37	5.60
20-B	X	6.56	X	6.20	5.43	X	5.89	5.39	6.45	X	6.37	5.30	6.54	X	6.43	5.54
21-B	X	6.49	X	6.12	5.58	X	5.54	5.47	6.43	X	6.24	5.48	6.53	X	6.30	5.43
22-G	X	6.78	X	6.43	5.65	X	6.02	5.47	6.48	X	6.51	5.34	6.71	X	6.52	5.52

*Postmortem inspection indicated slight pericarditis.
X Samples not obtained.

Appendix I. Moisture and temperature of longissimus and rectus femoris muscles (Group IV).

	Moisture (%)						2 hr temp. (°F)		Temperature (°F)		
	LD-R		LD-L	RF-R		RF-L			LD-R		LD-L
Animal	0 hr	24 hr	24 hr	0 hr	24 hr	24 hr	RF-R	RF-L	0 hr	45 min	45 min
1-G	71.45	72.09	72.58	75.00	74.48	76.07	X	X	104.0	104.4	X
2-G	71.93	73.24	73.91	75.21	74.61	74.78	X	X	102.9	103.7	X
3-B	75.33	72.01	71.76	72.92	74.11	74.70	X	X	104.6	105.6	X
4-B	72.93	73.88	74.01	74.65	75.78	76.06	X	X	103.3	102.9	102.7
5-G	71.52	73.44	73.98	75.58	76.58	76.51	X	X	103.7	104.0	102.2
6-G	72.82	71.82	72.08	75.18	75.96	76.70	X	X	104.0	104.0	103.4
7-G	72.36	72.78	72.24	75.41	74.85	75.53	X	X	103.2	102.6	103.1
8-G	72.23	72.84	73.10	75.23	76.58	76.37	X	X	103.6	103.8	102.2
9-G	72.86	74.01	74.38	75.78	76.96	77.30	X	X	104.2	104.2	104.2
10-B*	70.35	72.96	72.16	75.86	74.33	75.41	X	X	103.6	103.7	101.6
11-G	72.97	73.57	73.48	75.22	75.90	76.02	X	X	103.5	103.6	104.0
12-G	71.85	73.62	72.61	74.86	75.50	76.16	X	X	103.8	104.0	103.4
13-G	68.89	71.92	72.96	75.30	76.26	76.83	X	X	102.2	102.0	102.0
14-G	71.90	73.82	73.50	72.67	76.45	76.92	X	X	102.4	102.6	102.9
15-G	70.64	72.12	71.54	73.24	75.37	76.22	X	X	100.6	100.2	101.6
16-G	71.54	73.24	72.58	73.08	76.02	76.43	X	X	104.0	104.3	103.8
17-G	73.96	74.30	74.22	75.96	74.52	76.90	93.0	101.6	104.0	103.6	103.6
18-B	72.78	74.76	74.46	76.66	76.40	77.48	92.3	102.2	102.2	102.1	102.2
19-G	71.44	73.49	73.29	75.07	74.23	76.69	94.1	102.0	103.0	103.1	102.0
20-B	74.86	74.89	75.06	75.99	76.85	77.43	94.8	99.4	102.6	102.0	102.0
21-B	74.02	74.48	74.95	76.07	76.78	75.82	95.7	100.1	104.6	105.1	103.3
22-G	74.78	74.84	75.56	75.89	76.28	76.36	91.5	101.2	102.5	102.2	103.3

*Postmortem inspection indicated slight pericarditis.
X Samples not obtained.

Appendix J. Transmission values (Group IV).

Animal	LD-R	LD-L	RF-R	RF-L	BF	SS
1-G	12.0	8.0	7.0	11.8	7.5	9.5
2-G	6.2	5.8	6.2	6.2	4.8	6.5
3-B	74.5	54.0	5.0	32.0	7.0	11.2
4-B	36.8	16.5	22.8	21.0	14.0	22.0
5-G	53.0	66.5	30.2	29.5	21.2	28.5
6-G	85.2	38.8	19.0	18.0	14.0	31.5
7-G	41.5	24.2	14.5	14.2	15.0	24.8
8-G	56.0	19.2	12.0	13.0	10.2	18.5
9-G	22.8	13.0	13.8	12.0	10.5	20.0
10-B*	32.2	16.5	17.5	19.2	16.0	23.5
11-G	80.5	89.2	14.5	18.8	22.7	23.0
12-G	12.0	12.0	9.8	9.8	8.0	12.8
13-G	9.0	15.2	12.5	11.2	9.5	16.0
14-G	21.5	17.5	12.5	12.8	9.5	18.5
15-G	9.8	19.5	9.8	8.0	6.8	11.8
16-G	54.8	57.2	12.0	7.8	9.5	12.5
17-G	11.2	13.2	11.0	10.5	8.8	13.0
18-B	21.8	11.8	10.2	11.0	8.8	14.8
19-G	13.5	8.2	11.0	10.2	8.8	13.8
20-B	95.0	18.5	11.5	11.2	11.5	15.2
21-B	48.8	33.2	12.0	18.0	14.0	15.2
22-G	18.0	37.0	11.5	13.5	13.2	18.0

