ROLE OF THE ADRENAL CORTEX AND THYROID GLAND FUNCTION UPON SOME PHYSICAL AND CHEMICAL PROPERTIES OF PORCINE MUSCLE

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Robert A. Merkel
Major professor

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ABSTRACT

ROLE OF THE ADRENAL CORTEX AND THYROID GLAND FUNCTION UPON SOME PHYSICAL AND CHEMICAL PROPERTIES OF PORCINE MUSCLE

By David Glen Topel

This study consisted of three separate investigations and included 148 pigs. In Part I, the influence of thiouracil and Tapazole[1] feeding upon adrenal and thyroid weight, plasma 17-hydroxycorticosteroid (17 OHCS) levels, and some chemical and physical properties of the l. dorsi muscle was studied. Part II included the determination of plasma sodium, potassium and 17 OHCS levels, adrenal weights and extractability of muscle proteins and sodium and potassium content of the l. dorsi muscle from normal pigs and those exhibiting slight and severe PSE musculature. The influence of exogenous adrenocortical-like steroids upon plasma 17 OHCS, sodium and potassium levels and several porcine muscle characteristics was studied in Part III.

The goitrogens Tapazole and thiouracil were fed at various levels and for varying periods of time. Both drugs caused hypertrophy of the thyroid gland to approximately the same degree, but thiouracil had a more pronounced effect upon adrenal atrophy than Tapazole. L. dorsi muscle pH, myofibrillar and sarcoplasmic protein extractability, non-protein nitrogen and sodium and potassium levels were quite similar when Tapazole and thiouracil treated pigs were compared to controls. Thiouracil treatment, however, produced a pale, soft exudative (PSE) condition in the ham muscles, especially the gluteus medius in some pigs; whereas, Tapazole treatment resulted in normal colored, firm appearing ham musculature. Plasma 17 OHCS levels were lower than control values in both the Tapazole and thiouracil treated pigs, but these differences were not significant.

[1] 1-methyl-2 mercaptoimidazole

In Part II, significantly lower quantities of sarcoplasmic, myofibrillar proteins and NPN were extracted from PSE musculature than normal muscle. Considerable variation in the quantity of extractable protein was found between muscle samples in the slight PSE group. These extractability data indicate that muscle protein extractability is not entirely consistent with the visible PSE muscle characteristics.

Plasma 17 OHCS levels from pigs possessing severe PSE muscles were 3.3 γ/100 ml lower than the normal group. This difference was approaching significance ($P < .05$). Considerable variation existed within the three groups for plasma 17 OHCS which was apparently due to the variation from one individual to another in disposition prior to collecting the blood sample. Muscle pH was significantly different between the normal (pH 5.46), slight PSE (pH 5.35) and severe PSE (pH 5.18) groups. L. dorsi muscle area was significantly ($P < .01$) larger for the severe PSE group (4.64 sq. in.) than normal pigs. A highly significant correlation coefficient (-.43) was obtained between l. dorsi area and muscle firmness scores for the pigs in this study. Thus, the more muscular pigs probably are more predisposed to PSE muscle than poorly muscled pigs.

Administration of prednisolone or methyl prednisolone produced adrenal atrophy and lower levels of plasma 17 OHCS for each level and period of time studied in the experiments of Part III. Quantity of sarcoplasmic and myofibrillar proteins from glucocorticoid treated pigs, in all three experiments, was not significantly different from controls. Daily prednisolone injection (200 mg/day) for seven days resulted in significantly higher muscle NPN values. Methyl prednisolone at either 225 or 450 mg

per day for 21 and 25 days produced no significant differences in muscle NPN values. However, muscle samples were collected either 3 or 5 days after the last methyl prednisolone administration; whereas, muscle samples were collected 24 hours after the last prednisolone administration.

Marked differences in rate of post-mortem muscle pH fall were obtained between prednisolone treated and control pigs. Prednisolone was found to have a sodium retaining effect upon muscle while methyl prednisolone resulted in sodium dimunition. Neither drug significantly altered muscle potassium level of the l. dorsi muscle. Sodium and potassium levels in the plasma of pigs treated with prednisolone or methyl prednisolone were within the normal range and similar to values obtained for controls.

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By

DAVID GLEN TOPEL

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INTRODUCTION

The subject of porcine quality has recently received considerable attention, especially among European and American researchers. The term porcine quality referred to in this manuscript is defined as those physical, chemical and morphological factors which are related to palatability characteristics. While emphasis has been placed primarily upon measurement and improvement of the quantitative aspects of porcine carcasses such as degree of muscling, lean to fat ratio, etc. in the past two decades, the qualitative characteristics have received less attention. It had been assumed that quality was not a serious problem in porcine muscle probably because of the young slaughter age and processing methods employed.

Research findings within the last six to eight years indicate a relationship exists between the pork quality factors such as degree of firmness, color of muscle, intramuscular fat and the palatability factors. More recently the industry has become aware of the pale, soft, exudative (PSE) condition in porcine carcasses. This condition results in abnormally high shrinkage and poor water binding properties during processing. In addition, PSE pork has been shown to be less tender and juicy than normal pork.

Work by Ludvigsen (1953), Briskey and Wismer-Pedersen (1961a,b), Bendall and Wismer-Pedersen (1962) and Goldspink and McLoughlin (1964) showed a relationship between post-mortem pH and muscle temperature with the incidence of pale, soft, exudative pork. The PSE condition resulted when a rapid drop in pH occurred while muscle temperature remained above

37°C. Although data are available on various post-mortem chemical and physical changes in porcine muscle, the causal factors for the rapid pH drop remains largely unexplained.

It is known that the thyroid and adrenal glands, directly or indirectly, influence specific enzymes involved in oxidative and anaerobic pathways in muscle tissue (White et al., 1964). Little evidence is available concerning the influence of adrenal or thyroid hormones on physical or chemical properties of muscle and much of this work has been conducted with rats and dogs. Ludvigsen (1957) and Henry et al. (1958) reported data indicating that the thyroid and adrenal gland activity can alter physical and chemical properties of porcine muscle. No other data were found in the literature relating the activity of these two glands to ultimate physical and chemical properties of porcine muscles.

After reviewing these facts, this study was undertaken with the following experimental objectives:

1. To determine if there is a possible relationship of the porcine thyroid and adrenal glands with specific post-mortem muscle characteristics associated with the PSE condition.
2. To study the relationship between goitrogenic activity, adrenal size and plasma glucocorticoid levels. In addition, specific chemical and physical properties of the l. dorsi were studied.
3. To investigate the influence of exogenous adrenal glucocorticoids upon various chemical, physical and morphological properties of porcine muscle.

REVIEW OF LITERATURE

Role of the Thyroid Gland in Soft, Exudative Porcine Muscle

Ludvigsen (1953) reported a condition in Danish Landrace pigs which he called "muscular degeneration" (MD). He described the condition as alterations of the musculature, appearing macroscopically as a discoloration, the altered musculature having a pale or graying color. Freshly cut muscle was juicy and had an extremely sour smell. Ludvigsen noted that the condition was especially prevalent in the l. dorsi muscle, but that on occasion it was found in all muscles of a given animal (total MD). The pH of normal l. dorsi muscle was 6.8 to 7.0 approximately 45 minutes post-mortem, while values as low as 5.3 to 5.5 were found when "muscular degeneration" was observed.

Ludvigsen (1953) produced total MD by feeding 1 gram of methyl thiouracil daily for 10 days prior to slaughter. Likewise, if the same dose of methyl thiouracil was fed for 20 to 24 days, 2 to 2 1/2 months before slaughter, total MD will result as well as several secondary effects, such as loss of appetite, interruption of growth and exema . The author also reported that total MD muscle changes were counteracted when pigs were fed 2 grams of iodinated casein daily containing 2.7 to 3.0% free thyroxin for a period of 10 days, three weeks prior to slaughter. These experiments were conducted with pigs having total MD diagnosed by means of a muscular puncture.

Briskey (1963) fed methyl thiouracil for 10 days prior to slaughter and reported that the ham musculature from these pigs was pale, soft, and

exudative when compared to untreated controls. When pigs were exercised to reduce the glycogen level, quality was measurably improved.

Terrill et al. (1948, 1950) and Acevedo et al. (1948) reported no differences in physical or chemical composition of the carcasses from thiouracil fed pigs, but weight of the thyroid gland was significantly increased by this goitrogen. Carcass firmness was one of the physical characteristics observed; however, the authors apparently were referring to firmness of fat rather than skeletal muscle.

Thyroid-Adrenal Relationship

There is considerable evidence suggesting an interrelationship between the thyroid gland and adrenal cortex. Leblond and Hoff (1944) noted a decrease in adrenal gland size of rats receiving goitrogenic sulfa drugs or thiouracil. Baumann and Marine (1945) confirmed these findings and they noted involution of the adrenals to half their normal size in rats fed thiouracil for four months. Histological studies revealed adrenal involution involved all three zones of the cortex. The fasicular zone showed a greater amount of lipoid material than normally seen while the lipoid content of the remaining zones was reduced. The authors concluded that cortical involution was apparently a compensatory response for loss of thyroid secretion.

Maqsood (1950) reported high environmental temperature significantly decreased weight of the adrenal gland in male mice to about the same degree as thiouracil administration. Maqsood indicated the decrease in adrenal weight was probably due to a decrease in thyroid secretion rate which occurred during exposure to high environmental temperature.

McCarthy et al. (1959) provided thiouracil (0.1%), Tapazole (Lilly 0.001%, 0.005% and 0.01%), potassium perchlorate 1% in drinking water and fed a low iodine diet, and incorporated p-aminosalicylic acid (1%, 2%, 4%), p-aminobenzoic acid (2%, 4%), and sulfaguanidine (2%, 4%) in the diet of rats. After 12 weeks of treatment, atrophy of the adrenal gland was evident in all treatments except p-aminosalicylic acid.

McCarthy and Murpheree (1960) later reported the possibility of direct action of p-aminobenzoic acid on the adrenal gland as a more likely mechanism for induction of adrenal atrophy. The authors found an inhibition of the thyroid gland as indicated by reduced I^{131} uptake by the thyroid. Thus, further evidence is presented to associate goitrogenic activity with ability to induce adrenal atrophy, although the possibility of direct action of p-aminobenzoic acid on the adrenal gland coupled with thyroid inhibition cannot be excluded.

Lazo-Wasem (1960) also reported that 0.3% thiouracil fed to rats for 3 weeks brought about adrenal atrophy accompanied by thyroid and pituitary enlargement as compared to non-thiouracil fed controls. Pituitary ACTH content of thiouracil fed rats was less than 1/3 that found in controls. These data support the hypothesis that adrenal atrophy following thiouracil is probably associated with lowered ACTH titers.

Role of Adrenal Glucocorticoids in Muscle Disorders

In reviewing the literature, one recognizes a paucity of information regarding the hyper and/or hypo affects of glucocorticoid levels upon skeletal muscle characteristics.

Hyperfunction of the Adrenal Cortex. Hyperfunction of the adrenal cortex leads to hyperglycemic states in the human and other animals. This condition is usually referred to as hypercorticordism (Cushing's syndrome). Although the morphologic causes of hypercorticoidism is somewhat confused, therewas characteristically an augmented secretion of glucocorticoids (C-11-oxysteroids). In complications of spontaneous Cushing's syndrome, muscle weakness appears as a complaint in one half to more than 80% of the patients (Charles et al., 1952). On the other hand, the frequency of muscle weakness among patients receiving synthetic hydrocortisone type steroids in pharmacologic dosages has not been established. Severe or striking examples of muscle weakness have, however, been described during treatment with cortisone, 9-alpha fluorohydrocortisone, prednisone, and triamcinalone (Perkoff et al., 1959; Williams and Lond, 1959; MacLean and Schurr, 1959; and Harman, 1959).

Slight atrophy of the triceps surae muscles of the rat was produced by immobilization of one hind limb in a plaster of Paris cast. However, disuse atrophy was significantly aggravated when the immobilized animal was exposed to stressors and/or treated with cortisol (Bajusy, 1958).

Faludi et al. (1964) induced myopathy in dogs by injecting large doses of anti-inflammatory steroids (cortisol, prednisolone, methyl prednisolone, hexamethasone and triamcinolone). Weight loss and muscle atrophy occurred in all treated groups, but was most pronounced in the triamcinolone treated dogs. Marked differences existed between the treated groups in the capacity to decrease muscle size. Cortisol reduced muscle

size the least. Fiber thickness of the muscle changed least in the methyl-prednisolone treated group and was most pronounced in the triamcinolone group. The authors indicate that further investigation is necessary to understand the pathogenesis of this myopathy.

The mechanism responsible for hyperglycemia and muscle weakness caused by the glucocorticoids is in part explicable by the effect of these hormones upon gluconeogenesis. This was first inferred by Long et al. (1940) from their data with rats. They found that when either a potent adrenal cortical extract or one of the adrenal C-11-oxysteroids was administered to fasted normal, hypophysectomized, or adrenalectomized rats, urinary nitrogen excretion increased. Analysis indicated that 53 to 65% of the protein catabolized was converted to glucose. Ingle (1941) force-fed rats a high carbohydrate diet and observed that adrenal corticoid administration brought about a concommitant increase in urinary nitrogen along with hyperglycemia and glycosuria.

Studies with radioactive isotopes have clearly demonstrated the effect of adrenal C-11-oxysteroids upon gluconeogenesis. Welt et al. (1952) injected C^{14} labeled glucose at a constant rate into rats and estimated gluconeogenesis by comparing activities of injected and excreted glucose. They found that cortisone administration resulted in a sevenfold increase in glucose production from non-carbohydrate material. The origin of steroid myopathy from hypercorticoidism remains speculative. One current hypothesis is that it is related to losses of cell protein by the great increase in the rate of gluconeogenesis, thus causing muscle weakness.

Hypofunction of the Adrenal Cortex. In man atrophy of the adrenal cortex produces a syndrome known as Addison's disease. With few exceptions, the symptoms involve extreme muscular weakness as well as other abnormalities (Turner, 1955). According to Ludvigsen (1957) hypofunction of the thyroid and adrenal cortex appears to be the actual cause of the muscular changes characteristic of PSE pork. From his observations he concluded that these muscle changes resulted from an insufficiency of thyroid hormone in the blood and a reduced corticotrophin content of the anterior pituitary gland. The condition described by Ludvigsen in pigs may not at all be comparable to Addison's disease in man because death of the animal seldom occurs.

Ludvigsen (1957) also reported reduced lactic acid in venous blood of MD pigs after exercise and increased constriction of the arterioles and capillaries in such muscle. He postulated that this constriction is responsible for lack of rise in lactic acid in the blood during exercise which is normally expected.

Ludvigsen (1957) also suggested that adrenal cortical hormones have an influence upon vasomotor reactions. One of the symptoms he observed in MD pigs was vasoconstriction of the skeletal muscles during exercise, and since hydrocortisate has a striking vasodilating effect, Ludvigsen postulated the pituitary-adrenal cortex axis obviously plays an important role in the regulation of vasomotor reactions.

Wismer-Pedersen (1959) observed that the incidence of the condition described by Ludvigsen (1953, 1957) was very rare in Danish Landrace pigs and called the condition usually encountered "pale and watery."

Henry et al. (1958) submitted 12 pigs to high nutritional levels and found they were sensitive to such stress as evidenced by hypernatrenia. The authors indicated low quality pork (exudative myopathia) was involved with a hyperproduction of aldosterone and somatotropic hormone (STH), with concommitant deficiency of ACTH and glucocorticoids. The muscles were loaded with sodium and a potassium deficiency ensues. There was progressive disappearance of striation in the fibrillae and a loss of muscle pigment. Eventually decalcification, polyuria and general asthemia resulted.

Role of Adrenal Cortex in Amino Acid Metabolism

Wool and Weinshelbaum (1959) reported the function of cortisone and cortisol in addition to their participation in the regulation of protein metabolism, was the mobilization of endogenous protein. They incorporated C^{14} labeled amino acids into a protein fraction of diaphragms excised from adrenalectomized and normal rats and found less incorporation of C^{14}-amino acids in the protein fraction from normal rats compared with adrenalectomized rats.

Wool (1960) investigated this difference between normal and adrenalectomized rats by a rather indirect approach. It was found that cortisone decreased the rate of penetration into the muscle cell and decreased accumulation of C^{14}-amino acids by the cells of isolated rat diaphragm. Therefore, less amino acids might be available for protein synthesis. Adrenalectomy did not effect amino acid transport into the diaphragm cells;

however, adrenalectomy increased the rate of incorporation of radioactivity from L-phenylalanine into protein without a simultaneous effect on the rate of penetration of that amino acid.

Kaplan and Shimizu (1963) measured the effects of cortisol on the concentrations of free amino acids in muscle tissue. Administration of cortisol appeared to result in increased concentration of virtually all amino acids and urea in the muscle of both fasted and non-fasted rats. These findings indicate evidence against the hypothesis that cortisol exerts its effect at the muscle cell membrane. If cortisol decreases the permeability of muscle cells to amino acids which, in turn, results in diminution of the pool of amino acids available for activation and incorporation into protein, then less amino acids should have been found in the muscle cells when cortisol was administered.

Kostyo (1965) reported adrenal steroids caused an appreciable delay in the response of the muscle amino acid transport process both in vivo and in vitro. This suggests the interaction of the steroid with the muscle cell may be at some site other than the membrane transport system.

Ryan (1963) found a variable effect on rat muscle amino acids depending on length of administration of hydrocortisone. Twenty four hours after injection of hydrocortisone an increase in the free amino acids of plasma and muscle was found. These acids were decreased after 10 days of treatment with hydrocortisone.

Inhibitory Action of Steroids on Adrenal Steroidogenesis

Natural Steroids. The studies of Ingle and co-workers (1937, 1938) showed that prolonged treatment with corticosteroids caused atrophy and hypofunction of the adrenal cortex. The view has generally been accepted that the mechanism responsible for this action of corticosteroids is an inhibition of pituitary ACTH production (Boland and Headley, 1949). This concept of the mechanism of diminished adrenal function has found wide acceptance in the literature and the possibility of direct inhibition by corticosteroids has been raised only upon occasion.

