1969

Influence of Aldosterone on the Quality of Porcine Muscle.

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QUALITY OF PORCINE MUSCLE.

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INFLUENCE OF ALDOSTERONE ON THE QUALITY
OF PORCINE MUSCLE

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Animal Science

by
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B.S., Mississippi State University, 1963
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ABSTRACT

The influence of aldosterone and aldosterone-blocking agents on porcine muscle quality, and the possible role of aldosterone in the etiology of pale, soft, exudative (PSE) porcine musculature were studied. Preliminary investigations indicated that an intravenous injection of 1.5 mg. of aldosterone, followed by an hour and a half equilibration period, would produce PSE musculature. Also, an oral drench of 500 mg. of Aldactazide followed by a 30 min. equilibration period would prevent the development of PSE musculature resulting from a 30 min. stress period with an electric prod.

Thirty-four Poland China pigs, approximately 95 kg., were divided into three trials of 12, 10 and 12 pigs each and treated with 1.5 mg. aldosterone, 250 mg. Aldactone and 500 mg. Aldactazide, respectively. The pigs in the latter two trials were stressed for 30 minutes. The animals were slaughtered and serum and muscle (longissimus dorsi) samples were taken for sodium and potassium determinations. After chilling for 24 hrs. at 3°C, the carcasses were cut and subjective quality scores applied by a three-member panel. The l. dorsi muscle was sampled again for determinations of objective estimates of quality.

The injection of 1.5 mg. of aldosterone resulted in a highly significant ($P < .01$) decrease in Wisconsin Pork Quality Score from 2.8 to 1.8. A similar trend of a reduction in quality was obtained for the objective evaluations. Likewise, the oral administration of 500 mg. of Aldactazide significantly ($P < .05$) increased the Wisconsin Pork Quality Score from 1.2 to 2.5, and improved the objective measure of protein turbidity by reducing the percent transmission from 93.8 to 70.5. The oral administration of 250 mg. of Aldactone tended to improve the objective measures of quality, but not the subjective measures.

No statistically significant differences were obtained for either serum or muscle sodium or potassium values in this study.

It was postulated that a hyperproduction of aldosterone resulted in a depletion of muscle potassium, inactivating pyruvate kinase, causing a rapid accumulation of lactic acid, resulting in the development of PSE muscle. Results obtained did not substantiate the above hypothesis.

Four Hampshire pigs, approximately 95 kg., were subjected to treatments similar to those receiving aldosterone and Aldactazide in order to observe differences in pyruvate kinase activity of muscle and liver. Treatments failed to produce quality differences with regard to PSE conditions; therefore, data obtained on enzyme activity were inconclusive toward substantiating a relationship to PSE conditions.

INTRODUCTION

The influence of the endocrine system on the physiological and biochemical etiology of porcine muscle quality, particularly pale, soft, exudative (PSE) musculature, is rapidly becoming more evident. It has been suggested that selection for the "meat-type" hog, one with a greater portion of the carcass composed of ham and loin, has altered the hormonal homeostasis to such an extent that these animals cannot cope with even normal "stressors." The apparent failure of these animals to compensate for certain stress conditions has resulted in a reduction of muscle quality through the development of pale, soft, exudative musculature in their carcasses.

Research over the past 15 years shows many factors to be related to the development of PSE muscle. This condition has been shown to vary with breed and amount of muscling, as well as environmental conditions. It appears when there is a rapid drop in pH (<5.3) post-mortem while the carcass is at a high temperature ($>40^{\circ}\text{C}$). This results in a precipitation of the sarcoplasmic proteins onto the myofibrillar proteins; thereby reducing the ability of the muscle to bind water.

It has been observed in this condition that a rapid

post-mortem glycolytic rate occurred. Therefore, researchers have investigated some of the hormones which have been shown to influence the rate of glycolysis. Particular attention has been given to hormones of the thyroid and adrenal glands. Within the adrenal gland, major emphasis has centered around the glucocorticoids. However, Henry et al. (1958) reported that pigs with PSE musculature had hyperproduction of aldosterone and somatotropin with a concomitant deficiency of adrenocorticotropin and glucocorticoids.

The objectives of this study were to: 1) study the relationship between aldosterone and aldosterone-blocking agents toward PSE musculature, and 2) explore the role or mode of action of aldosterone in the etiology of PSE musculature.

LITERATURE REVIEW

Pork Muscle Quality

The problem of inferior muscle quality resulting from the development of pale, soft, exudative musculature was first reported by Ludvigsen (1954) as "muskeldegeneration." Since that time many workers have studied this condition in an attempt to elucidate the basic cause or causes. Extensive reviews have been published by Briskey (1957, 1963, 1964), Breidenstein (1963), Bray (1966) and Cassens (1966).

Porcine muscles have been found to have extremely variable post-mortem glycolytic rates (Briskey, 1963). Various types of ante-mortem stress immediately prior to slaughter have been shown to affect the post-mortem changes in porcine muscle (Briskey et al., 1959; Sayre et al., 1961, 1963a, b; Kastenschmidt et al., 1964, 1965). These changes resulted in a rapid accumulation of lactic acid post-mortem while the carcass was at a high temperature, and a subsequent development of the PSE condition (Briskey, 1964; Briskey et al., 1966). Bendall and Wismer-Pedersen (1962) reported that the drop in pH at this temperature caused a precipitation of the sarcoplasmic proteins onto the myofibrillar proteins, thereby reducing the

water-holding capacity of these muscles. However, Bodwell et al. (1966) reported that even though a loss in fibrillar water-binding capacity occurred as a consequence of a low pH and high temperature, a characteristic frequently encountered in soft, watery muscle, low fibrillar water-binding capacity by itself was not the primary causal factor in making pork muscle appear soft and watery.

In an attempt to understand what was responsible for the variability in the post-mortem glycolytic rate of pork muscle, Kastenschmidt et al. (1958) studied the levels of glycolytic intermediates and cofactors in "slow glycolyzing" muscles from Hampshire and Chester White animals and compared them to levels found in "fast-glycolyzing" muscles in Poland China animals. They found the adenine nucleotide levels were the primary regulator factors for phosphorylase, and that phosphorylase was the primary control site of post-mortem glycolysis. However, phosphofructokinase and pyruvate kinase were also involved in controlling post-mortem glycolysis. Significant differences in blood lactate or glucose levels were observed among the groups of pigs.

Hormonal Influences on PSE Muscle

It has been shown that development of PSE muscle was associated with various stress conditions prior to slaughter. Since any stress condition interferes with the

homeostatic state of the animal, and since the animal's hormonal systems tend to maintain homeostasis, PSE research has attempted to better characterize this relationship. Ludvigsen (1954) suggested that, since the PSE condition was more prevalent in pigs of breeds which have had intensive selection for the ability to grow muscle, there had been automatic selection of a high content of growth stimulating hormone (GSH) in the pituitary and thereby a lower content of thyroid stimulating hormone (TSH) and adrenocorticotrophic hormone (ACTH).

Limited and conflicting information is available on the effect of the thyroid hormones on PSE pork. Ludvigsen (1954) and Briskey (1963) reported that thiouracil-induced hypothyroidism in pigs resulted in pale, soft, exudative musculature. Judge et al. (1965) observed that pigs with severe PSE musculature had elevated plasma levels of protein bound I^{131} , indicating that thyroid hormone and thyroid gland status were related to various post-mortem muscle properties. However, Topel and Merkel (1966) using methylthiouracil and tapazole failed to confirm previous findings. These workers obtained only slight PSE musculature in ham muscles of some methylthiouracil-fed pigs, but no PSE condition occurred in those fed tapazole. Topel and Merkel (1966) also noted a decrease in adrenal gland weight as a result of these goitrogens, and concluded that it appeared the development of PSE musculature was not associated

with hypothyroidism per se, but may be the indirect result from the secondary effects of methylthiouracil upon the adrenal gland.

Two classes of hormones are produced by the adrenal gland. Catecholamines are produced by the medulla and steroids are produced by the cortex. In work concerned with the catecholamines, Trautman et al. (1963) showed that the oral administration of adrenalin prevented the development of the PSE condition, marked by reduced glycogen levels, and retarded pH reduction. Radouco-Thomas (1962) reported that combining adrenalin with ascorbic acid protected against the condition. Aberle and Merkel (1968) reported no beneficial effects from intramuscular injections of epinephrine. They attributed the failure to confirm previous findings to an insufficient period of time between injection and slaughter. Topel (1968) suggested a possible interaction between the catecholamines and the glucocorticoids on micro-circulatory integrity as related to PSE muscle. Merkel (1968) implied that insufficiency of the domestic pig's circulatory system affected his ability to respond to releases of adrenal medullary hormones.

Steroids produced by the cortex consist of two groups of structurally similar compounds having different physiological functions. Most of the attention in PSE research has been focused on the glucocorticoids, since these

hormones influence carbohydrate metabolism. Based on previous finding of muscle degeneration in rabbits (Germuth et al., 1951; Ellis, 1956) and myopathy in dogs (Faludi et al., 1964) following administration of various glucocorticoids, and since low ACTH levels have been implicated in PSE muscle, Topel and Merkel (1967) used prednisolone and methyprednisolone on pigs. They were successful in obtaining adrenal atrophy and lower 17-hydroxycorticosterone levels, but failed to produce PSE musculature. In previous work Topel et al. (1967) reported slightly lower levels of plasma 17-hydroxycorticosterone in pigs with severe PSE musculature. These workers attributed the failure to demonstrate this in the second experiment to the gluconeogenic effects of the glucocorticoids used upon the ultimate extent and rate of post-mortem anaerobic glycolysis.

The mineralocorticoids, which regulate mineral metabolism or electrolyte balance, have received little consideration in the study of PSE muscle. Henry et al. (1958) reported that pigs with PSE musculature had hyperproduction of aldosterone and somatotropin with a concomitant deficiency of adrenocorticotropin and glucocorticoids. However, Topel and Merkel (1966) and Topel et al. (1967), conducting research closely allied but not directly related to this problem, failed to support the findings of Henry.

