STUDIES ON A MYODEGENERATION SYNDROME IN SWINE

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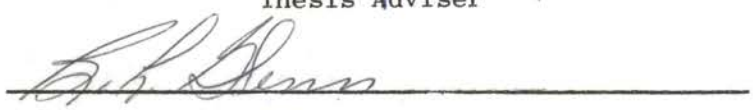

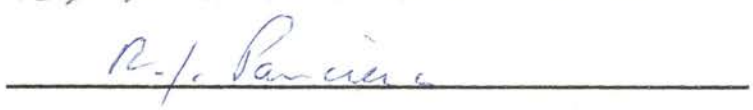

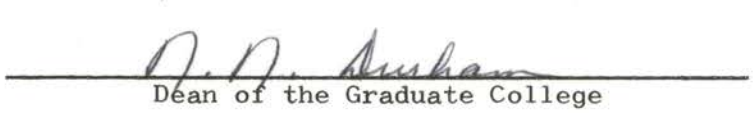

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CHAPTER I

INTRODUCTION

Massive areas of necrosis were observed in the musculature of several swine carcasses in the fall of 1967 during routine postmortem examination at the Oklahoma State University abattoir. The lesions were observed in the central areas of lumbar epaxial muscles and extended longitudinally for as much as several centimeters. Areas surrounding the central necrotic core of the muscle appeared to be relatively unaffected. More thorough inspection of the entire musculature in subsequent cases disclosed lesions of a similar nature but generally of smaller size in several muscles of the pelvic limb. Similar lesions were observed in the musculature of a limited number of swine with a history of acute death which were presented for necropsy examination in the Department of Pathology, College of Veterinary Medicine, Oklahoma State University.

The similarity in nature and distribution of muscle lesions in swine carcasses encountered at the abattoir, and in those found dead without apparent cause and examined at necropsy, suggested that the lesions represented a specific syndrome not previously recognized in local swine herds. Further investigation disclosed that the lesions were most often found in slaughter-age swine of heavily muscled body conformation. Frequently, affected animals had been subjected to unusual environmental stress shortly before the onset of illness or

death. It was also observed that the disease occurred in representatives of a single breed and further, there was a higher incidence in specific family groups. The indications of possible genetic relationship to susceptibility and the number of affected animals with similar lesions encountered over a relatively short period of time emphasized the possible economic importance of this peculiar muscle disease syndrome to the swine breeding and meat industry. The need for further investigation of this disease syndrome was apparent.

Various types of muscle disease syndromes in swine have been reported in the literature by several investigators in the United States and Europe. Muscle lesions reported in some of these syndromes bear similarity to those observed in the syndrome encountered at Oklahoma State University. None of the previously described syndromes were sufficiently similar in all respects to that observed in Oklahoma swine to suggest complete identity with the Oklahoma disease. However, there is sufficient similarity to suggest a close relationship relative to causal factors and perhaps they may represent different manifestations of the same disease.

It appears that the disease syndrome as observed in local swine herds and in some of the syndromes described in the literature occurs almost exclusively in heavily muscled individuals and is especially prevalent in herds in which selective breeding is practiced to develop swine of superior muscling for the consumer market. The majority of affected animals encountered at Oklahoma State University have come from a Yorkshire herd in which considerable inbreeding was practiced to establish a more rapidly maturing and a more heavily muscled blood line. Since this same objective is sought by other progressive swine breeders,

it is apparent that if predisposition to the disease is genetically linked to superior muscling, then predilection to the disease is increased. In view of this possibility, it is apparent that some means of detecting the more susceptible animals in breeding herds is desirable so that they can be removed. If adequate means for detecting susceptible animals were available, selective breeding for superior conformation and more desirable muscle:fat ratio could be accomplished with a more favorable economic outlook for the future in the swine industry.

The present investigation was conducted as a cooperative venture by the Department of Veterinary Pathology and the Institute of Animal Science and Industry, Oklahoma State University. The study was undertaken to more fully characterize the disease clinically, morphologically, and relative to certain biochemical parameters. A battery of serum determinations was performed for biochemical characterization of the disease. Subsequently, serum analyses for selected biochemical constituents were applied to a population deemed susceptible and a population deemed not susceptible.

CHAPTER II

REVIEW OF SELECTED LITERATURE

General Aspects of Myodegeneration in Swine

Various aspects of myodegeneration in swine were reviewed by Blaxter and McGill (1955). These authors listed three major factors, acting singly or in combination, which they believed to have a causal relationship to myodegeneration. The factors which they considered to be most commonly involved were: 1) A deficient diet, such as a relative lack of vitamin E or selenium, 2) added substances in the diet such as an excess of polyunsaturated fatty acids, and 3) unaccustomed muscular exertion or other stresses probably in association with inherited predisposition.

Lannek (1963) noted that muscular dystrophies in domestic animals were mainly restricted to the temperate climate zones of the world -- areas that are generally not deficient in natural protecting substances as tocopherols and selenium. He proved that a higher content of polyunsaturated vegetable fat (linoleic acid) is produced in plants grown at low temperatures. He further showed that pigs fed on grain containing a high content of polyunsaturated fatty acids developed a spontaneous muscular dystrophy unless tocopherols and selenium were proportionally increased in the diet.

Grant (1961) fed pigs a ration containing large amounts of polyunsaturated vegetable fat and fish oil. Pigs fed this diet developed

microangiopathy with associated myopathy. He concluded that these lesions represented another expression of Mulberry Heart Disease. Blaxter and McGill (1955) reported that swine fed a ration deficient in vitamin E and containing unsaturated fat favored the development of massive edema comparable to the exudative diathesis of chicks fed on a similar diet.

Myopathy in Swine Four to Nine Months of Age

Of several myopathic disorders encountered in swine, the syndrome designated as "fatal syncope", "enzootic apoplexy", "Herztod" or "Ludvigsen's total muscular degeneration" is considered to be of major economic importance to the swine industry in Denmark, Austria and parts of Germany. The syndrome was originally described as characterized by unexpected acute death in pigs which had not shown clinical signs of disease. Death in these pigs was attributed to acute myocardial degeneration. A recent description of the syndrome by Nieberle and Cohrs (1967) presents the clinical features which occur in the period immediately prior to death as a shock-like syndrome with associated asphyxia and tetanic cramps. Lesions which they described on necropsy examination were congestion and edema of the lungs, passive congestion of the thyroid and of the visceral organs, and myocardial degeneration in a tigroid stripe pattern. Additionally, large amounts of serous fluid in the pericardium and blood clots in the left ventricle were observed in many of the affected animals.

On microscopic examination, lesions of the heart were described as hyaline degeneration of bands of myocardial fibers with intervening areas of congestion and ischemia. Seventy per cent of the cases also

showed collapse of many of the follicles in the thyroid gland. This later lesion was believed by these authors to be almost pathognomic of the disease. Degenerative changes comparable to those in the heart were also present in skeletal muscles.

A disorder designated as "Ludvigsen's total muscle degeneration" characterized predominantly by degenerative changes in skeletal muscle was reported (Ludvigsen, 1957). Ludvigsen considered this to be a mild form of Herztod. Eikmeier (1965) found a high correlation between the increasing incidence of this milder form of the disease and selective breeding for greater muscle mass. The pigs most severly affected ranged from 80-110 kilograms in weight that are raised under husbandry conditions which afford little or no exercise. The disease syndrome was found to be almost nonexistent in breeding animals that were afforded better opportunity for liberal exercise.

Ludvigsen (1957) observed that incidence of the disease was higher in certain genetic strains of pigs and that exposure of the more highly susceptible pigs to only slight changes in husbandry or environmental conditions may precipitate developments of the syndrome. Acutely affected pigs developed signs of lethargy, fever, labored breathing, slight cyanosis, ischemia of areas in the skin, muscular tremors and weakness. On rare occasions, degenerative changes were found in all skeletal muscles, but usually only a single muscle or associated groups of muscles were involved. Muscles more commonly affected were the longissimus dorsi, psoas, gluteal, biceps femoris and the gracilis. Eikmeier (1965) reported that the areas of the longissimus dorsi muscle most frequently involved were located between the ninth and eleventh thoracic vertebrae and between the second and fourth lumbar vertebrae.

Innes and Saunders (1962) state that the mildly affected musculature may only be pale, edematous and have a sour odor while the lesions in more severly affected muscles exhibit a dull gray, chicken-flesh appearance typical of acute myodegeneration.

Nieberle and Cohrs (1967) describe the lesions in severely affected muscles as irregular opaquish gray patches and streaks resembling the lesion found in the heart of pigs dead from acute Herztod. Microscopically the lesions in different muscles vary from simple ischemia to marked degenerative changes in individual muscle fibers and groups of fibers. Changes observed in severely affected muscles include hyaline degeneration accompanied by lumpy fragmentation, focal finely dispersed fatty degeneration, granular disintegration, and myolysis. The myofibers may be widely separated by large quantities of edematous fluid containing numerous histiocytes. Fibrous connective tissue proliferation was present in older lesions which apparently had developed at some earlier period prior to death of the animal.

The etiology and pathogenesis of Herztod or Ludvigsen's total muscle degeneration is incompletely resolved. Of the many explanations offered, Ludvigsen's theory is considered to be most probable (Nieberle and Cohrs, 1967). He proposed that the selection of pigs for rapid growth and large muscle mass has inadvertently created a hormone imbalance. The hormone imbalance favors the growth hormone which in-turn has a depressant effect on thyroid stimulating hormone and adrenal corticotropic hormone of the anterior pituitary gland. This situation, frequently in association with an inadequate diet (carbohydrate rich and protein poor), favors an explosive release of thyroid hormone in an

emergency, thus creating a thyrotoxicosis which leads to hypoexemia and thyrotoxic circulatory collapse.

Porcine Stress Syndrome

Topel et al. (1968) described a condition in swine which they termed "Porcine Stress Syndrome". Swine which have been bred for rapid growth qualities, more efficient feed conversion, superior muscling and minimum backfat were more susceptible to development of the condition. Clinically, the disease was characterized by acute death in market age pigs and usually occurs during or immediately following stress conditions such as transporting, fighting, or other forms of exertion, gilts in estrus, or a sudden rise in atmospheric temperature. The clinical signs observed were similar to those of Ludvigsen's total muscle degeneration. Signs usually commenced as a muscle tremor followed by dypsnea, cyanosis, elevated body temperature, alternating areas of blanching and erythema in the skin, terminal collapse and death. Complete rigor occurred very rapidly, usually developing within two to five minutes after death.

The lesions associated with the Porcine Stress Syndrome as described by Topel et al. (1968) were pale, soft, and very moist appearing muscles most often involving the longissimus dorsi, gluteus medius and the biceps femoris muscles. These authors stated that the muscle fibers were unaltered morphologically. The absence of hyaline degeneration and necrosis in affected muscles indicates that the Porcine Stress Syndrome as described by Topel et al. is significantly different from Herztod at least with respect to the type of muscle lesions involved.

Other investigators have reported observations of lesions similar to those described by Topel et al., variously designated as "pale, soft exudative pork" and "transport muscle degeneration", in slaughter swine carcasses observed at abbatoirs for many years (Lannek, 1967; Judge, 1968). Judge (1968), in an effort to explain the cause of pale, soft exudative pork (PSE), postulated that chronic stress of husbandry practices and environment, especially with certain genetic strains of pigs, places excessive demands on the pituitary-adrenal system to such an extent that the animal may lose the capacity to resist further secondary stress. Experimental work of Judge and Stob (1963) suggested that chronic stress (daily injections of epinephrine in growing lambs) render the muscle relatively more sensitive to external stimuli than other body tissues. A high content of lactic acid in PSE muscle was believed to be a reflection of an inadequate circulatory system which does not allow the muscle to maintain local homeostasis after massive discharge of nervous impulses associated with the excitement of handling and slaughter (Judge, 1968). Frankel et al. (1963) proposed that a circulatory failure precipated by lactic acid build-up under hyperthermic conditions resulted in hypoxia of the muscle tissue. Biochemical alterations in PSE muscle include a rapid drop of pH, increasing quantities of lactic acid, loss of myoglobin, accelerated rates of anaerobic glycolysis and low levels of adenosine triphosphate and creatine phosphate (Sybesma, 1963; Lannek, 1967; and Judge, 1968).

Necrosis of the Longissimus Dorsi Muscle in Swine

Necrosis of the longissimus dorsi muscle in approximately five month old pigs was studied in Holland by Thoonen and Hooren (1960).

The clinical signs described included sudden development of unilateral or bilateral swelling of the longissimus dorsi muscle with associated shortening and arching of the back and difficult motility. Unilateral involvement or unequal involvement of the muscles on the two sides resulted in assymetry of posture or outline of the back area so that a lopsided posture was often a prominent feature. Affected pigs were extremely distressed, feverish, and inappetent. After several days, obvious distress disappeared and appetite returned to normal. Natural death from the disease was not mentioned, however, animals so affected remained deformed for the remainder of their lives. Postmortem examination at slaughter of one of the affected pigs disclosed that the central area of sections of the left longissimus dorsi muscle between third and thirteenth thoracic vertebrae (approximately 20 centimeters) and between the second and fifth lumbar vertebrae (approximately 10 centimeters) were necrotic. Similar areas of necrosis were observed in the right longissimus dorsi muscle between the first and fifth lumbar vertebrae (approximately 13 centimeters). A reddish zone of demarcation separated the dead from living muscle mass. The necrotic portions of the muscle were represented by an amorphous mass which was pale, dry, wax-like and chalky in appearance. Microscopically, the nuclei of interstitial cells and muscle fibers were pyknotic or absent. The sarcoplasm had disintegrated into lumpy fragments. Hemorrhages were present along the edges of the dead muscle. Histiocytes, leukocytes, fibroblasts and foreign body giant cells were present in the zone of demarcation between the dead and alive muscle tissue. Numerous areas of calcification were present in the necrotic tissue. Organizing fibrous connective tissue was present in the peripheral areas of the lesions encroaching upon and

producing atrophy of adjacent muscle fibers. Thoonen and Hooren (1960) did not speculate on the pathogenesis of the lesion; however, Nieberle and Cohrs (1967) suggested that ischemic infarction from thrombosis and embolism of the nutrient artery was responsible for massive coagulation necrosis of the longissimus dorsi muscle which occurs in otherwide healthy slaughter pigs.

Relationship of Serum Enzyme Levels and Muscle Degeneration

A balance between cell synthesis and breakdown normally maintains blood serum values of intracellular enzymes at relatively constant and generally low levels. Pathologic states which cause tissue damage may result in significant elevations in serum enzyme levels due to increased permeability of the cell membranes or as a result of complete breakdown of the cell structure (Mattenheimer, 1968). Overproduction of muscle and intracellular enzyme may also be a contributing factor to elevated serum levels as suggested by Farrell et al. (1966) in relation to elevated creatine phosphokinase levels in vitamin E deficient rabbits.