Synthetic Steroids. Péron et al. (1960) found that corticosterone inhibited in vitro steroidogenesis and similar findings with glucocorticoids by Fekete and Görög (1963) suggests that a direct adrenal inhibitory mechanism plays a physiological and pharmacological role, in addition to the regulatory mechanism controlling adrenal steroid function mediated by the pituitary gland.

Christy et al. (1956) reported that prednisolone administered for periods of one to two weeks appeared to be four or more times as effective as similarly administered cortisone in suppressing adrenocortical responsiveness.

Metabolism of Glucocorticoids

Schapiro and Katz (1959) injected C^{14} labeled hydrocortisone in rats and reported that the C^{14} activity was widely distributed in the tissues.

The results were variable from one experiment to another, but in most experiments radioactivity was higher in the posterior pituitary gland than in any other tissue. Activity in the anterior pituitary, liver and kidney was higher than that in muscle or brain.

Gold (1960) reported the biologic half-life of free 11-desoxycortisol in circulating plasma of man was 42 minutes (estimated as Porter-Silber chromogen).

Species Differences in Plasma Glucocorticoids

Kruger et al. (1965) reported normal level for free glucocorticoids in human plasma was approximately 16 to 18 γ/100 ml. These authors pointed out, however, that method of measurement and the time of day the sample is collected may influence results.

Bush (1953) studied the levels of corticosterone in the rat, rabbit, ferret, cat and Rhesus monkey. All species examined were found to secrete large amounts of 17-hydroxycorticosterone (17 OHCS) and/or corticosterone. The ratio of 17 OHCS to corticosterone secreted varied from <0.05 in rats to >20 in the Rhesus monkey, but did not vary appreciably between members of any one specie. Bush suggested that specie differences observed in adrenocortical secretion are genetically determined and cannot at present be related to any known differences in adrenocortical function. Observable physiological differences may, however, exist and caution should be taken in generalizing from ACTH results with rats.

Lindner (1959) reported normal levels of 17 OHCS for sheep to be 0.5 to 1.0 ug/100 ml plasma and Brush (1960) and Shaw et al. (1960) re-

ported 3 to 4 ug/100 ml plasma to be normal for cattle. No reported levels of 17 OHCS in pig plasma were found in the literature.

Influence of Housing and Exercise on Plasma 17 OHCS Levels and Adrenal Weights

Barrett and Stockham (1963) reported that rats housed in single cages, undisturbed for 18 hours prior to bleeding showed reproducible levels of 5.5 ug/100 ml plasma. Rats housed in groups of 20 under the same conditions exhibit mean levels of 9.5 ug/100 ml plasma. Non-specific stimuli such as environmental change, noise, handling, weighing, etc., all produced marked increases in plasma corticosterone levels which remained supernormal for at least 2 hours.

Cornil et al. (1965) showed that muscular exercise caused a significant fall in plasma cortisol level.

Addis et al. (1965) studied the influence of environmental temperature and humidity upon quality of the gluteus medius muscle and adrenal gland weight of the pig. No significant differences were reported for color, firmness scores and pH of the gluteus medius muscle or weight of the adrenal gland from these pigs.

Influence of 17 OHCS on Na and K Metabolism

The adrenal cortex secretes a spectrum of steroids with differing biological properties. At one end of the spectrum is cortisol, weak in its effects upon renal electrolyte function, but active in protein and carbohydrate metabolism. At the opposite extreme is aldosterone, active

in electrolyte regulation but having a negligible effect upon carbohydrate and protein metabolism in amounts normally secreted. Corticosterone is intermediate, sharing some of the biological activities of both the aforementioned steroids but less potent in both respects (Barger et al. 1958).

The work of Seldin et al. (1951) suggested that cortisol administered in very high doses induces sodium retention and increases potassium excretion. Cortisol is the dominant secretion of the adrenals in man and other species and it maintains a normal glomerular filtration rate (Mendelsohn and Pearson, 1955). However, it should be noted that cortisone action permits the organism to maintain the integrity of the regulatory mechanism so as to enable the nephron to adapt readily to changing salt loads (Ingle, 1952).

Davis and Howell (1953) studied the sodium retaining activity of cortisone in adrenalectomized dogs having acites. Administration of cortisone in physiological doses led to natridresis either by affecting the glomerular filtration rate, the rate of tubular reaborption of sodium or both.

Knowlton (1960) reported no impressive alterations in sodium or potassium content of the skeletal muscle from adrenalectomized rats injected with cortisone acetate in amounts sufficient to induce severe hypertension. In contrast, skeletal muscle from adrenalectomized rats injected with comparable amounts of desoxycorticosterone showed a marked increase in muscle sodium and reduction in potassium content.

Faludi et al. (1964) reported serum electrolytes were within normal limits throughout the experiment when cortisol, prenisolone, methyl prednisolone, dexamethasone and triamcinolone were administered to dogs at a dosage of 100 mg/day.

Swingle et al. (1958) indicated that adrenal steroids possessing potent glucocorticoid activity function as a homeostatic mechanism in the salt and water balance of the body by enabling the animal to freely transfer fluid and electrolyte from one body compartment to another. These authors indicated that aldosterone apparently will not function in this capacity.

Levels of Sodium and Potassium in Porcine Muscle

Kirton and Pearson (1963) reported a range of 1750-3070 ppm potassium and 350-470 ppm sodium (fresh basis) in samples of ground pork. Lawrie and Pomeroy (1963) and Gillette et al. (1965) reported a smaller range for the l. dorsi muscle, but these values were similar to those of Kirton and Pearson (1963).

Levels of Sodium and Potassium in Pig Serum

Bohstedt and Grummer (1954) reported a range of 314 to 336 mg % sodium in pig serum. Widdowson et al. (1956) reported normal values for serum sodium and potassium of the pig to be 331 ± 10 mg % and 23.4 ± 2.1 mg %, respectively. Similar findings were reported by Kornegay et al. (1964).

Extractability of Muscle Proteins

One of the first studies reported concerning the extractability of muscle proteins was by Deuticke (1932). He found that muscles which had been fatigued by stimulation, frozen and pulverized, imparted less protein to an extracting solution than those freshly extracted. Bate-Smith (1934) studied the effects of a series of extracting solutions. He found that with ammonium and lithium chlorides of adequate strength, no differences could be observed between the extractability of fresh and rigor muscle.

Bailey (1954) pointed out that the most direct explanation of this early work was that stimulation and rigor involve a change of state which is reflected in a loss of muscle protein solubility in some salt solutions but not all. In light of recent knowledge, they probably involve the combination of myosin and actin to give a less soluble complex. In freshly minced relaxed muscle, the ATP acts as a specific dissociating agent. In rigor or fatigued muscle, extraction is facilitated by salts which depolymerize the complex. Bailey concluded that, considering the large amount of recent work on the theory of contraction and rigor, this is probably an over simplified explanation.

From present theories of rigor, it might be thought the disappearance of ATP from the muscle is largely responsible for an "in vivo" aggregation of myosin and actin which retards protein extractability. According to Bailey (1954), this is incorrect. While ATP hastens the rate of solution, it does not increase the final yield, except when the

extracting solution has an ionic strength above 0.5. Crepax (1951) indicated that the action of ATP upon extractability of the muscle proteins is that of strengthening the dissociating action of electrolytes on the binding forces which hold the proteins in place within the muscle. The characteristic decrease in extractability of contracted muscles is not due to the hydrolysis of ATP which accompanies these contractions.

Bailey (1954) stated that extractability is not solely determined by solubility. He indicated that this is probably because the dissolution of F actin or F actomyosin threads, several microns long, is seriously impeded in a mechanical way by the insoluble conponents of muscle. The extractability of myosin and actin depends, in part, on the mutual combination of these proteins and the hindrance to diffusion by the surrounding insoluble muscle structures. A relaxed muscle, freshly minced, will yield free myosin, even on coarse grinding, but further comminution and stronger salt solutions will bring out large amounts of actomyosin. Homogenization must be continued to mechanically break not only the surrounding structures but to disperse the concentrated actin gel.

Considering the above facts, Bailey (1954) drew the following conclusions for muscle protein extractability: At any particular stage of rigor the extractability of the intracellular protein fraction appears to be determined by pH, ionic strength of extracting solution, type of extractant and by adequacy of grinding.

Jacob (1947) studied the effect of pH on extractability of sarcoplasmic proteins. He found that pH 7.7 was optimal for extraction of all

muscle proteins within the phosphate buffer range. At all pH values, a precipitate formed on dialysis. The quantity of precipitate varied considerably above pH 7.8, was least at 7.6 and became more abundant at lower pH values; however, the precipitate formed above pH 7.1 was soluble in 0.5M KCl.

Helander (1957) obtained the maximum protein solubility in the pH range of 6.5-9.0. Recovery was substantially the same within these limits. Therefore, pH 7.4 was selected for use in his studies. He also indicated that optimum ratio of solvent volume to tissue volume was 10:1.

Saffle and Galbreath (1964) reported that fat had no effect upon the extractability of salt-soluble proteins. Muscle pH had a significant effect on the amount of salt-soluble protein which could be extracted. As pH increased, amount of protein extracted increased. The amount of salt-soluble protein was 50% greater in prerigor beef than 48 hours post-mortem.

Borchert and Briskey (1965) found that liquid nitrogen freezing of prerigor muscle increased extractability of both sarcoplasmic and myofibrillar protein fractions as compared to controls which were chilled under normal conditions for 24 hours.

Protein Extractability of Normal and Soft Exudative Muscle

Bendall and Wismer-Pedersen (1962) showed that washed muscle fibrils obtained from PSE pork had a lower water retention at low ionic strength and much lower extractability at high ionic strength than fibrils from normal pork. Washed fibrils from PSE pork showed a greater protein con-

tent than similar fibrils from normal pork. The authors concluded that the greater fibrillar protein content in the watery fibrils was probably caused by a layer of denatured sarcoplasmic protein which was firmly bound to the surface of the myofilaments.

Hill (1962) reported values for the amount of sarcoplasmic, myofibril, stroma and NPN in porcine, bovine and ovine muscle. Results showed that stroma protein was highest in bovine muscle and lowest in porcine. He also reported differences between individual muscles for extractability of the protein fractions.

McLoughlin (1963) concluded that solubility of the sarcoplasmic and fibrillar proteins from exudative muscle was reduced at low and high ionic strengths, which was in agreement with Bendall and Wismer-Pedersen (1962). McLoughlin and Goldspink (1963a) and Goldspink and McLoughlin (1964) observed the effect of temperature and pH on the solubility of sarcoplasmic proteins. They concluded that color of post-rigor muscle could be maintained if the temperature of muscle was reduced to about 30°C before the pH approached 6.0.

Solubilities of sarcoplasmic and myofibrillar protein were determined by Sayre and Briskey (1963) at the time of slaughter, onset of rigor mortis, completion of rigor mortis and 24 hours post-mortem in muscles exhibiting a wide range of physiological conditions during the post-mortem period. Muscle protein solubility was grossly altered by conditions of both temperature and pH which existed at onset of rigor mortis or during the first few hours after death. Sarcoplasmic protein

solubility at 24 hours was decreased to 55% of that found at 0 hour in muscle groups exhibiting high temperature and low pH at the onset of rigor mortis. Conversely, only a 17% reduction of sarcoplasmic protein solubility was noted in groups with high pH at onset. Myofibrillar protein solubility ranged from no reduction during the first 24 hours after death when pH remained high at onset to 75% reduction in muscle with low pH and high temperature at the onset of rigor mortis. The 24 hour pH of muscle appeared to have only a minor influence on protein solubility. Sayre and Briskey (1963) concluded that protein solubility appeared to be one of the major factors affecting juice-retaining properties of muscle.

Partmann (1963) measured extractability of the actomyosin fraction to determine the degree of denaturation of fish muscle during freezer storage. The extractability of the structural proteins of rosefish and cod decreased at higher storage temperatures and with advancing storage time.

Scopes (1964) reported that sarcoplasmic proteins are denatured readily at pH values below 6.0 at 37°C and at higher temperatures independent of pH. He found that denaturation of sarcoplasmic protein <u>in situ</u> is associated with decreased myofibrillar solubility in KCl. Starch-gel electrophoresis indicated one major and several minor proteins were specifically denatured in conditions of low pH and high temperature. The major protein has been identified as ATP-creatine phosphotransferase.

Factors Which Influence Meat Hydration

Muscle Proteins and Water. A comprehensive review of the basic concepts of meat hydration has been reported by Hamm (1960). He stated that the "true hydration water" of muscle is the amount of water that attaches to protein by monomolecular and multimolecular adsorption. This water is bound directly to polar groups of proteins and makes up about 4 to 5% of the water in muscle. The physical properties of bound water are different from those of free water. Bound water has a lower vapor pressure and a lower dissolving power than normal water.

Hamm (1960) further stated that most of the water in muscle is free, chemically speaking. Apparently free water is mechanically immobilized by the network of the cellular protein membranes and protein filaments. Hamm concluded that changes in water-holding capacity of meat caused by changes of protein charges (i.e. by pH, ions, etc.) are not due to changes of true hydration water fixed to the polar groups of meat proteins.

He stated that the amount of free water "immobilized" within the tissue is influenced by the spatial structure of muscle. Tightening this spatial structure (network of proteins) decreases immobilized water and loosening the protein structure has the opposite effect. This so called "stereo effect" (Hamm, 1959) is extensively influenced by changes of protein charges. The presence of certain ions or adjustment to certain pH values greatly affects the spatial protein arrangement and consequently affects water-holding capacity. Hamm (1959) defines water-holding capacity of meat as the ability of meat to hold its own or added water during application of force (pressing, heating, grinding, etc.).

Water-Holding Capacity as Influenced by Specie, Age and Sex Differences. Schon and Stosiek (1958a,b) found an inherent difference in water-holding capacity between specie, age, and sex of meat animals. The reasons for these differences have not been elucidated. They utilized the adductor and l. dorsi muscles and found that pork has a greater water-holding capacity than beef. The water-holding capacity of bovine muscle increased in the order of steer to heifer to cow; however, no sex differences were observed for porcine muscle.

Muscle Location and Muscle Hydration. Swift and Berman (1959) reported considerable variation for glycogen content and buffering capacity among eight bovine muscles. However, all muscles showed characteristic patterns for pH changes and water retention; even though they contained residual glycogen when the ultimate pH was attained. The relatively high characteristic pH of certain muscles could not be attributed to lack of glycogen alone.

Urbin et al. (1962) reported variation within the cross-sectional area of the l. dorsi muscle when free moisture determinations were made by a modification of the Grau-Hamm procedure. The medial portion of the l. dorsi muscle had significantly lower free moisture values than the lateral portion.

Variation in juice retaining properties both between and within selected pork muscles was reported by Topel et al. (1965). The rectus femoris had the greatest difference in juice retaining properties between proximal and distal sections of the ham muscles studied.

Influence of Color and Hydrogen Ion Concentration on Meat Hydration. Water holding capacity of porcine muscle is positively related to both muscle color intensity and pH (Judge et al., 1958; Bate-Smith, 1948; Briskey et al., 1959, 1960; Lawrie, 1958; Wismer-Pedersen, 1959). Dark, firm muscle was higher in pH and lower in free water than pale, soft muscle.

The influence of pH on meat hydration is a typical example of the importance of protein net charge. The isoelectric point of muscle proteins is approximately pH 5.0. At the isoelectric point, the net charge of muscle proteins is minimal. At this pH, meat hydration has a sharp minimum. Normal pH of meat is about 5.5 which is close to the isoelectric point, consequently water holding capacity is quite low (Hamm, 1959).

According to Briskey et al. (1959), there were no significant differences in pH or the ratio of expressible water to total water among four muscle color classes of fresh hams. Hams were classified in four groups: 1) pale; 2) two-toned; 3) two-toned, normal and 4) dark color. Relative amounts of expressible water, however, increased significantly during the chilling process. This increase was greatest from muscles which possessed high concentrations of glycogen at the time of slaughter.

Briskey and Wismer-Pedersen (1961a) recorded continuous pH and temperature changes during post-mortem chilling. Carcasses that had a sharp, significant decrease to about pH 5.1, 1 1/2 hours post-mortem and a subsequent elevation to pH 5.3 to 5.6 possessed pale, exudative tissue with soft, inferior structure.

Briskey and Wismer-Pedersen (1961b) concluded that the pH-time sequence and subsequent development of watery or normal tissue was dependent on a number of factors. However, only the following three factors were emphasized: 1) amount of glycogen in muscle tissues at the time of slaughter; 2) the phosphorylase activity and state of glycogen in the tissue; and 3) the methylene-blue reduction activity of muscle tissues.

Temperature-pH Relationship Upon Meat Hydration. Wismer-Pedersen and Briskey (1961b) studied the effects of various combinations of temperature and acidity in relation to muscle structure. The authors suggested that pale, exudative pork could be produced by maintaining body temperature for an extended post-mortem period. Conversely, they indicated that rapid chilling of muscle samples, which had a low pH within 45 minutes of slaughter, prevented this development of soft, watery muscle.

Bendall and Wismer-Pedersen (1962) concluded the immediate cause of wateriness in pork was the combined effect of high temperature (37°C) and low pH on the muscle proteins which is in agreement with the suggestion of Wismer-Pedersen and Briskey (1961b). Bendall and Wismer-Pedersen (1962) also concluded that the depression and elevation of pH values which was reported by Briskey and Wismer-Pedersen (1961a) was a reversible phenomenon due to the effect of temperature on the pK values of charged groups of the fibrillar and sarcoplasmic proteins. They consequently refuted the suggestion of Wismer-Pedersen and Briskey (1961a) that the depression and elevation of pH was a factor in the development of soft, watery pork.

