69-4225

CONLEY, Gary Olen, 1933-

GENETIC VARIATION IN GASTRIC LESIONS OF SWINE.

Iowa State University, Ph.D., 1968
Agriculture, animal pathology

University Microfilms, Inc., Ann Arbor, Michigan

GENETIC VARIATION IN GASTRIC LESIONS OF SWINE

by

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A Dissertation Submitted to the
Graduate Faculty in Partial Fulfillment of
The Requirements for the Degree of
DOCTOR OF PHILOSOPHY

Major Subject: Animal Breeding

Approved:

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Iowa State University
Ames, Iowa

1968

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INTRODUCTION

Veterinarians and animal scientists recognized the occurrence of gastric ulcers in swine before the twentieth century. However, during the past decade the prevalence of these lesions seems to have increased. Several reports of death losses in swine herds as well as in testing stations indicate the disease is economically important.

The basic etiology of gastric ulcers is largely unknown in both laboratory and domestic animals. Acute forms of gastric lesions have been produced experimentally by numerous agents such as drugs, diets, physical traumas and psychological stresses. The function of these agents in the formation of naturally occurring ulcers is not certain.

An observed correlation among the incidence in relatives has led some investigators to suggest a genetic proclivity to gastric ulceration in swine. Selection for economic characters of swine during the past 10 years with its accompanying genetic changes could account for part of the increase in ulcer frequency. An equally obvious explanation is the change which has been made in feed preparation and diet additives. Also, increased use of autopsies and diagnostic facilities may have increased the reported frequency without an accompanying increase in the actual frequency.

The term ulcer is used to describe gradual disintegration and necrosis of basal layers of a mucous membrane. A gastric ulcer is defined as an ulcer occurring on the mucous membrane of the inner wall of the stomach, while a duodenal ulcer is defined as one existing on the mucous membrane of the duodenum. Ulcers on the mucous membrane of either the stomach or the duodenum are included in the term peptic ulcer. A lesion is described as any pathological or traumatic discontinuity of tissue with a circumscribed area of degeneration. The term erosion, while defined as a disintegration of structure or an eating away of tissue, is used interchangeably with lesion.

Additional terms are used by swine ulcer investigators to differentiate between ulcers occurring in separate stomach regions. The term esophagogastric ulcer is used to describe an ulcer occurring on epithelial tissue contained within the stomach in the area that is the continuation of the epithelial tissue of the esophagus. A fundic ulcer is defined as one occurring on the mucous membrane and secretory tissue of the fundus gland region. A pyloric ulcer is on the mucous membrane and secretory tissue of the pyloric gland region. The term gastric ulcer is used to designate ulcers on the mucous membrane covering the glandular regions of the stomach.

The main objective in the present investigation was to determine the importance of heredity as an underlying cause of swine ulcers. Incidental to this determination were analyses of the influence of environmental factors in the formation of gastric lesions.

REVIEW OF LITERATURE

The History of Gastric Lesions in Swine

The occurrence of gastric ulcers in swine has been reported occasionally during the last 70 years. The early reports were mostly of ulcers in the glandular regions of the stomach. McIntosh (1897) described the symptoms and post-mortem appearance of several cases of what were probably chronic gastric ulcers. Rosenow (1923) found peptic ulcers in pigs to be similar microscopically but not grossly to those found in man, and indicated that streptococci were one of the primary causes.

A survey was conducted by Jensen and Frederick (1939) to determine ulcer frequency in several species of domestic animals. They found a five percent incidence of ulcers in 20,000 pigs slaughtered in the midwestern United States. Most of these ulcers occurred in the glandular regions.

An incidence of 2.4 percent was reported by Kernkamp (1945) from examination of 754 stomachs. He reported these ulcers resembled those occurring in man and were found in pigs of all ages. The highest incidence occurred in pigs from 5 to 11 months of age. Kernkamp found most ulcers occurring on the greater curvature within the fundic gland region.

Ulcers found in pigs at the boar testing station at Ames, Iowa occurred primarily in the glandless esophagogastric region (Berg 1960) although some fundic ulcers were noted.

Berg suggested that the management practices of limited space and a high energy ration could have been the primary causes of these ulcers. The first report of esophagogastric ulcers in swine in Ireland was given by McErlean (1962) in 1956. By 1962, he found the condition occurring in 50 percent of apparently healthy pigs. Ŝenk and Ŝabec (1965) studied stomachs obtained from the autopsy of pigs which died while being fed in confinement in Yugoslavia from 1963 through 1965. In 1963, ulcers were found in only .9 percent of the pigs but by 1965 the incidence had increased to 12.8 percent of all pigs in the study. Sixty five of the pigs which were autopsied in 1965 had esophagogastric ulcers, in 40 of the 65 pigs the ulcer was the primary cause of death.