Henson (1966) found a significantly greater concentration of creatine phosphokinase (CPK) in skeletal muscle, myocardium, and the brain than in other body tissues. Data obtained by Meltizer (1968) suggested that increased cell membrane permeability or decrease serum clearance of CPK may be associated with stress. Increased serum CPK levels without loss of muscle mass have been observed in acute paroxysmal myoglobinuria, in some cases of acute polymyositis, following crushing injuries and strenous unaccustomed exercise. These findings are believed to be supportive evidence for "membrane leak" or abnormal

permeability of the sarcolemma (Farrell et al., 1966; Hess et al., 1964; Sodeman, 1961). Hess et al. (1964) postulated that the sarcolemma is more permeable to CPK than to other enzymes.

The degree of elevation of serum CPK levels associated with muscle damage depends upon the stage of the myopathic process at which the blood sample was taken. Henson et al. (1966) studied the activity of serum CPK and serum glutamic oxalacetic transaminase (SGOT) following plasmoid dehydroiodide injection into rabbits. They observed twenty per cent of the myofibers in the diaphragm were undergoing degenerative changes when serum CPK activity began to rise and that serum CPK and SGOT reached maximal activity between eight and twelve hours after poisoning with the myotoxin. Serum enzyme levels returned to normal forty-eight hours after injection. Milhorat (1966) correlated serum CPK activity with myopathic changes occurring in patients with Duchenne muscular dystrophy. Extensive hyaline degeneration and minimal amount of regenerative attempts were correlated with markedly high serum CPK activity. In the advanced stages of Duchenne muscular dystrophy when only a few muscle fibers remain, CPK activity was practically normal.

Hess et al. (1964) found the mean serum enzyme levels in fifty patients with pseudohypertrophic muscular dystrophy to be elevated as follows: CPK elevated 54.7 fold, SGOT elevated 2 fold, and LDH elevated 3.1 fold. These authors concluded that in most diseases of the muscle, a relationship exists between the maximal elevations of CPK, SGOT and lactic dehydrogenase (LDH).

Serums LDH and SGOT are present in a wide variety of organs. Particularly high concentrations of these enzymes are found in the liver and skeletal muscle. In myopathic diseases, Blencoe et al. (1960)

found a correlation between elevated serum levels of LDH and SGOT. They concluded that high levels of both LDH and SGOT indicate cellular destruction and are released from diseased muscle in proportionally equal amounts.

CHAPTER III

MATERIALS AND METHODS

The study was conducted in three phases as follows: Phase I consisted of preliminary characterization of the myodegeneration syndrome on the basis of gross and microscopic lesions and limited biochemical determinations in spontaneously affected individual swine which were encountered during postmortem examination at the abattoir, in those which had died unexpectedly and were presented for necropsy, and in affected swine which were still alive at the time of initial examination. Phase II consisted of a series of determinations of several biochemical constituents in blood serum in three different populations of swine to evaluate the applicability of biochemical parameters in further study of the disease. Phase III was a comparative study of the sequential changes in serum levels of selected biochemical constituents following a period of forced exercise in a group of swine representing a high incidence breed population versus a group representing a breed population in which no known cases of the disease had occurred.

Experimental Animals

The animals studied in Phase I consisted of 25 individual swine submitted to the Department of Veterinary Patholgy for diagnosis plus one additional pig examined clinically but which was not available for necropsy examination. Nineteen animals were submitted dead and six

animals were alive. All of these animals were of Yorkshire breeding stock originating in the swine herd of the Institute of Animal Science and Industry, Oklahoma State University. They ranged from 45 to 222 kilograms in weight and from three months to two years of age. The age of most frequent occurrence among the group was between four and five months. Animals studied in Phase II consisted of three genetically dissimilar groups of swine. Groups I and II consisted of 24 animals maintained under uniform husbandry practices by the Institute of Animal Science and Industry, Oklahoma State University. Group I consisted of 10 Yorkshire swine from the herd in which the syndrome was of frequent occurrence. Group II consisted of 14 Hampshires representing a strain of swine in which the disease had not been recognized. Group III consisted of 11 swine of mixed breeding from a nearby commercial herd that had no reported or observed incidence of the disease. The latter group of animals was raised under husbandry practices and a dietary regime that differed considerably from those in Groups I and II. All animals in each of the three groups were randomly selected from approximately three-month old pigs in the 40 to 50 kilogram weight range.

Animals studied in Phase III consisted of two Subgroups of seven pigs each selected from Groups I and II, respectively, which were used in Phase II. The pigs comprising Subgroups I (Yorkshires) and II (Hampshires) ranged from 84-98 kilograms in weight by the time Phase III of the study was conducted and had been continuously maintained under similar husbandry practices. The seven pigs in each subgroup were selected to include all pigs from each of Groups I and II having heavy muscled body conformation believed to be predisposing to the development of the myodegeneration syndrome and which also showed unusually high

levels of certain serum constituents determined in Phase II. The remainder of the pigs in Subgroups I and II were selected to include representative animals which showed intermediate and low levels of these same serum constituents.

General Experimental Procedure

Phase I: Preliminary Characterization of the Myodegeneration Syndrome

Twenty-five animals studied in Phase I were examined by necropsy. Gross lesions were recorded and representative tissue specimens were collected for microscopic examination. Specimens for microscopic study included selected areas of the muscle lesions and adjacent unaffected muscle, thyroid glands, adrenal glands, kidney and liver. Additionally brain tissue from two animals and the pituitary gland from one animal was examined microscopically. The tissues were fixed in 10 per cent buffered formalin, sections were cut at five microns thickness and stained with hematoxylin and eosin.

Blood samples for biochemical examination were obtained prior to necropsy of the six animals which were alive when first submitted for examination. A blood sample for biochemical examination was also obtained from one live pig with clinical signs of the myodegeneration syndrome but which did not succumb to the disease and was not examined by necropsy.

Individual case histories as well as clinical signs observed in those affected animals which were still alive when first noted by the herdsman and handlers were recorded. Several of the animals were found dead unexpectedly without prior clinical signs being noted. However, a

number of the animals which were submitted dead were being subjected to physical stress of handling or had been observed fighting at the time of or immediately prior to development of the first observed clinical signs.

Phase II: Biochemical Studies in Rested Swine

This phase of the study was designed to obtain comparative data on levels of selected serum constituents which might be of value in predicting individual swine that may be more susceptible to development of the myodegeneration syndrome. Data was obtained from three groups of swine representing (1) animals from a high-incidence herd (Group I), (2) animals from a herd with no recognized occurrence of the disease, but with husbandry practices and diet similar to those of the high incidence herd (Group II), and (3) animals from a herd with no recognized occurrence of the disease, but with different husbandry practices and diet (Group III). Three blood samples were obtained successively at two-week intervals from each animal in the three groups by venipuncture of the anterior vena cava (Hokanson et al., 1965). The animals were handled carefully so that samples were obtained with minimal disturbance to the animal and without prior stress of muscular exertion. All blood samples were collected during the early morning between 7:00 and 8:30 a.m. so that effects of high environmental temperature and prior activity were minimized. The blood samples were collected without anticoagulant and held under refrigeration for 2-4 hours before centrifugation to facilitate clot retraction and collection of serum. The serum was held under refrigeration until all determinations were completed.

The serum biochemical constituents measured were: sodium, chloride, phosphorous, magnesium, calcium, potassium, creatine phosphokinase, glutamic oxalacetic transaminase, glutamic pyruvate transaminase and total lactic dehydrogenase. All determinations were conducted in as near the same sequence and at as near the same time interval after collection of the sample as practical. In most incidences, all determinations were completed within 24 hours after sample collection. In these instances where greater time lapse was involved, the serum was stored in the frozen state.

Phase III: Biochemical Studies Following Forced Exercise

Blood samples for biochemical examination were collected from the seven Yorkshire pigs and the seven Hampshire pigs comprising Subgroups I and II, respectively, immediately prior to and at 2, 12, 24, and 48 hours following a ten minute period of forced exercise. Additional blood samples were collected at 96 hours from the Yorkshire group. Blood serum constituents were determined as described in Phase II. The serum values obtained in Phase II and those obtained immediately prior to exercise served as control for evaluation of alterations which occurred in the post exercise period.

Serum Assay Procedures

Serum sodium and potassium levels were determined using a Baird Model KY2 flame photometer according to procedures recommended by the manufacturer.[1] Serum calcium and chloride levels were determined using

[1]Baird-Atomic, Inc., Cambridge, Massachusetts.

a Model 301 Oxford titrator according to the manufacturer's recommended procedure.[2] Serum magnesium was determined as described by Natelson (1963). Serum phosphorus was determined as described by Goldenberg and Fernandez (1966). All serum enzyme determinations were made using reagent kits supplied by Sigma Chemical Company according to procedures recommended by the supplier.[3]

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[2]Oxford Laboratories, San Mateo, California.

[3]Sigma Chemical Company, 3500 DeKalb Street, St. Louis 18, Mo. Sigma Technical Bulletin Nos. 520 (1967), 505 (1964), and 500 (1965).

CHAPTER IV

RESULTS

Clinical Signs

Pigs most frequently affected by the myodegeneration syndrome were the more rapidly maturing pigs with exceptionally heavy musculature and in the age range of four to six months. The youngest pig affected by the disease encountered in this study was two months old and the oldest was two years of age. Clinical manifestations observed in the initial stages of acutely affected pigs included muscular tremor in local muscle groups which tended to spread rapidly until most skeletal muscles were involved, acute respiratory distress manifested by open-mouth, labored breathing with the tongue protruding, rapid development of cyanosis of mucous membranes, distended cutaneous vessels, and simultaneous or subsequent development of large irregular patches of ischemia intermingled with cyanotic areas of the skin particularly noticeable over the shoulders, jowl, brisket, and ventral abdomen. Subsequently, the majority of affected pigs developed a unilateral or bilateral swelling involving the longissimus dorsi muscles and also occasionally the gluteal and thigh muscles of the pelvic limb. The degree of swelling of these muscle groups varied from slight to marked.

During early stages in development of the clinical signs affected pigs were reluctant to move, appeared weak as manifested by wobbly gait and leaning against a fence or other available support, but usually

remained standing with an accentuated arched back posture until near terminus at which time they lost the ability to stand and usually lay on their side in a semicomatose state with an arched back posture and with paddling or running movements of the feet until death.

Death occurred in the majority of acute cases which were observed clinically in less than 30 minutes to several hours after development of initial clinical signs. In animals which were less acutely and less severely affected, death was delayed from one to several days after initial signs were noted. None of the animals observed in the acute syndrome recovered; however, three of the acutely affected animals were killed for necropsy examination within one to one and one-half hours after initial development of clinical signs. Rigor mortis developed very rapidly after death. In those which died within a few hours after onset of clinical signs, full development of rigor occurred within a few minutes after death.

Those animals which survived beyond the first few hours after onset of clinical signs usually continued to exhibit the arched back posture, reluctance to move, and altered gait. In those cases with more unilateral involvement of the loin musculature, the pig appeared to be "tucked in" on the affected side giving a lopsided appearance to the posture and walk.

Those animals with bilaterally swollen loin muscles stood with a marked arching of the back with hindquarters tucked up under the body so that they walked in an awkward stiff-legged fashion on the heels of the rear feet and the toes of the forefeet. Swelling of the loin muscles began to regress slowly in those pigs which survived beyond a few days after the acute attack. Regression of the swelling was usually

accompanied by marked atrophy and reduction in the loin muscle mass forming a concave depression lateral to the vertebral column, consequently the dorsal spinous processes appeared more prominent. Pigs with unilateral involvement of the loin that survived for longer periods continued to exhibit lateral curvature of the spine. One pig which apparently had survived an earlier attack was observed in the herd and had developed a marked edema of the hind legs.

Case Histories and Predisposing Factors

A stressful experience was usually implicated as a predisposing factor to development of the myodegeneration syndrome. The following are examples illustrating activities which precipitated attacks of the disease. A year-old boar died of the disease a few hours after his first service. Similarly, a ten-month old gilt that was being maintained in a pen adjacent to a boar was found dead during an estrus period. Fighting, particularly common when strange animals are mixed, appeared to be a major stressing factor in initiating development of the disease process. Two gilts, five and six months of age, died unexpectedly of the disease the following day after they were observed fighting with other gilts. A six-month old boar died of the disease following a similar fighting incident. The boar had been selected as a herd sire and had been placed in a pen with other herd sires. Following the fighting incident, the pig was observed to be in extreme distress and had a noticeable swelling of the right loin, altered gait, and walking in an oblique fashion. The pig subsequently lost all desire to move or eat. Twenty-four hours later he was discovered near death on his back in a 6-inch deep gutter, unable to rise. Rigor mortis

developed almost immediately after death. Several pigs, ten to twelve weeks of age, exhibited signs of dypsnea, mild cyanosis, and lethargy following fighting episodes which frequently occurred among pigs being placed together for the first time. This transient generalized distress and muscular weakness was believed to be a mild form of the disease and suggested that these pigs might succumb to later and more severe attacks of the disease.

Physical activity and excitement induced by working and handling operations were definite factors in initiating development of the myodegeneration syndrome. Ten per cent of 60 Yorkshire barrows randomly selected from the Animal Science and Industry herd that were involved in an experimental procedure developed an acute fatal attack of the disease syndrome during handling.[1] Exertion during trimming of the hooves of a six-month old gilt in preparation for a fair exhibit presumably initiated development of the disease syndrome and death. A further example occurred in a ten-month old female which was observed to have a swelling of the loin shortly after a sorting operation in the herd. Other management practices and environmental conditions which appeared to be predisposing to development of the myodegeneration syndrome in isolated cases were transfer from close confinement to open pasture, abrupt confinement with strange pigs, change to an unaccustomed ration, transportation stresses, and abrupt changes in environmental temperature.

Genetic predisposition to development of the syndrome was strongly

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[1]These pigs were rigidly restrained in a whole body counter for three consecutive 30-minute periods in a K-40 experiment for live-carcass evaluation.

implicated in that all cases encountered in this study occurred in a highly inbred Yorkshire breeding herd. Although a complete herd study was not made, an exceptionally high incidence of the disease was noted among littermate offspring from two sows in the herd and in a subsequent generation of one of these sows. In one litter, two of three gilts which were saved as herd sows developed the disease and the third farrowed offspring which succumbed to the disease. In the other litter, two of three littermates died of the disease. Complete evaluation of genetic history was not included as a part of this study; however, most if not all pigs among the herd which developed the myodegeneration syndrome were traceable to a single herd sire.