*Postmortem inspection indicated slight pericarditis.

Appendix J. Transmission values (Group IV).

Animal	LD-R	LD-L	RF-R	RF-L	BF	SS
1-G	12.0	8.0	7.0	11.8	7.5	9.5
2-G	6.2	5.8	6.2	6.2	4.8	6.5
3-B	74.5	54.0	5.0	32.0	7.0	11.2
4-B	36.8	16.5	22.8	21.0	14.0	22.0
5-G	53.0	66.5	30.2	29.5	21.2	28.5
6-G	85.2	38.8	19.0	18.0	14.0	31.5
7-G	41.5	24.2	14.5	14.2	15.0	24.8
8-G	56.0	19.2	12.0	13.0	10.2	18.5
9-G	22.8	13.0	13.8	12.0	10.5	20.0
10-B*	32.2	16.5	17.5	19.2	16.0	23.5
11-G	80.5	89.2	14.5	18.8	22.7	23.0
12-G	12.0	12.0	9.8	9.8	8.0	12.8
13-G	9.0	15.2	12.5	11.2	9.5	16.0
14-G	21.5	17.5	12.5	12.8	9.5	18.5
15-G	9.8	19.5	9.8	8.0	6.8	11.8
16-G	54.8	57.2	12.0	7.8	9.5	12.5
17-G	11.2	13.2	11.0	10.5	8.8	13.0
18-B	21.8	11.8	10.2	11.0	8.8	14.8
19-G	13.5	8.2	11.0	10.2	8.8	13.8
20-B	95.0	18.5	11.5	11.2	11.5	15.2
21-B	48.8	33.2	12.0	18.0	14.0	15.2
22-G	18.0	37.0	11.5	13.5	13.2	18.0

*Postmortem inspection indicated slight pericarditis.

Appendix K. Glycogen levels[1] of longissimus and rectus femoris (Group IV).

	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
Animal	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
1-G	34.6	44.2	42.6	34.2	4.2	X	61.6	47.4	2.8	34.4	38.6	46.0	31.4	X	X	25.3	X
2-G	33.5	35.5	35.5	19.6	1.4	X	49.8	28.0	1.8	21.6	28.0	35.3	20.7	1.6	31.8	12.3	1.4
3-B	33.5	29.4	26.1	3.7	1.8	X	30.6	4.1	1.6	24.9	18.5	24.3	6.9	2.1	26.7	4.1	1.8
4-B	41.8	46.2	46.2	31.5	3.3	X	68.9	51.3	2.6	45.8	X	53.8	X	18.8	53.2	X	15.2
5-G	44.0	49.9	46.9	15.4	1.7	X	57.2	23.5	3.3	25.5	X	28.5	X	3.8	34.7	X	7.1
6-G	47.7	45.5	45.5	23.5	1.9	X	58.7	33.7	1.9	37.0	X	36.6	X	3.9	36.9	X	3.4
7-G	31.6	35.7	36.6	21.6	0.5	X	56.5	44.0	3.3	33.7	X	40.5	X	16.2	35.1	X	6.3
8-G	49.9	53.2	52.4	26.6	1.6	X	68.2	49.0	7.3	39.2	X	44.9	X	8.2	45.0	X	4.9
9-G	32.4	35.9	25.6	10.2	1.8	X	50.3	26.1	2.2	27.2	X	30.8	X	3.1	39.7	X	3.7
10-B	33.7	37.8	39.9	25.7	3.1	X	55.5	42.1	8.8	24.1	X	24.6	X	5.6	34.1	X	4.5
11-G	34.9	36.5	19.2	3.8	1.6	X	39.7	10.4	2.4	26.0	X	17.6	X	2.5	21.3	X	2.7
12-G	45.6	41.6	40.0	26.1	0.8	56.2	52.3	27.7	1.2	42.6	X	X	40.2	3.8	X	17.9	2.8
13-G	58.5	48.0	54.1	41.2	6.5	71.0	67.8	52.5	12.9	45.7	X	X	41.4	5.8	X	33.8	6.1
14-G	36.6	35.7	25.8	7.5	0.8	41.6	39.1	13.9	1.0	24.8	X	X	15.9	6.7	X	2.8	3.0
15-G	34.8	38.2	45.4	33.1	3.8	45.0	45.8	28.8	5.9	29.9	X	X	29.0	3.4	X	16.1	4.8
16-G	38.8	33.5	37.1	8.7	2.4	44.8	37.5	8.1	2.4	39.1	X	X	27.7	5.0	X	5.5	2.4
17-G	X	52.6	50.8	29.4	1.2	X	59.8	31.2	3.2	X	32.5	X	38.0	5.7	X	12.5	4.5
18-B	X	54.9	54.9	28.2	2.0	X	59.0	43.5	3.2	X	40.7	X	29.3	2.6	X	23.0	2.4
19-G	X	62.1	62.1	31.8	1.8	X	64.5	47.7	1.6	X	40.4	X	38.6	6.3	X	23.4	4.1
20-B	40.1	39.0	32.9	6.6	1.8	X	65.4	43.8	3.5	X	44.0	X	35.9	8.2	X	23.7	5.5
21-B	X	49.5	48.3	14.8	1.0	X	47.1	17.0	2.0	X	30.1	X	21.6	2.9	X	6.6	1.8
22-G	X	57.1	59.2	37.2	5.1	X	54.7	33.6	1.8	X	47.8	X	34.2	4.9	X	17.8	2.6