Bendall et al. (1963) reported a further study on the rates of ATP turnover in relation to pH and onset of rigor mortis in muscle samples

removed immediately after slaughter and held at 37°C. Muscle which was eventually soft and watery showed a much faster rate of ATP turnover and a shorter lag period prior to the onset of rigor. The authors were unable to explain the differences in rates, which could not be altered by various pre-mortem treatments. The authors noted that muscle which was allowed to go into rigor at a constant temperature of 37°C became watery and pale. They concluded that the probable reason for soft, watery pork was a combination of high temperature and low pH and suggested that the condition could be prevented by cooling rapidly to 30°C or below.

Sayre et al. (1964) concluded that when onset of rigor mortis occurred at pH values below 5.9 with temperatures above 35°C, the l. dorsi muscle became pale and exudative. Conversely, the authors reported that if onset of rigor mortis occurred when pH values remained above pH 6.0, the muscle was dark and firm.

Bodwell (1964) held one side of pork carcasses at 37°C and normally chilled the opposite side. He reported the 37°C treatment rarely induced soft, watery muscle as had been expected from reports of previous workers. Bodwell concluded that a low pH at a high muscle temperature per se was not a causal factor in development of PSE pork.

Histological Factors Associated with Meat Hydration. Histologically there are several unusual features and abnormalities related to muscle pH (Lawrie, 1958). Muscles with an ultimate pH of 5.3 had discernable cross-striation but the muscle fibers were frequently twisted and broken. At pH 5.1 the protein gel of more than half of the fibers appeared to have

coagulated. At pH 4.9, all fibers were abnormal: some showed no cross-striations but had longitudinal markings; still others had cross-striations, but were both twisted and finely corrugated.

Wismer-Pedersen (1959) studied histological samples of porcine loin muscle possessing different pH values. No systematic differences in appearance of the fibers and cell structure were observed between muscles with high and low pH.

Variations in the distribution of water in bovine l. dorsi, semimembranosus, serratus ventralis and rectus abdominus muscles were histologically studied by Lockett et al. (1962). It was reported that extracellular space of muscle tissue was positively correlated with the water-protein ratio; whereas, intracellular water content was negatively correlated. The evidence indicates that, in muscles which characteristically contain a relatively high proportion of water to protein, the additional water is located in extracellular spaces.

McLoughlin and Goldspink (1963b) did not observe any histological changes characteristic of degeneration in the soft, watery muscles. Lawrie (1960) observed distinct histological differences between normal and exudative muscles in Landrace pigs. However, these differences appeared to be primarily related to the 24 hour post-mortem pH level rather than the soft, watery condition.

Bendall and Wismer-Pedersen (1962) reported histological information to support their conclusion that washed fibrils from soft, watery pork showed a greater protein content than similar fibrils from normal pork.

The authors concluded that greater fibrillar protein content in the watery fibrils was probably caused by a layer of denatured sarcoplasmic protein, which was firmly bound to the surface of the myosin filaments.

Cassens et al. (1963) used electron microscopy to follow changes in porcine muscle during the 24 hour post-mortem chilling period. The affect of ante-mortem subjection to elevated temperatures or to elevated temperatures and then chilling upon the rate and magnitude of change in post-mortem muscle color, texture and water binding was studied. Normal muscle exhibited a gradual disruption of sarcoplasmic components with little if any change in the myofibrils. Muscle that went into rigor rapidly at a low pH and high temperature ultimately appeared soft, pale and watery and electron micrographs revealed a rapid disruption of sarcoplasmic components and some disorganization of the myofilaments. Muscle that went into rigor rapidly at a high pH and a reduced temperature ultimately appeared dark, firm and dry and electron micrographs revealed a high degree of organization and preservation of myofibrillar structure.

Preslaughter Factors and Methods of Prevention of Soft Pork. Briskey et al. (1959a,b,c; 1960) reported a decrease in glycogen content of ham muscles from pigs subjected to exhaustive ante-mortem exercise. An inverse relationship was reported between initial glycogen level and 24 hour pH, water-binding capacity and color.

Sayre et al. (1961) subjected pigs to a 0 to 5°C environment for 30 to 40 minutes prior to slaughter. They reported that the treatment resulted in a decrease in initial muscle glycogen and post-mortem accumulation of lactic acid. Although color intensity of the chilled muscle

increased, water-binding capacity was not consistently affected. The authors concluded that factors other than initial glycogen content, 24 hour pH and rate of glycolysis were important in determining water-binding capacity.

Short-term excitement and exercise immediately prior to slaughter resulted in muscle with inferior water-binding properties. Long and short-term ante-mortem sucrose feeding produced elevated initial glycogen levels and resulted in muscles that were slightly soft and pale (Sayre et al., 1963a). These authors further concluded that total phosphorylase activity was not affected by pre-slaughter treatment and did not appear to be associated with rate of post-mortem glycolysis or with ultimate muscle characteristics.

Meyer et al. (1962) studied 20 Poland China and Chester White pigs and concluded that glucose tolerance was not an absolute indicator of post-mortem changes in muscle characteristics, but that there was a definite trend for those animals with a high glucose tolerance to have higher initial muscle glycogen levels and an ultimately inferior muscle quality.

Kastenschmidt et al. (1964) studied the effects of four ante-mortem environmental temperature treatments, which were as follows: 1) warm (42-45°C for 30-60 minutes); 2) cold (1-3°C for 30 minutes); 3) warm followed by cold treatment; and 4) cold followed by warm treatment. The warm treatment alone was reported to induce the development of extremely pale, soft and exudative muscle. The authors concluded that warm treatment followed by cold treatment resulted in dark, dry firm muscle. The other two treatments resulted in less marked changes.

Borchert and Briskey (1964) removed the wholesale ham immediately post-mortem and immersed it in liquid nitrogen (-195°C) for various periods of time. It was concluded that immersion in liquid nitrogen with subsequent equilibration and thawing at either -18°C or 4°C prevented the development of pale, soft, exudative muscle.

Effect of Electrolyte Content, Fat and Protein. Swift and Berman (1959) reported statistically significant correlations between water retention and pH, fat content and ratio of moisture to protein. A highly significant correlation was found between water retention and zinc content, in contrast to an inverse relationship found between water retention and either calcium or magnesium content. This information indicated that zinc differs in an important aspect from the two other ions. The possibility that zinc may participate in determining pH as a component of an enzyme system was pointed out.

Sodium chloride will increase water holding capacity by the influence of chloride ions rather than sodium ions (Hamm, 1959). Salt cross-linkages between peptide chains may be split off by binding of chloride ions and there is an increase of meat hydration of both the net charge and stereo effect (Hamm, 1959).

Sherman (1961) and Hamm (1959) indicate that polyphosphates and citrates influence water retention due to their relatively high ionic strength and to their influence on muscle pH. These salts function primarily to form strong complex compounds with alkaline earth metals. They eliminate bivalent cations in meat, especially magnesium ions.

EXPERIMENTAL METHODS

This study was divided into three separate investigations. Part I involved the influence of thiouracil and Tapazole administration upon adrenal and thyroid gland size, plasma 17 OHCS levels, and certain chemical and physical properties of the l. dorsi muscle. Part II includes the determination of plasma sodium and potassium content, 17 OHCS levels, adrenal weights, and pH, protein extractability, sodium and potassium content of the l. dorsi muscle from normal pigs and those exhibiting slight and severe PSE muscle. Part III concerned the effect of exogenous hormone like adrenal steroids upon plasma 17 OHCS, sodium and potassium content, and several porcine muscle characteristics.

Experimental Design and Pre-slaughter Treatment

Part I. Part I consisted of two separate experiments. In experiment 1, twelve Hampshire pigs were randomly assigned to two lots with three barrows and three gilts per lot. A normal finishing ration was fed to the pigs in Lot I (controls); those in Lot II were fed the finishing ration plus 35 gm Tapazole[1] per 100 pounds of feed from 160 to 210 lbs (slaughter weight).

Thirty-five crossbred (Hampshire x Yorkshire) barrows and gilts were randomly divided into five lots for experiment 2. The pigs in Lot I (controls) were fed a normal finishing ration. Each of the pigs in the other four lots received the same finishing rations plus the following goitrogenic compounds:

[1] 1-methyl-2-mercaptoimidazole.

Lot II - 0.5 gm Tapazole daily for 21 days

Lot III - 1.0 gm thiouracil daily for 21 days

Lot IV - 0.5 gm Tapazole daily for 10 days

Lot V - 1.0 gm thiouracil daily for 10 days

The animals ranged in weight from 190 to 210 lbs at slaughter.

Part II. Sixty market weight barrows and gilts representing four breeds (Yorkshire, Poland China, Hampshire and Landrace) were included in this phase of the study. The pigs were obtained from the Michigan Swine Improvement Station (East Lansing) and several Michigan swine producers. Slaughter weight ranged from 200 to 230 pounds.

Part III. Three experiments were included in this phase. In experiment 1, fifteen Hampshire barrows ranging from 208 to 219 pounds live weight were randomly divided among three lots. The pigs in Lot 1 served as controls, while those in Lots II and III were injected intramuscularly daily with 100 and 200 mg of prednisolone (delta-1-hydrocortisone), respectively, for seven days.

Experiment 2 included ten Hampshire and five Yorkshire pigs weighing 190 to 208 pounds. The pigs in Lot I served as controls; those in Lot II were injected intramuscularly with 100 mg of prednisolone daily for 10 days. Each pig in Lot III was fed 225 mg methyl prednisolone (6-methyl-delta-1-hydrocortisone) daily for 21 days. The methyl prednisolone was incorporated in 2 pounds of the finishing ration. Water was provided at libitum at all times while feed was provided at libitum each day but was removed at night to insure consumption

of the 2 pounds of feed containing methyl prednisolone each morning. The pigs in Lot II were slaughtered 24 hours after the last prednisolone injection; those in Lot III were slaughtered 5 days after the last hormone feeding.

Nine Yorkshire and nine Hampshire barrows and gilts ranging in weight from 180 to 215 lbs were randomly divided into three lots with 6 animals per lot in experiment 3. The pigs in Lot I served as controls while those in Lots II and III each received 450 mg methyl prednisolone daily for 10 and 25 days, respectively. The methyl prednisolone was fed with 2 lbs of finishing ration as previously described in experiment 2. These pigs were slaughtered 3 days after the last methyl prednisolone feeding. All pigs included in this study were held off feed approximately 12 hours prior to being slaughtered in the Meat Laboratory. However, water was provided ad libitum to all pigs.

Slaughter, Cutting and Sampling Procedure

The pigs were electrically stunned, bled and slaughtered in accordance with conventional procedures. Blood samples (250 ml) were collected in a glass centrifuge tube containing ammonium heparin immediately after sticking. The blood was immediately centrifuged for 40 minutes at 2600 rpm in a refrigerated centrifuge (4°C). The plasma was decanted into freezing jars, sealed, and stored at -30°C until assayed. All glassware was washed with sulfuric acid and rinsed six times with deionized water.

Samples of the l. dorsi muscle from the 5th or 6th lumbar region were excised from the left side of the uneviscerated carcass of pigs in

Lots I and II of experiment 2, in part III of the study. The samples were obtained approximately 40 minutes post-mortem for the initial pH determination. The carcasses were then eviscerated, split and placed in 4°C coolers. Samples for pH determination were removed from the l. dorsi muscle each half hour for 4 hours post-mortem. Ultimate pH determination was recorded 24 hours post-mortem. The carcasses from the other experiments were dressed in the usual manner and chilled for 24 hours at approximately 4°C.

Adrenal and Thyroid Weights

The adrenal and thyroid glands were removed from the carcass during evisceration, immediately trimmed of adhering tissues and weighed to the nearest 0.01 gm on the Mettler balance.

Carcass Measurements

The cutting procedure and carcass measurements obtained in part 1 were essentially as described by the Pork Evaluation Committee at the 1952 Reciprocal Meat Conference. No cutout data were obtained from pigs in the other phases of this study except loin eye area was obtained from pigs in part II.

Panel Evaluation of Muscle Color and Firmness

Muscle color and firmness characteristics of the right loin and ham from pigs in part I were subjectively evaluated 24 hours post-mortem by a five member panel. The panel visually rated each loin on a five point scale as follows: very dark (5), slightly dark (4), grayish pink (3),

slightly pale (2), or very pale (1). The panel scored the light ham muscles either grayish pink (3), slightly pale (2) or very pale (1) and the dark muscles as very dark (5), slightly dark (4) or grayish pink (3). Firmness scores for both ham and loin muscles consisted of firm (3), slightly soft (2) and very soft (1).

Muscle Sample Preparation

The section of the right l. dorsi muscle between the 10th and last thoracic vertebrae was excised from each carcass and trimmed of adhering tissue for protein extraction. The remaining sections of the l. dorsi muscle were later excised, trimmed of adhering tissues and ground five times through a 2 mm plate. The ground sample was sealed in glass jars and frozen for subsequent sodium and potassium analyses.

Muscle Protein Extraction Procedure

Sample preparation. The muscle samples were ground once through a 2 mm plate in a prechilled grinder to minimize heat denaturation. Samples were ground into a beaker and immediately covered with parafilm to prevent evaporation.

Protein extraction. Protein solubility of the l. dorsi muscle was determined by a modified method of the procedure described by Helander (1957) and Lawrie (1961). Ten grams of muscle were homogenized for 1 minute with 30 ml cold 0.03 M potassium phosphate buffer at pH 7.4. An additional 70 ml of 0.03 M buffer was added and the mixture was gently stirred for 30 minutes at 4°C. It was then centrifuged at 2600 rpm for

20 minutes (4°C). The supernatant was retained. The centrifugate was resuspended in 100 ml cold 0.03 M buffer solution, stirred and centrifuged twice more as described above. Total soluble nitrogen designated fraction (A) was determined on the combined supernatants and soluble non-protein nitrogen (NPN) designated fraction (C) from the supernatants after precipitation of the protein by 20% trichloroacetic acid. The difference (A-C) represented sarcoplasmic protein nitrogen.

The residues from extraction with 0.03 M buffer were suspended in 100 ml of a cold (4°C) mixture of KI (1.1 M) and potassium phosphate buffer (0.1 M) at pH 7.4. The mixture was stirred gently for one hour at 4°C and then centrifuged at 2600 rpm for 20 minutes (4°C). Extraction with the KI-phosphate buffer and subsequent centrifugation was repeated two additional times. The combined supernatants from the KI buffer extraction were assayed for total myofibrillar nitrogen. Nitrogen determinations of each fraction were made by the micro Kjeldahl procedure as outlined by A.O.A.C. (1960). Nitrogen values were expressed as mg of protein per gram of fresh tissue assuming a nitrogen content of 16.7% (Bailey, 1937).

Sodium and Potassium Analysis

Muscle sodium and potassium analysis. Sodium and potassium content of the *l. dorsi* muscle was determined by flame photometry utilizing the TCA extraction procedure of Mounib and Evans (1957) as modified by Kirton and Pearson (1963). A Beckman DU spectrophotometer with a model 9220 flame attachment was used for the analyses. The potassium and sodium

content was calculated from a standard curve determined by plotting the percent transmittance against the parts per million (ppm) of these electrolytes in the standard solution. The standards contained 3, 9, 15, 22.5, and 30 ppm potassium and 0.6, 1.8, 4.5, and 6.0 ppm sodium in a 2% TCA solution. These standards were run concurrently with each group of muscle samples analyzed. Sodium was read at 589 mu and potassium at 768 mu.

Plasma sodium and potassium analysis. Plasma sodium and potassium determinations were made by diluting 0.1 ml of plasma to 10 ml with deionized water containing 0.02% nonionic tergitol. Both sodium and potassium analyses were made from the same dilution of plasma. The standard solution contained sodium and potassium in a ratio of 10:1. The standards contained 5.0, 15.0, 25.0, 37.5, and 50.0 ppm sodium and 0.5, 1.5, 2.5, 3.75, and 5.0 ppm potassium. Other steps in the analysis were the same as those described for muscle sodium and potassium.

17-Hydroxycorticosteroid Assay of Porcine Plasma

Extraction of 17-hydroxycorticosteroids from porcine plasma. The 17-hydroxycorticosteroids (17OHCS) in porcine plasma were determined by a modified procedure of Peterson et al. (1957). Ten ml of plasma was carefully added to 50 ml of spectral grade methylene chloride (Merk and Co.) in a 500 ml separatory funnel. Extraction was carried out by gentle rotation for 10 minutes. The methylene chloride plus extracted hormones were transferred to glass stoppered centrifuge tubes. The sample was washed with 4 ml of cold (4°C) 0.1 N sodium hydroxide by vigorous shaking for 15 to 20 seconds. The alkali layer is then removed by aspiration.

Two 10 ml methylene chloride-hormone aliquots (for unknown and blank) were transferred to separate 40 ml ground glass-stoppered conical test tubes. To the unknown tubes 0.4 ml of phenylhydrazine-sulfuric acid-ethyl alcohol reagent was added to the methylene chloride extract; for blanks, 0.4 ml of blank reagent (Peterson's method) was added. The tubes were stoppered, shaken vigorously for 15 to 20 seconds and allowed to stand for 30 minutes. The supernatant methylene chloride phase was removed by aspiration and the phenylhydrazine-sulfuric acid-ethyl alcohol reagent was allowed to stand at room temperature for 10 hours for maximum color development.

Spectrophotometric analysis for 17-OHCS. The contents of the glass-stoppered tubes were transferred to micro cuvettes (Beckman 3.5 x 12.8 x 46.6). Absorbance of the colored products wasmeasured against the acid-alcohol blank at 410 mu in a Beckman DU Spectrophotometer. Ten ml of deionized water was run through the entire procedure to serve as reagent blank and 10 ml of water containing 8 γ cortisone acetate (United State Pharmacopeia) served as the standard.

The ethyl alcohol used in the study was 200 proof, USP, Rossville Gold Shield (Commercial Solvents Corp., Terre Haute, Indiana). It was found that this alcohol gave better results than that obtained by the method of Peterson et al. (1957). All glassware was scrupulously cleaned with soap, water and concentrated sulfuric acid and then rinsed six times with deionized water.