Gross Lesions

Pigs submitted for necropsy usually were in marked rigor with the back arched and with hind quarters tucked forward. A noticeable unilateral or bilateral enlargement of the longissimus dorsi muscle was usually present. Unilateral involvement produced an obvious assymetric conformation of the back. The visibly affected portion usually extended from the eighth or ninth thoracic vertebra to the fifth lumbar vertebra. Cyanosis of the tongue and mucous membranes and irregular cyanotic patches in the skin were usually present. Ischemic areas interspersed among the cyanotic patches were often present, especially in the younger animals which had been dead for only a short time. These blotchy discolorations of the skin were most noticeable over the ears, shoulders, brisket, jowl, and ventral abdomen. The major internal lesions observed were associated with the skeletal musculature and were quite varied in both severity and extent of involvement. In all instances, the most

severe lesions, and often the only grossly visible lesions, occurred in the longissimus dorsi muscles. Other muscles frequently involved to varying extent and degree included the biceps femoris, semimembranosis, semitendinosis, gracilis, and gluteus medius. Less frequently, lesions were found in the superficial gluteus, latissimus dorsi, and supraspinatus muscles.

There was a distinct tendency for the lesions to occur more consistently in the deeper, more central areas of the muscle and to extend longitudinally through the thicker portion of the body of the muscle. This feature was most prominent in the older lesions of the longissimus dorsi in which the lesion often formed a well-defined, central core-like area extending along the main body of the muscle. Lesions when present in other muscles were usually less prominent, less well-defined, and less consistent in locations within the muscle.

In those animals which died rapidly after onset of clinical symptoms, the lesions transverse section appeared as ill-defined, swollen, pale areas within the affected muscle with little or no congestion in the surrounding muscle tissue. In those cases in which lesions were more prominently developed, they appeared as ill-defined, irregular, pale areas, surrounded by an irregular, ill-defined dark reddish zone of congestion and hemorrhage of varying thickness. The pale areas were dull grayish white, dry in texture and wax-like in appearance with scattered reddish foci of congestion and hemorrhage often interspersed within the pale area (Figures 1 and 2). In the well-developed lesion, the pale area was more well defined by a more extensive and darker zone of hemorrhage and congestion was slightly yellowish in color, and somewhat friable in texture. Such areas presented a

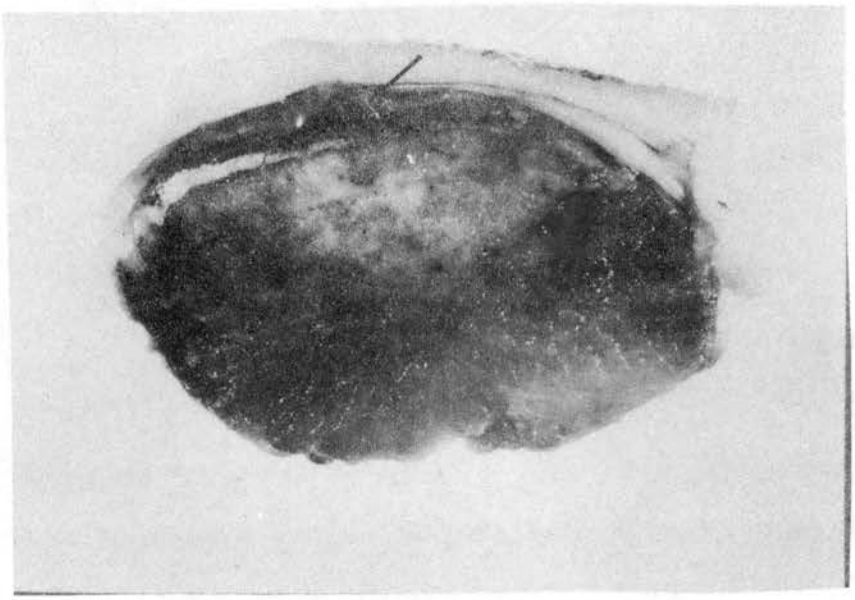

Figure 1. Transverse Section of the Longissimus Dorsi Muscle Showing the Myopathic Lesion in an Early State of Development and Pronounced Edema of the Adjacent Fascia (arrow)

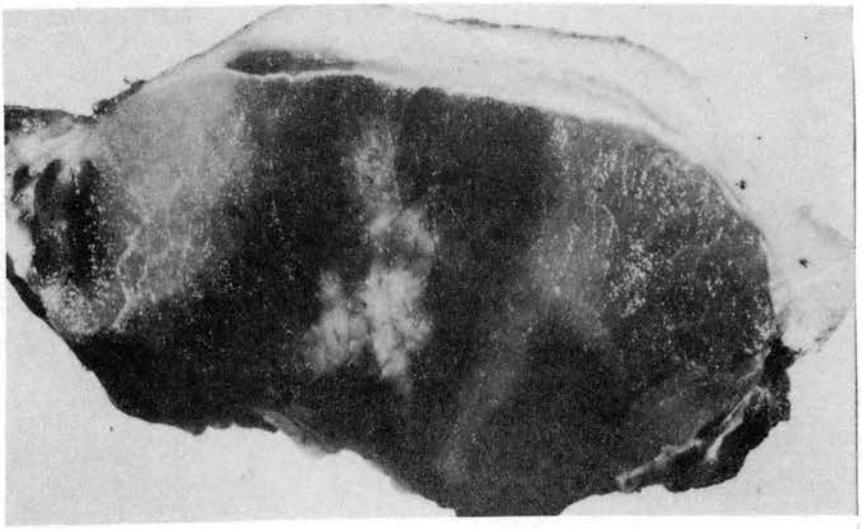

Figure 2. Transverse Section of the Longissimus Dorsi Muscle Showing the Lesion at a Slightly Later Stage of Development Than That of Figure 1

swollen appearance with more compact arrangement of muscle fiber bundles which were more prominently outlined by perimysial septae than in the surrounding unaffected muscle (Figure 2). The muscle tissue in areas immediately adjacent to the hemorrhagic and congested zone exhibited a compact appearance with loss of normal fibrilar structure had a slightly perceptible bluish tinging, was somewhat translucent in character, and had a slightly dry, waxy texture (Figure 2). Pronounced edema was a common feature in the intermuscular septae and fascial sheaths surrounding severely affected muscles.

Lesions which had been present for a much longer time were represented by well-defined pale areas having a compact, amorphous, structureless character, were light greenish-yellow in color and had a dry caseous cheese-like texture in the central area. The necrotic area was surrounded by a dense fibrous connective tissue envelope of varying thickness (Figure 3). Varying size whitish focal areas of calcification were scattered throughout the caseous central core of the lesion. Scattered streaks of fibrous connective tissue without caseous centers were observed in some muscles. These were interpreted to be the healed stage of prior lesions. Edema of intermuscular septae was observed occasionally in the central areas of some of these scarified muscles.

Other miscellaneous necropsy findings were inconstant and encountered in varying degrees among the affected animals. Excess serous fluid in the thoracic cavity and pericardial sac and mild to marked pulmonary edema and congestion was present in several animals. Moderate congestion of the liver, kidney, spleen, and adrenal cortex was a fairly consistent finding. The heart in a few cases was in systole at the time of necropsy, but no definite gross lesions were observed in the

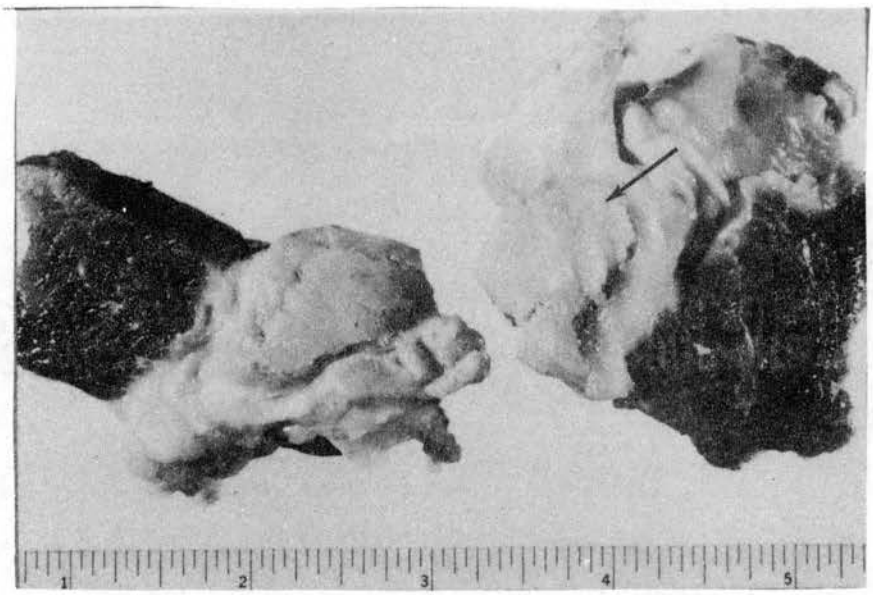

Figure 3. Transverse Section of Portions of the Longissimus Dorsi Muscle Showing an Old Lesion With Well Defined Central Necrotic Core (arrow) Surrounded by Fibrous Connective Tissue and Adjacent Unaffected Muscle Tissue

myocardium. Regional lymph nodes draining affected areas of the skeletal musculature were usually enlarged, markedly congested and edematous. Urine present in the urinary bladder was not visibly abnormal in color; however, one case among several which were checked in the laboratory showed a positive test for occult blood. All other organs and tissues appeared essentially normal.

Microscopic Lesions

Microscopic appearance of lesions in the skeletal musculature varied considerably with the stage of development of the lesion in different areas and in different muscle fibers in the same area. However, the general nature of the lesions was that of a rapidly progressing, marked degeneration and coagulation necrosis of individual muscle fibers.

The central areas of early lesions were markedly ischemic and, even in the earliest lesions observed, individual muscle fibers exhibited varying degrees of hyalinization and necrosis (Figures 4, 5, and 6). The majority of individual fibers were swollen; some were very markedly enlarged. In those fibers showing hyaline degeneration, the myofibrils tended to fuse into a homogeneous mass within the sarcolemma with indistinct to complete loss of cross striations. Muscle fibers in a slightly more advanced stage of degeneration showed beginning granular fragmentation of the hyalinized sarcoplasm. Further changes included progressive fragmentation and clumping of the sarcoplasm, accompanied by disruption of the sarcolemma. Most sarcolemmal nuclei were intact and in some areas they appeared to be more numerous as if undergoing proliferation. Peripheral areas of the lesion showed a diminishing

Figure 4. Histologic Section of a Muscle Lesion in an Early Stage of Development Showing Swollen Muscle Fibers With Varying Degrees of Hyalinization and Granular Degeneration. H&E. x 100

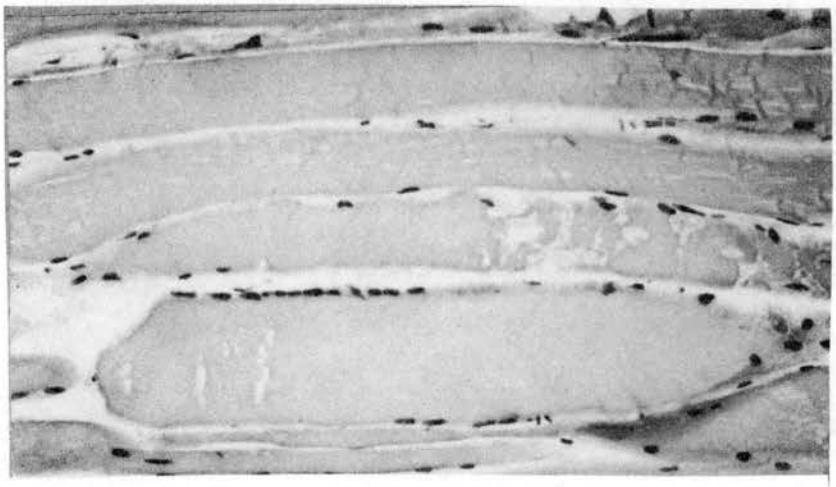

Figure 5. Higher Magnification of Muscle Fibers in Figure 4 Showing Increased Number of Sarcolemma Nuclei of a Markedly Swollen, Hyalinized Muscle Fiber. H&E. x 250

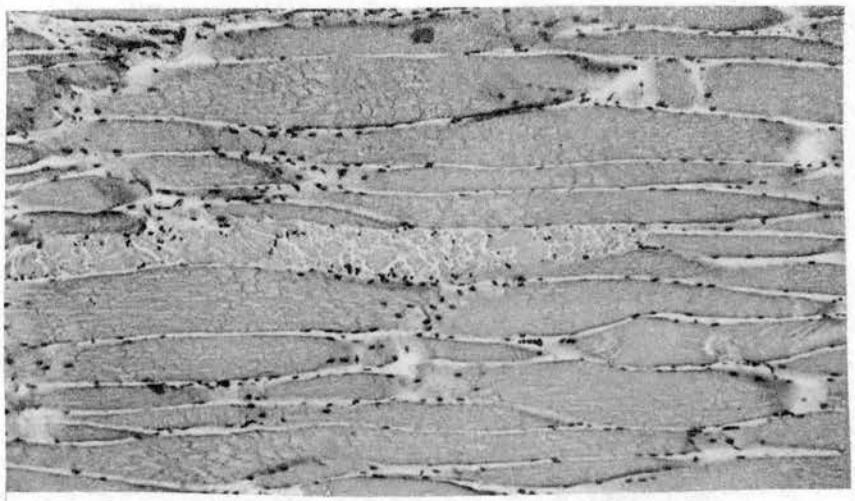

Figure 6. Histologic Section of Muscle Lesion in an Early Stage of Development Showing Necrotic Muscle Fiber With Fragmented Sarcoplasm Among Other Fibers Undergoing Hyalinization and Granular Degeneration. H&E. x 100

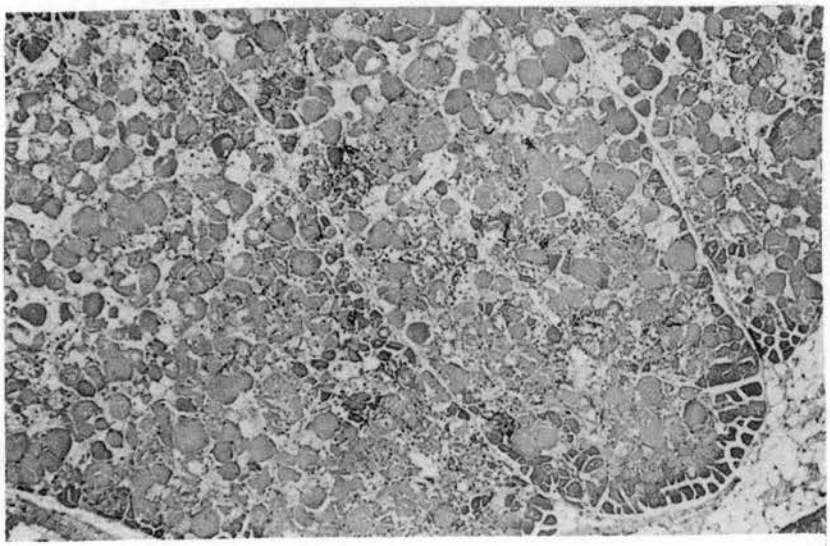

Figure 7. Histologic Section of the Central Area of a Muscle Lesion at a Slightly Later Stage of Development Than in Figures 4, 5, and 6 Showing Complete Necrosis of Muscle Fibers and Varying Degrees of Myolysis and Histiocytic Proliferation. H&E. x 40

number of affected fibers among normal or slightly swollen fibers and varying degrees of congestion and hemorrhage.