Recent Surveys of Lesion Incidence

Reports of deaths in test stations and individual herds led to surveys taken from packing plants. Hoekstra (1962) in a survey of 857 swine stomachs from a Wisconsin packing house found a 48.6 percent incidence of lesions with 22.6 percent of the stomachs having an ulcer. All of the lesions observed were in the esophageal region of the stomach.

Thoonen and Hoorens (1963) studied pigs fed in confinement and observed a 4.7 percent incidence of fatal hemorrhage in 1,322 pigs with 12.8 percent of the affected pigs having perforations. Hoorens et al. (1965) collected data from 24,000 pigs slaughtered in Belgium over a 12 month period at the rate

of approximately 100 per day or 2,000 per month. They found lesions in 34 percent of these stomachs with severe ulcers in .93 percent of the 24,000 stomachs. Only .01 percent of the stomachs had an observable lesion in the glandular region. Large seasonal variation was noted and the highest incidence was in March. Large variations were also noted between groups of pigs slaughtered at different plants on the same day or on different days at the same plant, with a 70 percent incidence in one group of 112 pigs. There were no significant breed, sex, or age differences in these data.

Stomachs from 3,753 swine slaughtered in Wisconsin, Illinois and Iowa were collected by Muggenburg et al.(1964). They found ulcers in 13 percent of the females and 19 percent of the barrows. The overall incidence of erosions was 60 percent with 27 percent of the lesions classified as ulcers. Large differences were found between farms but no significant differences were observed between breeds. The difference between the spring and fall season was not significant, but there were trends observed within the seasons. These same workers studied the stomachs of 753 pigs which were examined after death caused by other diseases. Only 2.5 percent of the 753 diseased pigs had erosions or ulcers of the gastric mucosa.

Ferrando et al.(1965) conducted a survey of 984 pigs slaughtered during April and May in France. The incidence of

"Ulcère du Caridia" (esophagogastric) ulcers was 34 percent in these pigs. These investigators suggested heredity as a possible cause of the lesions. They also felt that the transporting of pigs to market was a contributing factor.

Kowalczyk et al.(1966) observed ulcers occurring in several farm herds. In one herd over a four year period 21 percent of 384 lactating first parity sows showed ulcer symptoms with ulcers causing a death loss of 13 percent. No symptoms or deaths were noted during the next three years, but the incidence of ulcer symptoms was above 20 percent the following two years. These authors felt that diet, season, temperature, and the stress of confining gilts raised on pasture in farrowing crates could have been precipitating causes of these lethal ulcers.

Esophagogastric ulcers were found in 87 of 443 pigs from Indiana examined by Curtin et al.(1963). The incidence of these ulcers was significantly greater in the late spring and early winter than during other months. Also in that study, rapid growth of the pig appeared to be important in increasing the incidence of esophagogastric ulcers.

Characteristics of the Lesions

The reported incidence of ulcers in the glandular region of the stomach has not increased, over the past decade, in proportion to those in the non-glandular area. Ulcers occurring in the non-glandular area were characterized by Kowalczyk

et al.(1960) and referred to as esophagogastric. They found diagnosis in the living animal to be very difficult. The early stages of the esophagogastric ulcers had no apparent clinical symptoms while in later stages there were signs of anemia accompanied by tarry diarrhea.

Muggenburg et al.(1964) found mostly esophagogastric ulcers in the stomachs of 594 pigs. These authors characterized the ulcerative process as beginning at the epithelial surface and progressing in the following steps: (1) epithelial changes, (2) acute erosions, (3) subacute ulcers, (4) chronic ulcers and (5) scars.

A punched-out appearance of the esophageal region was observed by McErlean (1962) with the surrounding cardia unaffected. The ulcer penetrated the submucosa laying bare the underlying musculature and blood vessels. He felt keratinization of the esophageal region was the first stage in the degenerative process. Kowalczyk et al.(1960) had concluded that keratinization was not a precursor of esophagogastric ulcers.

Esophagogastric ulcers were described in a doctoral dissertation by Griffing (1963). He found 107 ulcers in the glandless portion of 610 stomachs examined. The typical esophagogastric ulcer in this study was described as a depressed or crater-like area with elevated edges. "The base of the ulcer was brown, rough, and covered with necrotic debris." The base of healed ulcers appeared white and smooth with proliferating connective tissue filling the crater. These ulcers

ranged in dimensions from 0.25 cm x 1.0 cm to 5.0 cm x 8.5 cm. Griffing found 24 specimens of fundic ulcers in these 610 pigs. The fundic ulcers appeared as inflammed and congested areas on the crests of the gastric folds. The dimensions of the fundic ulcers in his study varied from 0.12 cm x 0.75 cm to 0.5 x 2.0 cm. The ulcers examined in the present study correspond to the descriptions given by Griffing. However, the fundic ulcers were of greater length and had a higher incidence in the present study.