Muscle pH

A muscle sample weighing approximately 2.5 gm was homogenized for 1 minute in a Waring blendor containing 25 ml 0.005 M sodium iodoacetate for the pH determinations recorded every half hour for the first four hours post-mortem. Deionized water was used for the ultimate pH reading. Duplicate pH measurements were made with a Beckman, Model G pH meter or a Corning Model 12 pH meter.

Statistical Analysis

Analysis of variance was determined on the data from parts I, II and III. If a significant variance ratio was calculated between lots for a specific characteristic, Duncans Multiple Range Test was calculated and applied to the lot means. Simple correlation coefficients were determined on some data in part II (Steel and Torrie, 1960).

RESULTS AND DISCUSSION

There were no significant sex or breed differences between treatments or the chemical and physical characteristics studied in any of the six experiments. Thus, breeds and sexes within each treatment were combined for statistical analyses for the subsequent results and discussion.

Part I. The Influence of Hypothyroid Function Upon Pale, Soft, Exudative Porcine Muscle

Effect of goitrogenic drugs upon porcine muscle properties. Two goitrogens, thiouracil and Tapazole, were fed to pigs to produce hypothyroidism in order to study its affects upon porcine muscle characteristics. In experiment 1, Hampshire pigs were fed 35 gm of Tapazole per 100 lbs of feed from 160 lbs to slaughter weight. Thirty-five gm of Tapazole were selected for experiment 1 since Romach et al. (1962) reported 33.75 gm of Tapazole per 100 lbs ration completely blocked I^{131} uptake by the thyroid gland of pigs.

The results of this study are shown in Table 1. The ham and l. dorsi muscles from the five pigs fed Tapazole were firm and possessed normal color. They were similar to the controls for these characteristics.

Extractability of sarcoplasmic and myofibrillar protein fractions of the l. dorsi muscle was not significantly different for Tapazole treated and control pigs. Several workers (Bendall and Wismer-Pedersen, 1952; McLaughlin, 1963; McLaughlin and Goldspink, 1963b; Sayre and Briskey, 1963) have shown that porcine muscle which exhibits the PSE appearance 24 hours post-mortem possesses poor protein extractability in either high or low

ionic strength buffers. Extractability of the myofibrillar protein fraction was slightly higher for Tapazole treated pigs than controls. In addition, ultimate pH values between control and treated pigs were similar.

Table 1. Means[1] for sarcoplasmic and myofibrillar protein fractions, pH and muscle color and firmness scores of pigs fed Tapazole

	1. dorsi muscle characteristics				
Lot	Soluble sarcoplasmic protein[2]	Soluble myofibrillar protein[2]	pH	Color score[3]	Firmness score[4]
I Control	48.8	75.3	5.47	2.7	2.7
II Tapazole	45.2	84.2	5.54	3.0	3.0

[1]All means are statistically non-significant.
[2]mg per gm of fresh muscle.
[3]A five point scale was used with 1 indicating the lightest appearing muscle.
[4]A three point scale was used with 3 indicating the highest degree of firmness.

It is interesting to note that three of the five pigs fed Tapazole showed secondary effects such as loss of appetite, interruption of growth and edema. Similar secondary effects were observed by Ludvigsen (1953) among pigs fed thiouracil prior to slaughter. Briskey (1963) reported that Poland China pigs fed thiouracil produced soft, watery ham muscles which is in contrast to the effects of the goitrogen, Tapazole, fed in this study.

After observing the results of experiment 1, it was decided to compare the influence of Tapazole and thiouracil upon some physical and chemical properties of porcine muscle. In addition, the relationship

between hypofunction of the thyroid and adrenal cortex was studied since pork muscle quality as reported by Forrest et al. (1963) and Judge et al. (1959) is affected by seasonal temperature variation. These studies indicate that PSE muscle structure occurs most frequently among pigs slaughtered during seasons when temperature fluctuations are pronounced or those reared when environmental temperatures are high. These conditions no doubt provide a stress upon the pig.

The thiouracil level selected for the second experiment was the same as that used by Ludvigsen (1953) to produce PSE porcine muscle. Since Tapazole is considerably more potent than thiouracil in its goitrogenic activity (Premachandra et al., 1960), 0.5 gm of Tapazole per day was compared with 1 gm thiouracil per day in this study.

Protein extractability. There were no significant differences in extractability of the sarcoplasmic or myofibrillar protein fractions and non-protein nitrogen fraction of the l. dorsi muscle from treated or control pigs, Table 2. Results were similar to those reported for experiment 1. The l. dorsi muscle from pigs in Lot V (1 gm of thiouracil for 10 da) and Lot IV (0.5 gm of Tapazole for 10 da) yielded slightly higher quantities of extractable sarcoplasmic and myofibrillar protein fractions than controls. Values for extractable sarcoplasmic and myofibrillar protein fractions of the l. dorsi muscle from pigs in Lot I (controls), II (0.5 gm of Tapazole for 21 da) and III (1 gm thiouracil for 21 da) were nearly identical. No significant differences for pH or NPN values of the l. dorsi muscle were obtained between any of the five lots. From these results, it appears that neither thiouracil nor Tapazole affects the muscle properties characteristic of the PSE condition in the l. dorsi muscle of the pig.

Table 2. Means[1] and standard deviations of muscle protein fractions, NPN, pH, ham and loin muscle firmness and color scores, and thyroid and adrenal gland weights of pigs fed thiouracil and Tapazole

Chemical or physical characteristics	Lot I control		Lot II Tapazole 21 da.		Lot III thiouracil 21 da.		Lot IV Tapazole 10 da.		Lot V thiouracil 10 da.	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Myofibrillar prot. mg/gm	98.0	14.0	98.0	8.0	97.0	13.0	107.0	5.0	112.0	8.0
Sarcoplasmic prot. mg/gm	53.0	6.0	49.0	6.0	53.0	6.0	54.0	3.0	57.0	3.0
NPN mg/gm	4.0	0.4	4.7	0.3	4.6	0.2	4.7	0.5	4.7	0.1
pH, *l. dorsi*	5.7	0.1	5.7	0.2	5.7	0.1	5.9	0.1	5.8	0.4
L. ham muscle score[2]	2.3	0.5	2.7	0.5	2.1	0.7	2.6	0.6	1.9	0.6
D. ham muscle score[2]	3.4	0.3	3.1	0.1	3.4	0.3	3.6	0.2	3.6	0.4
Loin muscle score[2]	2.6	0.5	3.0	0.4	3.0	0.2	2.9	0.3	3.1	0.5
Ham firmness score[3]	2.1[a,b]	0.6	2.7[a]	0.5	1.7[b]	0.5	2.2[a,b]	0.3	2.1[a,b]	0.3
Loin firmness score[3]	2.4	0.4	2.9	0.1	2.5	0.5	2.8	0.2	2.5	0.3
Thyroid wt., gm	5.8[a]	1.0	9.3[b]	1.0	9.1[b]	2.0	6.4[a,b]	1.0	7.0[a,b]	1.0
Av. adrenal wt., gm	1.98	0.3	1.93	0.2	1.60	0.2	1.84	0.2	1.68	0.1

[1]Means for each characteristic with the same superscript are not significantly different from each other. Means with no superscripts indicate non-significance.
[2]A five point scale was used with 1 indicating the lightest appearing muscle.
[3]A three point scale was used with 3 indicating the highest degree of firmness.

The influence of these two goitrogens upon the ham muscles differed from those of the l. dorsi. As expected from the results of protein extractability data, visual color and firmness scores (panel of 5 judges) of the l. dorsi muscle from pigs in the five lots were not significantly different from each other. However, the light ham muscles from pigs in Lot III and V which were fed thiouracil were lighter in color than controls (Lot I) or the Tapazole fed pigs (Lots II and IV). Furthermore, ham muscle firmness scores for pigs fed 1 gm of thiouracil for 21 days were lower than controls and significantly ($P < .05$) lower than those fed 0.5 gm of Tapazole for 21 days. These differences in muscle firmness were not observed when the two goitrogens were fed for 10 days. The dark ham muscle scores were not significantly different between treatments.

No significant difference in thyroid weight was obtained between control and treated pigs when these two goitrogens were fed for 10 days; however, a highly significant ($P < .01$) increase in thyroid weight was obtained when they were fed for 21 days. The average weight of the thyroid gland from pigs receiving Tapazole and thiouracil for 21 days was 9.3 and 9.1 gm, respectively, as compared to 5.8 gm for controls. Acevedo et al. (1948) reported that continued administration of thiouracil caused hypertrophy of the thyroid gland.

Weights of the thyroid gland indicate that both Tapazole and thiouracil produced approximately the same degree of hypertrophy of the thyroid; however, slight evidence of PSE musculature was observed in some ham muscles, especially the gluteus medius, from thiouracil fed pigs but not from those fed Tapazole.

The evidence of PSE appearing musculature in hams from pigs fed thiouracil is in agreement with the findings of Briskey (1963). However, it should be pointed out that only some ham muscles seemed to be affected and no PSE characteristics were observed in the l. dorsi muscle by feeding thiouracil. Terrill et al. (1948, 1950) reported that feeding thiouracil to pigs did not significantly alter the physical or chemical composition of the carcass.

Thyroid-Adrenal Relationship

Feeding thiouracil for either 10 or 21 days decreased adrenal gland weight. Essentially no differences were observed when Tapazole was fed. The average adrenal gland weight from pigs in the control lot was 1.93 gm. Adrenal weights from pigs fed 1 gm of thiouracil daily for 21 days averaged 1.60 gm and those receiving the drug for 10 days averaged 1.68 gm. These values were not significantly different from controls; however, they approached significance ($P < .05$). Adrenal weights from pigs fed 0.5 gm of Tapazole for 10 and 21 days averaged 1.84 and 1.93 gm, respectively, which were similar to the controls. Considerable variation in adrenal gland weight was observed among pigs fed thiouracil. It appears this drug had a greater affect upon some individuals than others within the same lot.

The influence of thiouracil upon pig adrenal weights is in agreement with the adrenal cortex atrophy noted by Leblond and Hoff (1944), Baumann and Marine (1945) and Lazo-Wasem (1960) for thiouracil fed rats. Lazo-Wasem (1960) also reported that 0.3% thiouracil for three weeks brought about thyroid and pituitary gland hypertrophy as compared to non-thiouracil

treated rats. He reported the pituitary ACTH content of thiouracil fed rats was less than one third that of controls. These data support the hypothesis that adrenal atrophy following thiouracil administration is caused by lowered ACTH titers.

McCarthy et al. (1959) studied the influence of several goitrogens upon the adrenal cortex of the rat. These authors reported that both thiouracil and Tapazole when fed for 12 weeks produced atrophy of the adrenal gland. Adrenal atrophy resulting from Tapazole administration to rats as reported by McCarthy et al. (1959) was not observed among the pigs which received Tapazole for 3 weeks in this study.

Since Forrest et al. (1963) and Judge et al. (1959) reported seasonal influences upon PSE porcine muscle, it is interesting to note the work of Maqsood (1950). He reported that high environmental temperatures alone caused a significant decrease in rat adrenal gland weights which he indicated was probably due to the decrease in thyroid secretion rate occurring at these temperatures. Addis et al. (1965) found no significant differences in adrenal weights of the pig when subjected to temperatures ranging from 1.1 to 10.4°C. It appears from these data that high environmental temperature results in decreased adrenal gland weight but variation at lower environmental temperature (1.1 to 10.4°C) has little influence upon adrenal gland weight.

These data indicate that thiouracil and Tapazole have similar goitrogenic effects upon the thyroid gland of the pig but thiouracil appears to have a greater or more rapid appearing influence upon the adrenal gland than Tapazole. This difference may explain why some ham muscles from pigs

fed thiouracil were indicative of PSE musculature and these same ham muscles from Tapazole treated pigs were firm, dry and normal in color.

Carcass characteristics (Table 3) such as average fatback thickness, lean cuts, and loin eye area were quite similar between pigs in the five lots. Therefore, the degree of muscling or finish probably had little influence upon physical or chemical muscle characteristics determined in this study.

Sodium and potassium levels in the l. dorsi muscle were similar among all pigs in this study. These data indicate that if thiouracil has an effect upon the adrenal gland function, the mineralocorticoids are probably not the major hormones affected.

Because a rapid drop in pH occurs in muscle post-mortem which ultimately possesses PSE musculature (Wismer-Pedersen and Briskey, 1961a; Briskey and Wismer-Pedersen, 1961a; Goldspink and McLaughlin, 1964), the adrenal glucocorticoids might be involved via their gluconeogenic effects. Therefore, the free, 17-hydroxycorticosteroids (17 OHCS) in plasma were determined. Levels of these hormones from pigs fed the two goitrogens were lower than controls; however, the values were not significantly different.

It should be pointed out that it is difficult to collect blood samples from pigs without excitation. The degree and length of excitation was not completely controlled in this study. The procedure for collecting blood samples was quite uniform, but some pigs struggled more than others. The effect of excitation and struggling upon the secretion of 17 OHCS prior to collection of blood samples, therefore, may have affected these results in this study.

Table 3. Means[1] and standard deviations of fatback thickness, % lean cuts, loin eye area, muscle sodium and potassium and plasma 17 OHCS of pigs fed thiouracil and Tapazole

Chemical or physical characteristics	Lot I control		Lot II Tapazole 21 da.		Lot III thiouracil 21 da.		Lot IV Tapazole 10 da.		Lot V thiouracil 10 da.	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Fatback, in.	1.2	0.1	1.2	0.1	1.2	0.1	1.3	0.2	1.3	0.2
Lean cuts, %	53.0	1.0	54.0	2.0	52.0	4.0	51.0	2.0	54.0	1.0
Loin eye area, sq.in.	4.7	0.3	4.5	0.5	4.3	0.5	4.0	0.5	4.4	0.6
Na in muscle, ppm	374.0	25.0	364.0	24.0	369.0	34.0	371.0	23.0	342.0	34.0
K in muscle, ppm	4375.0	73.0	4354.0	137.0	4384.0	35.0	4351.0	110.0	4344.0	80.0
17 OHCS, γ/100 ml	19.0	4.0	16.0	5.0	16.0	1.0	13.0	3.0	14.0	3.0

[1]No significant differences between means in this table.

Part II. The Relationship of Plasma 17 OHCS Levels to Some Chemical and Physical Properties of Porcine Muscle

There is considerable evidence indicating that a rapid post-mortem pH drop concurrent with high temperature of the muscle (37°C or above) alters porcine sarcoplasmic and myofibrillar proteins to such a degree that diminished water binding and PSE musculature results (Ludvigsen, 1953; Wismer-Pedersen and Briskey, 1961b; Bendall and Wismer-Pedersen, 1962; Sayre and Briskey, 1963; Sayre et al., 1964; Goldspink and Mc Loughlin, 1964).

The effect of sudden ante-mortem environmental temperature change has shown that cold treatment (Sayre et al., 1961), heat treatment (Sayre et al., 1963) and combinations of the cold and heat treatments (Kastenschmidt et al., 1964) alter color and structure of porcine muscle. Since these treatments and environmental temperatures (Ludvigsen, 1953; Forrest et al., 1963; and Judge et al., 1959) provide stress conditions upon the pig, adrenal gland weights and plasma levels of free 17 OHCS were determined and their relationship to some physical and chemical properties of porcine muscle was studied.

Porcine muscles were subjectively divided into three groups based upon visual appearance of color, firmness and degree of exudation. Examples of the ham musculature representing each group are shown in plates I, II and III. Ham muscles, particularly the piriformis, gluteus medius and tensor faceae latae, shown in plate I are typical of the severe PSE group. Plates II and III show examples of slight PSE and normal musculature, respectively.

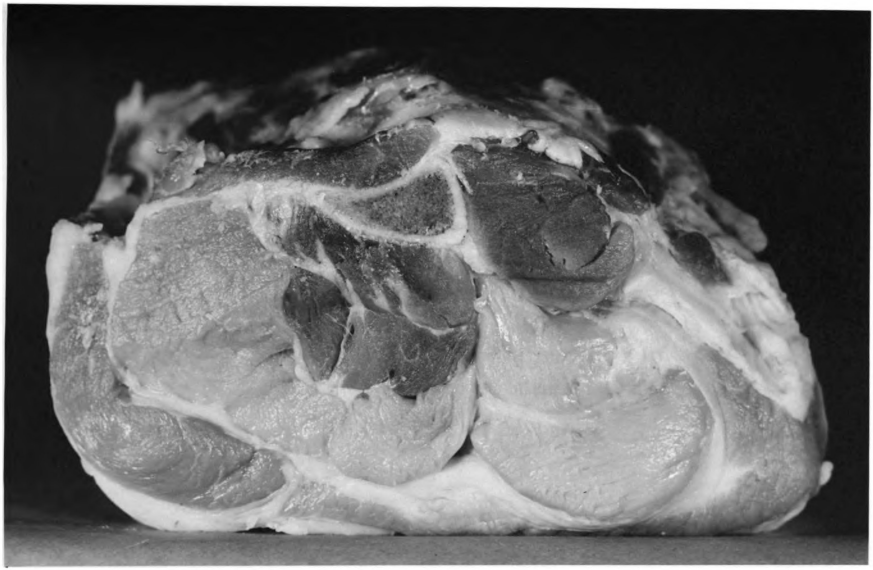


Plate I. Severe PSE.