1μ moles glucose equivalent/g frozen, powdered muscle.
X Samples were not obtained.

Appendix K. Glycogen levels[1] of longissimus and rectus femoris (Group IV).

	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
		15	45		24	15	45		24		15	45		24	45		24
Animal	0 hr	min	min	2 hr	hr	min	min	2 hr	hr	0 hr	min	min	2 hr	hr	min	2 hr	hr
1-G	34.6	44.2	42.6	34.2	4.2	X	61.6	47.4	2.8	34.4	38.6	46.0	31.4	X	X	25.3	X
2-G	33.5	35.5	35.5	19.6	1.4	X	49.8	28.0	1.8	21.6	28.0	35.3	20.7	1.6	31.8	12.3	1.4
3-B	33.5	29.4	26.1	3.7	1.8	X	30.6	4.1	1.6	24.9	18.5	24.3	6.9	2.1	26.7	4.1	1.8
4-B	41.8	46.2	46.2	31.5	3.3	X	68.9	51.3	2.6	45.8	X	53.8	X	18.8	53.2	X	15.2
5-G	44.0	49.9	46.9	15.4	1.7	X	57.2	23.5	3.3	25.5	X	28.5	X	3.8	34.7	X	7.1
6-G	47.7	45.5	45.5	23.5	1.9	X	58.7	33.7	1.9	37.0	X	36.6	X	3.9	36.9	X	3.4
7-G	31.6	35.7	36.6	21.6	0.5	X	56.5	44.0	3.3	33.7	X	40.5	X	16.2	35.1	X	6.3
8-G	49.9	53.2	52.4	26.6	1.6	X	68.2	49.0	7.3	39.2	X	44.9	X	8.2	45.0	X	4.9
9-G	32.4	35.9	25.6	10.2	1.8	X	50.3	26.1	2.2	27.2	X	30.8	X	3.1	39.7	X	3.7
10-B	33.7	37.8	39.9	25.7	3.1	X	55.5	42.1	8.8	24.1	X	24.6	X	5.6	34.1	X	4.5
11-G	34.9	36.5	19.2	3.8	1.6	X	39.7	10.4	2.4	26.0	X	17.6	X	2.5	21.3	X	2.7
12-G	45.6	41.6	40.0	26.1	0.8	56.2	52.3	27.7	1.2	42.6	X	X	40.2	3.8	X	17.9	2.8
13-G	58.5	48.0	54.1	41.2	6.5	71.0	67.8	52.5	12.9	45.7	X	X	41.4	5.8	X	33.8	6.1
14-G	36.6	35.7	25.8	7.5	0.8	41.6	39.1	13.9	1.0	24.8	X	X	15.9	6.7	X	2.8	3.0
15-G	34.8	38.2	45.4	33.1	3.8	45.0	45.8	28.8	5.9	29.9	X	X	29.0	3.4	X	16.1	4.8
16-G	38.8	33.5	37.1	8.7	2.4	44.8	37.5	8.1	2.4	39.1	X	X	27.7	5.0	X	5.5	2.4
17-G	X	52.6	50.8	29.4	1.2	X	59.8	31.2	3.2	X	32.5	X	38.0	5.7	X	12.5	4.5
18-B	X	54.9	54.9	28.2	2.0	X	59.0	43.5	3.2	X	40.7	X	29.3	2.6	X	23.0	2.4
19-G	X	62.1	62.1	31.8	1.8	X	64.5	47.7	1.6	X	40.4	X	38.6	6.3	X	23.4	4.1
20-B	40.1	39.0	32.9	6.6	1.8	X	65.4	43.8	3.5	X	44.0	X	35.9	8.2	X	23.7	5.5
21-B	X	49.5	48.3	14.8	1.0	X	47.1	17.0	2.0	X	30.1	X	21.6	2.9	X	6.6	1.8
22-G	X	57.1	59.2	37.2	5.1	X	54.7	33.6	1.8	X	47.8	X	34.2	4.9	X	17.8	2.6