Stresses and Secretions Involved in the Etiology of Lesions

Various environmental factors both alone and in combination were studied by Muggenburg et al.(1967) to determine their relationship to ulcer formation. These factors were transportation, fasting, crowding, mixing of pigs and length of time at the abattoir. All of these stresses, when in combination with others, significantly increased ulceration. However, neither transportation nor mixing was effective when used alone. Crowding did not increase the incidence of ulcers, but the total number of pigs in a pen had a large effect on fundic ulcers. A 90 percent incidence of fundic ulcers occurred in pigs grouped 30 and more to a pen, while pigs in groups of less than 16 showed no ulcers.

Medical practitioners have thought hypersecretion (especially of hydrochloric acid) by the gastric glands was the

basic cause of gastric ulcers. Recent research has contradicted this hypothesis. Three recent research studies cited by Davenport (1966, p. 105) found low acid secretion in human subjects having gastric ulcers. High concentrations of acid may have no effect on the gastric mucosa in some areas of the stomach. Code (1960) discussed the interplay of the various factors implicated in ulcer formation. He represented peptic ulcer formation as "a struggle between ulcer-promoting factors and ulcer-opposing factors". He felt that acid imbalance was the most important of these factors, and that either hypo-acidity or hyper-acidity would promote ulcer formation. Hollander (1954) showed that prolonged gastric hypersecretion would not cause ulceration. He concluded that the older hypothesis must be replaced by one including the interplay of secretions with the "two component mucous barrier". Anderson and Soman (1966) associated lowered acid and gastric secretions with the occurrence of gastric ulcers. They found ulcers associated with alkaline areas of the gastric mucosa. When the ulcers healed, these areas again became acid secreting.

Using 14 pigs weighing approximately 30 pounds, Huber and Wallin (1966) surgically created a gastric fistula or a Heidenhain pouch in each animal. Samples of gastric secretions were collected three hours after feeding. The buffering action of the ingested material and saliva caused large differences in pH between the pigs with the pouch and those with the fistula. The average value of pH for 21 samples from the

Heidenhain pouchs was 1.19 ± .06 while those from the gastric fistulas had an average of 2.69 ± .25. Samples of gastric juice taken from gastric fistulas, in a study by Muggenburg et al.(1966a), had pH values ranging from 1.1 to 6.9. Individuals pigs had average pH values varying from 2.5 to 3.6. Perry et al.(1966) found that stomach contents from pigs with esophagogastric ulcers had an average pH of 2.45 while the average from normal stomachs was 3.76. The average pH of stomach contents in a study by Mahan et al.(1966) was 4.86 with a significantly lower pH in those stomachs having an ulcer. Maxwell et al.(1967) found no significant differences in the pH of stomach contents between pigs having ulcers and normal pigs with all pigs having an average pH of 3.89. The range of pH values cited above agrees with those found in the present study.

Bicknell et al.(1967) showed that all pigs died within 7-36 days after surgical ligation of the extrahepatic bile duct. Death of these 13 pigs was due to bleeding and perforated esophagogastric ulcers. The pigs which died from fundic lesions in a study by Tournut et al.(1966) had severe pancreatic necrosis while only small groups of islet and acinar cells were necrotic in pigs with erosions. These findings indicate that either pancreatic or liver secretions may act as protective agents for the stomach wall. Reflux of the duodenal contents which contain these secretions is probably involved in this protection.

Protective Mechanisms of the Gastrointestinal Tract

Since gastric secretions are capable of digesting animal protein, there must be protective mechanisms to prevent autodigestion. Recent experimental evidence indicated that the gastric mucosa provides this protection. The protection furnished by the mucous barrier depends on its relationship to other factors. The buffering capacity of the mucous, the acidity of gastric contents, the amount of reflux of duodenal contents and the rate of mucous cell replacement are factors which may change the degree of protection.

Curtin and Goetsch (1966) studied alterations of gastric mucins associated with esophagogastric ulcers in pigs. The stomachs were classified as: (1) normal, (2) cornified, (3) eroded, (4) ulcerated or (5) healed. Included in the research were 800 pigs but only five stomachs from each classification were used. The physical characteristics of the mucins were different for ulcerated and normal pigs. The number of cells containing free acid groups was significantly higher in tissue taken from ulcerated stomachs. The healing of ulcers was preceded by the return of normal cellular constituents.