Aldosterone

Aldosterone was first isolated by Tait et al. (1952) from an amorphous fraction remaining from adrenal cortical extracts after substances possessing potent "glucocorticoid" activity had been removed. Simpson et al. (1953) elucidated the chemical structure, characterizing it as the 13-aldehyde of corticosterone. Voluminous literature has accumulated regarding the biochemistry and physiology of this hormone. Extensive reviews have been made by Laragh and Kelly (1964) and Ross (1965).

However, in spite of the wealth of information available on aldosterone, there is a paucity of knowledge regarding its influence on electrolyte metabolism in muscle, particularly porcine muscle. Therefore, this review will deal only with those generally accepted concepts which are pertinent to the role of aldosterone in porcine muscle quality.

Physiological Effects of Aldosterone

Aldosterone, produced by the zona glomerulosa of the adrenal cortex, functions in modifying the rate of transport and the distribution of sodium, potassium, and hydrogen ions across cellular membranes. Although it may possibly affect many if not all tissues, its most important function appears to be promotion of sodium retention and potassium elimination by renal tubular cells.

The extrarenal effects of aldosterone are not well defined and their physiological significance still remains questionable. However, certain experiments suggest that aldosterone may act to support both myocardial and circulatory function and may therefore be useful in treatment of circulatory failure. Ballard et al. (1960) and Tanz (1962) reported a positive inotropic effect of relatively low concentrations of the hormone on myocardial muscle in vitro. Bein and Jacques (1960) and Spink and Vick (1961) found that aldosterone administration protected various animals from endotoxic shock and that it may also potentiate the pressor effects of pressor agents in this type shock. Combination of aldosterone with angiotension was especially effective in this situation. Levy and Richards (1962) investigated the effect of aldosterone on the electrolyte content and the contractile properties of isolated rabbit atrial muscle. The hormone increased sodium and decreased potassium content of this muscle. Higher concentrations of aldosterone produced a slightly negative inotropic effect, whereas lower concentrations had no effect. However, Woodbury and Koch (1957), working from the basis that patients with Conn's syndrome (hyperproduction of aldosterone) showed an increased sodium and decreased potassium content in muscle, were unable to demonstrate similar ionic alterations following subcutaneous injections of 20 μ g of aldosterone into mice.

Therefore, it appears aldosterone influences tissues other than those of the kidney. However, the exact nature and effect of this influence are obscure.

Control of Aldosterone Secretion

The exact regulating mechanism of aldosterone secretion is another area in which many questions remain unanswered. Substances at present which are known to influence the rate of secretion include the plasma potassium concentration, corticotrophin (ACTH), and angiotension II (Ross, 1965). The latter is associated with the influence of blood pressure on the juxtaglomerular cells and their release of renin. Beck et al. (1957) reported that an increase in growth hormone increased the amount of aldosterone excreted in the urine, but its secretion could hardly constitute the normal mode of control. Mills et al. (1958) increased aldosterone secretion in dogs by obstructing the thoracic inferior vena cava. Vogt (1960) suggested the secretion of aldosterone to be under the influence of the central nervous system.

Abnormalities of Aldosterone Secretion

Aldosterone abnormalities are generally accompanied by the following: 1) disorders characterized by potassium wastage, 2) conditions in which there is progressive sodium retention and edema, and 3) states characterized mainly by arterial hypertension.

Primary aldosteronism (Conn's syndrome) arises from an oversecretion resulting from adrenal cortex adenoma. It is characterized by hypertension and severe potassium depletion, manifested by hypokalemic alkalosis and expressed by periodic muscular paresis or paralysis and occasional tetany. Sodium is retained within the intracellular compartment and although extracellular volume is expanded and plasma sodium concentrations tend to be high, edema does not occur (Ross, 1965).

Secondary aldosteronism means that adrenal hyperfunction is a consequence of stimulation from sources extraneous to the adrenal cortex. Adrenal adenomas are usually absent, and hypersecretion is bilateral. This type of hyperaldosteronism is associated with edematous states of other major disturbances such as nephrosis, cirrhosis, or heart failure, and is sometimes confused with oversecretion found in certain forms of arterial hypertension.

The role of aldosterone in "essential hypertension" needs further clarification. Elevated amounts of aldosterone have been found in urine of patients with severe benign and malignant "essential hypertension" and it has been suggested that human arterial hypertension could be caused by mild and chronic hyperaldosteronism (Genest et al., 1957). However, the amount of day-to-day variation of aldosterone excretion of individual patients despite fairly constant sodium intake, and the failure to find elevated

rates of excretion in patients with untreated mild essential hypertension, does not support this view. It is generally agreed, however, that aldosterone excretion may be elevated in severe benign hypertension and in malignant hypertension (Ross, 1965).

Aldosterone and Stress

Since aldosterone functions to regulate mineral homeostasis, and since stress from whatever source creates unnatural conditions which interfere with homeostasis, Simpson and Tait (1954) suggested that aldosterone may be involved in Selye's "General Adaptation Syndrome." Also, an increase in aldosterone excretion has been reported as a result of emotional stress (Lamson et al., 1956). Ross (1965) reported on various experiments in which aldosterone was used either alone or in combination with pressor substances to improve the survival rate of animals subjected to endotoxic shock.

Aldosterone Antagonists

Aldosterone antagonists are of two general types: 1) synthetic antagonists and 2) naturally occurring antagonists. Most of the synthetic antagonists are derivatives of the steroidal spirolactones, which block the action of aldosterone through reversible competitive inhibition. The naturally occurring antagonists are progesterone and derivatives, testosterone and derivatives, and

glucocorticoids. Progesterone functions as a reversible competitive inhibitor, blocking the sites of action of aldosterone. The mechanism whereby testosterone and the glucocorticoids modify the actions of aldosterone are still ambiguous.

Comprehensive reviews of aldosterone antagonists have been compiled by Kagawa (1964) and Ross (1965).

Sodium and Potassium Content of Pork

Numerous researchers (Kulwich et al., 1958, 1960, 1961; Kirton and Pearson, 1963; Kirton et al., 1963; Lawrie and Pomeroy, 1963; and Gillett et al., 1965) have investigated the variation existing in the sodium and potassium content of pork and its relation to the determination of lean muscle mass by K^40 . These workers found significant differences to exist in the sodium and potassium content among various muscles, and among various breeds. Gillett et al. (1965) reported that Hampshires contained more potassium and less sodium than Yorkshires on a per unit basis. They also reported that when extreme values were compared, differences as high as 34.7% potassium per kilogram of muscle were found.

Reports of sodium and potassium values in relation to porcine muscle quality are scant and conflicting. Henry et al. (1958) reported a large increase in sodium and a decrease in potassium in PSE muscle. They also reported an

increase in serum sodium among PSE pigs. Topel and Merkel (1966), using exogenous goitrogens to alter muscle quality, stated that the sodium and potassium levels in the longissimus dorsi muscle were similar and within normal ranges. However, these workers failed to produce quality differences with their treatments. In another experiment to study the relationship of 17-hydroxycorticosteroid levels to pork quality, Topel et al. (1967) found no significant differences in the sodium and potassium content of either muscles or plasma from various quality classifications. However, muscles from the severe PSE group had a higher mean sodium content than the DFD (dark, firm, dry) group. Beecher et al. (1968), studying the light and dark portions of the semitendinosus muscle, found that the level of potassium was similar in both portions, while sodium concentrations were significantly ($P < .01$) greater in the semitendinosus dark portion than in the light portion. The workers also stated that other workers (Drahota, 1961; Sreter and Woo, 1963; Swift and Berman, 1959) had observed higher concentrations of potassium and lower concentrations of sodium in white than in red muscle.

Pyruvate Kinase

A review of literature concerning pyruvate kinase is pertinent because of its role in glycolysis and its potassium dependency.

A review by Boyer (1962) reported that pyruvate kinase was widely distributed and appeared to be a constituent of all living cells. Studies have been reported which show the presence of the enzyme in nineteen different tissues from eleven animal species ranging from protozoa to mammals and from erythrocytes of six species, as well as yeast, molds, and the seeds or tissues of eight plants.

This enzyme catalyzes the following reaction:

Under normal conditions, this reaction is considered to be essentially irreversible, thereby acting as a rate controlling step in glycolysis.

However, the role of pyruvate kinase in glucose synthesis is also feasible. Lardy and Ziegler (1945) first demonstrated that pyruvate kinase catalyzed a significant formation of phosphoenolpyruvate from ATP and pyruvate. Krimsky (1959) confirmed this finding. Additional support to the reversibility of this reaction was indicated by observations of Von Korff and Twedt (1957) that in the presence of mitochondria carrying out oxidative phosphorylation, a ratio of phosphoenolpyruvate to pyruvate of 10 to 1 was maintained. McQuate and Utter (1959) stated that although the equilibrium of the reaction lies far to the right, the maximum initial velocity of the forward reaction has been reported as only 150 to 200 times that of the back reaction.

The dependency of pyruvate kinase activity upon univalent cations such as K^+ , Rb^+ , NH_4^+ or Cs^+ , divalent cations such as Mg^{+2} or Mn^{+2} , and the non-activating or inhibitory influence of Na^+ , Li^+ and Ca^{+2} have been demonstrated by several workers (Kachmar and Boyer, 1953; Suelter et al., 1966; Mildvan and Cohn, 1965; Mildvan and Cohn, 1966). Therefore, the presence or absence of potassium in a biological system could act as a controlling factor on glycolysis or gluconeogenesis.

The influence of certain hormones on pyruvate kinase activity has received minor attention. Weber et al. (1965) reported that enzyme activity decreased to 18% of the normal value in liver cells of diabetic rats. Treatment with insulin increased activity to 424% of the value observed in untreated diabetics. They also reported that hepatic pyruvate kinase activity was not affected by glucocorticoid treatment. Kimberg and Yielding (1962) found that diethylstilbestrol inhibited rabbit muscle pyruvate kinase activity by 60%. A lesser degree of inhibition was obtained by 17β -estradiol, equilin, equilenin, 17α -estradiol, and phenanthridine. Other steroids including deoxycorticosterone but not aldosterone were tested and found to be ineffective in inhibiting enzyme activity. It was pointed out, however, that this was a model system in vitro and that any biological importance is only a matter of speculation.