[1] μ moles glucose equivalent/g frozen, powdered muscle.
X Samples were not obtained.

Appendix L. Lactic acid levels[1] of longissimus and rectus femoris (Group IV).

Animal	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
1-G	24.2	29.0	38.6	48.1	103.8	X	21.0	32.1	90.0	11.9	8.0	15.9	42.1	X	X	48.6	X
2-G	38.0	38.2	48.7	59.0	96.0	X	20.3	36.3	93.8	21.3	12.9	16.7	37.0	82.8	20.9	42.8	79.8
3-B	56.0	60.0	77.6	99.9	97.4	X	70.2	101.7	102.6	37.0	43.8	40.2	80.2	67.7	41.9	77.9	75.0
4-B	36.6	38.2	53.8	62.7	98.6	X	18.9	44.6	95.6	28.2	X	22.6	X	75.4	28.1	X	89.9
5-G	47.8	44.6	51.0	73.3	71.6	X	35.8	59.9	78.4	35.0	X	39.8	X	75.8	42.7	X	78.0
6-G	37.0	38.2	37.0	61.7	90.0	X	22.8	52.4	99.2	26.5	X	31.3	X	71.1	34.6	X	76.7
7-G	51.9	63.6	66.9	78.2	90.0	X	29.9	48.4	96.8	32.6	X	29.7	X	71.9	27.7	X	76.0
8-G	41.5	44.5	50.6	69.7	95.0	X	27.9	44.3	100.8	23.2	X	27.2	X	82.5	25.5	X	83.6
9-G	47.8	65.3	57.8	92.4	82.4	X	29.6	48.7	92.4	19.1	X	41.2	X	69.4	19.3	X	67.5
10-B	35.5	55.0	53.1	61.4	91.0	X	25.0	36.3	92.8	29.0	X	28.7	X	67.6	29.1	X	66.1
11-G	44.4	55.5	72.1	95.1	100.8	X	41.0	89.4	89.0	20.3	X	64.4	X	68.1	51.9	X	74.1
12-G	38.8	44.2	45.0	64.6	79.0	40.5	32.5	67.1	94.0	17.9	X	X	32.0	56.5	X	82.3	84.0
13-G	37.9	39.8	47.4	51.3	101.0	10.5	20.5	24.0	86.4	8.8	X	X	20.3	59.4	X	40.7	66.3
14-G	45.5	48.4	63.1	78.0	85.4	38.1	36.5	76.6	90.8	31.9	X	X	47.6	74.7	X	88.0	76.2
15-G	41.3	46.7	48.9	50.6	77.7	38.7	41.7	62.8	84.6	20.3	X	X	33.0	75.2	X	66.3	80.6
16-G	40.6	48.8	60.5	101.8	97.2	33.8	43.9	99.1	90.2	20.3	X	X	40.0	78.3	X	69.4	81.0
17-G	X	20.7	34.6	55.4	84.0	X	30.0	73.3	89.4	X	20.0	X	32.4	75.2	X	99.0	89.3
18-B	X	18.9	26.2	63.4	102.6	X	13.5	35.4	97.8	X	10.3	X	26.1	63.6	X	41.6	65.1
19-G	X	21.6	31.4	57.0	81.5	X	21.0	34.5	85.2	X	19.8	X	32.7	74.7	X	49.2	75.2
20-B	47.4	55.0	57.0	88.4	78.8	X	21.3	42.6	85.4	X	22.9	X	37.5	66.3	X	78.9	67.0
21-B	X	28.8	39.4	59.6	75.4	X	34.9	54.0	100.8	X	26.1	X	49.9	70.5	X	73.1	69.8
22-G	X	20.3	25.0	49.6	105.6	X	28.9	47.6	111.2	X	12.0	X	29.2	77.0	X	61.5	78.8

[1] μ moles/g frozen, powdered muscle.
X Samples were not obtained.