Central areas of the lesion at a later stage in development showed complete necrosis of all fibers, progressive myolysis of individual necrotic fibers, more marked proliferation of sarcolemmal nuclei, increase in histiocytic cells, and beginning proliferation of fibroblasts (Figure 7). Peripheral areas of the lesions at this stage showed more marked degeneration and necrosis of affected fibers, more pronounced hemorrhage, and more marked proliferation of histiocytic cells, lymphocytes and macrophages. Very few eosinophils were present in the peripheral inflammatory zone; however, neutrophils were fairly numerous in this area of some lesions.

Lesions which were of longer duration showed a well-defined central area of necrotic muscle tissue surrounded by a layer of newly forming fibrous connective tissue of varying thickness. Fibrous tissue projected into the peripheral area of the necrotic tissue accompanied by numerous histiocytic cells, macrophages, multinucleate giant cells, and varying numbers of lymphocytes (Figures 8, 9, and 10). Varying size foci of calcification were scattered throughout the necrotic muscle tissue in the central area of the lesion. Viable muscle fibers immediately adjacent to the outer edges of the lesion showed degrees of atrophy resulting from encroachment by fibrous connective tissue from the periphery of the lesion.

Microscopic lesions other than in skeletal muscle were minimal. Marked congestion was present in lymph nodes draining affected muscle areas. Miscellaneous findings in other organs corresponded to the gross observations of pulmonary congestion and edema, moderate congestion of

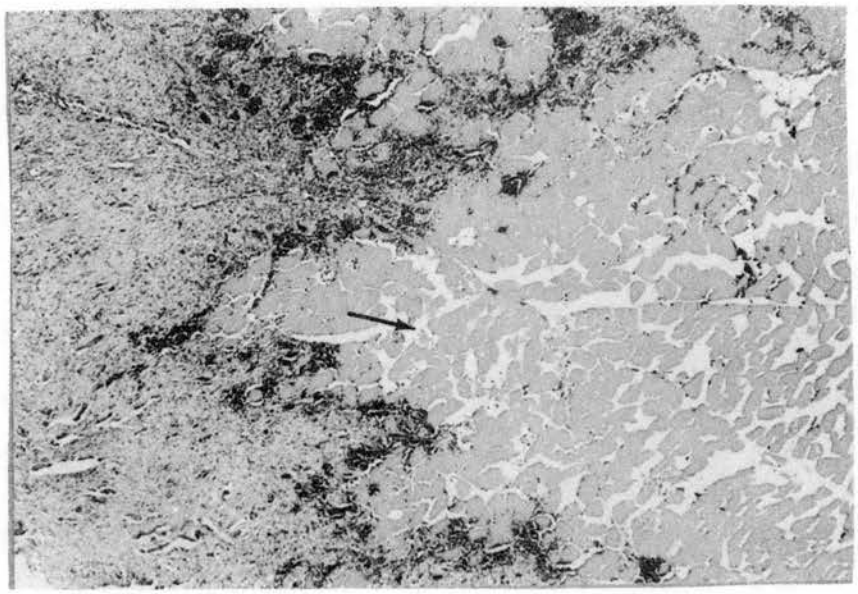

Figure 8. Histologic Section of an Older Muscle Lesion Showing Necrotic Center (arrow) Surrounded by Inflammatory Cells and Fibrous Connective Tissue. H&E x 40

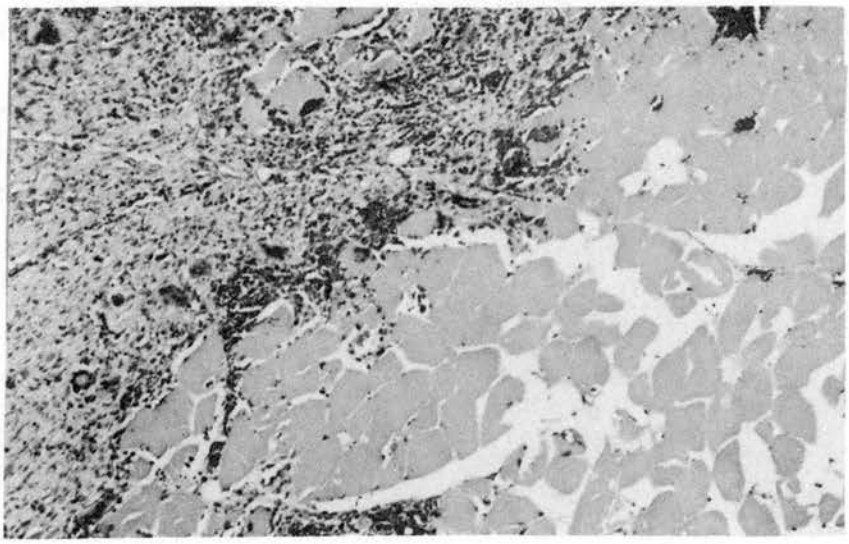

Figure 9. Higher Magnification of Figure 8 Showing Peripheral Invasion of Necrotic Muscle Tissue by Inflammatory Cells and Fibrous Connective Tissue. H&E x 100

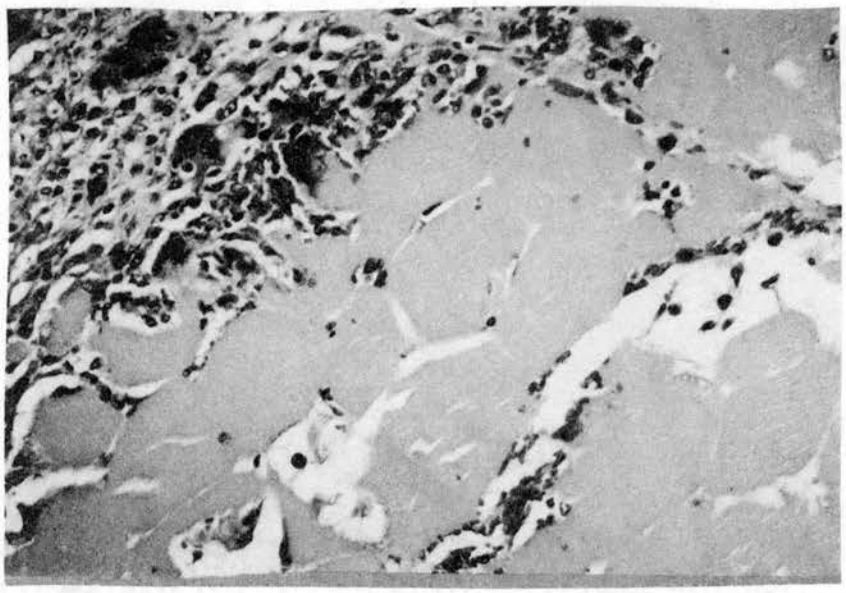

Figure 10. Higher Magnification of Figure 9 Showing Multinucleate Giant Cells, Macrophages, Lymphocytes, and Immature Fibroblasts Invading Peripheral Area of Necrotic Muscle Tissue. H&E x 250

the liver, kidneys and spleen and moderate to marked congestion of the adrenal cortex which was especially more pronounced in the zona reticularis. No significant lesions were observed in the thyroid gland or other tissues.

Results of Blood Serum Analysis

Phase I: Serum Values in Pigs With the Spontaneously Occurring Disease

The blood serum constituents measured in pigs with the spontaneously occurring disease included creatine phosphokinase (CPK), serum glutamic oxalacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), total lactic dehydrogenase (LDH), phosphorus, calcium, magnesium, chloride, sodium, and potassium. Results of the serum analyses in the seven animals with spontaneously occurring disease from which blood samples were obtained prior to death are presented in Table I.

The most marked changes in serum constituents observed in pigs with the spontaneously developed disease occurred in the enzymes with elevations in CPK values being the most outstanding. The degree of elevation in CPK values paralleled the degree and extent of myopathy observed postmortem. Pig No. 79-8, which had the highest CPK value, also had the most severe and extensive muscle lesions. The three pigs, Nos. 44-1, 91-3, and 2-4, which had the lower CPK values, had the mildest and least extensive muscle lesions. Serum SGOT values were mildly to markedly elevated in all pigs and, as with CPK, the degree of elevation tended to parallel the degree of severity of the muscle lesions. Serum LDH values were mildly to markedly elevated in five of the seven pigs

TABLE I

PHASE I: LEVELS OF VARIOUS BLOOD SERUM CONSTITUENTS IN PIGS WITH THE SPONTANEOUSLY OCCURRING DISEASE

Serum Constituent	Pig No.[1] 44-1	79-9	79-8	91-3	2-4	69-4	M-8917
CPK[2] Sigma units/ml	364	8,900	17,600	910	650	5,400	1,399
SGOT[3] Sigma-Frankel/ml	159	1,100	1,600	150	204	86	159
SGPT[3] Sigma-Frankel units/ml	34	128	118	34	36	56	32
LDH[4] Berger-Broida Units/ml	2,220	1,680	58,800	1,575	1,080	3,360	2,288
P mg%	13.6	12.1	24.7	12.0	10.4	7.6	.....
Ca mG%	9.8	9.5	9.4	7.4	10.2	7.6	.....
Mg m Eq/L	.....	2.6	3.3	2.4	3.4	2.1	.....
Cl m Eq/L	94.5	98.6	95	97	.....	100	.....
Na m Eq/L	144	140	133	141	141	140	.....
K m Eq/L	11.8	8.6	10.8	9.0	7.4	12.0	.....

[1]Pig Nos. 44-1, 79-9, 91-3, and 2-4 were slaughtered within 1½ hrs. after developing acute clinical signs of the disease. Pig No. 2-4 had only mild clinical signs at time of slaughter but had a history of an earlier attack of the disease approximately one month previously. Pig Nos. 79-8 and M-8917 both died of the disease; however, No. 79-8 had much more severe myopathy at necropsy. Pig No. 69-4 had unilateral, marked swelling of the loin muscles but survived.

[2]Sigma Units = mμ moles creatine phosphorylated/min.

[3]Sigma-Frankel Unit = 4.82×10^{-4} μ moles glutamate formed/min.

[4]Berger-Broida Unit = 4.8×10^{-4} μ moles pyruvate formed/min.

checked, the highest value being extremely elevated and occurred in the animal which had the most severe lesions; however, there was a lesser degree of correlation of LDH values with severity of lesions in the remaining animals than was observed with CPK and SGOT. Serum SGPT was significantly elevated in the two animals with the most severe lesions and was slightly elevated in the one animal which survived the disease but was normal in the remaining four pigs.

None of the non-enzyme serum constituents measured showed consistent elevations in all animals; although serum potassium values ranged from high normal to moderate elevation among all seven animals checked. Significantly high potassium values were present only in three animals, but the highest values did not occur in the animals with the most severe lesions. Serum phosphorus ranged from high normal to mildly elevated in all except the animal with the most severe muscle lesions, in whcih it was markedly elevated. Serum chloride was generally in the lower range of normal, but was not considered significantly low. Serum calcium values were normal in all except two animals with less severe lesions in which it was slightly below normal. Serum sodium and magnesium values were essentially normal in all animals.

Phase II: Studies of Serum Constituents in Rested Swine

Blood serum constituents measured in three blood samples collected successively at two week intervals from the three groups of swine used in this phase of the study were the same constituents as were measured in affected animals of Phase I. Results of the serum analyses in the three groups, totaling 35 animals, are summarized in Table II. Values

TABLE II

PHASE II: RANGES AND AVERAGE OF SERUM CONSTITUENT VALUES OBTAINED IN THREE BLOOD SAMPLES COLLECTED AT TWO-WEEK INTERVALS IN RESTED SWINE

Serum Constituent	Group I-Yorkshire		Group II-Hampshire		Group III-Mixed	
	Range	Avg.	Range	Avg.	Range	Avg.
CPK	0-670	93	0-660	84	0-545	73
SGOT	26-97	42	18-145	57	7-196	39
SGPT	13-40	26	13-48	29	14-42	23
LDH	615-2100	1044	210-1950	1126	390-2020	998
P	6.7-12.6	8.7	5.8014.0	9.2	7.8-12.1	9.8
Ca	8.6-12.6	10.3	7.1-13.0	10.2	8.6-11.6	9.5
Mg	1.3-3.9	2.1	1.3-3.4	2.4	1.4-2.9	2.2
Cl	89-108	101	93-108	101	98-113	102
Na	131-146	138	126-142	135	130-145	138
K	4.7-9.5	6.2	4.4-8.0	5.7	4.8-8.5	5.8

for each serum constituent in each sample from individual animals are given in Tables IX, X, and XI of the Appendix.

In general, there was considerable variation in values of each of the serum constituents in blood samples obtained at two-week intervals both in the same animal as well as in different animals of the same group. There were no significant differences in values for any of the serum constituents measured among the three groups of animals studied when compared on a group basis; however, Pig No. Y79-8, from the high incidence Yorkshire group, which later died of the disease had consistently higher CPK values than were obtained in other animals in each of the three groups.

Phase III: Levels of Serum Constituents Following Forced Exercise

The principal alterations in levels of serum constituents following a 10-minute forced exercise period in the two groups of swine used in Phase III of this study were associated with the CPK, LDH, and SGOT enzymes. Significant alterations in serum levels of these three enzymes occurred in certain pigs, predominantly in Subgroup I composed of Yorkshire swine from the high incidence herd, with only limited changes occurring in pigs of Subgroup II composed of Hampshire swine from the low incidence herd. Results of serum analyses for CPK, LDH, and SGOT in the two subgroups are graphically illustrated in Figures 11, 12, and 13. A tabulation of these results is presented in Table VI, VII, and VIII of the Appendix.