Seasonal variations in pyruvate kinase activity of rat tissue have been reported by Tanaka et al. (1967), being highest during winter and lowest during summer. Temperature and light variability was eliminated by controlled environment. In addition these workers reported the isolation of two types of pyruvate kinase. Type M, having a molecular weight of 250,000, was present in muscle, brain, heart, liver, kidney, fat pads and leucocytes. Type L, having a molecular weight of 208,300, was present only in liver and erythrocytes. The level of type L enzyme varied greatly under various physiological conditions, whereas that of type M changed only slightly.

MATERIALS AND METHODS

Preliminary investigations, involving 14 pigs (12 Poland China, 1 Hampshire and 1 Duroc), were conducted in order to select dosage levels and equilibration periods to be used in this study.

Following the preliminary investigations, 34 Poland China pigs, approximately 95 kg., were divided into three trials during the fall of 1968 to study the influence of aldosterone, Aldactone and Aldactazide on pork muscle quality.

Treatments

Trial 1. Twelve pigs were randomly allotted to slaughter and treatment groups. Because of limited physical facilities, six pigs (three controls and three treated) were slaughtered on separate days. Approximately 18 hrs. before treatment, the pigs were hauled to the meats laboratory together to minimize stress effects due to transportation. Treated pigs were injected in the ear vein with 1.5 mg. of aldosterone dissolved in 10 ml. of physiological saline. Control pigs were injected with 10 ml. of physiological saline. All pigs were allowed to remain undisturbed for an hour and a half equilibration period immediately prior to slaughter.

Trial 2. Ten pigs were randomly allotted to slaughter groups of six and four pigs, respectively. The group of six pigs was divided into two equal treatment groups of three pigs each; while the group of four pigs was divided into two equal treatment groups of two pigs each. Animals were transported to the meats laboratory about 18 hrs. before treatment as in Trial 1. One treatment group of each slaughter group received an oral drench of 250 mg. of Aldactone, while the other two treatment groups received distilled water. After a 30 min. equilibration period, pigs were stressed for 30 min. using an electric prod, and slaughtered immediately.

Trial 3. Twelve pigs were randomly allotted to slaughter and treatment groups. Six pigs (three controls and three treated) were slaughtered on separate days. About 18 hrs. before treatment, the pigs were hauled to the meats laboratory as in previous trials. Treated pigs received an oral drench of 500 mg. of Aldactazide, while the controls received a drench of distilled water. After a 30 min. equilibration period, the pigs were stressed for 30 min. and then slaughtered.

Carcass Handling and Sampling

Following the administration of the various treatments, all animals were handled similarly. The pigs were slaughtered in the Louisiana State University Meats

Laboratory and dressed according to conventional procedures. Special consideration was given to minimizing the time intervals from exsanguination to chill room. Serum samples for sodium and potassium determinations were collected at the time of exsanguination, taking care to avoid contamination. Muscle samples for sodium and potassium determinations were taken from the longissimus dorsi muscle at the 12th rib just prior to placing the carcass in the chill room. Upon entering the chill room, temperature of ham and loin muscles, and pH of the loin muscles were recorded every 15 min. for the first hour, every 30 min. for the next 2 hrs., every hour for the next 3 hrs., and at 24 hrs. after exsanguination. Temperature was measured with a YSI (Yellow Spring Instrument) Model 47 tele-thermometer. Measurements of pH were accomplished by inserting a semi-micro combination electrode (Corning) into the longissimus dorsi muscle and recording the value, adjusted for muscle temperature, from a Leeds & Northrup Model 7401 pH meter. After the carcasses had chilled for 24 hrs. at 3°C, they were cut according to conventional procedures. Subjective quality scores were applied according to Wisconsin Pork Quality Standards (Anon., 1963) by a three-member panel. Following cutting and the application of the subjective quality scores, the l. dorsi muscle was sampled again for objective measures of quality, namely water binding capacity and protein turbidity. All samples were processed as soon

as they were collected.

Sodium and Potassium Determination

Serum and muscle sodium and potassium were determined utilizing a Coleman Model 21 Flame Photometer with values being recorded from a direct reading scale panel (Cat 21-400) on a Coleman Model 22 Galv-0-Meter.

Serum Preparation. Serum samples were prepared for sodium and potassium determinations according to the procedures outlined in the Operating Directions for the Coleman Model 21 Flame Photometer (Coleman Manual D-332, 1962).

Muscle Preparation. Muscle samples were trimmed to remove all external fat and connective tissue. Samples were ground through a 5mm. plate of a laboratory size food grinder which had been rinsed several times with distilled water, several times with deionized distilled water, and several times with acetone. Cations were removed from the muscle by the TCA (trichloroacetic acid) extraction procedure of Mounib and Evans (1957), as modified by Kirton and Pearson (1963). TCA extraction was carried out on triplicate samples and readings obtained from appropriate dilutions. Direct readings were mathematically converted so as to be expressed as ppm.

Water Binding Determination

Muscle samples were trimmed of all external fat and connective tissue and ground through a 5mm. plate of a laboratory size food grinder. Water binding was determined using a modification of the procedure by Wierbicki et al. (1957). Duplicate 25 gram samples of fresh muscle were placed above a fritted disc in the upper portion of specially designed, calibrated centrifuge tubes. Vented rubber stoppers were placed in the tubes. Tubes were immersed to 0.9 their length in a 70°C water bath for 30 min., after which they were allowed to cool at 25°C for 10 min., and then centrifuged at 170 x g for 10 min. The volume of fluid expressed during heating and centrifugation was read directly. This volume as a ratio to the gm. of moisture in the sample was expressed as percent moisture loss.

Protein Turbidity Determination

Samples were taken from the trimmed, ground muscle used to supply samples for water binding determinations. Protein turbidity was determined by the transmission value method described by Hart (1962). Duplicate 10 gm. samples were homogenized in 30 ml. of cold distilled water for 5 min. in a cold room at 4°C. The homogenate was transferred to a centrifuge tube, made to a total volume of 40 ml. with cold distilled water, thoroughly mixed, and held at 4°C for 24 hrs. It was then centrifuged for 20 min. and filtered

through Whatman #52 filter paper. One ml. of the clear filtrate was mixed with 5 ml. of a citric acid-sodium phosphate buffer (9.35 ml. of 0.2 M Na_2HPO_4 plus 10.65 ml. of 0.1 M citric acid) at pH 4.6. This mixture was held at 20°C for 30 min. The protein turbidity of this mixture was read as percent transmission in a Beckman DB spectrophotometer which was standardized against a blank of 1 ml. of muscle extract in 5 ml. of distilled water. Normal muscle was expected to give a transmission value of 0-20 while severe PSE muscles had values of 80-100.

Statistical Analyses

This experiment was designed as a randomized complete block with more than one observation per treatment (Steel and Torrie, 1960). However, in order to take advantage of physical facilities and minimize temperature variation and time from exsanguination to chill room, data were collected on paired pigs (a control and a treated) within a block. This imposed a 3 x 2 factorial arrangement upon the randomized complete block design, with periods (paired pigs) and treatments as main effects and slaughter dates as blocks. The variables for trials 1 and 3 were analyzed using the above analysis. However, due to disproportionate subclass numbers in Trial 2, these data were analyzed by least-squares procedure (Harvey, 1960). Means and standard errors are cited in the text; and individual observations

are presented in the appendices.

Source of Compounds

The treatment compounds in this study were obtained from the following sources:

Aldosterone - Mann Research Laboratories
136 Liberty St.
New York, N.Y. 10006

Aldactone and Aldactazide -
G. D. Searle & Co.
P. O. Box 5110
Chicago, Ill. 60680

Pyruvate Kinase Trial

Four Hampshire pigs, approximately 95 kg., were used to observe differences in the activity of pyruvate kinase from the liver and muscle of normal and PSE pigs. Two pigs received, as nearly as possible, identical treatment to those of Trial 1 and two pigs received the treatment of Trial 3. Within 5 min. after exsanguination, samples were removed from the liver and l. dorsi muscle, and chilled as rapidly as possible in crushed ice. Samples were ground through a 5mm. plate of a laboratory size food grinder, the head of which had been chilled in a blast freezer at -29°C.

The enzyme was isolated and assayed according to the procedure described by Tanaka et al. (1967). Three gm. of liver was homogenized in 10 ml. of cold (4°C) KCl solution (0.15M) containing MgSO_4 (0.005 M) and EDTA (0.001 M).

The homogenate was centrifuged at 105,000 x g for 60 min.; and the supernatant used for assay of pyruvate kinase activity and protein determination. Simultaneously, 1.5 gm. of muscle tissue was homogenized in 13.5 ml. of the above solution, centrifuged as above, and the supernatants used as described with the liver. Pyruvate kinase was assayed by measurement of pyruvate formed from phosphoenolpyruvate in the presence of ADP with 2,4-dinitrophenylhydrazine, according to a modification of the method of Kimberg and Yielding (1962). The composition of the assay mixture was as follows: 50 μ M tris (hydroxymethyl)-aminomethane, 200 μ M phosphoenolpyruvate, 200 μ M ADP, 5 μ M MgSO_4 , 100 μ M KCl, total volume 1.0 ml., pH 7.5. The addition of 0.1 ml. of supernatant to 1.0 ml. of assay mixture started the reaction. After 3 min. incubation at 37°C, 0.2 ml. of the reaction mixture was added to 0.5 ml. of 2,4-dinitrophenylhydrazine (0.0125% in 2 N HCl) and allowed to stand 10 min. at 37°C. Then 1.2 ml. of 2.4 N NaOH containing 0.001 M EDTA was added. After allowing 10 min. for full color development, optical density was measured at 510 $\text{m}\mu$ on a Beckman DB spectrophotometer. Blanks were run in identical fashion, only omitting ADP from the assay mixture. A standard curve was obtained using sodium pyruvate in the following concentrations (μ M/ml.): 4.5, 8.4, 10.5, 42, 84.