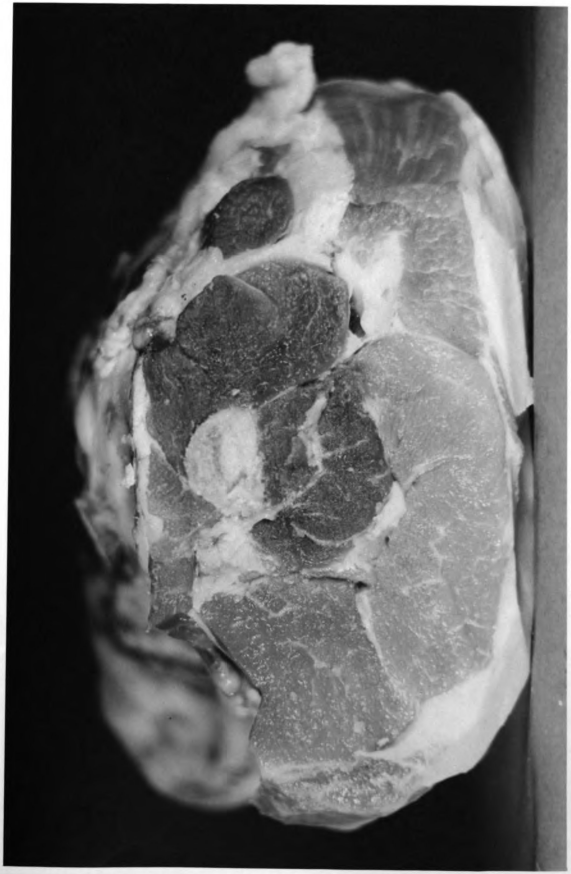


Plate II. Slight PSE.

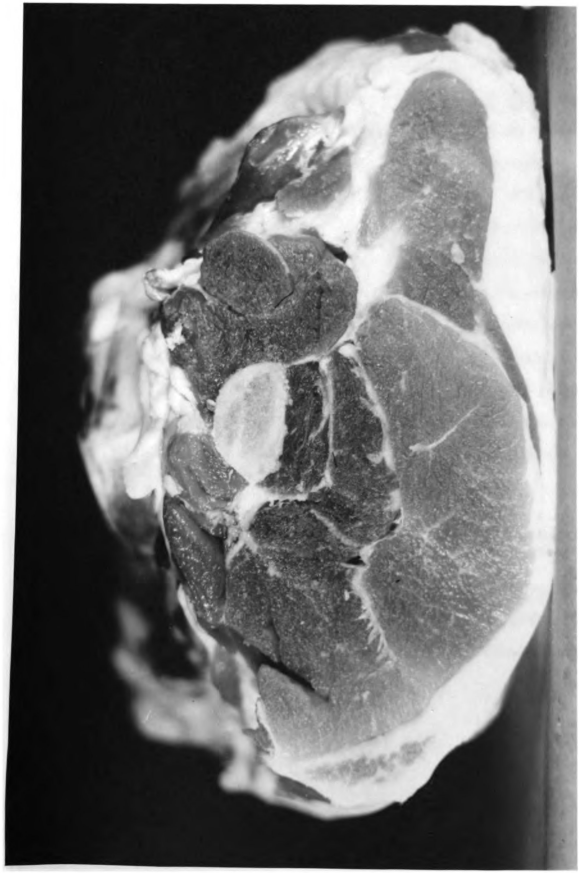


Plate III. Normal.

Protein extractability. Significantly ($P < .05$) lower quantities of sarcoplasmic and myofibrillar protein were extracted from l. dorsi muscles of the severe PSE group than the normal group (Table 4). These data are in agreement with the results of Bendall and Wismer-Pedersen (1962); Sayre and Briskey (1963) and Goldspink and McLoughlin (1964) who reported that PSE muscle possesses poor protein extractability in both high and low ionic strength buffers. However, the above authors did not report or discuss extractability characteristics of slight PSE muscle as studied in this experiment. Slight PSE muscle extractability did not differ significantly from the other two muscle groups for myofibrillar protein, but it had a significantly ($P < .05$) higher quantity of sarcoplasmic protein than the severe PSE muscle. Although the quantity of sarcoplasmic protein extracted from the slight PSE muscle was lower than that found in the normal muscle group, the difference was not significant.

Table 4. Means[1] and standard deviations of some chemical and physical characteristics obtained from normal pigs and those exhibiting slight or severe PSE musculature.

Chemical and physical characteristics	Severe PSE group		Slight PSE group		Normal group	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Sarcoplasmic prot., mg/gm	39.0[a]	6.0	50.0[b]	10.0	54.0[b]	10.0
Myofibrillar prot., mg/gm	48.0[a]	11.0	70.0[a,b]	20.0	95.0[b]	15.0
pH, l. dorsi	5.18[a]	0.10	5.35[b]	0.10	5.46[c]	0.11
NPN, mg/gm	3.2[a]	0.5	3.7[a]	0.7	4.3[b]	0.6
Plasma, 17 OHCS γ/100 ml	17.1	5.8	17.8	5.7	20.4	6.4
Adrenal weight, gm	1.98	0.32	2.04	0.45	2.07	0.32

[1]Means for each characteristic with the same superscript are not significantly different from each other. Means with no superscripts are not statistically significant.

Considerable variability in extractability of sarcoplasmic and myofibrillar protein fractions was found among pigs in the slight PSE group. Some l. dorsi muscles in this group had protein extractabilities as high as muscles in the normal group while others extracted similar to muscles classed severe PSE. This is indicated by the large standard deviation obtained for the sarcoplasmic and myofibrillar protein values in this group. These results indicate that sarcoplasmic and myofibrillar protein extractability is not entirely consistent with the three visual classifications of porcine muscle as normal, slight or severe PSE.

Bodwell (1964) reported that high temperature and low pH combination per se did not result in PSE muscle as expected from the results of Bendall and Wismer-Pedersen (1962), Sayre et al. (1963), and Goldspink and Mc Loughlin (1964). However, Bodwell (1964) did find that a high temperature-low pH combination resulted in decreased water holding capacity. Thus, the proteins responsible for hydration in porcine muscle were apparently altered by this treatment. Hamm (1959) reported myofibrillar proteins account for the major water binding sites of muscle and sarcoplasmic proteins have a much lower capacity for water binding.

It is interesting to note that several muscles which appeared firm and normal or dark in color possessed protein extractability values which were much lower than other muscles in this group. A few of these muscles had extractabilities of sarcoplasmic and myofibrillar protein fractions similar to those in the severe PSE group. While the severe PSE muscles possessed low protein extractability with both high and low ionic strength buffers the variability in protein extraction between muscles in this group was not as extreme as those of the other two groups.

McLoughlin and Goldspink (1963a) reported the pale color characteristic of PSE muscle may result from the masking of muscle pigment by precipitated protein (poor protein extractability). This conclusion is not in agreement with the finding of this study since low extractability values were observed even among the normal or dark colored muscles. These data indicate the difficulty encountered in objective and subjective classification of porcine muscle to facilitate study of the causative factors associated with PSE muscle.

McLoughlin and Goldspink (1963) reported the change in extractability of porcine muscle proteins occurred between 45 minutes and 24 hours postmortem. All carcasses appeared relatively normal 45 minutes after slaughter but changes in texture, color and water holding were manifest during subsequent cooling of the carcass. From these observations, it may be concluded that the alterations in PSE muscle proteins were not due to degeneration of the muscle ante-mortem, but were produced by the postmortem changes.

The processes responsible for these protein alterations are still unexplained. Bendall and Wismer-Pedersen (1962) reported that loss of protein solubility in high ionic strength buffers is due to adsorption of denatured sarcoplasmic proteins onto the myofibrils. Reduced extractability of the sarcoplasmic protein fraction in the present study was accompanied by reduced myofibrillar protein extractability. The denatured sarcoplasmic protein (not extractable) remaining in the centrifugate may possibly have adsorbed on the myofibrillar proteins reducing their extractability. It might also be postulated that reduced solubility of

myofibrillar proteins in PSE muscle is due to denaturation of these proteins by the same processes involved in denaturation of the sarcoplasmic proteins.

Ultimate pH. Low pH values of the l. dorsi muscle were obtained in severe PSE muscles 24 hours post-mortem. The pH reading was significantly different between all three groups. These pH values were 5.18, 5.35 and 5.46 for the severe PSE, slight PSE and normal muscle, respectively. In one instance, an ultimate pH value of 4.87 was found in a severe PSE l. dorsi muscle. Low ultimate pH values were also recorded in the l. dorsi muscle by Lawrie (1958) and McLoughlin and Goldspink (1963b). These workers found that low pH was associated with marked exudation. However, in the present study, considerable exudation was observed in some of the post-rigor muscle samples without the occurrence of abnormally low ultimate pH values. This supports the work of Wismer-Pedersen (1959), Wismer-Pedersen and Briskey (1961a) and Briskey and Wismer-Pedersen (1961a). These authors reported PSE muscle resulted when post-mortem pH fall is rapid with or without the development of abnormally low ultimate pH. Therefore, rate of post-mortem pH fall appears to be more important than ultimate pH in the development of PSE muscle.

Non-protein nitrogen. The NPN content of the l. dorsi muscle was significantly ($P < .05$) lower in the severe and slight PSE group than normal pigs. No significant difference was found between the mean NPN values for severe or slight PSE muscle. It should be pointed out that the buffer volume recovered after extraction varied between and within groups,

but was usually lowest for muscles from the severe PSE group. Thus the buffer solution is apparently bound in some manner to the non-extractable proteins of the homogenized muscle. These volume differences are used in calculating extractability values of the NPN fraction as well as the sarcoplasmic and myofibrillar protein fractions and may, therefore, account for some of the differences in NPN or protein fractions obtained for the three muscle groups.

McLoughlin (1963) reported higher NPN values for porcine muscle with an ultimate pH of 5.5 than those with an ultimate pH of 5.05. In the same study with a different group of pigs, the NPN-pH relationship was less well defined.

The physiological role of muscle NPN components is not completely understood. However, two dipeptides included in muscle NPN, carnosine and anserine, have been studied by Davey (1960). The distribution of carnosine and anserine varies from muscle to muscle. Red muscle (rich in myoglobin) containslittle or no carnosine and anserine; whereas, white muscles such as the l. dorsi contain a higher concentration of these dipeptides. The concentration of carnosine and anserine appears to be more closely related (inversely) to the respiratory activity of the muscle than the myoglobin concentration. The importance of the buffering capacity of these dipeptides in the physiological pH range of 6.5 to 7.5 has been assessed by Davey (1960). The dipeptides can contribute as much as 40% to the buffering capacity in this pH range in resting, living muscle. Therefore, the dipeptides are important in stabilizing pH which would fall rapidly during muscle excitation due to lactic acid accumulation.

From Davey's observations (1960) the lower NPN values found in the severe PSE muscle group may play a role in the rapid pH drop of the PSE muscle reported by Briskey and Wismer-Pedersen (1961a). Further work on the buffering capacity of muscle NPN components must be conducted before definite conclusions can be made.

Plasma 17 OHCS levels. Plasma levels of 17 OHCS of the severe PSE group were 3.3 γ/100 ml lower than the normal muscle group, but only 0.7 γ/100 ml lower than the slight PSE group (Table 4). These differences were not statistically significant, but the levels between the severe PSE and normal group were approaching significance ($P < .05$). Considerable variation (rather large standard deviation) was observed in these hormone levels, especially in the severe PSE group. Five of the twenty pigs in the severe PSE group had plasma 17 OHCS levels below 10.6 γ/100 ml. No such low values were observed in the normal group. However, some plasma 17 OHCS levels in the severe PSE group were as high or higher than the average value found in the normal group.

The high 17 OHCS values of some pigs in the severe PSE group and the large variation of these hormones between pigs within the other groups may be explained by the method used to collect the blood samples. As was pointed out in Part I, it is extremely difficult to collect a blood sample from pigs without stressing them.

Adrenal gland weight, plasma 17 OHCS levels and protein extractability data from carcasses with normal appearing muscle are presented in appendix IX. Most carcasses in this group which possessed lower than average myofibrillar and sarcoplasmic protein extractability also had lower than

average plasma 17 OHCS levels and some had smaller adrenal gland weights. This indicates a relationship exists between the adrenal glucocorticoids and protein extractability properties of post-mortem muscle.

From these data it might be observed that adrenal weight is somewhat indicative of plasma 17 OHCS levels. However, when the average adrenal weights for the three muscle groups were compared, the values appear quite similar. The average adrenal weights for pigs in the normal, slight and severe PSE groups were 2.07, 2.04 and 1.98 gm, respectively. It may be pointed out, however, that in all three groups, pigs with very small adrenal weights usually had lower than average levels of plasma 17 OHCS.

It appears from these data that lower levels of plasma glucocorticoids are probably associated with the ultimate physical and chemical properties of post-mortem skeletal muscle. This concurs with the work reported by Ludvigsen (1957) and Henry et al. (1958). They found that pigs with PSE muscle possessed lower levels of ACTH in the pituitary gland and concluded that a lower adrenal cortex output of glucocorticoids was involved in the PSE condition.

The mode of action of the glucocorticoids in the development of PSE muscle has not been elucidated. Since lactic acid accumulates rapidly in post-mortem PSE muscle, it seems feasible that the lower plasma 17 OHCS levels might influence this phenomenon by their effect on carbohydrate metabolism. Engle (1953) reported the 11-oxycorticosteroids inhibited the hexokinase catalyzed reaction. If 17 OHCS levels are low, the reaction converting glucose to glucose-6-PO_4 is accelerated. If this

occurred in PSE muscle, more pyruvate available for lactic acid production may result.

Ludvigsen (1957) suggested that adrenal glucocorticoids influence PSE muscle by their vasodilatory action. Furthermore, one of the major symptoms he observed in MD pigs was skeletal muscle vasoconstriction and decreased lactic acid in ear vein blood after exercise as compared to exercised controls. Forrest (1965) reported a significant increase in PCO_2 and decreased pH of blood collected anaerobically from pigs with PSE musculature indicating the circulatory system is altered.

Hydrocortisate has been shown to have striking influence on regulation of the vasomotor mechanism (Schayer, 1964). The results of these investigations indicate the adrenal cortex apparently plays a role in removing lactic acid from muscle after exercise or excitation. The lower glucocorticoid levels in the plasma of PSE pigs may play a role in the vasomotor response to accumulation of lactic acid in porcine muscle after excitation.

The role of adrenal glucocorticoids in amino acid metabolism and protein synthesis is of interest in discussing PSE porcine muscle. Wool and Weinsheldaum (1959) and Wool (1960) found that cortisone and cortisol participate in the mobilization of endogenous protein. Kaplan and Shimizu (1963) and Kostyo (1965) reported that cortisol and other glucocorticoids increased concentrations of virtually all amino acids and urea in skeletal muscle. These authors suggested the adrenal glucocorticoids cause an appreciable delay in the response of the muscle amino acid transport process.

Turner (1960) indicated that adrenalectomized animals not only have an increased protein synthesis but also have a diminished rate of protein catabolism. This accounts for the lowered nitrogen excretion in fasted adrenalectomized animals. The lower levels of plasma 17 OHCS and muscle NPN observed in the severe PSE group than in the normal group of pigs indicate the glucocorticoid involvement upon pig muscle NPN is similar to that found in the rat by Wool and Weinshelbaum (1959), Wool (1960), Kaplan and Shimizu (1963) and Kostyo (1965).

Sodium and potassium levels. Results for the l. dorsi muscle and plasma sodium and potassium levels are presented in Table 5. The severe PSE muscle group had a mean sodium and potassium content of 397 and 4217 ppm, respectively, as compared to 387 ppm sodium and 4360 ppm potassium for normal muscle. These differences were not significant and are in agreement with the work of Briskey et al. (1959) but disagree with the work of Henry et al. (1958). The latter author reported a large increase in sodium and a decrease in potassium in PSE muscle. It should be pointed out that they only used 12 pigs in their study.

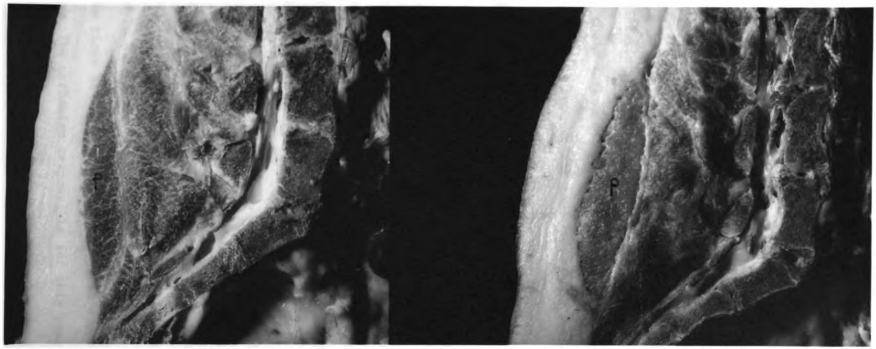
Table 5. Means[1] and standard deviations of muscle and plasma sodium and potassium levels and l. dorsi area of pigs possessing normal or slight and severe PSE muscle.

Chemical or physical characteristics	Severe PSE group		Slight PSE group		Normal group	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Muscle Na, ppm	397.0	59.0	395.0	40.0	387.0	50.0
Muscle K, ppm	4217.0	361.0	4208.0	351.0	4360.0	258.0
Plasma Na, mg %	325.0	32.0	363.0	32.0	347.0	32.0
Plasma K, mg %	25.0	3.0	27.0	4.0	27.0	4.0
l. dorsi area, sq. in.	4.6[b]	0.6	4.5[b]	0.5	3.9[a]	0.5

[1]Means for each characteristic with the same superscript are not significantly different from each other. Means with no superscripts are not

The average plasma potassium values for the three groups ranged from 25 ± 3 to 27 ± 4 mg % and from 325 ± 32 to 363 ± 32 for sodium. These values were not significantly different and are in the normal range reported by Widdowson and McCance (1956) and Bohstedt and Grummer (1954).

Carcass muscling. Area of the l. dorsi muscle, which is indicative of total muscling in the pork carcass, was significantly ($P < .01$) larger for the severe PSE group (4.64 sq. in.) than normal pigs (3.94 sq. in.). A highly significant correlation coefficient (-.43) was obtained between l. dorsi area and muscle firmness score for the pigs in this study. This indicates that the more muscular pigs have a greater predisposition to soft, exudative muscle than pigs with a lesser degree of muscling. Vitlo (1965) reported a similar relationship ($r = -.44$) between loin eye area and muscle firmness score. It may be that selection for the "meat-type" pig has resulted in unintentional selection for characteristics associated with development of PSE muscle.