Dragstedt (1961) pointed to the buffering action of ingesta as a major factor in protection of the stomach wall. The presence of food triggered a secretory response and failure of the empty stomach to stop this secretion was considered a factor, in ulcer formation.

Production of Lesions by Extrinsic Agents

Gastric lesions have been produced by many agents in several species of animals. These lesions are usually acute ulcers and not the chronic form seen in most clinical cases. Rothenbacher (1965) found the stress produced by moving into strange buildings and the restraining of gilts in the last stages of pregnancy would produce acute esophagogastric ulcers in the gilts and also in their unborn pigs. He concluded that a hereditary factor played an important etiological role in ulcer formation. Fasting and restraint produced fundic lesions in 14 of 16 young pigs in an experiment by Tournut et al. (1966). These lesions were formed within 24 hours by the restraint of pigs in a corset and had started healing 24 hours after their release.

Muggenburg et al.(1966a,b) in two experiments produced acute ulcers in experimental pigs by injections of histamine or reserpine. At the highest dosages these drugs caused ulcers in the fundic gland region.

In an experiment conduced by Riker et al.(1967) pigs were exposed to temperatures of 29.4°C and 18.3°C with a third group rotated between these two temperatures. The pigs exposed to the alternating temperatures had significantly more esophagogastric ulcers and lesions than the pigs in either constant temperature.

Diet as an Etiological Agent in Lesion Formation

Previous studies have indicated an ulcerogenic effect due to the physical form of the diet fed. Corn in varying degrees of fineness was fed to pigs in an experiment by Mahan et al. (1966). The results indicated that small particle size was ulcerogenic. Including polythylene cubes in the feed did not decrease the ulcerogenic effect. These authors suggested a genetic predisposition influenced the response of pigs to ulcerogenic diets.

Chamberlain et al. (1967) studied the effect of pelleting and fasting on ulcer formation in pigs. The same ration ground to the same fineness was used in all groups. The pelleting of feed resulted in a significant increase of esophagogastric ulcers with an accompanying increase in feed efficiency. Perry et al. (1966) summarized the results of six feeding trials in which they had used gelatinized corn as a ulcerogenic diet. They reported that a modification of the starch was probably responsible for the ulcerogenic effect.

The influence of nutritional factors in the formation of esophagogastric ulcers was studied in several experiments by Reese et al. (1966). The feeding of ten different feed additives had no effect on ulcer formation in these studies. Several grains were ground through the same size hammermill screen and then fed in mixtures and alone. Wheat and oats fed alone and oats in all combinations significantly reduced

ulcer formation. There appeared to be no association between pH of the stomach contents and ulcers in these data. Maxwell et al. (1967) found that oat hulls included in an ulcerogenic corn ration protected against ulceration.

Nafstad et al. (1967) found that feeding rations containing ten percent of a 36 percent soybean meal resulted in completely normal stomachs. None of the feed additives tested had any effect except cod liver oil and casein both of which proved to be ulcerogenic. Low fat diets did not protect the pigs in this study but the workers concluded that unsaturated dietary fats were involved in ulcer formation. McErlean (1962) found no differences in incidence between commercial and home-mixed feeds (many contained whey and/or skim milk). He quoted Grant as having found that stored grain containing auto-oxidized unsaturated cereal fats caused an increase in gastric ulceration.

The Influence of Biological Organisms

Bixby (1964) concluded that Candida albicans contributed to esophagogastric ulceration in pigs. This fungus was consistently present in the pigs showing ulcers. Later research by Stedham et al. (1967) failed to support these findings, with no differences found in Candida albican growth between those stomachs containing ulcers and those free of ulcers. Supplemental sugar did not change the growth pattern of the Candida or the ulceration of the stomachs. Curtin et al.

(1963) were consistently able to isolate Candida albicans from the surface of the ulcers, with fungi mycelia found invading the superficial tissue of the ulcers.

Griffing (1963) in a study of stomachs from 610 pigs found a small but nonsignificant increase in ulcers in stomachs containing Candida albicans, but concluded they were secondary to the ulcerations. The gastric contents of those pigs having ulcers had a lower free and total acidity than those with normal stomachs.

Postmortem examinations of 70 ulcerated stomachs by Rothenbacher et al.(1963) did not reveal any pathogenic microorganisms associated with the ulcers. Death in these pigs was usually due to hemorrhaging at the ulcer site.