One unit of enzyme was expressed as the amount of

enzyme which catalyzes the formation of 1 μ mole of pyruvate per minute under the above conditions. The specific activity was expressed as units of enzyme per mg. of protein.

Protein was obtained by multiplying 6.25 times the mg. of nitrogen determined by microkjeldahl and direct nesslerization.

RESULTS AND DISCUSSION

Preliminary Investigations

Since a hyperproduction of aldosterone had been reported in the etiology of PSE porcine musculature (Henry et al., 1958), it was of interest to determine if PSE musculature could be produced by the administration of this hormone, as well as prevented by the administration of an aldosterone-blocking agent.

The literature indicated that the normal daily secretion rate of aldosterone in man varied from 70 to 300 μg , and that plasma concentration of aldosterone in man ranged from 5 to 200 $\mu\text{g}/100\text{ ml}$. (Ross, 1965). Also, indications were that a delay of 45 to 90 minutes was necessary for aldosterone to become effective (Ross, 1965). Therefore, these preliminary investigations, using various dosages of aldosterone and various periods of equilibration, revealed that PSE musculature could be produced using a 10 ml. physiological saline injection containing 1.5 mg. of aldosterone, with an hour and a half equilibration period.

Preliminary investigations with an aldosterone-blocking agent were limited to the use of Aldactazide. Aldactazide was a combination of equal quantities of Aldactone (spironolactone) and hydrochlorothiazide, which act

synergistically. Aldactone effects diuresis by blocking, through competitive inhibition, the sodium- and water-retaining and potassium-excreting effects on the distal renal tubules of aldosterone. Hydrochlorothiazide, a potent diuretic agent, promotes excretion of sodium and water primarily by inhibiting their absorption by the proximal renal tubules; and also it promotes excretion of potassium. The recommended dosage of Aldactazide for an adult man ranged from 50 to 200 mg./day. Therefore, the use of various dosages and equilibration periods indicated that in this study an oral drench of 500 mg. of Aldactazide in distilled water and a 30 min. equilibration period prevented the development of PSE musculature resulting from a 30 min. stress period with an electric prod.

Proposed Role of Aldosterone

The primary function of aldosterone is to increase sodium retention and potassium excretion in the renal tubules of the kidney. In the reversal of glycolysis to form glucose-6-phosphate, ATP, available from aerobic oxidation of a fraction of lactic acid (via pyruvic acid and the citric acid cycle), must be provided to reverse the pyruvic kinase and phosphoglyceric kinase reactions. However, there is a rate limiting factor; pyruvic kinase is potassium dependent (White et al., 1964). PSE muscle occurs when there is a rapid rate of pH decline at a high

temperature. Just before and at the time of exsanguination a certain amount of aerobic glycolysis is occurring within the muscle, therefore, oxidative phosphorylation and the production of ATP. Likewise, since considerable anaerobic glycolysis has occurred, the ratio of pyruvate to phosphoenolpyruvate is probably very high. Perhaps then in normal muscle, sufficient potassium is present for pyruvate kinase activity, reversing glycolysis and allowing a portion of the lactic acid from preslaughter exercise, excitement, etc., to go back into the Embden-Meyerhof pathway as other compounds, thereby decreasing the rate of pH drop post-mortem. In PSE muscle, aldosterone production has caused an increased excretion of potassium, reducing its availability for the reversal of glycolysis. Subsequently, pre-slaughter lactic acid would remain, plus the lactic acid which accumulates during post-mortem glycolysis, consequently a more rapid drop of pH at a high temperature.

Results of Trial 1

In this trial, an injection of 1.5 mg. of aldosterone was used to produce PSE musculature in Poland China pigs. Figures 1 and 2 show representative hams and loins from the control and aldosterone treated pigs, respectively. Note the uniform color, firm texture, and dry appearance of the face of the ham in Figure 1 as compared to the pale color, soft texture and watery appearance displayed by the

FIGURE 1. HAM AND LOIN OF CONTROL FOR TRIAL 1

FIGURE 2. HAM AND LOIN OF ALDOSTERONE
TREATED FOR TRIAL 1

muscles in the lower portion of the ham in Figure 2. Due to the lighting on these figures, it is difficult to detect differences between the 1. dorsi muscles, however, close examination revealed more evidence of moisture on the surface of this muscle in Figure 2 than in Figure 1.

Means and standard errors for subjective and objective quality evaluations are presented in Table 1. The injection of 1.5 mg. of aldosterone resulted in a highly significant ($P < .01$) reduction in porcine muscle quality as measured by the Wisconsin Quality Score. The control animals had a quality score of 2.8 compared to 1.8 for the treated group. No significant differences were obtained for the objective measures of quality; however, trends were similar. The control group had a higher water binding capacity, losing 34.5% moisture, compared to 37.3% for the treated group. Likewise, the 29.4% transmission value from the protein turbidity test for the controls indicated a greater sarcoplasmic protein content in that group than in the treated group (40.6% transmission), reflecting superior quality.

Table 2 contains the means and standard errors for serum and muscle sodium and potassium for this trial. No statistically significant differences were obtained for either serum or muscle sodium and/or potassium. Serum sodium was slightly higher for the controls (157.8 meq./l) than for the aldosterone treatment (156.3 meq./l); whereas

TABLE 1. MEANS AND STANDARD ERRORS FOR SUBJECTIVE^a
AND OBJECTIVE QUALITY EVALUATIONS

Item	Control		Aldosterone	
	Mean ^b	S.E. ^c	Mean	S.E.
Wisc. Qual. Score**	2.8	±0.1	1.8	±0.1
Water binding (% H ₂ O loss)	34.5	±1.0	37.3	±1.0
Protein turbidity (%T)	29.3	±8.6	40.6	±8.6

** (P < .01)

^aWisconsin Pork Quality Standards: 1 = PSE,
3 = normal.

^bn = 6

^cStandard deviation of mean (standard error).

TABLE 2. MEANS AND STANDARD ERRORS
FOR SODIUM AND POTASSIUM

Item	Control		Aldosterone	
	Mean ^a	S.E. ^b	Mean	S.E.
Serum Na ⁺ , meq./l	157.8	± 1.4	156.3	± 1.4
Serum K ⁺ , meq./l	5.9	± 0.2	5.6	± 0.2
Muscle Na ⁺ , ppm	433.3	±15.3	455.1	±15.3
Muscle K ⁺ , ppm	4,337.8	±59.8	4,265.4	±59.8

^an = 6

^bStandard deviation of mean (standard error).

potassium was lower for the treated group (5.6 meq./l) than for the controls (5.9 meq./l). Muscle sodium content was higher for the treated group (455.1 ppm) than for the controls (433.3 ppm), while muscle potassium content was lower for the treated group (4,265.4 ppm vs. 4,337.8 ppm).

There was little or no relationship between temperature and pH values and the development of PSE muscle in this study. Therefore, values are recorded individually in appendix tables 7, 8, and 9, and not incorporated into the text.

Results of Trial 2

An oral drench of 250 mg. of Aldactone, a dosage equivalent to 500 mg. of Aldactazide, was used in an attempt to prevent the production of PSE musculature in Poland China pigs resulting from a 30 min. stress period with an electric prod. Representative hams and loins from the control group and the Aldactone treated group are shown in Figures 3 and 4, respectively. The hams and loins in these figures are quite comparable with regard to their expression of PSE characteristics. Disregarding the effect of lighting in Figure 4, the texture appeared to be somewhat softer than that of Figure 3, as evidenced by the protruding muscles in the lower portion of the face of the ham. This was unexpected since Aldactone was supposed to prevent the development of PSE musculature.

FIGURE 3. HAM AND LOIN OF CONTROL FOR TRIAL 2

FIGURE 4. HAM AND LOIN OF ALDACTONE
TREATED FOR TRIAL 2

No significant differences were obtained for either the subjective or objective evaluations of quality (Table 3). The mean Wisconsin Quality Score was essentially the same for the treated group (2.1) as for the controls (2.0). However, objective measures indicated a slight advantage in quality for treatment. The treated group had a slightly lower percent moisture loss (42.6 vs. 45.1) and a lower protein turbidity value (68.0% T vs. 92.3% T).

Table 4 contains the least squares means and standard errors for sodium and potassium content of serum and muscle. No statistically significant differences were found for any of these measurements. Serum sodium was higher in the treated group, while serum potassium was higher in the control group. Muscle sodium was lower in the treated group (470.0 ppm vs. 477.6 ppm), while muscle potassium was higher in the treated group (4,209.5 ppm vs. 4,113.0 ppm).

Results of Trial 3

An oral drench of 500 mg. of Aldactazide was used to prevent the development of PSE musculature in Poland China pigs resulting from stress with an electric prod. Figures 5 and 6 represent the hams and loins of the control and Aldactazide treated pigs, respectively. Examination of Figure 5 reveals that the muscles in the outer portion of

TABLE 3. LEAST-SQUARES MEANS AND STANDARD ERRORS
FOR SUBJECTIVE^a AND OBJECTIVE QUALITY
EVALUATIONS

Item	Control		Aldactone	
	Mean ^b	S.E. ^c	Mean	S.E.
Wisc. Qual. Score	2.0	±0.5	2.1	±0.5
Water binding (% H ₂ O loss)	45.1	±1.6	42.6	±1.6
Protein turbidity (% T)	92.3	±9.1	68.0	±9.1

^aWisconsin Pork Quality Standards: 1 = PSE,
3 = normal.

^b_n = 5

^cStandard deviation of mean (standard error).

TABLE 4. LEAST-SQUARES MEANS AND STANDARD
ERRORS FOR SODIUM AND POTASSIUM

Item	Control		Aldactone	
	Mean ^a	S.E. ^b	Mean	S.E.
Serum Na ⁺ , meq./l	165.1	± 2.4	166.8	± 2.8
Serum K ⁺ , meq./l	8.6	± 1.1	7.9	± 1.1
Muscle Na ⁺ , ppm	477.6	±15.9	470.9	±15.9
Muscle K ⁺ , ppm	4,113.0	±73.8	4,209.5	±73.8

^an = 5

^bStandard deviation of mean (standard error).