Elevations in CPK values after the exercise period were most dramatic in Pig No. Y79-8 of Subgroup I which also had the highest

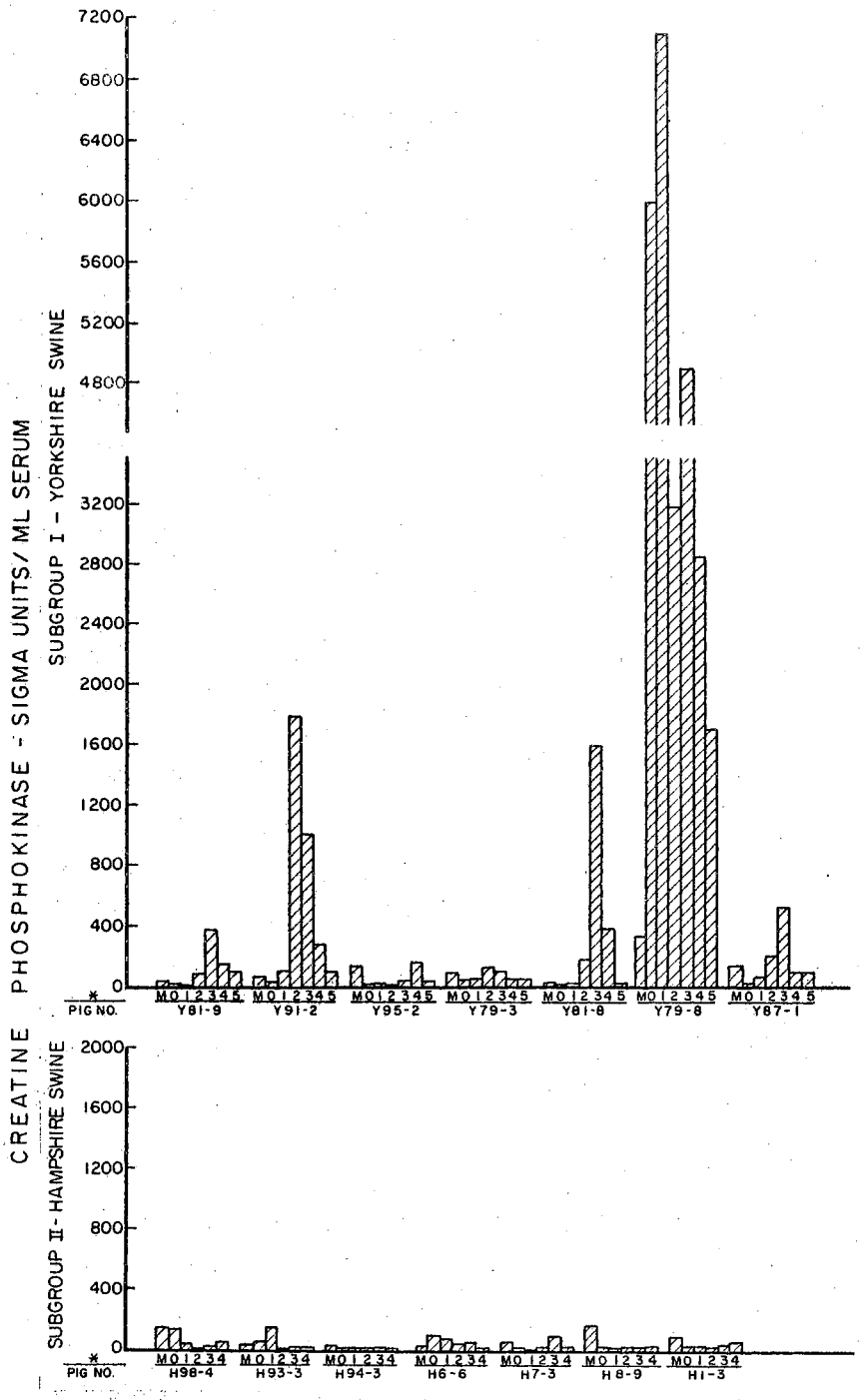

Figure 11. Serum Creatine Phosphokinase Levels in Subgroups I (Yorkshire) and II (Hampshire) Swine Prior to and at Intervals Following Forced Exercise

*M = Mean value from Phase II; 0 = immediately prior to exercise 1, 2, 3, 4, 5 = 2, 12, 24, 48, and 96 hrs., respectively, post exercise

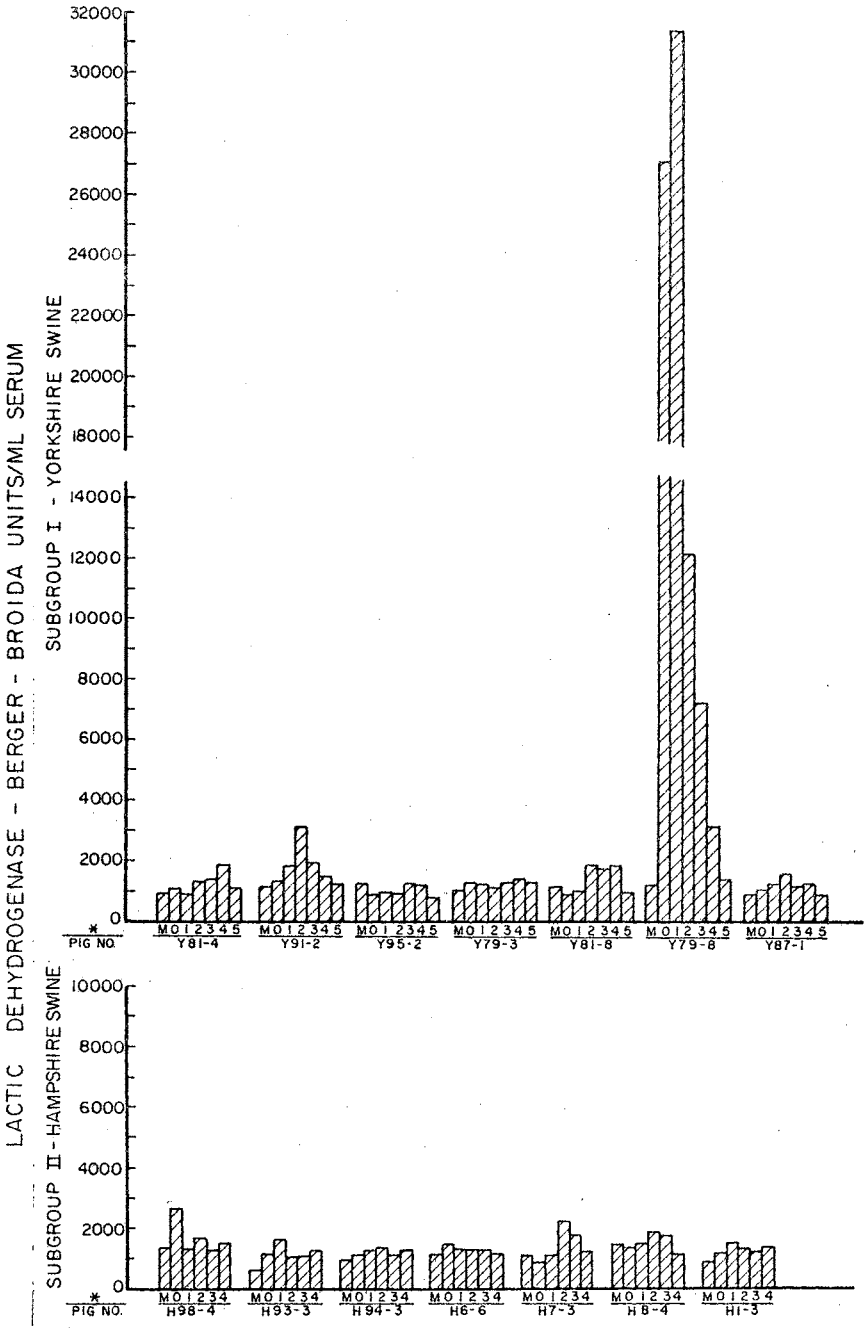

Figure 12. Serum Lactic Dehydrogenase Levels in Subgroups I (Yorkshire) and II (Hampshire) Swine Prior to and at Intervals Following Forced Exercise

*M = Mean value from Phase II. 0 = immediately prior to exercise 1, 2, 3, 4, 5 = 2, 12, 24, 48, and 96 hrs., respectively, post exercise

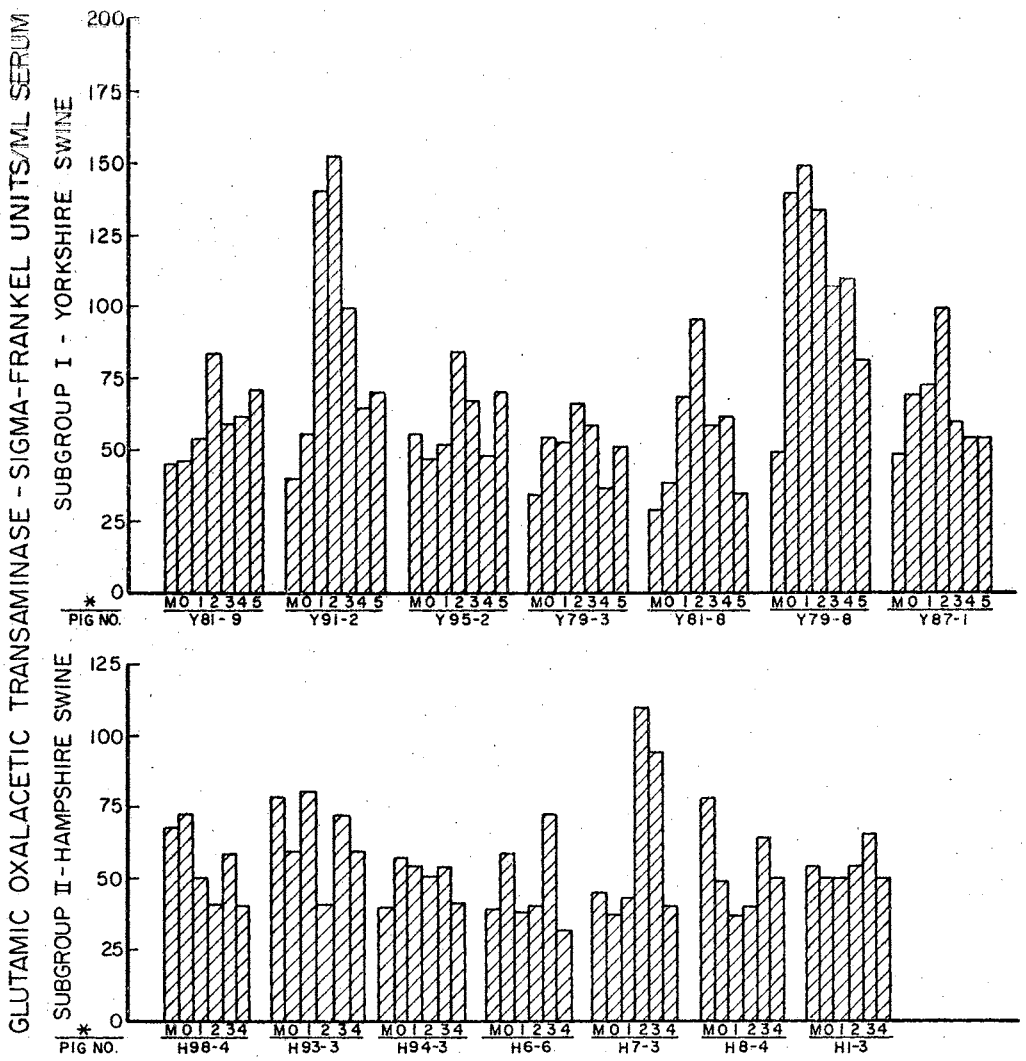

Figure 13. Serum Glutamic Oxalacetic Transaminase Levels in Subgroups I (Yorkshire) and II (Hampshire) Swine Prior to and at Intervals Following Forced Exercise

*M = Mean value from Phase II; 0 = immediately prior to exercise 1, 2, 3, 4, 5 = 2, 12, 24, 48, and 96 hrs., respectively, post exercise

average elevation in serum CPK values of all animals used in the Phase II experiment (Figure 11). This pig had a very high CPK value in the blood sample obtained immediately prior to exercise and was observed to have marked swelling of the loin muscle immediately after the exercise period. The high CPK value in this pig immediately prior to the forced exercise period, as compared to the values obtained in the Phase II experiment, suggested that this animal was probably already affected by the myodegeneration syndrome prior to exercise in the Phase III experiment. Support for this suggestion is provided in that the CPK value of 6,000 in this pig at the zero bleeding time was comparable to the intermediately high values observed in some of the clinically affected pigs studied in Phase I.

Moderately high elevations in serum CPK occurred in two additional animals of Subgroup I. One of these pigs, No. Y91-2, died unexpectedly of the disease one week later. The other pig, No. Y81-8, was inadvertently sold for market and was unavailable for further observation. Two additional pigs, No Y81-9 and Y87-1 in Subgroup I, showed mild elevations in serum CPK following exercise but did not develop clinical signs of the disease prior to elimination from the herd. Altogether, five of the seven pigs in Subgroup I developed elevations in serum CPK following the exercise period which were all above the pre-exercise levels in the same pig and well above the CPK levels after exercise in each of the seven pigs of Subgroup II. The two pigs of Subgroup I which later died of the disease, Nos. Y79-8 and Y91-2, showed peak elevations in serum CPK levels at the second hour and twelfth hour, respectively, following the exercise period, while the remaining three pigs, which showed only mild to moderate CPK elevations but which did

not develop the disease, showed peak elevations at 24 hours after exercise.

Postexercise elevations in serum LDH levels in pigs of Subgroup I were comparable to serum CPK elevations in that the two pigs, Nos. Y79-8 and Y91-2 which later died of the disease, showed elevations in LDH significantly above the pre-exercise levels (Figure 12). Both of these animals showed peak LDH levels at the comparable postexercise interval as their peak CPK levels. None of the remaining pigs in Subgroup I nor none of those in Subgroup II developed significant elevations in serum LDH during the postexercise period.

Each of the seven pigs in Subgroup I showed increasing postexercise levels of SGOT which peaked at 12 hours postexercise and then regressed; however, only two of the seven pigs in Subgroup I showed significant peak elevations in SGOT above that of the remaining five animals of the subgroup (Figure 13). The two pigs, Nos. Y78-9 and Y91-2 with elevated SGOT were the same pigs which showed the most marked postexercise elevations in serum CPK, were the only ones which showed elevations in serum LDH, and were the only ones which later died of the disease. Each of the seven Hampshire swine comprising Subgroup II showed postexercise fluctuations in SGOT levels but only one pig, No. H7-3, showed a pattern of increased SGOT levels which peaked at the 12-hour postexercise interval similar to that exhibited by all animals of Subgroup I.

Serum phosphorus and potassium were the only remaining serum constituents measured in the Phase III experiment which showed a consistent pattern of elevation in the postexercise period over the pre-exercise levels in pigs of either Subgroup I or II, although considerable random fluctuations occurred in other serum constituents

in individual animals among both subgroups. Results of analyses of all other serum constituents which included SGPT, phosphorus, calcium, magnesium, chloride, sodium, and potassium are presented in Tables IX through XV of the Appendix.