Appendix M. Glucose 6-phosphate levels[1] of longissimus and rectus femoris (Group IV).

Animal	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
1-G	9.09	5.42	4.12	6.38	9.63	X	0.57	1.81	9.20	5.08	2.01	1.56	1.65	X	X	2.36	X
2-G	4.18	2.07	1.08	2.51	4.36	X	0.90	0.54	4.80	2.24	0.81	0.14	0.41	2.56	0.14	0.95	0.54
3-B	3.37	3.41	3.05	4.40	3.95	X	3.55	4.58	4.71	4.67	4.12	1.69	2.97	3.50	0.68	3.40	3.50
4-B	3.84	1.61	1.25	2.59	7.78	X	0.18	0.63	7.04	3.68	X	0.53	X	7.66	0.44	X	10.74
5-G	3.93	2.14	1.52	5.54	6.66	X	1.25	4.16	7.27	4.74	X	1.33	X	8.74	2.11	X	5.77
6-G	4.56	2.04	1.30	4.56	6.99	X	0.49	2.04	8.12	3.19	X	0.56	X	6.80	0.76	X	5.83
7-G	7.04	4.40	2.98	5.55	6.23	X	1.08	1.22	7.88	6.81	X	0.61	X	13.92	0.87	X	10.68
8-G	5.63	3.11	1.83	6.13	9.40	X	0.81	1.97	10.94	4.23	X	0.90	X	9.01	0.66	X	8.36
9-G	6.90	4.56	2.74	4.36	4.87	X	0.61	1.67	5.22	2.98	X	0.71	X	6.38	0.30	X	4.07
10-B	4.89	5.96	2.67	4.21	7.69	X	0.82	0.92	7.69	3.52	X	0.71	X	8.20	0.40	X	7.98
11-G	6.13	4.61	6.13	7.45	6.69	X	4.82	5.88	6.84	4.62	X	1.88	X	3.47	2.74	X	2.98
12-G	5.08	3.42	1.25	3.35	4.98	1.04	0.52	3.14	5.76	1.55	X	X	0.18	7.02	X	2.76	5.63
13-G	5.20	3.73	2.05	2.80	8.80	0.84	0.84	0.48	10.65	1.41	X	X	0.30	7.54	X	1.79	7.74
14-G	6.13	4.84	4.39	5.76	5.27	5.14	4.24	5.43	5.12	5.05	X	X	2.40	7.45	X	1.70	2.48
15-G	6.65	4.13	2.04	2.67	7.79	3.34	1.99	4.62	7.36	4.15	X	X	0.65	7.34	X	2.50	7.55
16-G	6.67	6.15	3.57	6.55	6.62	4.47	3.31	6.48	6.21	3.42	X	X	2.08	5.73	X	5.96	5.12
17-G	X	2.56	0.72	5.63	8.22	X	0.51	4.57	9.75	X	3.20	X	1.71	9.82	X	3.90	5.25
18-B	X	1.29	0.76	3.40	7.44	X	0.38	0.33	6.80	X	0.16	X	0.58	4.67	X	1.43	5.26
19-G	X	1.75	0.31	2.98	6.20	X	0.31	0.51	5.52	X	2.50	X	0.70	14.57	X	1.70	9.06
20-B	8.12	5.75	3.19	6.99	7.14	X	0.41	0.82	6.50	X	1.75	X	1.50	6.40	X	3.30	6.01
21-B	X	1.31	1.02	4.96	6.45	X	1.02	4.44	7.73	X	2.39	X	1.86	6.58	X	3.13	3.72
22-G	X	0.91	0.28	1.60	8.12	X	0.45	2.64	7.58	X	0.45	X	0.40	7.38	X	2.50	5.40

[1] μ moles/g frozen, powdered muscle.
X Samples were not obtained.

Appendix N. ATP levels[1] of longissimus and rectus femoris (Group IV).