Heredity as a Factor in Ulcer Formation

Methods have been developed for estimating the relative importance of heredity and environment as factors in ulcer formation. Researchers used the frequency of ulcers in relatives to give liability values for the disease. Doll and Kellock (1951) studied 409 families in which one or both of the parents had either gastric or duodenal ulcers. They concluded that hereditary factors were important in determining the development of peptic ulcers. They also found that the relatives of patients with duodenal or gastric ulcers had a higher than average incidence of ulcers occurring in the same site. They concluded that gastric ulcers and duodenal ulcers

were inherited separately. Remelli et al.(1964) analyzed data from 100 families affected by duodenal ulcer and compared these families to those collected by Doll and Kellock. They determined the frequency in sibships of the original subject (propositi) and from this determined a relative risk for the disease. The frequency in the sibships was 32.5 percent ± 2.5 independent of sex and corrected for age. The relative risks were 5.8 for all families, 16.7 in families where both parents were affected, 4.0 with just the father affected and 2.9 when just the mother was affected. These authors concluded that duodenal ulcers have a multifactorial genetic basis with several additive genes each having a weak effect. Falconer (1965) discussed a method for estimation of heritability and applied it to the data of Doll and Kellock. This method used the "liability" to the disease as the scale of measurement with a threshold as the point on the scale where the disease is recognized. His estimate of heritability for gastric and duodenal ulcers, which he pooled, was .37 ± .06.

Research concerning ulcer formation in animals and man has given conflicting results. These differences could have been caused by the opposite reaction of different gastric areas to environmental and secretory stresses. Also many phases of environment which are not recognized may be affecting the results. Many factors probably play a part in determining the incidence of ulcers in most experiments with the agents under the researchers control having only a minor effect.

DATA COLLECTION

Description of the Herd Used in the Study

Stomachs from 2112 pigs were collected for this study from pigs produced at the Bilsland Memorial Farm in a project[1] designed to investigate the genetic effects of paternal irradiation. The sow herd originated in 1959 from purebred Duroc and Hampshire gilts purchased as littermate pairs from 39 Iowa Breeders. In subsquent years, pairs of replacement females were selected from first and second parity litters produced within the herd. Boars were purchased in littermate pairs each season and used for only one breeding season. One boar of each littermate pair was exposed to 300r X-irradiation at least five months before breeding. The irradiated and non-irradiated sires and their respective progeny will be referred to in this study as the two treatment levels. Approximately 60 boars and 300 females were mated in each of two seasons each year. The sows farrowed litters in March and April and again in September and October.

Each litter was farrowed and grown to 154 days in an 8' x 16' pen. The male pigs were castrated before 14 days of

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[1]Project No. 1424 of the Iowa Agricultural and Home Economics Experiment Station, Ames, Iowa. The project was supported by the U.S.A.E.C. Contract AT(11-1)-707.

age, and the litters were weaned at 42 days of age by removing the dam from the pen. The pigs were self-fed a pelleted ration throughout the period. The ration fed during the period from approximately 60 to 154 days is given in Table 1. The litters were removed from individual pens at 154 days of age and the individual pigs weighed and placed into four different groups according to weight. Backfat was measured at three sites by using a metal probe at the time of weighing. Pigs having a weight greater than or equal to 86 kg. were

Table 1. Finishing ration used at Bilsland Memorial Farm

Ingredient	Percentage by weight
Corn # 2 yellow - ground fine	72.247
Dura Bond	1.993
Molasses - (blackstrap)	4.983
Soybean meal, solvent, 44% protein	11.709
Fish solubles; 50% solids, 32% protein	2.491
Meat and bone scraps, 50% protein	2.491
Dehydrated alfalfa meal, 17% protein	2.491
Ground limestone, 38% Ca	.448
Dicalcium phosphate, 26% Ca, 18% Phos	.598
Salt, iodized	.399
Trace mineral mixture (swine)	.150[a]
Vitamin B-12 - 15 milligrams	
Vitamin D2 - 1 million IU	
Riboflavin - 3 grams	
Pantothenic acid - 5 grams	
Niacin - 12 grams	
Auromycin - 20 grams	

[a]To furnish the following amounts of specified minerals in P.P.M.; iron 70.4, copper 4.8, cobalt 1.6, zinc 81.6 manganese 56.8.

slaughtered within the following week. Pigs weighing less than 86 kg. were fed in groups until they weighed at least 86 kg. and then taken as a group to slaughter. Ninety pigs were fed together in each pen. Pigs were slaughtered in February through May or in August through November.

Groups of 45 slaughter weight pigs were weighed and moved to loading pens where they were held until the next day. Feed and water were furnished free-choice during this period. The groups were hauled approximately 60 miles to the packing plant where they were held without feed approximately 18 hours before being slaughtered. This caused the pigs to be fasted for 20 or more hours preceding slaughter. Usually pigs were slaughtered once a week, but when large numbers of pigs were reaching market weight several groups were slaughtered in the same week.