Serum phosphorus (Table X) and potassium (Table XV) both were increased during the postexercise period over the immediate pre-exercise levels in all seven of the pigs of Subgroup I. The peak levels of phosphorus occurred in one pig at 2 hours, the peak level of potassium in one pig at 12 hours, while all other pigs in the Subgroup showed peaks of both constituents. However, there was very little correlation between the peak serum potassium levels and the various serum enzymes which were elevated.

Although the serum phosphorus and potassium levels fluctuated considerably in some pigs of Subgroup II during the postexercise period, there was no consistent pattern of elevation among the group. The higher potassium values in individual samples from pigs of Subgroup II occurred in samples which were partially hemolyzed.

In addition to the foregoing serum constituents, 17-hydroxy corticosteriod assays were made on the majority of blood samples obtained from pigs utilized in Phase III.[1] Results of these assays were reported to be within normal range.

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[1]The 17-hydroxy corticosteroid assays were performed by Dr. V. K. Ganjam, Department of Physiology and Pharmacology, Oklahoma State University, whose work was supported by National Institutes of Health, National Institute of Child Health and Human Development, Research Grant No. HD-00636.

CHAPTER V

DISCUSSION AND CONCLUSIONS

The myodegeneration syndrome described in this study appears to be a separate and distinct myopathic disease entity from the majority of myopathic syndromes previously reported in swine. The "Spiernecrose bij Varkens" (muscle necrosis in pigs) syndrome described in slaughter pigs in Holland by Thoonen and Hoorens (1960) appears very similar, if not identical, to the myodegeneration syndrome described in the present study. Thoonen and Hoorens described the principal clinical signs, gross lesions, and microscopic appearance of the lesions observed in their cases which seem to be identical in practically all aspects to those observed in affected pigs reported in this study. Their report, however, did not mention the occurrence of lesions in muscle groups other than the longissimus dorsi and did not include serum analyses for biochemical alterations in affected pigs nor any reference to relationship of body conformation or possible genetic influence on predisposition to the disease.

Although genetic predisposition to the myodegeneration syndromes in swine seems to be unquestionable, the fundamental causes and interrelationship of possible causal factors remains to be determined. The myodegeneration syndrome described in the present study can be defined as an acutely developing and usually fatal myopathy which occurs more commonly in immature market age and young adult swine of superior

muscling. It is characterized clinically by acute respiratory distress, shock-like cardiovascular insufficiency, muscle tremor, swelling of loin musculature, and death usually within one to several hours after initial clinical signs with rapid development of postmortem rigor. The lesions are characterized by acutely developing muscle degeneration and necrosis confined principally to the central areas of the loin muscles and other large skeletal muscles.

On the basis of the frequent occurrence of swollen loin muscles in acutely affected pigs, which was observed to occur very rapidly in at least one pig immediately after exercise, together with the microscopic changes observed in early lesions, it appears that initially at least, the enlarged muscle results from marked continued contracture of the muscle in "cramp-like" fashion. This prolonged contracture undoubtedly causes marked ischemia and resultant hypoxia within the muscle which may be responsible for rapid death in the central areas of the muscle with rapid development of the histologic features of progressive degeneration and necrosis of muscle fibers. This explanation of the sequence of events provides a satisfactory and logical explanation for the rapid enlargement of the loin muscle as well as location of the degenerative lesion in the central area of the muscle. Since rigor develops rapidly in affected animals, it is reasonable to assume that it also develops rapidly in local muscle areas suddenly deprived of blood supply. Local rigor plus "postmortem" swelling of muscle fibers within the local lesion, subsequent congestion peripheral to the lesion, and edema of the perimuscular fascia would account for continued enlargement of the loin muscle until resolution and healing of the lesion and contracture of

scar tissue with associated atrophy of adjacent muscle fibers was well advanced.

Other myopathic syndromes previously reported in slaughter age swine and variously designated as "fatal syncope", "enzootic apoplexy", "Herztod", "Ludvigsen's total muscle degeneration", "Porcine Stress Syndrome", and "pale soft exudative pork", although similar to each other in certain respects, do not seem to be the same entity as the myodegeneration syndrome described in the present study. However, it may be that all of these conditions are related to some as yet undetermined fundamental defect or disturbance in muscle metabolism, circulatory deficiency, endocrine imbalance, neurogenic, or neurocirculatory defect, differing only in degree of the defect. Thus, manifestations could vary considerably in individual animals. Whatever the basic defect or multiplicity of defects which may be involved, there appears to be a genetic predisposition to development of the myopathic changes, since a greater incidence occurs among pigs in closely related blood lines bred for heavy muscle conformation and rapid maturity. There is no doubt that husbandry methods and unaccustomed exercise are precipitating factors in development of the clinical manifestations and degenerative changes in muscle which characterizes the various syndromes.

Various theories have been advanced regarding possible fundamental causes which may be involved in some of the myopathic conditions previously reported in slaughter age swine. Sybesma and Hart (1965) proposed that hypoxia of muscle tissue induced by unaccustomed stress of muscular exertion and transport tiring was a major factor in causing muscle degeneration in slaughter swine. Their experimental studies

related blood volume and heart weights to total muscle mass in three groups of pigs representing breeds of different body conformation. Results of their study showed increased degree of muscle degeneration in those pigs having the widest ratio of total muscle mass to blood volume and heart weight. Sybesma and Hart concluded from the results of their investigation that hypoxia of muscle was one of the etiologic factors of muscle degeneration in swine. They further stated that the less favorable balance between blood volume and muscle mass predisposed to the fatal shock-like clinical manifestations reported in pigs during or following severe exercise or transport stress.

Ludvigsen (1957) considered the conditions designated as "fatal syncope", "enzootic apoplexy", "Herztod", and "Ludvigsen's total muscle degeneration" to be different expressions of the same entity. Nieberle and Cohrs (1967) in their discussion of the etiology and pathogenesis of the disease consider it extremely probable that a circulatory deficiency resulting from a disturbance of internal secretion and hormone regulation is a major factor in fatal cases. They also suggested the possibility that a primary disturbance in the autonomic nervous system may be involved. Ludvigsen (1957) suggested that the condition is the result of an inherited imbalance between three hormone systems -- the hypophyseal-adrenocortical system, the hypophyseal-thryoid system, and a growth hormone -- in which growth hormone is increased, causing a depressant effect on the thyroid stimulating and adrenal corticotrophic hormones.

In addition to varying degrees of myodegeneration, Nieberle and Cohrs (1967) and Dunne (1964) stated that collapse of the thyroid

follicles is a characteristic histologic lesion observed in approximately 70 per cent of the fatal cases of Herztod. The presence of this lesion apparently is interpreted as supporting evidence of a relative hypothyroidism in affected pigs. The thyroid lesion, considered to be an almost pathognomonic feature in acute cases of Herztod, was not observed in any of the cases of the myodegeneration syndrome encountered in the present study, even though marked skeletal muscle lesions were present.

The exact mechanism by which the circulatory collapse and myodegeneration are brought about in Herztod is poorly understood, although it is clear that unaccustomed muscular exertion is the immediate precipitating cause or "triggering" factor.

Several authors indicate that nutrition is probably of considerable importance as a predisposing factor in Herztod; however, there is little agreement regarding the specific dietary factors that may be involved or the exact manner in which they influence predisposition to the disease (Nieberle and Cohrs, 1967; Innes and Saunders, 1962; Blaxter and McGill, 1955). Dietary factors which have been attributed a causal role, either individually or in combination, include excess of carbohydrate and deficiency of protein, vitamin A, vitamin B_1, tocopherol, and trace elements.

The general clinical signs of acute respiratory distress, ischemic and erythematous blotches of the skin, rapid death and rapid development of rigor mortis described by Topel et al. (1968) in pigs with the condition designated "Porcine Stress Syndrome" (PSS) are similar to those observed in pigs acutely affected by the myodegeneration syndrome reported in the present study. However, specific muscle lesions other

than varying degrees of paleness and edema of various muscle groups were not observed in pigs with PSS. In experimental studies, they noted a greater decrease in blood pH, increase in serum phosphorus, and increase in serum potassium in stress-prone versus control groups following a period of forced exercise. In a further report, Topel (1968) indicated that stress-prone pigs showed less tendency toward elevation of 17-hydroxycorticosteroids in plasma following exercise than did control pigs.

Certain of the clinical features and biochemical alterations in serum reported in PSS pigs are similar to those observed in pigs with the myodegeneration syndrome reported in the present study. However, the muscle changes reported by Topel et al. (1968) in PSS pigs bear no real similarity to the lesions characteristic of the myodegeneration syndrome. The postmortem changes designated as "pale, soft exudative pork" of some swine carcasses after slaughter (Lannek, 1967; Judge, 1968) appear to be similar to those described in pigs with PSS.

There is general agreement among investigators of the various myodegeneration syndromes, PSS and PSE, that predisposition to development of these myopathic conditions and postmortem muscle changes is genetically determined. The current trend among swine breeders is toward selection of breeding stock of heavier muscling and rapid maturity. Since there is a distinctly greater incidence of these conditions among this type of pig, it is probable that susceptibility to the trait is being increased by intense selection for these more economically desirable features.

On the basis of the biochemical parameters used during investigation of the myodegeneration syndrome described in the present study, it

appears that serum CPK, LDH, and SGOT are the best indicators not only in pigs with already existing muscle lesions, but also as indicators of the more susceptible pigs, particularly if the determinations are made within a few hours following muscular exertion. Although further work is indicated, these results suggest that serum analysis for these three enzymes before and after muscular stress may offer possibilities as a means of selecting the more susceptible pigs for elimination from the breeding herd.

The observations and results derived from this study shed little or no light on the basic causes of myodegeneration syndromes of swine. However, they do suggest further avenues of study which might lead to better understanding of the etiology and pathogenesis relative to biochemical alterations responsible for development of the lesions. Possible approaches to further study should include: 1) further evaluation of serum enzymes as indicators of susceptible pigs including studies of LDH isozymes; 2) investigations of blood glucose and other parameters concerning carbohydrate metabolism; 3) investigations regarding possible defects in the circulatory system in susceptible pigs including cardiac output, blood volume studies, and microcirculation of the skeletal musculature; 4) investigation of possible membrane defects of muscle as well as possible generalized membrane defects in susceptible animals, and 5) thorough investigation of the genetic relationships in high incidence blood lines.

CHAPTER VI

SUMMARY

The primary objective of the investigation was to characterize more fully the clinical features, gross and microscopic lesions, and biochemical alterations in the serum relative to a spontaneously occurring myodegeneration syndrome in swine. The study was divided into three phases as follows: 1) Case histories, clinical signs and lesions were studied in 25 affected Yorkshire swine. Biochemical alterations in serum were studied in seven of these animals. 2) Levels of various serum constituents were evaluated at successive intervals in 10 Yorkshire (high incidence group), 14 Hampshire (low incidence group), and 11 mixed-breed (control group) swine 3-4 months of age. 3) Alterations in serum constituents were evaluated in 7 Yorkshire (high incidence group) and 7 Hampshire (low incidence group) swine at successive intervals following forced exercise.

The observations and results derived from this study suggested that the myodegeneration syndrome is a separate and distinct myopathic disease entity from the majority of myopathic syndromes previously reported in swine. It appears to be very similar to the disease designated as "muscle necrosis in pigs" previously reported in Holland. Predisposition to the disease appears to be genetically determined.

The myodegeneration syndrome is an acutely developing and usually fatal myopathy which occurs more commonly in immature market age and

young adult swine. It is characterized clinically by acute respiratory distress, shock-like cardiovascular insufficiency, swelling of the loin musculature, death of most affected animals, and rapid development of rigor mortis. The lesions are characterized by acutely developing muscle degeneration and necrosis confined principally to the central areas of the loin muscles and other large skeletal muscles.

Serum CPK, LDH, and SGOT proved to be the best biochemical parameters for evaluating the severity of the disease in affected animals and as possible indicators of the more highly susceptible pigs. Serum phosphorus and potassium consistently became elevated in the susceptible Yorkshire pigs during the postexercise period.

Cause of the disease was undetermined; however, acute attacks are precipitated by unaccustomed muscular exertion. The underlying basic cause may be related to either a fundamental defect in muscle metabolism, circulatory deficiency, neurogenic or neurocirculatory defect, endocrine imbalance, or combinations of these.

SELECTED BIBLIOGRAPHY

Benjamin, Maxine M. Outline of Veterinary Clinical Pathology, 2nd ed. Ames: The Iowa State University Press, 1962.

Blaxter, K. L., and R. F. McGill. "Muscular Dystrophy". Vet. Rev. Annot., Vol. 1 (1955), 91-114.

Blincoe, Clifton, and D. W. Marble. "Blood Enzyme Interrelationships in White Muscle Disease." Am. J. Vet. Res., Vol. 21 (1960), 866-869.

Cooper, C. C., R. G. Cassens, and E. J. Briskey. "Capillary Distribution and Fiber Characteristics in Porcine Muscle." A Report to the 60th Annual Meeting of the American Society of Animal Science, July 28-August 1, 1968, Oklahoma State University, Stillwater, Oklahoma.

Dunne, Howard W., and David C. Kradel. "Acute Circulatory Failure and Hepatosis Diaetetica". In Diseases of Swine, 2nd ed. Ed. Howard W. Dunne. Ames: Iowa State University Press, 1964, pp. 715-720.

Eikmeier, Von H. "Muskeldegeneration, Muskeldystrophie, Weibfleishigkeit des Schweines". Deutsche Tierarztliche Wochenschrift, Vol. 24 (1964), 675-677.

Farrell, Philip M., Edward L. Eyerman, and Louis L. Tureen. "Comparison of Plasma Creatinine Phosphokinase Changes in Nutritional and Genetic Muscular Dystrophy in the Chicken." Annals of the New York Academy of Science, Vol. 138, Art. 1 (September 9, 1966), 102-111.

Frankel, H. M., J. P. Ellis, Jr., and S. M. Cain. "Development of Tissue Hypoxia During Progressive Hyperthermia in Dogs." Amer. J. Physiol., Vol. 205 (1963), 733. (Quoted by Judge, 1968).

Ganjam, V. K. Personal Communication. 1968.

Goldenberg, Harry, and Alberto Fernandez. "Simplified Method for the Estimation of Inorganic Phosphorus in Body Fluids." Clinical Chemistry, Vol. 12 (1966), 871-884.

Grant, C. A. "Dietetic Microangeopathy in Pigs". Acts vet. Scand., Suppl. 3 (1966), 2-5. (Quoted by Dunne, 1964).

Henson, J. B., and R. R. Rao. "A Comparison of Serum Creatine Phosphokinase and Serum Glutamic Oxalacetic Transaminase in Skeletal Muscle Necrosis." Canadian Journal of Comparative Medicine, Vol. 30 (1966), 157-160.