Carcass with normal muscle

Carcass with PSE muscle

Plate IV. This plate was included to illustrate how the PSE musculature can be detected in the split carcass. Observation of the exposed muscles at the lumbosacral junction, particularly the piriformis (designated P in the photographs) usually indicates the presence of PSE musculature in the carcass. The piriformis muscle of the carcass on the left is normal in color, dry, firm and in close proximity with the subcutaneous fat. The piriformis muscle of the carcass on the right is pale in color, exudative, soft and has visibly separated from the subcutaneous fat.

Part III. The Influence of Exogenous Adrenocortical -like Steroids Upon the Adrenal Gland and Various Chemical and Physical Properties of Porcine Muscle

Three separate experiments are included in Part III; however, the objective of each was to block adrenal secretion of glucocorticoids by administering large doses of prednisolone or methyl prednisolone and study their influence upon chemical and physical properties of porcine muscle. Every attempt was made to prevent excitation of the pigs prior to slaughter. Results of the three experiments are shown in Tables 6, 7 and 8, and will be discussed together.

Adrenal gland atrophy. Atrophy was produced by administering either prednisolone (delta-1-hydrocortisone) or methyl prednisolone (6-methyl-delta-1-hydrocortisone). Both drugs significantly decreased weight (25 to 29%) of the adrenal gland, to approximately the same degree, even though levels ranged from 100 mg/day for seven days to 450 mg/day for 25 days.

Ingle et al. (1937, 1938), Boland and Headly (1949) and several other researchers working with laboratory animals showed that prolonged treatment with corticosteroids caused atrophy and hypofunction of the adrenal cortex. Christy et al. (1956) administered prednisolone and cortisone to humans for one to two weeks and found prednisolone to be four or more times as effective as cortisone in suppressing plasma 17 OHCS levels. The mechanism responsible for this reaction of corticosteroids is not completely understood. The view has generally been accepted that

corticosteroids exhibit pituitary ACTH production (Boland and Headly, 1949) and reduced adrenal function is due to absence of the ACTH stimulus. However, Péron et al. (1960) and Fekete and Görög (1963) suggested that corticosteroids have a direct adrenal inhibitory mechanism in addition to the regulatory mechanism mediated by the pituitary gland.

Plasma 17 OHCS levels. Plasma 17 OHCS were markedly reduced when adrenal atrophy was produced. In experiment 1, the plasma 17 OHCS levels from pigs injected with 100 or 200 mg prednisolone for 7 days were significantly decreased by 8.0 and 10.1 γ/100 ml, respectively. The blood sample was collected 24 hours after the last injection which indicates a rapid metabolic turnover of these hormones. In experiments 2 and 3, prednisolone and methyl prednisolone were administered in various concentrations and for longer periods of time than in the previous experiment. Injection of 100 mg of prednisolone for 10 days blocked the secretion (7.6 γ/100 ml lower than controls) of plasma 17 OHCS to approximately the same degree as the seven day injection. Blood samples were collected 24 hours after the last prednisolone injection in these two experiments. However, when 225 mg of methyl prednisolone were fed for 21 days and the plasma sample collected 5 days after the last hormone feeding, the plasma 17 OHCS were 2.7 γ/100 ml lower than controls. This indicates that the pig adrenal cortex apparently slowly regresses after prednisolone withdrawal. This is further supported by experiment 3 when 450 mg of methyl prednisolone was fed for 10 and 25 days and the blood sample collected three days after the last prednisolone feeding. As shown in Table 8,

plasma 17 OHCS levels were 5.3 and 5.0 γ/100 ml lower than controls for the 10 and 25 day treated pigs, respectively.

The normal level of free plasma 17 OHCS in the pig appears to be similar to those reported for humans (16 to 18 γ/100 ml) by Kruger et al. (1965). These values are considerably higher than those found in cattle (3 to 4 γ/100 ml) by Brush (1960) and Shaw et al. (1960) or in sheep (0.5 to 1.0 γ/100 ml) by Lindner (1959). Bush (1953) suggested that specie differences observed in adrenocortical secretion are genetically determined and at present cannot be related to any known differences in adrenocortical function. Further work is needed to more fully illucidate the so-called normal levels of 17 OHCS and other adrenal hormones in porcine blood.

Muscle proteins. The influence of prednisolone administration upon the extractability of muscle proteins was determined in all three experiments. Many physiological reactions of adrenocortical steroids have been thoroughly investigated but little is known about their effect upon muscle tissues per se. Widespread degeneration of skeletal muscle following administration of massive doses of cortisone to rabbits has been noted repeatedly (Ellis, 1956; Germuth et al., 1951).

The quantity of sarcoplasmic and myofibrillar proteins extracted from the prednisolone treated pigs in all three experiments was not significantly different from controls. This indicates that if degeneration occurred in porcine l. dorsi muscle from large doses of prednisolone, the degeneration is not due to decreased quantities of extractable protein.

The quantity of sarcoplasmic and myofibrillar proteins from pigs injected with 100 and 200 mg of prednisolone for 7 or 10 days in experiments 1 and 2 was slightly higher than controls. However, when either 225 mg or 450 mg of methyl prednisolone was fed daily for 10, 21 and 25 days, generally extractability of sarcoplasmic and myofibrillar proteins was slightly lower than controls. It should be pointed out that some pigs fed 225 to 450 mg of methyl prednisolone possessed l. dorsi muscles which were very exudative but darker in color than normal. In addition, some l. dorsi muscles from the treated pigs showed slight exudation while others appeared firm and dry 24 hours post-mortem.

Table 6. Means[1] and standard deviations of chemical and physical characteristics from control pigs and those injected with prednisolone for 7 days

Chemical or physical characteristic	Lot I control		Lot II 100 mg Pred./day		Lot III 200 mg Pred./day	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Av. adrenal wt., gm	1.96[a]	0.2	1.45[b]	0.3	1.49[b]	0.3
Plasma 17 OHCS γ/100 ml	21.8[a]	6.4	13.8[b]	6.3	11.7[b]	4.2
Sarcoplasmic prot., mg/gm	53.0	6.0	56.0	6.0	53.0	8.0
Myofibrillar prot., mg/gm	92.0	14.0	96.0	4.0	93.0	12.0
NPN, mg/gm	3.7[a]	0.2	4.3[a,b]	0.6	4.4[b]	0.4
pH, l. dorsi	5.62[a]	0.16	5.52[b]	0.07	5.47[b]	0.04
Na in plasma, mg %	358.0	22.0	342.0	34.0	337.0	34.0
K in plasma, mg %	27.7	0.8	30.4	4.0	32.4	4.0

[1]Means for each characteristic with the same superscript are not significantly different. Means with no superscript are not significant.

Table 7. Means[1] and standard deviations of chemical and physical characteristics from control pigs and those fed or injected with methyl Prednisolone

Chemical and physical characteristic	Lot I control		Lot II 100 mg Pred./day for 10 days[2]		Lot III 225 mg Methyl Pred./day for 21 days[3]	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Av. adrenal wt., gm	2.06[a]	0.2	1.52[b]	0.2	1.58[b]	0.3
Plasma 17 OHCS, γ/100 ml	13.4[a]	2.9	5.8[b]	0.86	10.7[a]	3.3
Sarcoplasmic prot. mg/gm	49.0	5.0	48.0	6.0	45.0	5.0
Myofibrillar prot. mg/gm	78.0	9.0	86.0	17.0	72.0	17.0
NPN, mg/gm	4.6	0.2	4.9	0.3	4.7	0.1
pH, l. dorsi	5.47	0.12	5.49	0.09	5.37	0.16
Na in plasma, mg %	365.0	19.0	360.0	26.0	351.0	15.0
K in plasma, mg %	31.2[a]	1.7	33.7[a]	5.0	24.0[b]	1.4
Na in l. dorsi, ppm	332.0[a]	16.0	369.0[b]	8.0	319.0[c]	10.0
K in l. dorsi, ppm	4326.0	184.0	4070.0	68.0	4103.0	61.0

[1]Means for each characteristic with the same superscript are not significantly different. Means with no superscript are not significant.
[2]Intramuscular injection.
[3]Incorporated with the feed.

Table 8. Means[1] and standard deviations of chemical and physical characteristics from control pigs and those fed methyl prednisolone for 10 and 25 days.

Chemical and physical characteristic	Control		450 mg Methyl Pred./day for 10 days		450 mg Methyl Pred./day for 25 days	
	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
Av. adrenal wt., gm	2.21[a]	0.2	1.59[b]	0.16	1.53[b]	0.2
Plasma 17 OHCS, γ/100 ml	12.2[a]	1.3	6.9[b]	3.1	7.2[b]	2.9
Sarcoplasmic prot., mg/gm	48.0	5.0	45.0	5.0	49.0	8.0
Myofibrillar prot., mg/gm	89.0	14.0	78.0	9.0	87.0	7.0
NPN, mg/gm	4.5	0.4	4.5	0.5	4.4	0.1
pH, l. dorsi	5.41	0.09	5.41	0.08	5.46	0.08
Na in plasma, mg %	346.0	16.0	344.0	8.0	344.0	9.0
K in plasma, mg %	27.4	4.2	30.1	2.0	28.3	4.6
Na in l. dorsi, ppm	323.0[a]	25.0	313.0[a,b]	40.0	288.0[b]	27.0
K in l. dorsi, ppm	4263.0	146.0	4121.0	241	4142.0	276.0

[1]Means for each characteristic with the same superscript are not significantly different. Means with no superscript are not significant.

Non-protein nitrogen. NPN values determined on the l. dorsi muscles from pigs injected with 100 and 200 mg of prednisolone for 7 days were higher than controls. The increase was significant ($P < .05$) for the 200 mg injected group. This is in agreement with the work of Kaplan and Shimizu (1963) who report that virtually all amino acids and urea in muscle of fasted and non-fasted rats were increased by administration of cortisol.

When high doses of methyl prednisolone were fed to pigs for 10 to 25 days and the muscle sample obtained 3 to 5 days after the last hormone feeding, the NPN values were similar to controls. Ryan (1963) also found a variable effect upon muscle amino acids in rats, depending on the length of administration of hydrocortisone. Twenty-four hours after injection of hydrocortisone, an increase in the free amino acids of plasma and muscle was found. These acids were slightly decreased after 10 days of treatment with hydrocortisone. Because the pigs in this phase of the study were slaughtered 3 to 5 days after the last hormone administration, it was not possible to determine if the changes in NPN values resulted from the high doses of methyl prednisolone or adrenal atrophy follows prolonged prednisolone feeding.

Muscle pH. Ultimate pH values of the 1. dorsi muscle of treated pigs were similar to those for controls. Only pH values of muscles from pigs injected with either 100 or 200 mg of prednisolone daily were significantly different from controls. Since rates of pH fall has been reported (Briskey and Wismer-Pedersen 1961a) to be associated with PSE muscle as discussed

in Part II and plasma glucocorticoid levels might be associated with PSE muscle, the effect of prednisolone administration upon post-mortem rate of pH fall was studied. The l. dorsi muscle from pigs injected with 100 mg of prednisolone for 10 days was compared with controls. The results are shown in Figure 1.

Initial pH was taken 40 minutes post-mortem and it is interesting to note that similar initial pH readings were found between control and treated pigs, but pH of the controls was slightly higher. One-half hour later, pH of the control group dropped considerably faster than prednisolone treated pigs, but after one hour both groups had nearly identical muscle pH (approx. 6.0) values. The major changes occurred between 1 and 2 1/2 hours after the initial pH reading. During this time, the pH of the control group dropped considerably faster than the prednisolone treated pigs. Between 2 1/2 and 4 hours, pH values of the control group remained relatively constant; whereas, pH of the prednisolone treated pigs continued to decrease, but very slightly. The ultimate pH values of the two groups were very similar (control 5.53 and prednisolone treated pigs 5.50). The l. dorsi muscle appeared firm and normal to dark in color in both groups.

It appears from these data that the affects of prednisolone upon skeletal muscle results in a retardation of the rate of post-mortem pH fall. It may be possible that prednisolone has an influence on the buffer systems of the muscle since higher levels of muscle NPN were obtained when prednisolone was administered. Davey (1960) demonstrated that the NPN components, carnosine and anserine, influence buffering capacity in muscle.

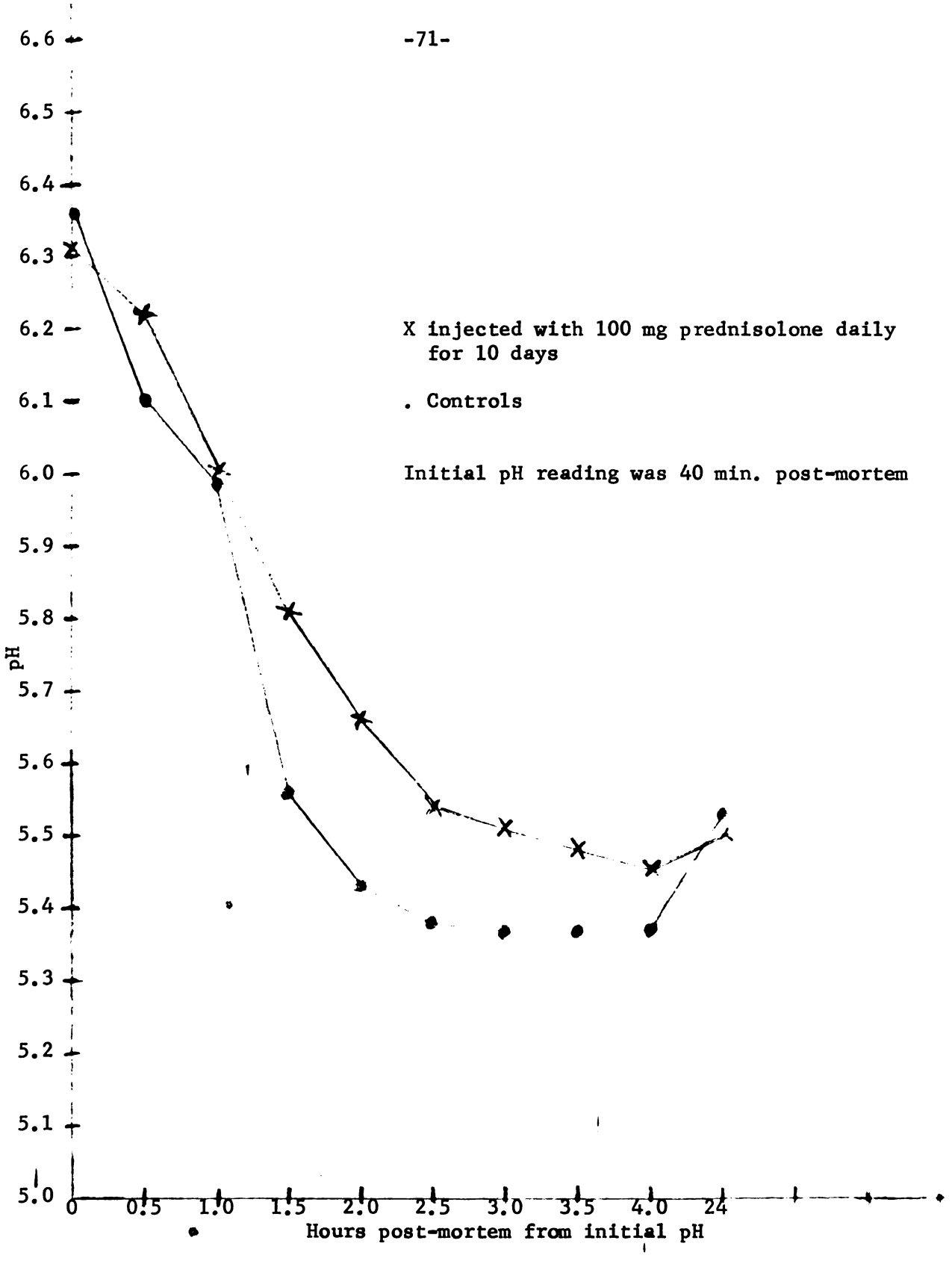


Figure 1. Post-mortem pH curve.

However, the buffering capacity of the NPN fraction in muscle needs further study before any definite conclusions can be made about its influence upon rate of post-mortem pH fall.

Plasma and muscle sodium and potassium content. Sodium and potassium levels were determined in the l. dorsi muscle and plasma in experiments 2 and 3 and on the plasma in experiment 1. When working with glucocorticoids, it is important to keep in mind their effect upon electrolyte balance is variable. The results are affected by dosage, time of administration, specie and possibly by other variables not yet elucidated (Ingle, 1950). Results with pigs in this study indicate that prednisolone affects muscle electrolytes differently than methyl prednisolone. When 100 mg of prednisolone were injected for 10 days, muscle sodium content increased significantly, but feeding 225 and 450 mg of methyl prednisolone daily for 21 and 25 days, respectively, significantly decreased the sodium content of muscle. When 450 mg of methyl prednisolone were fed for 10 days, muscle sodium decreased but not significantly. It should be mentioned, however, that the muscle samples for sodium analyses in the methyl prednisolone experiment were obtained 3 to 5 days after the last administration of the drug. Adrenal atrophy definitely resulted from this treatment and was manifest when the animals were slaughtered. Therefore, it is difficult to evaluate these findings since adrenal atrophy may also have influenced the electrolyte changes.

Lyster et al. (1957) found a slight sodium retention with prednisolone administration and sodium and water diuresis activity during methyl prednisolone treatment with rats.