The data were collected from pigs marketed in the fall of 1966 and the spring of 1967. Weights taken at the farm on the day preceding slaughter were recorded for all pigs. The data from the fall of 1966 consisted of records from 854 pigs. All records of a sire's progeny were removed when there were less than five progeny recorded. Also removed were records which were incomplete or in disagreement with existing herd records for sex or weight. The data from the spring of 1967 consisted of records from 1258 pigs, with 1132 records remaining after removal of records for the reasons previously given.

Collection of the Stomachs

Stomachs were collected at the packing plant where approximately 360 pigs were processed each hour. The stomachs were identified by attaching numbered metal tags corresponding to tattoo numbers on the pigs, and carried back to the farm laboratory for evaluation. Samples of gastric contents were obtained by puncturing the stomach and allowing liquid to drain into a 40 ml. test tube. The stomachs were opened along the lesser curvature and the mucosa exposed by inverting the stomach. Gross visible lesions were identified and the stomach given a score for each of three regions. These regions are shown in Figure 1 as taken from Sisson and Grossman (1953 p. 491).

Scoring the Lesions in Three Stomach Regions

The lesions of each area of the stomach had distinguishing characteristics. The esophageal area consists of nonglandular epithelium surrounding the esophageal orifice and varies from 6 to 10 cm. in diameter. The lesions of this area appeared as a sloughing of tissue with the remaining tissue stained and crusted. The base of the lesion was usually smooth with blood clots adhering to the edges. Lesions representing the types which occurred in this region are shown in Figure 2a. Stomachs which showed no sloughing of tissue would receive a score of zero while stomachs which had the

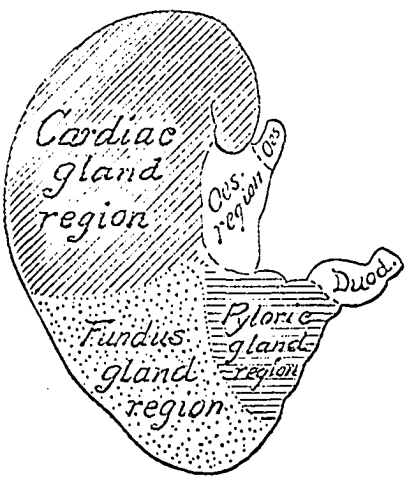


FIG. 422.—DIAGRAM OF ZONES OF MUCOUS MEMBRANE OF STOMACH OF PIG.

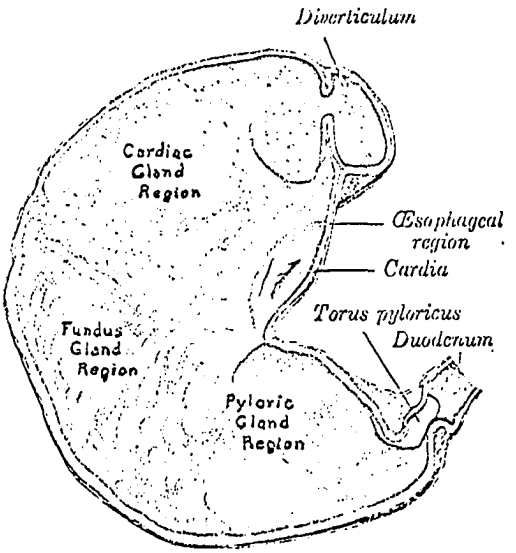


FIG. 422a.—FRONTAL SECTION OF STOMACH OF PIG.

Figure 1. Gastric regions of the pig's stomach

entire area sloughed would receive a score of 15. Lesions involving more than half the total area which showed signs of bleeding and were deeper than .5 cm. would also be given a score of 15.

The fundus gland region includes from 1/4 to 2/3 of the area of the gastric mucosa. This glandular region is composed of a mixture of three cell types which secrete mucous, enzymes and hydrochloric acid. The area was heavily folded in the empty stomach but appeared smooth when the stomach had been distended. The characteristic lesion observed in this region was a linear erosion, as shown in Figure 2b, along the crest

of a fold. Small oval punctate lesions were also seen in this area. A normal fundic region was given a score of zero. Regions having linear damage exceding 15 cm. in length and .5 cm. in depth with blood evident in the crevice would receive a score of 15. Also fundic regions with 15 or more punctate lesions would receive a score of 15.