Hess, Joseph W., Roderick P. MacDonald, Richard J. Frederick, Robert N. Jones, Joann Neely, and David Gross. "Serum Creatine Phosphokinase (CPK) Activity in Disorders of Heart and Skeletal Muscle." Annals of Internal Medicine, Vol. 61 (December, 1964), 1015-1027.

Hokanson, J. F., and A. J. Luedke. "Techniques of Venipuncture." In Diseases of Swine, 2nd ed. Ed. Howard W. Dunn. Ames: Iowa State University Press, 1964, pp. 786-790.

Innes, J. R. M., and L. Z. Saunders. Comparative Neuropathology. New York and London: Academic Press, 1962, 171-172.

Judge, M. D. "Environmental Stress and Meat Quality." A Report to the 60th Annual Meeting of the American Society of Animal Science, July 28-August 1, 1968, Oklahoma State University, Stillwater, Oklahoma.

Judge, M. D., and M. Stob. "Stress During Growth. I. Effects on physiology, carcass composition and carcass quality of lambs." J. Animal Sci., Vol. 22 (1963), 1059.

Lanneck, N. "Muscular Dystrophy in Pigs, Etiological Aspects." 17th World Veterinary Congress Proceedings, Vol. 2 (1963), 1263-1267.

Ludvigsen, J. "On the Hormonal Regulation of Vasomotor Reactions During Exercise With Special Reference to the Action of Adrenal Cortical Steriods." Acts Endocrinol, Vol. 26 (1957), 406. (Quoted by Nieberle and Cohrs, 1967).

Mattenheimer, Herman. The Theory of Enzyme Tests. New York: The Boehringer Mannheim Corporation, 1968, 5.

Meltzer, Herbert. "Creatine Kinase and Aldolase in Serum: Abnormality Common to Acute Psychoses," Science, Vol. 159 (22 January 1968), 1368-1370.

Milhorat, A. T., S. A. Shafig, L. Goldstone. "Changes In Muscles Structure in Dystrophic Patients, Carriers and Normal Siblings Seen By Electron Microscopy; Correlation with Levels of Serum Creatine Phosphokinase (CPK)." Annals of the New York Academy of Science, Vol. 138, Art. 1 (September 9, 1966), 246-291.

Natelson, Samuel. Microtechniques of Clinical Chemistry, 2nd ed. Springfield: Charles C. Thomas, 1963, 292-297.

Nieberle and Cohrs. Textbook of the Special Pathological Anatomy of Domestic Animals. Revised by Paul Cohrs, translated by R. Crawford. London, Oxford, Edinburgh, New York, Toronto, Paris, Braunschweig: Pergamon Press, 1967, pp. 19-21.

Sodeman, William A., ed. Pathologic Physiology: Mechanisms of Disease, 3rd ed. Philadelphia, London: W. B. Saunders Company, 1961, 1076-1077.

Sybesma, W. "Present Position of Enterse Disease (Muscular Degeneration of Pigs); Practical Possibilities of Reducing Losses." Tydschr. Diergineesk., Vol. 88, afl. 17, 1963.

Sybesma, W., and P. C. Hart. "Bloeduolume en Vleesdegeneratie bij varkens." Tijdschr. Dierganeesk., Vol. 90 (1965), 1116-1121.

Thoonen, J., and J. Hoorens. "Spiernecrose Bij Varkens." Vlaams diergeneesk., Vol. 29 (1960), 205-209.

Topel, David E. "Observation of the Porcine Stress Syndrome." A Report to the 70th Annual Meeting of the American Society of Animal Science, July 28 and August 1, 1968. Oklahoma State University, Stillwater, Oklahoma.

Topel, D. G., E. J. Bickwell, K. W. Preston, L. L. Christian, and C. Y. Matsushima. "Porcine Stress Syndrome." Modern Veterinary Practice (May, 1968), 40-60.

APPENDIX

TABLE III

PHASE II: LEVELS OF BLOOD SERUM CONSTITUENTS IN RESTED YORKSHIRE SWINE IN THREE SAMPLINGS AT TWO-WEEK INTERVALS

Serum Constituent	Sample No. and Avg.	Pig No. Y81-9	Y87-1	Y78-3	Y95-2	Y79-3	Y82-1	Y91-2	Y79-8	Y89-5	Y86-2	Y81-8	Total Average
CPK Sigma units /ml	1	24	326	51	53	151	74	....	670	101	....	0	
	2	85	59	44	372	56	131	57	180	0	20	49	
	3	20	32	38	21	47	20	78	160	20	21	29	
	Avg.	43	139	44	149	85	75	68	337	40	21	26	93
SGOT Sigma-Frankel units/ml	1	34	46	41	58	36	70	....	16	39	....	27	
	2	30	38	44	76	31	37	29	45	16	26	44	
	3	74	64	24	35	34	97	50	86	23	21	16	
	Avg.	46	49	36	56	34	68	40	49	26	24	29	42
SGPT Sigma-Frankel units/ml	1	20	27	27	29	33	27	....	33	27	....	29	
	2	20	25	16	30	26	24	26	31	13	27	33	
	3	40	21	21	24	24	39	30	33	26	14	18	
	Avg.	27	24	21	28	28	30	28	32	22	21	27	26
LDH Berger-Broida units/ml	1	615	990	910	795	980	880	....	1060	735	....	585	
	2	1020	865	1050	1740	1015	1345	1015	1265	960	1085	2100	
	3	1090	895	930	1125	1045	1020	1200	1230	1000	960	825	
	Avg.	908	917	963	1220	1013	1082	1108	1185	898	1023	1170	1044

Table III (Continued)

Serum Constituent	Sample No. and Avg.	Pig No. Y81-9	Y87-1	Y78-3	Y95-2	Y79-3	Y82-1	Y91-2	Y79-8	Y89-5	Y86-2	Y81-8	Total Average
P mg%	1	9.4	9.6	12.6	9.3	9.9	9.5	....	10.7	9.3	....	8.3	
	2	8.9	8.5	8.1	10.8	9.2	7.9	9.0	8.4	8.1	7.0	7.5	
	3	9.4	8.3	8.6	9.8	8.1	8.3	8.4	6.9	9.1	6.7	7.4	
	Avg.	9.2	8.8	9.7	9.9	9.0	8.5	8.7	8.7	8.8	6.8	7.7	8.7
Ca mg%	1	12.2	11.4	12.6	11.6	12.0	11.1	....	11.6	11.1	....	10.9	
	2	10.4	8.5	8.6	9.5	10.5	9.2	8.6	10.0	10.0	10.2	11.0	
	3	10.8	10.3	9.7	10.2	10.8	9.6	10.3	9.5	10.8	10.0	9.6	
	Avg	11.1	10.0	10.3	10.4	11.1	9.9	9.5	10.4	1.1	10.1	10.5	10.3
Mg m Eq/L	1	2.22	1.81	3.90	2.26	2.56	2.02	....	2.39	1.72	....	2.10	
	2	2.04	1.78	2.17	2.24	2.04	2.43	1.98	2.08	2.14	1.61	2.20	
	3	1.86	2.21	1.86	1.81	3.08	1.86	1.89	2.50	1.28	1.28	1.65	
	Avg.	2.04	1.93	2.64	2.10	2.56	2.10	1.94	2.32	1.71	1.45	1.98	2.07
Cl m Eq/L	1	102	104	104	99	98	99	....	104	94	....	95	
	2	101	99	103	89	102	103	98	103	97	99	103	
	3	102	104	105	106	102	108	102	104	104	100	106	
	Avg.	102	102	104	98	101	103	100	104	98	100	101	101
Na m Eq/L	1	144	144	145	143	141	142	....	141	140	....	142	
	2	131	141	141	134	139	139	137	146	137	141	131	
	3	134	139	136	136	132	138	138	134	137	134	134	
	Avg.	136	141	141	138	137	140	138	140	138	138	136	138
K m Eq/L	1	6.9	7.0	8.0	5.9	7.8	6.2	....	7.8	4.2	....	5.2	
	2	5.7	5.0	6.0	8.0	6.9	6.0	7.0	9.5	5.3	6.0	6.4	
	3	6.5	5.8	5.9	5.8	5.5	6.9	6.2	4.9	6.1	4.6	4.7	
	Avg.	6.4	5.9	6.6	6.6	6.7	6.4	6.6	7.4	5.2	5.3	5.4	6.2

TABLE IV

PHASE II: LEVELS OF BLOOD SERUM CONSTITUENTS IN RESTED HAMPSHIRE SWINE IN THREE SAMPLINGS AT TWO-WEEK INTERVALS

Serum Constituent	Sample No. and Avg.	Pig No. H98-4	H93-3	H3-5	H94-3	H1-3	H8-4	H94-4	H1-6	H96-5	H7-3	H99-4	H6-6	H95-8	H96-4	Total Average
CPK Sigma units /ml	1	174	24	84	...	144	....	73	660	63	104	62	28	68	28	
	2	214	56	49	31	101	284	95	14	16	30	214	0	159	101	
	3	78	37	96	35	17	41	39	24	34	31	22	74	27	15	
	Avg.	155	39	76	33	87	163	70	233	38	55	99	34	85	48	84
SGOT Sigma-Frankel units/ml	1	82	67	80	...	63	....	58	145	84	68	38	51	64	64	
	2	36	54	32	24	58	86	58	25	40	32	36	44	64	48	
	3	84	112	108	53	41	71	41	58	38	34	48	18	40	34	
	Avg.	67	78	73	39	54	79	52	76	54	45	41	38	56	49	57
SGPT Sigma-Frankel units/ml	1	27	24	32	...	20	....	24	44	34	48	25	47	44	31	
	2	24	28	26	13	27	25	24	26	24	24	24	24	26	25	
	3	34	48	41	22	30	30	40	24	41	21	18	21	36	24	
	Avg.	28	33	33	17	26	27	29	31	33	31	22	31	35	27	29
LDH Berger-Broida units/ml	1	1410	210	1030	...	270	....	555	1860	1110	1020	885	825	900	345	
	2	1095	630	1095	960	1065	1680	1395	1000	1440	1120	1095	1100	1590	820	
	3	1665	960	1950	960	1170	1230	1680	1175	1980	1045	1020	1465	1290	790	
	Avg.	1390	600	1358	960	835	1455	1210	1345	1510	1062	1000	1130	1260	652	1126
P mg%	1	5.8	9.7	14.0	...	10.6	....	11.5	11.5	10.3	11.7	10.1	6.6	11.2	11.7	
	2	8.4	8.7	9.6	8.1	9.2	8.6	7.2	8.8	9.5	8.8	8.4	9.1	9.3	7.8	
	3	8.7	8.7	9.5	9.4	8.4	9.4	8.5	10.8	8.2	8.6	8.0	8.5	8.9	7.5	
	Avg.	7.6	9.0	11.0	8.8	9.4	9.0	9.0	10.4	9.3	9.7	8.8	8.0	9.8	9.0	9.2

Table IV (Continued)

Serum Constituent	Sample No. and Avg.	Pig No. H98-4	H93-3	H3-5	H94-3	H1-3	H8-4	H94-4	H1-6	H96-5	H7-3	H99-4	H6-6	H95-8	H96-4	Total Average
Ca mg%	1	11.2	12.4	12.2	....	11.6	....	11.1	11.1	12.5	13.0	12.3	11.9	11.7	13.0	
	2	9.5	9.8	7.1	7.5	9.0	10.2	8.3	9.1	11.0	9.7	9.5	9.6	9.0	9.9	
	3	9.7	10.1	9.9	9.0	9.7	10.3	10.0	11.0	9.9	9.7	8.8	8.9	9.6	10.4	
	Avg.	10.1	10.8	9.7	8.3	10.1	10.3	9.8	10.4	11.1	10.8	10.2	10.1	10.1	11.1	10.2
Mg m Eq/L	1	2.14	2.31	3.02	....	2.39	....	2.06	3.11	2.81	2.98	3.36	3.06	3.36	2.98	
	2	2.27	2.43	2.40	2.17	2.08	2.27	2.69	2.04	2.08	2.43	2.27	2.08	2.27	2.27	
	3	1.35	2.11	2.16	2.41	3.36	1.96	2.11	2.11	2.62	1.38	2.62	3.19	2.21	2.04	
	Avg.	1.92	2.28	2.53	2.29	2.61	2.12	2.29	2.42	2.50	2.26	2.75	2.78	2.61	2.43	2.41
Cl m Eq/L	1	93	98	99	....	100	....	100	102	102	99	97	95	102	99	
	2	97	101	102	100	98	101	104	104	95	99	97	96	98	102	
	3	108	106	104	106	106	102	104	102	102	106	106	108	104	106	
	Avg.	99	102	102	103	101	101	103	103	100	101	100	100	101	102	101
Na m Eq/L	1	132	138	138	....	140	....	135	135	142	136	141	137	141	136	
	2	136	133	126	131	130	134	137	137	133	138	136	138	134	138	
	3	138	134	137	132	133	137	133	136	132	132	136	130	134	133	
	Avg.	135	135	134	132	134	136	135	136	136	135	138	135	136	136	135
K m Eq/L	1	5.2	6.3	6.2	....	5.7	....	5.4	7.8	5.9	6.0	6.8	5.8	8.2	6.0	
	2	6.2	5.7	6.0	7.0	5.9	5.5	5.1	5.8	6.1	5.1	6.2	6.0	8.0	5.2	
	3	6.2	5.8	6.6	4.8	4.8	4.7	5.8	4.6	4.4	5.0	4.7	5.0	4.5	4.4	
	Avg.	5.8	5.9	6.2	5.9	5.4	5.1	5.4	6.0	5.5	5.4	5.9	5.6	6.9	5.2	5.7