Animal	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
1-G	1.66	1.63	0.96	0.71	--	X	3.26	1.76	--	3.22	3.12	3.46	1.80	X	X	0.91	X
2-G	2.38	2.78	2.51	0.36	--	X	4.94	1.98	--	3.64	4.33	4.46	2.97	--	4.66	1.15	0.09
3-B	1.44	1.21	0.54	--	--	X	0.76	--	--	1.98	1.49	2.23	0.34	0.22	1.76	0.06	0.07
4-B	2.68	2.86	2.41	1.07	--	X	4.29	2.59	--	3.61	X	3.30	X	--	3.39	X	--
5-G	2.14	2.77	2.23	0.57	--	X	3.13	0.90	--	1.78	X	1.73	X	--	1.56	X	--
6-G	2.85	2.77	2.69	0.73	--	X	3.67	1.30	--	3.15	X	2.45	X	--	1.99	X	--
7-G	0.47	0.54	1.02	--	--	X	3.59	1.62	--	3.06	X	2.30	X	0.65	2.24	X	0.33
8-G	3.02	1.90	1.42	--	--	X	3.32	1.44	--	2.86	X	2.55	X	--	2.96	X	--
9-G	1.77	1.52	1.22	0.25	--	X	4.72	1.17	0.01	2.55	X	2.69	X	--	3.40	X	--
10-B	1.79	2.16	2.78	1.13	--	X	5.14	2.68	--	1.99	X	2.14	X	0.32	3.24	X	0.22
11-G	2.38	1.93	0.30	0.01	--	X	2.38	--	--	2.68	X	1.16	X	0.06	1.22	X	0.06
12-G	2.21	2.56	2.30	1.19	--	4.23	3.76	1.04	--	3.85	X	X	3.95	0.25	X	0.66	--
13-G	3.31	3.36	2.89	1.77	--	4.52	8.15	3.62	--	4.02	X	X	3.50	0.24	X	2.08	0.22
14-G	0.83	0.76	--	--	--	1.21	1.13	0.05	--	3.15	X	X	1.40	0.32	X	--	0.11
15-G	2.10	1.93	1.88	1.44	--	2.93	2.30	0.93	--	4.15	X	X	2.50	0.21	X	0.80	0.33
16-G	2.36	1.52	1.16	0.20	--	3.42	1.94	0.14	0.07	3.42	X	X	1.19	--	X	0.07	--
17-G	X	3.62	3.39	0.50	--	X	4.01	1.05	--	X	4.20	X	2.11	--	X	0.15	0.15
18-B	X	2.80	2.57	0.94	--	X	2.65	3.26	--	X	4.46	X	3.29	0.05	X	1.70	0.05
19-G	X	5.03	4.72	1.54	--	X	5.55	2.98	--	X	2.90	X	2.65	0.43	X	1.30	0.22
20-B	2.57	1.96	0.82	0.31	--	X	5.45	2.78	--	X	3.55	X	2.15	0.38	X	0.60	0.20
21-B	X	3.24	2.39	--	--	X	2.10	--	--	X	3.19	X	1.49	--	X	0.11	--
22-G	X	3.41	3.35	1.42	--	X	3.07	0.85	--	X	3.30	X	3.25	0.29	X	0.70	--

[1] μ moles/g frozen, powdered muscle.
X Samples were not obtained.
--Levels were not detectable.

Appendix 0. Creatine phosphate levels[1] of longissimus and rectus femoris (Group IV).