Potassium content of the l. dorsi muscle was not significantly altered by prednisolone treatment. Plasma levels of sodium and potassium were within the normal range for all prednisolone and and methyl prednisolone treated pigs except the potassium level from those fed 225 mg of methyl prednisolone. Plasma potassium levels in these pigs were significantly lower than controls. No other significant differences were obtained. Faludi et al. (1964) also found that plasma electrolytes were within the normal range for dogs treated with prednisolone and methyl prednisolone. The marked changes in muscle sodium and the normal plasma sodium values in pigs injected with these hormones indicate that these hormones function to some degree at least at the muscle level. Swingle et al. (1958) indicated potent glucorticoids function in a homeostatic mechanism in salt and water balance of the body by enabling the animal to freely transfer fluid and electrolytes from one body compartment to another. It appears from the data in the present study that prednisolone and methyl prednisolone influence the sodium homeostatic mechanism in the pig by keeping plasma electrolytes near normal levels by mobilizing muscle sodium.

SUMMARY

The results of this study were obtained from three separate investigations with 148 pigs. In Part I, the influence of thiouracil and Tapazole feeding upon adrenal and thyroid weight, plasma 17-hydroxycorticosteroid (17 OHCS) levels, and some chemical and physical properties of the l. dorsi muscle was studied. Part II included the determination of plasma sodium, potassium, and 17 OHCS levels, adrenal weights and the extractability of muscle proteins, pH, and sodium and potassium content of the l. dorsi muscle from normal pigs and those exhibiting slight and severe PSE musculature. In Part III, the influence of exogenous prednisolone and methyl prednisolone treatment upon plasma 17 OHCS, sodium and potassium levels and several porcine muscle characteristics was observed.

Tapazole and thiouracil (Part I) both produced hypertrophy of the thyroid gland to approximately the same degree, but thiouracil had a more pronounced effect upon adrenal atrophy than Tapazole. L. dorsi muscle pH, myofibrillar and sarcoplasmic protein extractability, non-protein nitrogen and sodium and potassium levels were quite similar between goitrogen treated and control pigs. Thiouracil treatment, however, produced a pale, soft, exudative (PSE) condition in the ham muscles of some pigs; whereas, Tapazole treatment resulted in normal colored, firm appearing hams in all pigs. Plasma 17 OHCS levels were lower than controls for both Tapazole and thiouracil treated pigs, but these differences were not significant.

Significantly lower quantities of sarcoplasmic and myofibrillar protein were extracted from PSE musclature than normal muscle, Part II. Also,

normal muscle possessed significantly higher NPN values than the severe PSE group. Considerable variation in the quantity of extractable protein was found between muscle samples in the slight PSE group. Plasma 17 OHCS levels from animals possessing severe PSE muscle were 3.3 γ/100 ml lower than the normal group. This difference was approaching significance ($P < .05$). Muscle pH in the severe and slight PSE muscle was significantly lower than normal muscle. Also, a highly significant correlation coefficient (-.43) was obtained between *l. dorsi* muscle area and muscle firmness scores for the pigs included in this phase of the study.

Pigs fed or injected with either prednisolone or methyl prednisolone (Part III), showed adrenal atrophy and lower levels of plasma 17 OHCS at the levels of administration and durations studied in these experiments. Quantities of sarcoplasmic and myofibrillar proteins extracted from glucocorticoid treated pigs in all three experiments were not significantly different from controls. Daily prednisolone injection (200 mg/day) for seven days resulted in significantly higher muscle NPN values. Marked differences in rate of post-mortem muscle pH fall were obtained between prednisolone treated and control pigs. Also, prednisolone was found to have a sodium retaining effect upon muscle while methyl prednisolone resulted in muscle sodium dimunition. Neither drug significantly altered potassium level of the *l. dorsi* muscle.

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APPENDIX

Appendix I. Thyroid Study I

Tattoo	Av. adrenal weight	Soluble sarcoplasmic protein	Soluble myofibrillar protein	Soluble non-protein nitrogen	pH	Color score	Firmness score
			Lot I - Control animals				
17A	1.47	38.9	36.7	3.12	5.12	1	1
28A	1.73	51.9	92.1	3.93	5.49	3	3
35A	1.74	47.1	58.2	3.63	5.50	3	3
36A	1.80	49.8	76.1	3.72	5.65	3	3
15A	1.66	46.0	95.4	3.70	5.55	3	3
16A	1.66	58.0	93.5	3.34	5.55	3	3
			Lot II - Tapazole treated animals				
17E	1.73	44.5	80.7	3.56	5.42	3	3
18E	2.00	46.4	87.4	3.59	5.52	3	3
05E	1.49	47.0	80.4	4.31	5.54	3	3
07E	1.37	46.4	82.9	4.05	5.49	3	3
09E	1.36	48.3	97.3	4.49	5.80	3	3
08E	1.54	38.8	76.2	4.52	5.51	3	3

Appendix II. Thyroid Study II

Tattoo	Date	No.	Lot	Wt. right adr.	Wt. left adr.	Breed 1=York 2=Hamp	Fibrillar protein mg/g	Sarcoplasmic protein mg/g
Controls								
07	8-12-64	301	1	1.91	2.18	1	105.62	63.99
09	8-17-64	302	1	2.18	2.14	1	109.12	56.56
07	9- 1-64	303	1	1.61	1.49	1	101.81	48.43
05	9- 1-64	304	1	1.69	1.70	1	69.56	48.81
09	9- 1-64	305	1	1.41	1.47	2	95.81	47.98
06	9- 1-64	306	1	2.41	2.43	2	92.50	50.62
05E	8-12-64	307	1	2.20	2.29	2	112.69	56.75
Tapazole-3 weeks								
15	8-17-64	308	2	1.39	1.58	1	98.62	52.70
06E	8-17-64	309	2	1.85	1.97	1	109.12	62.07
15E	8-28-64	310	2	2.01	1.89	1	106.62	49.50
17E	9- 1-64	311	2	1.98	2.20	2	98.37	48.88
X28	8-29-64	312	2	2.21	2.17	2	100.25	48.81
05X	8-29-64	313	2	1.71	1.92	1	82.50	42.37
06X	8-29-64	314	2	1.96	2.23	1	96.75	45.25
Thiouracil-3 weeks								
07E	8-17-64	315	3	1.65	1.74	1	117.25	62.00
08E	8-17-64	316	3	1.52	1.31	1	96.81	53.69
05E	8-17-64	317	3	1.23	1.17	1	112.00	59.88
29E	8-25-64	318	3	1.57	1.59	1	96.69	48.19
08E	9- 1-64	319	3	1.71	2.17	2	85.50	47.89
15E	9- 1-64	320	3	1.91	1.85	2	80.06	44.69
19E	9- 1-64	321	3	1.46	1.64	2	96.81	59.25
Tapazole-10 days								
08E	8-28-64	322	4	1.42	1.52	1	99.62	50.56
07E	8-28-64	323	4	1.96	2.16	1	101.87	56.56
05E	8-28-64	324	4	1.88	1.66	2	114.43	58.07
18E	8-28-64	325	4	1.96	2.04	1	109.31	52.88
06E	8-28-64	326	4	1.72	1.84	2	105.50	51.50
17E	8-28-64	327	4	1.76	1.63	1	112.31	59.75
09E	8-28-64	328	4	2.01	2.24	1	108.18	52.37
Thiouracil-10 days								
26	8-25-64	329	5	1.45	1.52	1	111.12	54.63
16	8-25-64	330	5	1.46	1.63	1	112.25	53.75
17	8-25-64	331	5	1.72	1.86	1	114.18	55.56
19	8-25-64	332	5	1.60	1.66	1	122.25	65.06
18	8-25-64	333	5	1.72	1.92	1	116.06	56.37
27	8-25-64	334	5	1.86	1.87	2	95.43	58.44
25	8-25-64	335	5	1.69	1.70	2	114.00	59.00

Appendix II. Thyroid Study II (continued)

Tattoo	Date	NPN mg/g	Na in muscle ppm	K in muscle ppm	Sex 1=M 2=F	pH	17-OHCS α/100 ml	Wt. of thyroid gland
Controls								
07	8-12-64	4.83	345	4416	1	5.45	16.92	5.98
09	8-17-64	3.92	363	4336	1	5.60	24.86	4.95
07	9- 1-64	5.00	356	4407	2	5.67	18.00	5.42
05	9- 1-64	4.01	379	4502	2	5.68	16.90	9.33
09	9- 1-64	4.60	376	4342	1	5.77	26.86	4.92
06	9- 1-64	4.80	424	4270	1	5.92	13.76	5.30
05E	8-12-64	3.94	380	4356	2	5.60	17.57	4.98
Tapazole-3 weeks								
15	8-17-64	4.70	362	4301	1	5.60	15.92	11.94
06E	8-17-64	4.52	359	4312	1	5.55	17.90	8.92
15E	8-28-64	5.60	404	4402	2	5.80	10.90	8.05
17E	9- 1-64	4.70	392	4475	1	6.12	18.60	9.64
X28	8-29-64	4.36	354	4493	2	5.50	11.63	9.17
05X	8-29-64	4.64	345	4407	2	5.50	26.10	8.89
06X	8-29-64	4.70	336	4090	2	5.50	17.26	8.56
Thiouracil-3 weeks								
07E	8-17-64	4.45	330	4406	1	5.85	14.54	11.90
08E	8-17-64	4.23	346	4468	1	5.59	17.90	10.40
05E	8-17-64	4.74	327	4363	2	5.58	17.90	12.86
29E	8-25-64	4.74	394	4372	2	5.91	18.16	6.95
08E	9- 1-64	4.70	380	4306	2	5.68	16.00	5.78
15E	9- 1-64	4.90	406	4340	1	5.67	16.85	5.81
19E	9- 1-64	4.66	402	4435	1	5.93	17.62	10.14
Tapazole-10 days								
08E	8-28-64	4.72	349	4375	2	5.80	8.00	6.24
07E	8-28-64	3.62	397	4385	2	5.80	16.80	5.60
05E	8-28-64	4.91	346	4248	2	6.04	14.40	5.25
18E	8-28-64	4.82	401	4445	1	6.00	9.60	7.25
06E	8-28-64	4.79	359	4486	1	6.00	14.40	4.65
17E	8-28-64	5.63	358	4166	1	6.00	16.00	8.47
09E	8-28-64	4.60	388	4356	1	5.80	17.60	7.68
Thiouracil-10 days								
26	8-25-64	4.74	356	4449	2	6.19	16.90	6.38
16	8-25-64	4.74	318	4273	2	5.92	21.98	6.85
17	8-25-64	4.74	403	4458	1	4.74	12.36	4.22
19	8-25-64	4.65	293	4290	1	6.05	13.00	7.06
18	8-25-64	4.65	338	4368	1	5.62	10.40	7.29
27	8-25-64	4.70	338	4300	1	5.90	16.80	9.22
25	8-25-64	4.74	354	4276	1	6.08	12.36	8.26

Appendix II. Thyroid Study II (continued)

Tattoo	Date	Fat-back (in.)	Lean cuts	Loin eye (sq.in)	Ham firm-ness score	Ham light muscle score	Ham dark muscle score	Loin firm-ness score	Loin color score
Controls									
07	8-12-64	1.56	54.6	4.74	3.0	3.0	3.3	3.0	3.0
09	8-17-64	1.28	53.7	4.31	3.0	3.0	3.0	3.0	3.0
07	9- 1-64	1.16	55.4	4.40	1.5	1.8	3.8	2.3	2.5
05	9- 1-64	1.06	54.6	5.18	1.5	1.8	3.5	2.0	2.5
09	9- 1-64	1.23	52.2	5.04	1.8	2.0	3.0	2.0	1.5
06	9- 1-64	1.33	52.8	4.80	2.0	2.0	4.0	2.5	3.3
05E	8-12-64	1.21	53.0	4.73	2.5	2.5	3.5	2.5	3.0
Tapazole-3 weeks									
15	8-17-64	1.46	54.8	4.05	3.0	3.0	3.0	3.0	3.0
06E	8-17-64	1.43	53.6	3.91	3.0	3.0	3.0	3.0	3.0
15E	8-28-64	1.36	50.3	4.18	2.2	1.8	3.4	3.0	3.0
17E	9- 1-64	1.26	55.3	4.92	1.8	2.2	3.2	3.0	3.0
X28	8-29-64	1.10	56.1	5.50	3.0	3.0	3.3	3.0	4.0
05X	8-29-64	1.25	55.4	4.67	3.0	3.0	3.0	2.8	2.8
06X	8-29-64	1.03	57.8	4.70	3.0	3.0	3.0	2.8	2.8
Thiouracil-3 weeks									
07E	8-17-64	1.43	45.8	4.23	2.0	2.8	3.3	3.0	3.3
08E	8-17-64	1.36	56.7	3.72	2.2	3.0	3.0	3.0	3.0
05E	8-17-64	1.23	55.4	4.01	1.2	2.0	3.3	3.0	3.3
29E	8-25-64	0.96	54.3	4.98	1.2	1.2	3.2	1.8	2.8
08E	9- 1-64	1.23	55.4	4.68	1.3	1.0	3.8	2.5	3.3
15E	9- 1-64	1.26	54.2	4.90	1.5	2.0	3.8	1.8	3.0
19E	9- 1-64	1.43	48.7	3.80	2.5	2.8	3.8	2.8	2.8
Tapazole-10 days									
08E	8-28-64	1.13	49.7	3.81	2.4	2.8	3.4	3.0	2.8
07E	8-28-64	1.13	50.1	4.95	2.8	3.0	3.4	3.0	2.8
05E	8-28-64	1.20	53.5	4.16	2.0	3.0	3.6	2.4	3.4
18E	8-28-64	1.60	49.6	3.20	1.8	1.4	3.8	3.0	3.2
06E	8-28-64	1.03	54.2	3.95	2.2	3.0	3.4	3.0	3.2
17E	8-28-64	1.64	47.5	3.81	2.4	2.8	4.0	2.8	2.6
09E	8-28-64	1.56	54.2	4.24	2.4	2.2	3.6	2.4	2.6
Thiouracil-10 days									
26	8-25-64	1.30	56.1	4.45	2.2	2.0	3.4	2.6	3.2
16	8-25-64	1.30	55.3	4.04	2.6	2.0	3.4	2.2	3.4
17	8-25-64	1.13	55.7	4.21	1.6	1.0	3.0	2.8	2.6
19	8-25-64	1.60	53.6	4.65	2.0	1.0	3.2	2.8	2.8
18	8-25-64	1.43	54.9	5.45	2.0	2.8	4.0	2.6	3.4
27	8-25-64	1.66	50.5	3.37	2.4	2.4	4.0	2.8	2.6
25	8-25-64	1.10	55.6	4.95	2.0	2.2	4.2	2.0	4.0

Appendix III. Study II, Firm Group

Tattoo	Date	Wt. right adrenal	Wt. left adrenal	Breed[a]	Fibrillar protein mg/g	Sarcoplasmic protein mg/g	NPN mg/g	Na in muscle ppm	K in muscle ppm
07E	10-22-63	1.60	1.63	1	123.37	62.06	3.75	416	4348
17E	10-29-63	1.89	2.16	1	107.75	57.87	3.50	477	4319
08E	11- 5-63	2.21	2.46	1	111.06	60.25	4.41	448	4165
16E	11- 5-63	1.84	2.04	2	100.50	67.00	4.46	335	4354
25E	11-12-63	1.79	1.90	1	111.37	51.31	5.02	431	4579
26E	11-12-63	1.68	1.65	2	103.18	63.18	3.85	383	4346
#1	1-14-64	2.32	2.22	2	103.00	72.31	4.26	416	4128
#2	1-14-64	1.74	1.85	2	108.87	73.18	4.54	439	4388
18E	1 -21-64	1.74	1.83	1	97.68	51.06	4.73	325	4608
06E	2-17-64	2.53	2.35	3	79.18	46.93	3.05	393	4275
08E	2-17-64	2.57	2.90	3	78.12	51.37	3.81	391	4793
05E	2-17-64	1.80	2.04	3	89.87	41.87	4.58	405	4140
17E	2-18-64	2.53	2.24	3	70.81	35.00	4.82	404	4161
05E	2-25-64	1.69	1.99	4	115.81	40.81	5.35	335	4675
08E	2-25-64	2.17	2.41	4	80.93	43.31	5.43	382	4311
09E	2-25-64	2.43	2.43	4	108.18	53.43	4.40	343	4722
15E	2-26-64	1.80	2.07	4	78.62	67.06	4.10	305	4644
18E	2-26-64	1.92	1.94	4	81.75	48.93	4.99	289	4261
37E	4- 7-64	1.78	1.97	3	77.37	45.68	2.83	396	3681
09E	4-21-64	2.42	2.34	4	88.81	51.30	4.15	439	4304

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix III. Study II, Firm Group (continued)

Tattoo	Date	Sex[b]	pH	Free 17-OHCS γ/100 ml plasma	Na in plasma mg %	K in plasma mg %	Firmness	Loin eye area
07E	10-22-63	2	5.49	26.80	347	31.5	3	3.85
17E	10-29-63	2	5.60	15.84	305	26.8	3	
08E	11- 5-63	1	5.55	17.60	410	40.0	3	3.02
16E	11- 5-63	1	5.51	37.92	291	21.6	3	
25E	11-12-63	1	5.44	21.92	400	28.2	3	
26E	11-12-63	2	5.48	23.84	391	35.5	3	
#1	1-14-64	1	5.55	22.08	320	22.6	3	4.53
#2	1-14-64	1	5.49	28.96	315	29.5	3	5.25
18E	1-21-64	2	5.48	20.48	330	26.7	3	4.88
06E	2-17-64	1	5.29	12.88	355	26.7	3	3.68
08E	2-17-64	2	5.25	18.72	336	27.5	3	4.10
05E	2-17-64	1	5.30	22.08	345	27.9	3	3.74
17E	2-18-64	1	5.52	20.68	331	26.9	3	3.38
05E	2-25-64	1	5.49	13.60	370	21.7	3	3.92
08E	2-25-64	2	5.52	17.28	368	24.5	3	3.90
09E	2-25-64	1	5.56	20.64	320	24.4	3	3.52
15E	2-26-64	1	5.50	13.92	397	29.6	3	4.02
18E	2-26-64	2	5.49	10.08	348	28.0	3	4.02
37E	4- 7-64	2	5.21	17.12	349	30.8	3	4.26
09E	4-21-64	2	5.52	26.24	331	22.7	3	3.12