The pyloric region includes from 1/8 to 1/4 of the gastric area and surrounds the opening of the stomach into the duodenum. The lesions of this area were circular and much larger than those observed in other areas of the stomach. A typical pyloric lesion is shown in Figure 2c. They appeared shallow but histological examination revealed greater tissue damage than in deeper appearing fundic lesions. Most of these lesions were crimson and had blood and clotted blood clinging to the edges. Deep bleeding ulcers were found occasionally on the Torus Pyloricus, a knob of tissue in the opening to the duodenum. Lesions greater than six cm. in diameter with signs of bleeding were given a score of 15 as were the deep ulcers on the Torus Pyloricus.

Visual appraisal was used to assign individual scores for each of the three regions based on the limits and descriptions given above.

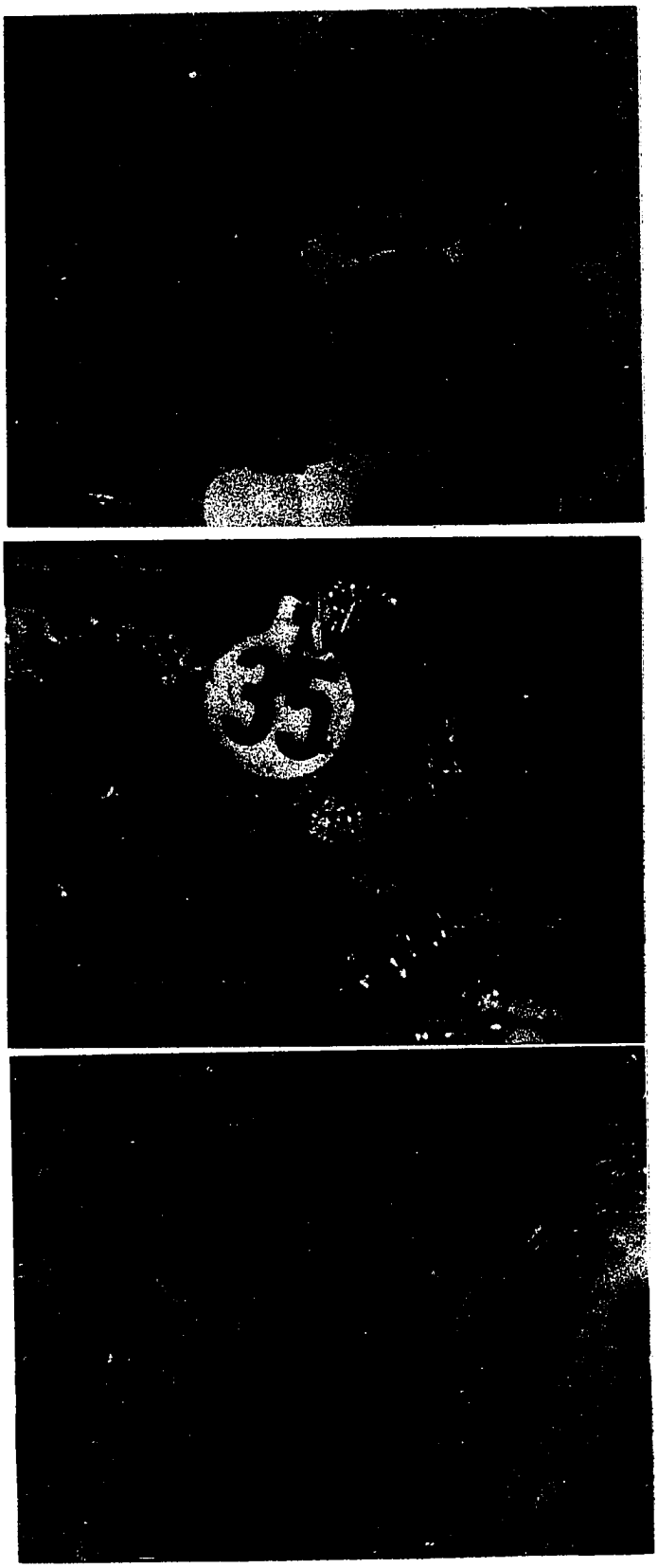
The incidence of pyloric lesions was less than .06 with only .02 of the pigs having a score greater than one for this region. Pyloric lesion data were not studied due to the very low incidence and a failure to detect variation which could be

Figure 2. Lesions characteristic of those occurring in the esophageal region, fundus gland region, and pyloric gland region

a. Shows lesion typical of those occurring in the esophageal portion of the stomach

b. Shows a linear fundic lesion on a fold of the fundus gland region of the gastric mucosa

c. Shows a large circular ulcer occurring in the pyloric gland region of the stomach



attributed to any of the factors to be studied.