Animal	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
1-G	1.71	1.05	0.85	1.15	--	X	1.30	1.25	--	0.37	0.35	0.66	--	X	X	--	X
2-G	0.09	0.13	--	--	--	X	2.65	--	--	1.39	3.24	4.06	0.34	--	0.95	0.34	--
3-B	--	--	--	--	--	X	--	--	--	0.13	0.47	0.61	0.41	0.37	0.34	0.24	0.30
4-B	0.89	0.54	0.18	0.27	--	X	3.31	0.63	--	1.65	X	1.19	X	--	0.79	X	--
5-G	0.80	0.80	0.54	0.33	--	X	1.25	0.33	--	0.20	X	0.31	X	--	0.55	X	--
6-G	0.73	0.57	0.41	0.33	--	X	0.73	0.33	--	0.93	X	0.26	X	--	0.15	X	--
7-G	0.54	0.27	0.27	--	0.09	X	2.03	0.34	0.05	--	X	0.56	X	--	0.10	X	--
8-G	0.34	0.61	0.27	--	--	X	1.35	0.38	--	1.28	X	--	X	--	0.71	X	--
9-G	--	--	--	--	--	X	0.56	--	--	0.61	X	0.41	X	0.18	0.20	X	0.18
10-B	0.49	0.10	0.41	0.21	0.28	X	3.39	0.30	--	0.41	X	0.36	X	--	0.68	X	--
11-G	--	--	--	--	--	X	--	--	--	1.03	X	--	X	0.12	--	X	0.12
12-G	0.55	--	0.16	0.23	--	2.14	0.26	0.16	--	1.25	X	X	0.60	0.17	X	--	--
13-G	0.53	--	--	--	--	5.73	1.89	0.34	--	7.74	X	X	0.89	0.30	X	0.30	0.45
14-G	0.08	0.15	0.15	--	--	0.30	0.61	0.09	--	--	X	X	--	--	X	0.35	0.11
15-G	0.34	0.37	0.26	0.21	--	0.42	0.21	0.20	--	--	X	X	--	--	X	--	--
16-G	--	--	--	0.20	--	0.47	--	0.20	--	2.61	X	X	0.45	--	X	0.30	--
17-G	X	0.35	--	0.05	--	X	0.51	0.05	--	X	0.90	X	--	--	X	--	0.17
18-B	X	2.57	1.36	0.14	--	X	2.95	0.28	--	X	9.08	X	0.58	0.27	X	0.42	0.27
19-G	X	4.73	2.16	--	--	X	3.70	0.11	--	X	0.95	X	--	--	X	0.15	0.11
20-B	0.30	0.30	--	--	--	X	5.65	0.10	--	X	--	X	--	0.54	X	--	--
21-B	X	1.02	0.40	--	--	X	0.17	--	--	X	1.22	X	0.48	--	X	0.27	--
22-G	X	2.33	0.68	--	--	X	0.45	--	--	X	0.65	X	--	--	X	--	--

[1] μ moles/g frozen, powdered muscle.
X Samples were not obtained.
--Levels were not detectable.

Appendix P. Glucose-1-phosphate, fructose-6-phosphate and glucose levels[1] of longissimus and rectus femoris (Group IV).

Animal	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
Glucose-1-phosphate																	
2-G	0.11	0.14	0.11	0.33	--	X	0.12	0.06	--	0.13	0.12	0.03	0.14	--	0.04	0.22	0.05
3-B	0.11	0.27	0.22	--	--	X	0.14	0.04	--	0.03	0.60	0.52	0.49	1.43	0.27	0.48	1.58
9-G	0.14	0.14	0.13	0.81	0.13	X	0.08	0.32	0.25	0.36	X	0.13	X	0.46	0.09	X	0.44
11-G	0.31	0.38	0.45	0.67	0.53	X	0.38	0.70	0.46	0.64	X	0.26	X	0.46	0.66	X	0.43
13-G	0.19	0.22	0.29	0.35	0.23	0.40	0.39	0.33	0.33	1.06	X	X	1.05	3.19	X	1.83	2.60
16-G	0.13	0.68	0.73	0.36	0.35	0.20	0.16	0.32	0.29	1.89	X	X	1.34	0.18	X	2.01	0.23
18-B	X	X	X	X	X	X	X	X	X	X	0.33	X	0.64	1.24	X	0.64	1.28
21-B	X	X	X	X	X	X	X	X	X	X	0.74	X	0.84	1.71	X	0.91	0.94
Fructose-6-phosphate																	
2-G	6.01	5.06	3.53	3.20	1.88	X	3.71	2.65	2.04	4.35	4.29	2.43	2.97	1.22	1.73	3.19	0.45
3-B	5.28	4.93	3.99	1.93	1.73	X	4.49	2.09	2.08	4.94	5.71	4.68	2.69	1.68	3.45	2.06	1.60
9-G	4.96	4.19	2.86	2.38	1.96	X	2.78	2.84	1.95	3.90	X	2.09	X	3.28	1.69	X	2.16
11-G	4.88	3.91	2.72	2.54	2.51	X	4.41	2.73	2.44	4.47	X	2.64	X	2.20	3.51	X	1.83
13-G	5.80	4.91	3.22	2.41	3.23	2.49	2.44	2.19	3.60	3.38	X	X	1.57	2.28	X	2.71	2.34
16-G	6.78	5.17	3.51	2.83	2.86	5.23	4.66	2.96	2.38	4.09	X	X	2.75	2.26	X	2.43	2.19
18-B	X	X	X	X	X	X	X	X	X	X	1.74	X	2.26	2.44	X	3.19	2.85
21-B	X	X	X	X	X	X	X	X	X	X	4.08	X	3.07	3.77	X	2.41	2.37
Glucose																	
2-G	1.66	1.85	1.98	3.02	4.93	X	0.71	1.56	4.19	1.05	1.36	0.60	0.68	3.41	0.34	1.18	3.13
3-B	2.30	2.41	2.77	4.37	5.61	X	2.76	4.36	6.26	1.55	2.62	2.21	3.72	5.32	1.78	3.88	4.90
9-G	3.27	3.38	3.31	3.40	5.07	X	1.65	2.32	4.32	2.23	X	1.82	X	3.93	1.04	X	5.23
11-G	2.86	2.95	4.11	5.27	7.09	X	2.82	3.88	6.07	2.27	X	2.85	X	4.59	2.43	X	4.79
13-G	1.89	2.07	2.14	2.81	7.39	0.55	1.54	0.88	5.53	1.05	X	X	0.76	3.24	X	1.82	3.74
16-G	1.80	2.58	2.95	4.73	6.24	1.76	2.49	4.91	6.58	1.81	X	X	2.50	4.33	X	4.27	4.91
18-B	X	X	X	X	X	X	X	X	X	X	0.31	X	0.65	2.13	X	1.37	2.86
21-B	X	X	X	X	X	X	X	X	X	X	1.70	X	1.96	3.21	X	3.09	3.79