FIGURE 5. HAM AND LOIN OF CONTROL FOR TRIAL 3

FIGURE 6. HAM AND LOIN OF ALDACTAZIDE
TREATED FOR TRIAL 3

the ham face were lighter in color, softer in texture and exuding more juice than those in Figure 6. Also, the l. dorsi muscle of Figure 5 was soft and watery compared to that of Figure 6.

The oral administration of 500 mg. of Aldactazide significantly ($P < .05$) increased the Wisconsin Pork Quality Score from 1.2 for the controls to 2.5 (Table 5). Although not statistically significant, a decrease in protein turbidity from 93.8% T for the controls to 70.5% T for the treated group indicated an improvement in quality. However, no differences were obtained for water-holding capacity (43.9% for controls vs. 43.1% for treated).

Means and standard errors for sodium and potassium of serum and muscle are shown in Table 6. Values varied erratically. Serum and muscle sodium contents were higher in the treated group (164.3 meq./l, 451.7 ppm) than in the controls (159.8 meq./l, 414.8 ppm). Likewise, serum and muscle potassium contents were lower in the treated group (7.1 meq./l, 3,944.8 ppm) than in the controls (8.0 meq./l, 3,968.2 ppm). However, none of these values differed significantly.

A significant ($P < .05$) period effect was obtained for serum potassium in this trial (appendix table 28).

TABLE 5. MEANS AND STANDARD ERRORS FOR SUBJECTIVE^a
AND OBJECTIVE QUALITY EVALUATIONS

Item	Control		Aldactazide	
	Mean ^b	S.E. ^c	Mean	S.E.
Wisc. Qual. Score*	1.2	±0.2	2.5	±0.2
Water binding (% H ₂ O loss)	43.9	±1.2	43.1	±1.2
Protein turbidity (% T)	93.8	±8.2	70.5	±8.2

*(P < .05)

^aWisconsin Pork Quality Standards: 1 = PSE,
3 = normal.

^bn = 6

^cStandard deviation of mean (standard error).

TABLE 6. MEANS AND STANDARD ERRORS
FOR SODIUM AND POTASSIUM

Item	Control		Aldactazide	
	Mean ^a	S.E. ^b	Mean	S.E.
Serum Na ⁺ , meq./l	159.8	± 5.0	164.3	± 5.0
Serum K ⁺ , meq./l	8.0	± 0.5	7.1	± 0.5
Muscle Na ⁺ , ppm	414.8	±13.3	451.7	±13.3
Muscle K ⁺ , ppm	3,968.2	±66.6	3,944.8	±66.6

^an = 6

^bStandard deviation of mean (standard error).

Discussion of Trials

The production of PSE musculature in Trial 1 with the injection of aldosterone and the prevention in Trial 3 with Aldactazide are in agreement with work of Henry et al. (1958), reporting pigs with PSE musculature had hyperproduction of aldosterone. The failure to report an increase in the Wisconsin Pork Quality Score, and the small advantage cited in objective measures of quality for the treatment in Trial 2, can possibly be attributed to the fact that Aldactone alone lacked the synergistic capabilities expressed by Aldactazide.

The objective measures of quality, even though they were not statistically significant, were in accord with expected results. Aldosterone decreased the water binding capacity of the l. dorsi muscle while the blocking agents increased this measurement, characteristics which have been shown to be related to PSE and normal musculature, respectively (Wisner-Pedersen, 1959; Wisner-Pedersen and Briskey, 1961; Bendall and Wisner-Pedersen, 1962). Protein turbidity values also tended to substantiate the production of PSE musculature with aldosterone and the prevention with a blocking agent. This procedure is based on the fact that in exudative meat the sarcoplasmic proteins have been denatured, resulting in a lower quantity of water soluble proteins than in normal meat. Therefore, the percent transmission will be higher for PSE musculature than for

normal muscle. According to Hart (1962), transmission values for normal meat are expected to be 0-20% while those for PSE meat are between 80-100%. This study revealed that these values were indicative only if extremes in each classification were encountered. However, turbidity values for intermediate quality score muscle did not array in a linear order. This is in agreement with Ockerman and Cahill (1968) who reported that if a given percent transmission value was used to separate acceptable and unacceptable quality, a large percent of the carcasses disqualified by this procedure also would be rated unacceptable by visual means. However, some carcasses rated unacceptable by visual means would not necessarily be rated unacceptable by this procedure.

No statistically significant differences were obtained for sodium and potassium contents of serum or muscle for any of the trials. Serum sodium values were higher in the controls in Trial 1 and in the treated groups of Trials 2 and 3. Since aldosterone is supposed to increase sodium retention, this was unexpected, but it is in agreement with serum values reported by Henry *et al.* (1958) and plasma values reported by Topel *et al.* (1967). The occurrence of lower serum potassium values for the aldosterone treatment of Trial 1 agrees with Henry *et al.* (1958) and Topel *et al.* (1967). However, the higher values for the controls of Trials 2 and 3 are contradictory to the results of the

above authors.

Muscle sodium and potassium values for Trials 1 and 2 show small differences, but are in agreement with results reported by Henry et al. (1958), and show the same trends as data reported by Topel et al. (1967). However, those of Trial 3 are contradictory to the above reports.

The results of these trials with regard to the potassium content of muscle failed to support the proposed role of aldosterone in the etiology of PSE musculature. However, it should be kept in mind that the procedures used in this study were measuring total potassium in the muscle. No attempts were made to estimate intracellular or extracellular potassium components. It is possible that an alteration in intracellular potassium concentration could occur which was not detected with the present techniques employed.

Furthermore, a point for consideration is the magnitude of the decrease in potassium concentration necessary to inactivate the pyruvate kinase reaction in porcine muscle. If this was known, it would give an indication of the sample size necessary to detect these differences.

Pyruvate Kinase Trial

In view of the fact that the differences in muscle potassium content did not support the proposed role of aldosterone in the etiology of PSE musculature, it was of

interest to observe the pyruvate kinase activity of pigs. Four Hampshire pigs (intermediate in susceptibility to PSE musculature) were subjected to treatments similar to Trials 1 and 3, and the pyruvate kinase activity was measured. None of the pigs developed PSE musculature, all having a Wisconsin Pork Quality Score of 3. Table 7 contains the activity of pyruvate kinase found in the muscle and liver of these pigs. The specific activity of the muscle was greater for the aldosterone treated animal (5,158.3) than for the aldosterone control (4,509.8), while that of the Aldactazide control (6,670.9) was greater than the treated (4,240.6). The reverse was true for the activity of the liver enzyme, 289.9 vs. 348.7 and 364.3 vs. 533.1, respectively. Results from these studies are not conclusive and cannot be extrapolated to the previous animals since differences in muscle quality were not consistent with other treatments. However, Kastenschmidt et al. (1968) reported that mass action ratios calculated from means of appropriate intermediates afforded consideration for a control in post-mortem glycolysis at the pyruvate kinase step. Therefore, some indication as to the activity of pyruvate kinase in pork muscle is of value.

TABLE 7. PYRUVATE KINASE ACTIVITY
OF PORK MUSCLE AND LIVER

Treatment	Specific Activity ^a	
	Muscle	Liver
Aldosterone Control	4,509.8	348.7
Aldosterone Treated	5,158.3	289.9
Aldactazide Control	6,670.9	364.3
Aldactazide Treated	4,240.6	533.1

^a μ M Pyruvate/ml./min./mg. Protein.

SUMMARY

A study was designed to investigate the influence of aldosterone and aldosterone-blocking agents (Aldactone and Aldactazide) on porcine muscle quality, and to obtain some insight into the role or mode of action of aldosterone in the etiology of PSE musculature. Fourteen pigs were used in preliminary investigations to establish dosage levels and equilibration periods for the compounds used. Following this, 34 Poland China pigs, approximately 95 kg., were divided into three trials of 12, 10 and 12 pigs each, and treated with aldosterone, Aldactone and Aldactazide, respectively. The pigs in the latter two trials were stressed for 30 minutes. Animals were slaughtered; serum and muscle sodium and potassium samples were taken; carcasses were chilled; and subjective and objective quality evaluations were determined.

Pyruvate kinase activity of porcine muscle and liver was obtained from four animals.

Under the conditions of this experiment, the following conclusions seem justifiable:

1. The intravenous injection of 1.5 mg. of aldosterone resulted in a highly significant ($P < .01$) reduction in Wisconsin Pork Quality Score, and a trend toward

decreased quality as measured by objective methods.

2. The oral administration of 500 mg. of Aldactazide caused a significant ($P < .05$) increase in Wisconsin Pork Quality Score and a decrease in protein turbidity, both indicating superior muscle quality.

3. The oral administration of 250 mg. of Aldactone tended to improve muscle quality as measured by objective methods but not as measured by the subjective method.

4. Serum and muscle sodium and potassium contents were not affected significantly by any of the treatments imposed. The differences obtained were small and somewhat inconsistent.

5. Aldosterone was postulated to influence PSE conditions through its effect on muscle potassium, inactivating pyruvate kinase. However, muscle potassium values failed to substantiate this postulation. It was entirely possible that sampling methods for potassium were inadequate to properly evaluate this theory.

RECOMMENDATIONS FOR FUTURE RESEARCH

Since aldosterone and aldosterone-blocking agents were found to affect PSE muscle conditions, further study is needed to elucidate the exact mechanisms involved.

Areas of considerable interest would be:

1. The influence of aldosterone on muscle electrolytes.
2. Improved techniques for studying alterations of intracellular and extracellular electrolytes in muscle.
3. The influence of pyruvate kinase isozymes on ante-mortem and post-mortem glycolytic rates as related to muscle quality.
4. The influence of aldosterone on the activity of cyclic AMP and phosphorylase in various tissues.
5. The incorporation of radioisotopes, involving electrolytes and glycolytic intermediates, to elucidate the basic cause or causes of PSE musculature.