TABLE V

PHASE II: LEVELS OF BLOOD SERUM CONSTITUENTS IN RESTED MIXED-BREED SWINE IN THREE SAMPLINGS AT TWO-WEEK INTERVALS

Serum Constituent	Sample No. and Avg.	Pig No. 1	2	3	4	5	6	7	8	9	10	11	Total Average
CPK Sigma units /ml	1	33	97	14	31	77	33	190	106	36	545	490	
	2	32	52	63	28	52	43	73	54	92	24	48	
	3	7	0	54	10	14	13	36	15	20	4	18	
	Avg.	24	50	44	23	48	30	100	58	49	191	185	73
SGOT Sigma-Frankel units/ml	1	40	24	32	46	62	41	90	37	47	196	59	
	2	43	38	35	34	48	38	36	48	25	22	22	
	3	38	20	26	26	18	10	7	19	30	7	19	
	Avg.	40	27	31	35	43	30	44	35	34	75	33	39
SGPT Sigma-Frankel units/ml	1	39	15	20	27	27	26	37	20	27	42	22	
	2	14	15	17	17	25	19	20	17	17	18	16	
	3	24	20	26	27	26	24	21	23	21	24	18	
	Avg.	26	17	21	24	26	23	26	20	22	28	19	23
LDH Berger-Broida units/ml	1	820	795	780	990	930	890	1470	1230	390	1040	735	
	2	1380	1090	1020	1100	1250	1120	1260	2020	1120	940	1200	
	3	1020	900	855	840	810	870	915	870	810	645	825	
	Avg.	1073	928	885	977	997	960	1215	1373	773	875	735	998
P mg%	1	10.4	8.5	12.1	11.1	9.4	9.4	10.4	9.9	8.6	11.5	10.4	
	2	10.1	10.3	11.5	8.4	8.3	9.5	9.3	9.1	10.0	9.1	7.8	
	3	9.3	9.3	9.5	9.7	8.9	9.5	10.0	12.1	11.5	11.3	11.0	
	Avg.	9.9	9.4	11.0	9.7	8.8	9.5	9.9	10.4	10.0	10.6	9.1	9.8

Table V (Continued)

Serum Constituent	Sample No. and Avg.	Pig No. 1	2	3	4	5	6	7	8	9	10	11	Total Average
Ca mg%	1	8.6	....	9.1	9.3	9.7	11.2	8.2	8.4	11.6	9.0	9.3	
	2	10.3	10.5	9.9	9.3	9.0	9.3	9.3	9.4	11.0	9.3	9.3	
	3	9.4	10.3	9.9	9.3	10.3	8.6	9.0	8.6	9.5	10.0	8.8	
	Avg.	9.4	10.4	9.6	9.3	9.7	9.7	8.8	8.8	10.7	9.4	9.1	9.5
Mg m Eq/L	1	2.60	1.93	2.78	2.92	2.18	2.36	2.60	2.32	2.14	2.43	2.57	
	2	2.30	1.58	2.08	2.20	2.08	1.40	2.17	1.55	2.17	2.17	2.11	
	3	1.96	1.73	2.07	1.90	2.35	2.18	1.69	1.83	1.76	2.07	1.86	
	Avg.	2.29	1.75	2.31	2.34	2.20	1.98	2.15	1.90	2.02	2.22	2.18	2.15
Cl m Eq/L	1	....	....	....	112	94	113	101	....	....	102	103	
	2	104	100	104	104	100	100	102	100	102	99	100	
	3	107	104	100	102	98	100	104	100	98	98	98	
	Avg.	106	102	102	106	97	104	102	100	100	99	100	102
Na m Eq/L	1	133	....	....	144	134	145	135	137	140	130	139	
	2	139	138	140	138	134	138	137	137	135	133	135	
	3	145	139	141	143	139	139	140	137	139	140	137	
	Avg.	139	139	141	141	136	140	137	137	138	134	137	138
K m Eq/L	1	5.0	....	....	6.3	5.8	7.5	5.9	5.5	5.0	8.5	6.0	
	2	5.6	5.6	7.0	7.3	5.8	6.9	6.4	5.9	5.2	5.0	5.8	
	3	4.8	5.5	7.0	5.6	5.8	4.9	4.9	5.1	4.9	5.9	5.3	
	Avg.	5.1	5.5	7.0	6.4	5.8	6.4	5.7	5.5	5.0	6.5	5.7	5.8

TABLE VI

PHASE: III: SERUM CREATINE PHOSPHOKINASE LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUP OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr		2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroups I-Yorkshires (High Incidence)	Y81-9	10	10 Minute Exercise Period	14	85	380	145	99	43
	Y91-2	33		104	1800	1000	280	89	68
	Y95-2	15		22	17	39	170	38	149
	Y79-3	40		57	127	92	58	48	85
	Y81-8	11		19	180	1600	390	31	26
	Y79-8	6000		7100	3180	4900	2850	1700	337
	Y87-1	15		71	310	530	93	91	139
Subgroup II-Hampshires (Low Incidence)	H98-4	150		41	10	24	47	....	155
	H93-3	49		145	13	16	25	....	39
	H94-3	13		16	9	17	8	....	33
	H6-6	106		75	40	44	9	....	34
	H7-3	8		13	28	86	24	....	55
	H8-4	21		16	24	23	33	....	162
	H1-3	23		29	19	35	51	....	87

Footnotes apply to Tables VI through XV.

[a]Blood sample obtained immediately prior to exercise.

[b]Average from three successive blood samples, Phase II.

TABLE VII

PHASE III: SERUM LACTIC DEHYDROGENASE LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period	2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	1,050		930	1,365	1,395	1,830	1,065	908
	Y91-2	1,395		1,800	3,100	1,960	1,470	1,215	1,108
	Y95-2	875		945	975	1,215	1,170	780	1,220
	Y79-3	1,240		1,240	1,090	1,260	1,380	1,260	1,013
	Y81-8	875		990	1,860	1,740	1,860	945	1,170
	Y79-8	27,060		31,350	12,700	7,200	3,150	1,470	1,185
	Y87-1	1,175		1,230	1,560	1,170	1,275	840	917
Subgroup II-Hampshire (Low Incidence)	H98-4	2,620		1,360	1,660	1,320	1,490	...	1,390
	H93-3	1,170		1,620	1,080	1,080	1,240	...	600
	H94-3	1,125		1,250	1,380	1,110	1,270	...	960
	H 6-6	1,440		1,350	1,320	1,350	1,140	...	1,130
	H 7-3	795		1,110	2,280	1,770	1,215	...	1,062
	H 8-4	1,365		1,470	1,850	1,785	1,080	...	1,455
	H 1-3	1,155		1,500	1,305	1,200	1,380	...	835

TABLE VIII

PHASE III: SERUM GLUTAMIC OXALACETIC TRANSAMINASE LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period	2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	47		54	84	58	62	71	46
	Y91-2	56		140	152	99	64	70	40
	Y95-2	47		52	84	67	48	71	56
	Y79-3	54		52	67	58	37	52	34
	Y81-8	38		64	96	58	62	35	29
	Y79-8	140		150	134	113	110	82	49
	Y87-1	70		73	100	60	54	54	49
Subgroups II-Hampshire (Low Incidence)	H98-4	72		50	42	58	40	..	67
	H93-3	59		76	41	72	58	..	78
	H94-3	57		54	50	54	41	..	39
	H 6-6	58		37	40	72	32	..	38
	H 7-3	37		43	110	94	41	..	45
	H 8-4	48		37	40	64	50	..	79
	H 1-3	50		50	54	66	50	..	54

TABLE IX

PHASE III: SERUM GLUTAMIC PYRUVIC TRANSAMINASE LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period	2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	16		16	24	33	25	50	27
	Y91-2	26		50	49	49	33	35	28
	Y95-2	12		38	20	26	24	35	28
	Y79-3	26		38	34	42	31	38	28
	Y81-8	12		31	34	38	26	18	27
	Y79-8	38		31	34	42	34	45	32
	Y87-1	26		24	27	31	30	35	24
Subgroup II-Hampshire (Low Incidence)	H98-4	64		38	39	39	32	..	28
	H93-3	43		48	42	46	32	..	33
	H94-3	36		42	38	44	35	..	18
	H 6-6	10		35	35	48	31	..	31
	H 7-3	24		30	42	46	26	..	31
	H 8-4	30		26	26	34	30	..	28
	H 1-3	30		32	38	38	36	..	26

TABLE X

PHASE III: SERUM PHOSPHORUS LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period					Avg. Values Phase II
			2 hr	12 hr	24 hr	48 hr	96 hr	
Subgroup I-Yorkshire (High Incidence)	Y81-9	7.1	7.1	9.2	6.9	7.7	8.5	9.2
	Y91-2	7.8	11.4	8.4	8.2	7.3	8.5	8.7
	Y95-2	7.6	7.2	8.0	8.3	8.5	8.3	9.9
	Y79-3	8.2	7.6	7.6	7.1	8.0	9.0	9.0
	Y81-8	6.8	7.6	7.3	6.6	6.8	9.8	7.7
	Y79-8	7.2	8.5	8.7	7.4	7.1	8.9	8.7
	Y87-1	8.0	6.2	8.3	6.9	8.8	8.5	8.8
Subgroup II-Hampshire (Low Incidence)	H98-4	9.4	8.1	9.1	8.5	8.7	...	7.6
	H93-3	8.1	8.6	8.3	7.8	8.3	...	9.0
	H94-3	8.1	8.5	8.2	8.7	8.5	...	8.8
	H 6-6	8.5	8.4	9.0	9.0	8.5	...	8.0
	H 7-3	8.8	8.5	11.4	9.5	9.3	...	9.7
	H 8-4	8.5	8.5	9.2	9.6	8.8	...	9.0
	H 1-3	8.4	9.0	9.3	8.8	9.1	...	9.4

TABLE XI

PHASE III: SERUM CALCIUM LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr		2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	7.8	10 Minute Exercise Period	8.2	8.3	7.4	7.0	7.9	11.1
	Y91-2	7.9		9.3	7.4	7.5	8.3	7.8	9.5
	Y95-2	7.5		7.8	6.8	7.3	7.2	7.5	10.4
	Y79-3	7.8		7.9	7.0	6.5	7.5	8.5	11.1
	Y81-8	7.8		9.8	7.5	7.2	7.7	7.0	10.5
	Y79-8	6.5		7.8	7.4	7.2	7.4	7.0	10.4
	Y87-1	8.3		7.5	7.0	7.8	7.2	9.8	10.0
Subgroup II-Hampshire (Low Incidence)	H98-4	7.8		6.9	7.0	7.7	7.3	...	10.1
	H93-3	8.3		8.4	8.4	6.5	7.5	...	10.8
	H94-3	8.2		7.3	6.4	6.6	8.6	...	8.3
	H 6-6	7.7		8.0	7.2	7.0	7.1	...	10.1
	H 7-3	7.0		7.0	6.5	7.3	7.2	...	10.8
	H 8-4	8.2		8.4	7.1	7.4	7.2	...	10.3
	H 1-3	7.9		7.6	9.5	6.6	9.6	...	10.1

TABLE XII

PHASE III: SERUM MAGNESIUM LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period	2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroups I-Yorkshire (High Incidence)	Y81-9	1.35		1.91	2.26	9.96	2.03	1.69	2.04
	Y91-2	1.13		1.35	1.30	0.90	1.80	1.69	1.94
	Y95-2	1.58		0.90	0.85	0.73	1.58	2.26	2.10
	Y79-3	1.63		1.75	1.13	1.24	1.30	2.08	2.56
	Y81-8	3.71		1.13	1.13	0.68	2.31	2.37	1.98
	Y79-8	2.42		0.68	1.18	1.13	1.13	1.41	2.32
	Y87-1	2.48		1.13	1.07	1.18	2.54	1.75	1.93
Subgroup II-Hampshire (Low Incidence)	H98-4	1.69		3.94	1.52	2.64	1.69	....	1.92
	H93-3	1.64		1.58	1.86	1.63	1.69	....	2.28
	H94-3	1.75		1.63	1.69	1.97	1.58	....	2.29
	H 6-6	2.31		4.51	1.80	1.92	1.30	....	2.78
	H 7-3	2.59		1.86	1.80	1.01	1.41	....	2.26
	H 8-4	3.56		2.14	3.94	1.30	2.82	....	2.12
	H 1-3	3.08		1.58	2.03	0.85	2.31	....	2.61

TABLE XIII

PHASE III: SERUM CHLORIDE LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr		2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	100	10 Minute Exercise Period	105	100	96	99	98	102
	Y91-2	97		105	98	100	103	101	100
	Y95-2	99		98	100	102	100	101	98
	Y79-3	100		100	98	103	98	104	101
	Y81-8	102		105	103	97	104	102	101
	Y79-8	100		104	100	101	105	101	104
	Y87-1	105		104	101	103	97	104	102
Subgroup II-Hampshire (Low Incidence)	H98-4	98		99	100	97	96	...	99
	H93-3	99		97	97	101	98	...	102
	H94-3	98		98	98	103	94	...	103
	H 6-6	99		96	97	99	95	...	100
	H 7-3	100		99	100	99	99	...	101
	H 8-4	99		101	108	100	94	...	101
	H 1-3	97		100	101	100	96	...	101

TABLE XIV

PHASE III: SERUM SODIUM LEVELS IN HGIH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period 2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	134	129	135	137	138	137	136
	Y91-2	131	130	131	139	142	141	138
	Y95-2	131	130	133	140	145	138	138
	Y79-3	130	132	134	138	146	148	137
	Y81-8	130	130	129	133	145	140	136
	Y79-8	134	130	129	136	144	130	140
	Y87-1	138	131	134	143	143	148	141
Subgroup II-Hampshire (Low Incidence)	H98-4	137	142	143	143	137	...	135
	H93-3	136	135	138	139	137	...	135
	H94-3	134	135	141	138	144	...	132
	H 6-6	136	141	140	141	140	...	135
	H 7-3	141	131	134	138	141	...	135
	H 8-4	134	139	145	144	139	...	136
	H 1-3	137	145	142	140	141	...	134

TABLE XV

PHASE III: SERUM POTASSIUM LEVELS IN HIGH INCIDENCE (YORKSHIRE) AND LOW INCIDENCE (HAMPSHIRE) GROUPS OF PIGS BEFORE AND AFTER EXERCISE

Groups	Pig No.	0 hr	10 Minute Exercise Period	2 hr	12 hr	24 hr	48 hr	96 hr	Avg. Values Phase II
Subgroup I-Yorkshire (High Incidence)	Y81-9	4.4		4.0	6.2	5.8	5.3	5.3	6.4
	Y91-2	4.1		5.9	4.6	5.9	6.6	5.2	6.6
	Y95-2	4.1		3.9	4.1	5.5	5.8	5.5	6.6
	Y79-3	5.3		4.4	4.6	5.7	6.6	6.4	6.7
	Y81-8	4.4		4.0	4.8	4.4	5.8	6.3	5.4
	Y79-8	4.7		4.2	4.5	4.8	5.8	6.2	7.4
	Y87-1	6.4		3.8	4.4	6.2	5.9	7.5	5.9
Subgroup II-Hampshire (Low Incidence)	H98-4	8.4		7.1	7.0	6.4	6.1	...	5.8
	H93-3	6.5		6.5	5.0	6.3	6.4	...	5.9
	H94-3	6.0		5.6	5.7	6.3	7.6	...	5.9
	H 6-6	7.4		5.2	5.9	5.7	5.1	...	5.6
	H 7-3	6.0		4.4	9.7	6.4	5.9	...	5.4
	H 8-4	4.5		4.7	5.7	6.6	5.7	...	5.1
	H 1-3	5.8		5.6	5.5	7.2	6.6	...	5.4

VITA

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