[1] μ moles/g frozen, powdered muscle.
X Samples were either not analyzed or were not obtained.
--Levels were not detectable.

Appendix Q. ADP and AMP levels[1] of longissimus and rectus femoris (Group IV).

Animal	LD-R postmortem time					LD-L postmortem time				RF-R postmortem time					RF-L postmortem time		
	0 hr	15 min	45 min	2 hr	24 hr	15 min	45 min	2 hr	24 hr	0 hr	15 min	45 min	2 hr	24 hr	45 min	2 hr	24 hr
ADP																	
2-G	2.97	3.01	3.01	2.22	0.49	X	1.42	2.65	0.53	1.58	1.33	1.09	2.10	0.56	1.33	1.76	0.40
3-B	3.09	3.01	2.33	0.61	0.49	X	2.93	0.68	0.47	2.45	2.01	1.59	1.25	0.78	2.18	1.01	0.70
9-G	7.24	6.10	7.42	7.30	7.94	X	2.89	6.54	7.18	1.80	X	5.14	X	0.28	4.26	X	0.45
11-G	6.54	6.68	7.31	8.67	9.65	X	6.72	7.73	9.09	1.55	X	7.00	X	0.28	6.79	X	0.28
13-G	4.23	4.86	4.23	4.23	8.18	2.12	3.32	2.06	4.58	1.72	X	X	2.11	1.09	X	3.04	0.62
16-G	5.55	6.12	5.78	5.72	7.44	4.63	5.49	5.78	7.61	2.34	X	X	2.65	4.75	X	1.09	4.92
18-B	X	X	X	X	X	X	X	X	X	X	0.95	X	1.37	0.71	X	2.08	0.42
21-B	X	X	X	X	X	X	X	X	X	X	1.49	X	1.72	0.60	X	0.77	0.24
AMP																	
2-G	0.38	0.40	0.48	0.06	0.01	X	0.24	0.34	--	0.18	0.19	0.28	0.49	0.08	0.45	0.61	0.08
3-B	0.53	0.56	0.58	--	--	X	0.54	--	0.04	0.34	0.74	0.82	0.70	0.28	0.70	0.45	0.24
9-G	0.62	1.50	0.66	0.67	0.56	X	1.42	0.82	0.56	0.57	X	0.53	X	0.36	0.49	X	0.28
11-G	0.54	0.56	0.56	0.50	0.31	X	0.64	0.65	0.48	0.57	X	0.78	X	0.11	0.76	X	0.36
13-G	0.37	0.57	0.34	0.31	0.26	0.11	0.14	0.14	0.23	0.04	X	X	0.08	0.04	X	0.24	0.20
16-G	0.29	0.40	0.40	0.06	0.29	0.26	0.60	0.20	0.20	0.20	X	X	0.43	0.11	X	0.08	0.23
18-B	X	X	X	X	X	X	X	X	X	X	0.09	X	0.18	--	X	0.27	0.09
21-B	X	X	X	X	X	X	X	X	X	X	0.09	X	0.21	0.14	X	0.15	0.06

[1] μ moles/g frozen, powdered muscle.
X Samples were either not analyzed or not obtained.
--Levels were not detectable.