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APPENDIX

TABLE 1. SUBJECTIVE AND OBJECTIVE QUALITY EVALUATIONS
FOR INDIVIDUAL ANIMALS FOR TRIAL 1

Animal No.	Treatment	Wisconsin Quality Score ^a	Water Binding ^b	Protein Turbidity ^c
8-1	Control	3	32.05	62.8
2-1	Control	2	34.59	78.3
2-11	Control	3	24.12	25.0
2-5	Aldosterone	1	32.16	100.0
6-0	Aldosterone	2	30.27	37.0
8-8	Aldosterone	1.5	30.41	94.5
2-2	Control	3	41.35	2.0
9-1	Control	3	38.34	3.0
7.4	Control	3	36.76	4.5
7-8	Aldosterone	2	45.10	7.5
7-3	Aldosterone	2	42.70	2.3
7-7	Aldosterone	2	42.96	2.5

^aWisconsin Pork Quality Standards: 1 = PSE,
3 = normal.

^bExpressed as percent moisture loss.

^cExpressed as percent transmission.

TABLE 2. SUBJECTIVE AND OBJECTIVE QUALITY EVALUATIONS
FOR INDIVIDUAL ANIMALS FOR TRIAL 2

Animal No.	Treatment	Wisconsin Quality Score ^a	Water Binding ^b	Protein Turbidity ^c
10-4	Control	2	36.98	92.5
11-5	Control	3	38.11	59.0
9-4	Control	2	45.90	99.0
9-3	Aldactone	3	36.64	48.0
14-5	Aldactone	2	45.00	94.0
9-5	Aldactone	3	36.33	32.5
7-6	Control	3	45.63	86.5
14-4	Control	1	50.20	99.0
7-11	Aldactone	1	46.93	91.0
7-1	Aldactone	1.5	46.13	91.5

^aWisconsin Pork Quality Standards: 1 = PSE,
3 = normal.

^bExpressed as percent moisture loss.

^cExpressed as percent transmission.

TABLE 3. SUBJECTIVE AND OBJECTIVE QUALITY EVALUATIONS
FOR INDIVIDUAL ANIMALS FOR TRIAL 3

Animal No.	Treatment	Wisconsin Quality Score ^a	Water Binding ^b	Protein Turbidity ^c
1-4	Control	1	47.89	98.0
7-7	Control	1	45.64	92.3
1-5	Control	1	43.49	89.0
3-6	Aldactazide	3	38.93	42.5
3-4	Aldactazide	3	45.36	60.5
2-3	Aldactazide	2	43.89	91.5
3-7	Control	1	39.99	95.0
1-4a	Control	2	46.89	93.0
1-6	Control	1	39.45	95.3
1-3	Aldactazide	1.5	41.64	92.0
15-1	Aldactazide	3	44.00	81.5
14-13	Aldactazide	2.5	45.07	55.0

^aWisconsin Pork Quality Standards: 1 = PSE,
3 = normal.

^bExpressed as percent moisture loss.

^cExpressed as percent transmission.

TABLE 4. SERUM AND MUSCLE SODIUM AND POTASSIUM
FOR INDIVIDUAL ANIMALS FOR TRIAL 1

Animal No.	Treatment	Serum ^a		Muscle ^b	
		Na ⁺	K ⁺	Na ⁺	K ⁺
8-1	Control	155	4.50	404.62	3,996.56
2-1	Control	154	5.50	423.68	4,084.38
2-11	Control	155	4.90	414.40	4,365.48
2-5	Aldosterone	157	5.20	484.46	4,335.35
6-0	Aldosterone	159	4.60	423.42	4,069.77
8-8	Aldosterone	151	5.40	398.64	3,809.92
2-2	Control	158	6.70	437.74	4,484.76
9-1	Control	159	6.70	394.65	4,484.57
7-4	Control	166	7.00	524.83	4,611.09
7-8	Aldosterone	162	6.80	472.42	4,591.73
7-3	Aldosterone	155	5.40	473.72	4,240.96
7-7	Aldosterone	154	6.20	477.98	4,544.84

^aExpressed as meq./l.

^bExpressed as ppm.

TABLE 5. SERUM AND MUSCLE SODIUM AND POTASSIUM
FOR INDIVIDUAL ANIMALS FOR TRIAL 2

Animal No.	Treatment	Serum ^a		Muscle ^b	
		Na ⁺	K ⁺	Na ⁺	K ⁺
10-4	Control	165	7.25	467.70	4,140.41
11-5	Control	160	6.90	529.58	3,887.44
9-4	Control	170	8.00	460.61	3,913.74
9-3	Aldactone	172	7.10	573.60	3,843.71
14-5 ^c	Aldactone	172	10.20	452.35	4,118.86
9-5	Aldactone	158	5.85	472.49	4,152.89
7-6	Control	158	7.25	478.35	4,135.96
14-4 ^c	Control	168	12.70	505.60	4,383.39
7-11	Aldactone	166	8.40	473.56	4,209.31
7-1	Aldactone	175	8.65	417.97	4,475.72

^aExpressed as meq./l.

^bExpressed as ppm.

^cStuck before the end of 30 min. stress period
because animal was dying.

TABLE 6. SERUM AND MUSCLE SODIUM AND POTASSIUM
FOR INDIVIDUAL ANIMALS FOR TRIAL 3

Animal No.	Treatment	Serum ^a		Muscle ^b	
		Na ⁺	K ⁺	Na ⁺	K ⁺
1-4 ^c	Control	164	9.85	402.40	4,051.69
7-7 ^c	Control	161	8.30	440.59	4,072.59
1-5	Control	166	5.52	428.35	3,792.91
3-6	Aldactazide	158	8.35	476.52	4,252.79
3-4	Aldactazide	166	10.00	423.00	3,878.61
2-3	Aldactazide	149	5.70	458.86	4,083.89
3-7	Control	172	7.60	377.95	3,824.05
1-4a ^c	Control	132	10.00	421.58	4,038.08
1-6	Control	164	6.70	418.16	4,030.20
1-3	Aldactazide	175	6.85	412.11	4,209.75
15-1	Aldactazide	172	7.35	498.18	3,527.14
14-13	Aldactazide	166	4.15	441.63	3,716.81

^aExpressed as meq./l.

^bExpressed as ppm.

^cStuck before the end of 30 min. stress period
because animal was dying.

TABLE 7. TEMPERATURES AND pH VALUES OF INDIVIDUAL ANIMALS
AT VARIOUS INTERVALS POST-MORTEM FOR TRIAL 1

Animal No.	Treatment	Items ^a	Intervals Post-Mortem ^b												
			0	0.25	0.50	0.75	1	1.5	2	2.5	3	4	5	6	24
8-1	Control	Ham	39.0	39.0	39.0	39.0	38.5	37.5	33.9	32.0	30.0	27.0	24.0	22.0	9.0
		Loin	40.0	40.0	39.5	38.0	36.0	35.5	30.4	28.0	26.0	23.0	20.0	18.0	8.0
		pH	6.40	6.00	5.95	5.80	5.75	5.70	5.80	5.80	5.80	5.70	5.75	5.75	5.95
2-1	Control	Ham	41.0	41.0	41.0	40.0	39.0	38.0	34.0	32.0	30.0	27.0	24.0	22.0	8.5
		Loin	41.0	40.0	39.5	37.0	35.5	33.5	28.0	26.0	24.0	21.0	18.5	17.0	7.5
		pH	6.05	5.90	5.90	5.80	5.75	5.80	5.80	5.75	5.80	5.80	5.90	5.75	5.90
2-11	Control	Ham	39.0	39.0	40.0	39.0	37.2	36.3	33.6	31.5	30.0	26.0	22.0	20.5	9.0
		Loin	37.0	36.0	36.0	33.0	30.6	28.0	25.8	23.0	22.0	19.0	17.0	16.0	7.4
		pH	6.60	6.55	6.40	6.20	6.05	5.65	5.75	5.70	5.65	5.75	5.65	5.60	5.95
2-5	Aldos- terone	Ham	41.0	41.0	41.0	40.5	40.0	39.0	35.5	34.0	31.0	28.0	25.0	22.0	7.5
		Loin	40.0	39.5	38.0	36.0	34.0	30.0	25.6	24.0	26.0	19.0	16.5	15.0	6.8
		pH	5.85	5.85	5.75	5.70	5.50	5.45	5.55	5.65	5.80	5.80	5.80	5.85	6.00
6-0	Aldos- terone	Ham	41.0	40.5	41.0	41.0	40.0	39.0	36.0	34.5	32.0	28.5	25.0	22.5	7.8
		Loin	40.0	40.0	39.0	38.0	35.0	32.5	27.8	26.0	23.9	20.5	18.0	17.0	7.0
		pH	6.65	6.50	6.30	6.10	5.95	5.90	5.80	5.85	5.90	5.85	5.90	5.85	6.00
8-8	Aldos- terone	Ham	41.0	42.0	42.0	41.0	50.0	37.4	36.0	33.5	32.0	28.4	26.0	24.0	11.5
		Loin	41.0	40.0	39.0	36.0	33.6	30.0	28.0	26.0	25.0	22.0	20.2	18.6	10.0
		pH	5.80	5.80	5.70	5.80	5.75	5.75	5.70	5.70	5.70	5.80	5.70	5.70	6.10
2-2	Control	Ham	40.0	39.9	40.0	39.5	39.0	37.0	35.5	33.5	31.0	27.5	24.0	21.0	5.9
		Loin	39.1	38.0	37.0	35.0	33.0	29.0	26.0	23.0	20.5	17.0	14.4	13.0	4.0
		pH	6.35	6.50	6.35	6.30	5.85	6.00	6.00	5.70	5.80	5.80	5.89	5.55	5.60