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4= Landrace
[b]Sex: 1 = male, 2 = female

Appendix IV. Study II, Slight PSE Group

Tattoo	Date	Wt. right adrenal	Wt. left adrenal	Breed[a]	Fibrillar protein mg/g	Sarcoplasmic protein mg/g	NPN mg/g	Na in muscle ppm	K in muscle ppm
06E	10-22-63	2.17	2.16	1	108.18	59.18	4.57	388	4647
09E	10-29-63	1.89	1.91	1	82.37	60.43	2.90	468	4313
18E	11- 5-63	2.09	2.06	2	103.00	65.37	3.92	425	4400
05E	11-12-63	1.44	1.44	2	88.56	55.56	3.53	362	4664
07E	11-12-63	1.10	1.40	2	59.62	56.87	3.85	346	4611
05E	12-31-63	2.18	2.43	2	106.37	68.31	4.07	433	4469
07E	12-31-63	2.07	2.35	2	71.75	65.75	3.85	379	4369
25E	1-21-64	1.54	1.60	3	48.01	48.56	3.13	367	4294
07E	2-17-64	1.80	1.82	3	74.18	49.81	4.59	380	4283
07E	2-25-64	2.30	2.63	4	52.75	46.56	4.75	392	4539
15E	3-10-64	2.35	2.00	3	54.37	32.00	4.00	340	3739
17E	3-10-64	2.71	2.39	3	45.96	43.06	3.21	373	3811
39E	4- 7-64	1.53	1.78	3	75.88	46.50	2.53	510	3696
38E	4- 7-64	1.27	1.81	3	59.56	46.50	2.62	398	3571
06E	4-21-64	2.91	2.89	4	55.68	37.81	3.72	385	3590
19E	4-21-64	2.37	2.69	4	82.75	50.68	2.78	390	4010
08E	4-21-64	1.79	1.96	4	44.05	44.37	3.45	367	4295
15E	4-21-64	2.42	2.64	4	50.37	45.20	3.40	398	4183
539X	2- 1-65	2.19	2.47	3	72.93	37.18	4.74	414	4301
28	3-21-65	1.68	1.75	2	71.68	44.00	5.30	391	4385

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix IV. Study II, Slight PSE Group (continued)

Tattoo	Date	Sex[b]	pH	Free 17-OHCS γ/100 ml plasma	Na in plasma mg %	K in plasma mg %	Firmness	Loin eye area
06E	10-22-63	1	5.45	15.88	325	28.4	2	4.88
09E	10-29-63	1	5.32	12.60	408	31.5	2	4.00
18E	11- 5-63	1	5.55	21.52	313	25.7	2	4.22
05E	11-12-63	1	5.30	11.32	372	33.8	2	4.46
07E	11-12-63	2	5.19	15.68	349	31.2	2	
05E	12-31-63	2	5.31	21.28	395	34.0	2	4.96
07E	12-31-63	1	5.29	28.64	375	37.6	2	4.32
25E	1-21-64	1	5.30	12.56	350	28.6	2	4.69
07E	2-17-64	1	5.25	31.68	368	24.0	2	4.55
07E	2-25-64	1	5.49	17.76	408	25.3	2	3.57
15E	3-10-64	2	5.23	20.32	350	26.5	2	
17E	3-10-64	2	5.25	17.92	370	26.3	2	
39E	4- 7-64	1	5.30	16.32	363	27.2	2	3.71
38E	4- 7-64	2	5.29	14.04	331	23.8	2	4.04
06E	4-21-64	2	5.42	23.20	360	26.9	2	5.35
19E	4-21-64	2	5.39	18.24	400	26.7	2	4.48
08E	4-21-64	2	5.50	22.84	315	22.5	2	4.67
15E	4-21-64	2	5.49	13.80	310	21.0	2	4.41
S39X	2- 1-65	1	5.40	9.92	411	29.3	2	4.78
28	3-21-65	1	5.30	11.32	394	26.5	2	5.41

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix V. Study II, Severe PSE Group

Tattoo	Date	Wt. right adrenal	Wt. left adrenal	Breed[a]	Fibrillar protein mg/g	Sarcoplasmic protein mg/g	NPN mg/g	Na in muscle ppm	K in muscle ppm
05E	10-29-63	2.37	2.31	2	85.12	51.12	3.54	433	4469
07E	1-28-64	2.26	2.38	4	32.16	38.68	2.96	326	4701
05E	1-28-64	2.22	2.40	4	42.43	41.68	3.72	347	4377
08E	1-28-64	2.13	2.22	4	48.34	37.00	4.06	353	4567
015E	2- 3-64	2.07	2.14	3	43.75	38.00	4.17	325	4636
016E	2- 3-64	2.35	2.48	3	54.50	46.50	3.46	403	4288
018	2- 3-64	2.44	2.63	3	50.25	40.93	3.13	382	4292
29	2- 3-64	1.83	1.93	3	42.31	36.56	3.15	364	4097
17E	2-26-64	1.85	1.82	4	48.80	41.12	3.62	304	4749
18E	3-10-64	1.90	2.04	3	49.93	45.06	2.90	362	3991
09E	3-10-64	2.34	2.16	3	40.18	33.68	3.17	338	3891
45E	4- 7-64	1.53	1.68	3	31.39	42.31	2.55	451	3493
46E	4- 7-64	1.37	1.60	3	46.75	35.35	2.77	393	3662
36E	4- 7-64	1.99	2.12	3	49.92	48.06	2.42	474	3611
18E	4-21-64	1.95	2.18	4	59.73	44.94	2.53	451	4304
E15X	2- 1-65	1.45	1.38	3	36.31	23.56	2.82	503	3986
06A	3-21-65	2.21	2.24	2	63.18	37.93	3.45	511	4547
36S	3-21-65	1.75	1.66	3	55.75	31.81	3.60	396	4269
15	3-21-65	1.84	1.79	2	44.12	31.25	3.69	432	4099
35S	3-21-65	1.19	1.38	3	48.81	36.06	3.87	392	4323

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix V. Study II, Severe PSE Group (continued)

Tattoo	Date	Sex[b]	pH	Free 17-OHCS γ/100 ml plasma	Na in plasma mg %	K in plasma mg %	Firmness	Loin eye area
05E	10-29-63	2	5.10	26.72	305	31.2	1	4.78
07E	1-28-64	1	4.87	18.56	295	20.5	1	4.55
05E	1-28-64	1	5.03	22.08	322	19.7	1	4.84
08E	1-28-64	1	5.10	16.64	352	18.9	1	4.92
015E	2- 3-64	1	5.17	8.12	327	25.4	1	4.74
016	2- 3-64	1	5.27	25.44	348	28.7	1	4.77
018	2- 3-64	1	5.08	10.40	357	26.4	1	4.35
29	2- 3-64	1	5.06	17.76	329	23.1	1	3.80
17E	2-26-64	2	5.29	12.80	411	30.2	1	3.03
18E	3-10-64	2	5.23	17.28	400	29.5	1	
09E	3-10-64	2	5.23	27.04	342	29.9	1	
45E	4- 7-64	1	5.31	9.92	360	25.7	1	4.06
46E	4- 7-64	2	5.25	22.72	325	25.1	1	4.93
36E	4- 7-64	2	5.30	16.00	377	29.5	1	4.43
18E	4-21-64	2	5.50	10.24	356	26.1	1	5.03
E15X	2- 1-65	1	5.40	20.80	382	23.6	1	3.81
06A	3-21-65	1	5.02	15.84	353	23.7	1	5.88
36S	3-21-65	1	5.28	19.68	400	31.5	1	5.18
15	3-21-65	1	5.05	8.80	385	26.3	1	5.50
35S	3-21-65	1	5.13	16.16	332	21.6	1	4.95

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix VI. Study III, Prednisolone-Experiment 1

Tattoo	Date	Av. wt. adrenal	Breed[a]	Fibrillar protein mg/g	Sarcoplasmic protein mg/g	NPN mg/g	Na in muscle ppm	K in muscle ppm
05E	3-30-64	2.31	2	112.69	56.75	3.94	374	4356
06E	3-30-64	1.88	2	80.81	55.31	3.79	385	4136
09E	3-30-64	2.03	2	76.93	40.63	3.87	403	4010
08E	3-30-64	1.61	2	98.81	56.58	3.76	370	3816
15E	3-30-64	2.01	2	91.37	56.28	3.46	375	4058
25E	3-30-64	1.35	2	98.62	56.83	3.46	345	4005
26E	3-30-64	1.05	2	89.37	55.20	4.24	344	4089
27E	3-30-64	2.00	2	96.75	61.17	4.75	382	4198
28E	3-30-64	1.44	2	95.87	46.03	5.05	344	4059
29E	3-30-64	1.42	2	100.18	61.61	3.92	346	4206
35E	3-30-64	1.34	2	108.62	59.41	5.09	335	4163
36E	3-30-64	1.38	2	81.81	45.18	4.00	333	4180
37E	3-30-64	2.00	2	97.37	59.45	4.15	367	4040
38E	3-30-64	1.16	2	79.06	44.22	4.81	367	3945
39E	3-30-64	1.58	2	102.06	60.36	4.04	352	4117

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix VI. Study III, Prednisolone-Experiment 1 (continued)

Tattoo	Date	Sex[b]	pH	Free 17-OHCS γ/100 ml plasma	Na in plasma mg %	K in plasma mg %	Lot
05E	3-30-64	1	5.60	17.44	390	28.6	1
06E	3-30-64	1	5.60	28.00	356	28.3	1
09E	3-30-64	1	5.90	29.44	350	28.1	1
08E	3-30-64	1	5.50	18.72	368	26.8	1
15E	3-30-64	1	5.50	15.40	329	26.9	1
25E	3-30-64	1	5.60	10.00	392	29.2	2
26E	3-30-64	1	5.55	15.40	310	28.9	2
27E	3-30-64	1	5.50	12.48	361	26.8	2
28E	3-30-64	1	5.55	7.48	336	29.5	2
29E	3-30-64	1	5.40	23.84	312	38.0	2
35E	3-30-64	1	5.50	13.92	332	33.5	3
36E	3-30-64	1	5.40	12.64	292	37.3	3
37E	3-30-64	1	5.50	16.16	375	25.0	3
38E	3-30-64	1	5.50	11.20	320	31.4	3
39E	3-30-64	1	5.45	4.96	368	35.0	3

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix VII. Study III, Prednisolone-Experiment 2

Tattoo	Date	Av. wt. adrenal	Breed[a]	Fibrillar protein mg/g	Sarcoplasmic protein mg/g	NPN mg/g	Na in muscle ppm	K in muscle ppm
27E	10-27-64	1.54	2	58.40	44.30	4.46	344	4135
08E	10-20-64	1.28	2	96.62	55.18	5.17	374	4041
09E	10-20-64	1.78	2	101.62	53.75	5.40	391	3982
16E	10-15-64	1.42	2	85.12	41.06	4.88	371	4050
17E	10-15-64	1.59	2	92.81	48.93	4.99	366	4145
02E	10-27-64	2.01	1	91.37	56.28	4.85	338	4406
28E	10-27-64	2.26	1	67.06	41.00	4.30	339	4335
45E	11- 3-64	1.86	2	71.18	51.80	4.69	331	4308
46E	11- 3-64	2.32	2	82.18	45.87	4.69	335	4545
47E	11- 3-64	1.87	2	82.31	51.12	4.80	318	4040
05E	11-10-64	1.18	1	60.95	43.50	4.77	336	4109
07E	11-10-64	1.33	2	73.31	43.75	4.55	364	4068
06E	11-10-64	1.74	1	96.62	51.25	4.86	303	4020
15E	11-17-64	1.76	1	52.53	38.75	4.85	303	4149
16E	11-17-64	1.93	2	80.18	50.60	4.85	290	4172

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix VII. Study III, Prednisolone-Experiment 2 (continued)

Tattoo	Date	Sex[b]	pH	Free 17-OHCS γ/100 ml plasma	Na in plasma mg %	K in Plasma mg %	Lot
27E	10-27-64	2	5.35	6.56	385	26.7	2
08E	10-20-64	2	5.62	6.24	358	40.3	2
09E	10-20-64	1	5.51	6.56	386	34.0	2
16E	10-15-64	2	5.50	4.92	322	35.9	2
17E	10-15-64	1	5.50	4.88	350	32.0	2
02E	10-27-64	2	5.30	15.88	376	31.2	1
28E	10-27-64	1	5.50	16.96	390	29.6	1
45E	11- 3-64	1	5.59	9.56	338	29.5	1
46E	11- 3-64	2	5.60	12.28	365	33.0	1
47E	11- 3-64	2	5.40	12.32	356	33.0	1
05E	11-10-64	1	5.15	7.84	352	26.5	3
07E	11-10-64	2	5.40	6.64	329	25.3	3
06E	11-10-64	1	5.60	13.52	346	24.7	3
15E	11-17-64	2	5.30	12.24	366	23.0	3
16E	11-17-64	2	5.40	13.64	365	23.4	3

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix VIII. Study III, Prednisolone-Experiment 3

Tattoo	Date	Av. wt. adrenal	Breed[a]	Fibrillar protein mg/g	Sarcoplasmic protein mg/g	NPN mg/g	Na in muscle ppm	K in muscle ppm
06E	3-16-65	1.72	2	70.06	35.37	5.56	344	4348
39E	3-16-65	1.30	2	71.63	43.56	4.27	243	4363
09E	3-17-65	1.72	1	81.12	50.56	4.72	284	4275
17E	3-17-65	1.69	1	69.93	51.81	4.29	331	4056
08E	3-17-65	1.52	1	86.62	48.31	4.25	332	3865
07E	3-17-65	1.63	1	90.50	45.87	4.35	344	3823
08E	3-30-65	1.27	2	81.43	43.62	5.42	292	4184
05E	3-30-65	1.60	1	89.25	56.43	4.53	342	3872
07E	3-30-65	1.63	1	88.81	62.00	4.85	272	3880
06E	3-30-65	1.32	1	81.75	43.00	3.19	282	4000
38E	3-31-65	1.79	2	84.97	43.70	3.20	271	4551
35E	3-31-65	1.57	1	101.06	45.43	5.41	271	4368
16E	3- 2-65	2.16	1	107.62	52.25	4.31	371	4318
06E	3- 2-65	2.41	2	78.43	43.12	4.05	322	3990
28E	3-16-65	1.97	1	80.25	44.81	4.64	325	4339
17E	3-16-65	2.00	2	82.50	46.62	5.48	315	4363
36E	3-31-65	2.34	2	79.50	45.62	4.45	313	4205
39E	3-31-65	2.35	2	108.26	57.00	4.52	293	4366

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix VIII. Study III, Prednisolone-Experiment 3 (continued)

Tattoo	Date	Sex[b]	pH	Free 17-OHCS γ/100 ml plasma	Na in plasma mg %	K in plasma mg %	Lot
06E	3-16-65	2	5.25	4.08	355	28.3	2
39E	3-16-65	2	5.39	10.72	336	28.2	2
09E	3-17-65	2	5.45	5.88	342	30.4	2
17E	3-17-65	1	5.43	10.96	342	31.1	2
08E	3-17-65	1	5.46	5.88	335	29.1	2
07E	3-17-65	1	5.50	4.12	355	33.6	2
08E	3-30-65	2	5.50	8.96	346	22.6	3
05E	3-30-65	2	5.45	6.56	360	29.5	3
07E	3-30-65	1	5.60	4.00	347	34.5	3
06E	3-30-65	1	5.46	11.52	334	32.0	3
38E	3-31-65	1	5.35	8.24	342	27.6	3
35E	3-31-65	2	5.40	4.04	338	23.6	3
16E	3- 2-65	1	5.48	11.88	355	30.2	1
06E	3- 2-65	1	5.53	10.00	314	22.0	1
28E	3-16-65	1	5.49	11.56	354	31.2	1
17E	3-16-65	2	5.31	13.32	351	31.9	1
36E	3-31-65	2	5.30	13.28	346	26.5	1
39E	3-31-65	2	5.40	13.20	357	23.0	1

[a]Breed: 1 = York, 2 = Hamp, 3 = Poland, 4 = Landrace
[b]Sex: 1 = male, 2 = female

Appendix IX. Adrenal weights, myofibrillar and sarcoplasmic protein values and plasma 17-OHCS levels from animals possessing normal muscle

Number	Wt. right adrenal gms	Wt. left adrenal gms	Myofibrillar protein mg/g	Sarcoplasmic protein mg/g	Plasma 17-OHCS γ/100 ml
1	1.60	1.63	123.4	62.1	26.8
2	1.89	2.16	107.8	57.8	15.8
3	2.21	2.46	111.1	60.3	17.6
4	1.84	2.04	100.5	67.0	37.9
5	1.79	1.90	111.4	51.3	21.9
6	1.68	1.65	103.2	63.2	23.8
7	2.32	2.22	103.0	72.3	22.1
8	1.74	1.85	108.9	73.2	28.9
9	1.74	1.83	97.7	51.1	20.5
10	2.53	2.35	79.2	46.9	12.8
11	2.57	2.90	78.1	51.4	18.7
12	1.80	2.04	89.9	41.9	22.1
13	1.59	1.49	68.8	36.7	14.1
14	1.70	1.72	67.1	50.0	17.7
15	2.53	2.24	70.8	35.0	20.7
16	1.89	1.87	68.4	37.7	11.5
17	1.69	1.99	115.8	40.8	13.6
18	2.17	2.41	80.9	43.3	17.3
19	2.47	2.43	108.2	53.4	20.6
20	1.80	2.07	78.6	67.1	13.9
21	1.92	1.94	81.7	48.9	10.8
22	1.57	1.78	76.7	39.3	14.5
23	1.78	1.97	77.4	45.6	17.1
24	1.44	1.71	43.6	37.8	17.9
25	2.37	2.73	83.7	51.3	17.4