TABLE 7 (cont'd.) TEMPERATURES AND pH VALUES OF INDIVIDUAL ANIMALS
AT VARIOUS INTERVALS POST-MORTEM FOR TRIAL 1

Animal No.	Treatment	Item ^a	Intervals Post-Mortem ^b												
			0	0.25	0.50	0.75	1	1.5	2	2.5	3	4	5	6	24
9-1	Control	Ham	39.1	40.0	40.0	40.0	40.0	39.0	37.0	35.0	33.0	29.0	20.1	22.5	6.0
		Loin	38.0	35.4	34.0	33.0	31.0	27.5	24.0	21.0	19.0	15.0	13.0	12.0	4.0
		pH	6.10	5.90	5.70	5.70	5.80	5.60	5.45	5.30	5.45	5.40	5.20	5.20	5.45
7-4	Control	Ham	42.5	42.9	42.8	42.0	41.9	40.0	38.0	36.5	34.5	31.0	28.0	25.5	7.0
		Loin	41.5	41.0	39.5	37.5	36.8	32.4	29.0	26.5	24.0	21.0	19.0	17.5	4.0
		pH	6.10	5.95	5.80	5.60	5.50	5.50	5.45	5.50	5.50	5.45	5.50	5.50	5.50
7-8	Aldos- terone	Ham	39.0	39.0	38.0	36.9	38.0	38.0	36.5	34.5	32.0	28.0	24.5	20.0	5.9
		Loin	38.9	38.8	38.0	36.0	35.0	30.0	27.0	23.5	20.0	16.5	13.8	12.0	4.0
		pH	6.00	5.75	5.65	5.40	5.35	5.40	5.40	5.40	5.30	5.40	5.40	5.40	5.50
7-3	Aldos- terone	Ham	39.9	40.0	40.0	40.0	39.5	37.9	36.5	34.0	32.2	28.5	25.0	22.0	6.0
		Loin	38.7	39.0	37.0	34.0	32.5	28.1	26.0	22.0	20.0	15.8	13.0	11.5	3.0
		pH	6.20	6.00	5.70	5.50	5.50	5.45	5.40	5.40	5.40	5.15	5.28	5.28	5.50
7-7	Aldos- terone	Ham	40.0	40.0	39.8	39.2	38.2	37.0	35.0	33.0	30.0	26.7	23.0	20.0	6.0
		Loin	39.0	37.0	35.0	33.0	30.5	28.0	24.0	21.5	18.8	15.5	13.0	12.0	4.0
		pH	6.30	6.20	6.35	6.15	6.15	5.90	6.00	5.95	5.85	5.60	5.30	5.45	5.45

^aHam and loin values are temperature, expressed as °C. pH values are from the loin.

^bExpressed in hrs. after the carcass entered the cooler.

TABLE 8. TEMPERATURES AND pH VALUES OF INDIVIDUAL ANIMALS
AT VARIOUS INTERVALS POST-MORTEM FOR TRIAL 2

Animal No.	Treatment	Item ^a	Intervals Post-Mortem ^b												
			0	0.25	0.50	0.75	1	1.5	2	2.5	3	4	5	6	24
10-4	Control	Ham	43.8	43.2	43.5	52.5	42.0	40.0	38.0	35.0	33.0	29.0	26.0	22.5	6.5
		Loin	42.8	40.0	36.5	34.2	33.0	28.0	25.0	21.1	19.0	16.0	14.0	12.0	4.5
		pH	5.55	5.55	5.45	5.35	5.35	5.35	5.35	5.35	5.35	5.35	5.40	5.35	5.25
11-5	Control	Ham	45.0	45.0	45.0	45.0	44.5	43.0	41.0	39.0	36.8	32.2	39.0	25.0	7.0
		Loin	43.0	43.5	42.8	40.0	38.0	34.0	30.0	27.0	24.0	20.0	17.5	15.5	5.0
		pH	5.95	5.45	5.50	5.45	5.50	5.50	5.60	5.60	5.50	5.50	5.50	5.45	5.35
9-4	Control	Ham	45.0	45.0	45.0	45.0	44.0	42.0	40.0	37.2	35.0	30.0	25.5	21.5	6.0
		Loin	42.0	41.0	39.9	38.0	35.0	31.0	28.0	24.2	21.5	17.5	15.0	13.0	4.5
		pH	5.30	5.18	5.20	5.20	5.30	5.30	5.30	5.30	5.30	5.30	5.20	5.20	5.25
9-3	Aldactone	Ham	43.0	43.0	43.0	43.0	42.0	40.0	38.5	36.5	34.0	31.0	27.0	24.0	7.5
		Loin	42.0	41.0	38.0	37.5	34.0	30.0	26.5	23.5	21.0	17.0	15.0	13.0	5.0
		pH	6.40	6.35	6.35	6.10	5.91	5.61	5.75	5.65	5.60	5.50	5.50	5.50	5.60
14-5	Aldactone	Ham	44.0	44.0	44.0	43.9	43.0	42.0	39.5	37.5	35.0	31.0	28.0	24.0	7.0
		Loin	42.0	41.0	40.0	37.0	34.0	30.5	26.2	23.2	20.5	17.0	24.0	13.0	4.5
		pH	5.60	5.20	5.20	5.20	5.20	5.25	5.30	5.30	5.30	5.40	5.40	5.40	5.20
9-5	Aldactone	Ham	45.5	45.5	45.5	45.0	44.8	43.5	41.0	39.0	37.0	34.0	29.9	26.5	13.5
		Loin	43.2	43.0	41.0	39.0	37.0	34.0	30.8	28.0	26.0	23.0	20.5	18.5	11.1
		pH	6.35	5.88	5.89	5.85	5.80	5.70	5.65	5.55	5.55	5.50	5.50	5.45	5.50
7-6	Control	Ham	45.0	44.8	44.0	43.0	42.0	39.0	37.0	33.5	31.0	27.5	24.5	21.5	6.5
		Loin	45.0	43.5	39.0	35.0	33.0	28.5	25.8	22.0	20.5	17.0	15.0	13.5	5.0
		pH	6.20	5.90	5.80	5.50	5.41	5.40	5.40	5.40	5.45	5.25	5.25	5.25	5.00

TABLE 8 (cont'd.). TEMPERATURES AND pH VALUES OF INDIVIDUAL ANIMALS
AT VARIOUS INTERVALS POST-MORTEM FOR TRIAL 2

Animal No.	Treatment	Item ^a	Intervals Post-Mortem ^b												
			0	0.25	0.50	0.75	1	1.5	2	2.5	3	4	5	6	24
14-4	Control	Ham	44.7	44.0	44.5	44.0	43.0	42.0	39.0	38.0	35.5	31.0	27.0	24.0	7.0
		Loin	43.0	43.0	40.5	39.5	37.0	34.0	30.5	29.0	26.5	22.5	20.0	17.0	6.0
		pH	6.00	6.00	5.30	5.25	5.25	5.25	5.29	5.30	5.30	5.30	5.30	5.30	4.90
7-11	Aldactone	Ham	45.5	45.0	45.0	44.8	44.0	42.0	40.0	37.5	36.0	31.0	27.2	24.0	7.0
		Loin	44.0	44.0	42.0	39.0	35.0	31.5	28.0	25.0	23.0	18.5	16.0	14.0	5.0
		pH	6.01	6.00	5.90	5.90	5.50	5.50	5.40	5.35	5.35	5.40	5.40	5.30	5.00
7-1	Aldactone	Ham	44.0	44.0	44.0	44.0	43.5	42.5	36.5	34.5	33.0	30.0	27.0	24.0	7.0
		Loin	38.0	41.0	39.0	36.5	34.0	31.0	26.0	23.0	21.5	18.0	16.0	13.5	5.0
		pH	6.00	5.30	5.30	5.35	5.30	5.30	5.38	5.38	5.40	5.40	5.30	5.30	4.95

^aHam and loin values are temperature, expressed as °C. pH values are from the loin.

^bExpressed in hrs. after the carcass entered the cooler.

TABLE 9. TEMPERATURES AND pH VALUES OF INDIVIDUAL ANIMALS
AT VARIOUS INTERVALS POST-MORTEM - FOR TRIAL 3

Animal No.	Treatment	Item ^a	Intervals Post-Mortem ^b												
			0	0.25	0.50	0.75	1	1.5	2	2.5	3	4	5	6	24
1-4	Control	Ham	44.0	43.1	42.0	41.0	41.0	39.8	38.0	35.8	33.5	30.0	25.8	23.5	6.0
		Loin	42.0	41.1	39.9	38.2	37.0	32.5	29.5	26.2	23.9	20.0	17.0	15.0	6.0
		pH	5.70	5.75	5.75	5.70	5.90	5.85	5.85	5.85	5.85	5.80	5.65	5.70	5.75
7-7	Control	Ham	44.0	43.8	43.8	43.0	43.0	41.0	39.0	37.0	35.5	31.3	29.0	26.0	9.0
		Loin	42.0	38.0	37.0	33.0	32.5	28.2	25.0	22.0	20.0	16.0	15.0	13.0	5.0
		pH	5.90	5.82	5.80	5.90	5.85	5.80	5.75	5.75	5.65	5.65	5.65	5.65	5.70
1-5	Control	Ham	45.0	45.0	45.0	45.0	45.0	44.8	43.5	42.0	39.0	36.0	32.0	30.0	15.5
		Loin	44.0	44.0	43.0	42.5	39.0	36.0	33.0	30.5	26.0	23.0	20.5	19.5	13.0
		pH	6.10	6.10	6.10	6.10	6.05	6.00	6.00	5.90	5.95	5.90	6.00	5.90	5.90
3-6	Aldacta- zide	Ham	42.9	43.0	43.0	42.1	42.0	40.0	38.0	36.0	33.8	30.0	27.2	23.5	7.5
		Loin	42.0	40.0	37.6	34.0	31.0	27.0	23.0	20.0	18.0	15.0	13.5	11.5	5.8
		pH	5.90	5.80	5.90	5.85	6.00	5.90	5.90	5.90	5.75	5.70	5.60	5.60	5.70
3-4	Aldacta- zide	Ham	43.0	42.7	42.5	42.0	41.0	39.0	37.0	35.0	33.0	30.1	26.0	23.0	8.0
		Loin	41.9	38.0	38.0	35.0	32.5	29.0	26.0	23.5	21.5	19.0	16.0	14.0	6.0
		pH	6.20	6.10	6.00	6.00	6.00	6.00	5.90	5.90	5.85	5.90	5.85	5.95	5.95
2-3	Aldacta- zide	Ham	42.5	43.5	43.0	43.0	42.0	50.0	38.0	35.8	32.5	30.0	26.0	24.0	7.9
		Loin	42.0	38.5	35.0	34.5	30.5	27.0	24.0	22.0	19.0	17.0	14.5	13.5	5.5
		pH	6.30	5.75	5.90	5.90	5.75	5.60	5.60	5.55	5.65	5.60	5.65	5.70	5.75
3-7	Control	Ham	43.0	43.0	42.9	43.0	42.0	40.0	38.9	37.0	36.0	31.0	28.0	24.0	7.0
		Loin	40.2	40.3	38.0	37.0	34.0	30.1	28.2	25.5	24.0	19.0	16.0	13.5	5.0
		pH ^c	5.90	5.90	5.95	5.95	--	--	--	--	--	5.55	--	--	--

TABLE 9 (cont'd.). TEMPERATURES AND pH VALUES OF INDIVIDUAL ANIMALS
AT VARIOUS INTERVALS POST-MORTEM FOR TRIAL 3

Animal No.	Treatment	Item ^a	Intervals Post-Mortem ^b												
			0	0.25	0.50	0.75	1	1.5	2	2.5	3	4	5	6	24
1-4a	Control	Ham	44.5	45.0	43.5	42.1	42.9	41.1	39.0	36.0	34.5	29.0	25.5	24.0	7.0
		Loin	42.0	40.0	36.0	33.0	32.0	28.3	25.0	21.6	19.5	15.0	13.0	12.0	5.0
		pH	6.10	--	--	--	--	--	--	--	--	5.55	--	--	--
1-6	Control	Ham	44.5	45.0	45.0	44.5	44.5	43.5	42.0	39.5	33.5	34.0	31.0	29.0	15.0
		Loin	43.9	43.0	42.0	39.0	37.0	33.9	31.8	28.0	26.0	22.0	20.0	18.5	13.0
		pH	--	--	--	--	--	--	--	--	--	5.50	--	--	--
1-3	Aldacta- zide	Ham	43.5	43.5	43.2	42.5	42.0	38.1	37.9	36.5	35.0	31.0	27.0	24.2	7.0
		Loin	42.0	42.1	40.2	36.9	35.0	28.0	27.1	24.9	22.0	18.5	15.0	13.0	5.0
		pH	5.70	5.65	5.90	5.72	5.90	--	--	--	--	5.60	--	--	--
15-1	Aldacta- zide	Ham	44.0	43.8	43.2	43.1	42.9	41.7	40.0	38.0	35.5	31.5	27.5	23.0	6.9
		Loin	42.0	40.0	38.0	37.0	35.2	31.5	29.0	26.0	22.5	18.0	16.0	14.0	5.0
		pH	--	--	--	--	--	--	--	--	--	5.75	--	--	--
14-13	Aldacta- zide	Ham	44.0	44.5	44.0	43.2	42.0	50.0	38.0	35.0	32.5	30.0	26.0	23.0	8.2
		Loin	42.0	41.2	38.0	36.0	32.8	29.9	26.0	22.0	20.0	18.0	15.0	13.0	5.8
		pH	--	--	--	--	--	--	--	--	--	5.50	--	--	--

^aHam and loin values are temperature, expressed as °C. pH values are from the loin.

^bExpressed in hrs. after the carcass entered the cooler.

^cSome pH values not obtained due to mechanical difficulties.

TABLE 10. ANALYSIS OF VARIANCE FOR WISCONSIN
QUALITY SCORE FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	0.52
Periods	2	0.02
Treatments	1	3.52**
P x T	2	0.27
Error	5	0.12

**($P < .01$)

TABLE 11. ANALYSIS OF VARIANCE FOR WATER BINDING
FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	337.19**
Periods	2	17.82
Treatments	1	22.39
P x T	2	10.17
Error	5	6.54

**($P < .01$)

TABLE 12. ANALYSIS OF VARIANCE FOR PROTEIN TURBIDITY
FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	11,768.80**
Periods	2	200.22
Treatments	1	387.60
P x T	2	824.14
Error	5	447.37

**($P < .01$)

TABLE 13. ANALYSIS OF VARIANCE FOR SERUM SODIUM
FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	44.08
Periods	2	2.58
Treatments	1	6.75
P x T	2	33.25
Error	5	11.68

TABLE 14. ANALYSIS OF VARIANCE FOR SERUM POTASSIUM
FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	6.31**
Periods	2	0.12
Treatments	1	0.24
P x T	2	0.58
Error	5	0.19

**($P < .01$)

TABLE 15. ANALYSIS OF VARIANCE FOR MUSCLE SODIUM
FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	4,489.97
Periods	2	723.73
Treatments	1	1,423.98
P x T	2	2,193.74
Error	5	1,412.44

TABLE 16. ANALYSIS OF VARIANCE FOR MUSCLE POTASSIUM
FOR TRIAL 1

Source	d.f.	Mean Squares
Blocks	1	439,488.86**
Periods	2	20,394.68
Treatments	1	15,715.87
P x T	2	73,645.47
Error	5	21,498.20

**(P < .01)

TABLE 17. LEAST-SQUARES ANALYSIS OF VARIANCE FOR
WISCONSIN QUALITY SCORE FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	1.53
Periods	2	0.14
Treatments	1	0.02
P x T	2	0.39
Error	3	1.03

TABLE 18. LEAST-SQUARES ANALYSIS OF VARIANCE
FOR WATER BINDING FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	129.28*
Periods	2	13.48
Treatments	1	14.74
P x T	2	22.33
Error	3	11.60

*(P < .05)

TABLE 19. LEAST-SQUARES ANALYSIS OF VARIANCE
FOR PROTEIN TURBIDITY FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	693.78
Periods	2	79.40
Treatments	1	1,323.14
P x T	2	1,088.04
Error	3	350.61

TABLE 20. LEAST-SQUARES ANALYSIS OF VARIANCE
FOR SERUM SODIUM FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	0.50
Periods	2	19.29
Treatments	1	6.25
P x T	2	85.05
Error	3	26.17

TABLE 21. LEAST-SQUARES ANALYSIS OF VARIANCE
FOR SERUM POTASSIUM FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	3.85
Periods	2	5.05
Treatments	1	1.02
P x T	2	1.17
Error	3	5.00

TABLE 22. LEAST-SQUARES ANALYSIS OF VARIANCE
FOR MUSCLE SODIUM FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	2,728.76
Periods	2	1,508.25
Treatments	1	99.95
P x T	2	4,576.02
Error	3	1,070.16

TABLE 23. LEAST-SQUARES ANALYSIS OF VARIANCE
FOR MUSCLE POTASSIUM FOR TRIAL 2

Source	d.f.	Mean Squares
Blocks	1	184,212.36
Periods	2	18,807.48
Treatments	1	20,930.86
P x T	2	27,870.88
Error	3	23,095.65

TABLE 24. ANALYSIS OF VARIANCE FOR WISCONSIN QUALITY
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	0.00
Periods	2	0.52
Treatments	1	5.33*
P x T	2	0.02
Error	5	0.35

*(P < .05)

TABLE 25. ANALYSIS OF VARIANCE FOR WATER BINDING
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	5.55
Periods	2	12.18
Treatments	1	1.66
P x T	2	11.64
Error	5	7.98

TABLE 26. ANALYSIS OF VARIANCE FOR PROTEIN TURBIDITY
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	120.33
Periods	2	0.96
Treatments	1	1,624.01
P x T	2	28.74
Error	5	403.20

TABLE 27. ANALYSIS OF VARIANCE FOR SERUM SODIUM
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	24.08
Periods	2	92.33
Treatments	1	60.75
P x T	2	252.00
Error	5	147.48

TABLE 28. ANALYSIS OF VARIANCE FOR SERUM POTASSIUM
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	2.14
Periods	2	12.72*
Treatments	1	2.58
P x T	2	0.16
Error	5	1.67

*(P < .05)

TABLE 29. ANALYSIS OF VARIANCE FOR MUSCLE SODIUM
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	301.10
Periods	2	853.70
Treatments	1	4,080.03
P x T	2	225.06
Error	5	1,055.84

TABLE 30. ANALYSIS OF VARIANCE FOR MUSCLE POTASSIUM
FOR TRIAL 3

Source	d.f.	Mean Squares
Blocks	1	51,541.97
Periods	2	49,893.91
Treatments	1	1,645.72
P x T	2	104,395.72
Error	5	26,636.48

VITA

Ferdinand Ludvic Passbach, Jr., was born in Natchez, Mississippi, November 21, 1941, and received his elementary education at Cathedral Elementary School. He attended Cathedral High School, Natchez, Mississippi, and graduated in May, 1959.

In September, 1959, he enrolled at Loyola University, New Orleans, Louisiana, where he majored in biology until May, 1960. The following September, he enrolled at Mississippi State University, where he received the degree of Bachelor of Science in Agriculture in May, 1963.

From September, 1963, until August, 1965, he attended Mississippi State University as a Graduate Assistant in the Animal Husbandry Department. He was awarded the degree of Master of Science in Agriculture in August, 1965.

In September, 1965, he accepted a position as Associate in the Department of Animal Science at Louisiana State University and began working toward his doctorate. Two years later he relinquished this position for one as a Graduate Assistant so that he could more actively pursue his degree. Since that time he has been engaged in fulfilling the requirements for a Doctor of Philosophy in the Department of Animal Science with a minor in biochemistry.

EXAMINATION AND THESIS REPORT

Candidate: **Ferdinand Ludvic Passbach, Jr.**

Major Field: **Animal Science**

Title of Thesis: **Influence of Aldosterone on the Quality of Porcine Muscle.**

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

July 1, 1969