The distributions of lesion scores of all stomachs collected for both seasons are presented in Table 2. The apparent excess in the end class was caused by the limitations of the scoring system rather than an actual excess of similar

Table 2. Distribution of lesion scores

Score	Esophageal	Fundic
0	1515	1369
1	120	134
2	65	76
3	47	50
4	24	32
5	22	28
6	13	24
7	8	9
8	11	14
9	5	12
10	11	19
11	4	2
12	9	7
13	3	2
14	2	3
15	26	104
Total	1885	1885

lesions.

Dr. M. B. Dockerty, surgical pathologist at the Mayo Clinic, made histological examinations of lesions which had been previously scored by visual appraisal. The histological examination revealed that the gross lesion score was not an accurate indication of the histological severity of the lesions.

Collection of Gastric Contents and pH Measurement

Gastric juice from the stomachs of 639 pigs was collected and evaluated for pH. A Corning pH meter with a single glass electrode made pH evaluation possible for stomachs containing as little as 10 ml. of liquid. The first samples were collected at the packing plant within 30 minutes after the animals were killed. These samples were packed in dry ice and returned to the laboratory for evaluation. Additional samples were taken from the same stomachs after returning to the laboratory and the pH readings compared to the earlier samples. A comparison between the two sampling times was made using 100 stomachs, and no significant differences were found between the times of sampling. All subsequent samples were taken at the laboratory. Sample pH values were recorded twice from independent readings and the results averaged unless a wide discrepancy was noted, in which case an additional reading was taken for those samples.

Carbon dioxide was used as an anesthetic before slaughter at the plant. Mullenax and Dougherty (1963) in a study of the physiologic responses of swine to inhaled carbon dioxide found a rapid lowering of the blood pH beginning one minute after the start of inhalation. In order to determine the effect of carbon dioxide inhalation on gastric pH, a small packing plant was used to obtain data from 36 pigs stunned with a rifle rather than carbon dioxide. There was no significant

difference in the mean pH of gastric samples taken at the two plants.

Stress as a Factor in Lesion Incidence

Pigs in some litters were being used in avoidance learning experiments. These pigs were subjected to avoidable electric shock with the object of teaching them to avoid the shock by responding to another stimulus, an electric buzzer. To explore the effects of psychological stress on ulcer incidence, 215 pigs were stressed, half of which had been tested in the avoidance learning chamber at 150 days of age. The 215 pigs were subjected to unavoidable electric shock in the same test chamber. The pigs were restrained in the test chamber and subjected to 10 periods of 60 seconds of intermittent shock. The total stress consisted of loading handling, restraining and shocking. The pigs were stressed at one day, two days, three days, or five days preceding slaughter. Comparing pigs to unstressed littermates gave no indication of a change in lesion incidence, lesion score or gastric pH due to the stress.

METHODS, RESULTS AND DISCUSSION

Lesion Incidences

The incidence of lesions for this study was calculated as the fraction of pigs in a group which had at least one lesion. The least squares means of lesions adjusted to a constant age of 182 days are shown in Table 3. The differences between the mean incidences of the Duroc and Hampshire breed were not significant. The two breeds differed significantly in their 154 day weight and because of this were different in their average slaughter age. When the means of the two breeds were not adjusted for the regression of lesion incidence on slaughter age the Hampshire breed had a significantly higher incidence of esophagogastric lesions, but there was no significant difference in fundic lesion incidence. The failure to demonstrate differences in lesion incidence between these two breeds would indicate that genetic control of lesion formation may be small relative to the environmental factors which affected both breeds alike. The means for barrows and gilts were similar with the differences being nonsignificant. The differences in lesion incidences between the two seasons studied were large, accounting for 18 percent of the variance in esophagogastric lesion incidence and 6 percent of the variance in fundic lesion incidence.

The overall incidence of fundic lesions in the present data was much higher than was reported by Muggenburg *et al*.

Table 3. Lesion incidence least squares means and standard errors

Classification	Number of pigs	Esophagogastric lesions	Fundic lesions
Duroc	892	.15 ± .01	.26 ± .01
Hampshire	961	.17 ± .01	.23 ± .01
Barrows	1022	.15 ± .01	.26 ± .01
Gilts	831	.16 ± .01	.23 ± .01
Fall 1966	721	.04 ± .01	.15 ± .01
Spring 1967	1132	.24 ± .01	.31 ± .01

(1964) and Hoekstra (1962) from surveys of more than 4,500 pigs in the midwestern United States. The reason for the higher incidence was not readily apparent, but collection of weight and backfat data at 154 days and the subsequent mixing of pigs into large groups in the present study might have been factors. Muggenburg et al.(1967) found that, while crowding did not increase incidence of ulcers, placing pigs in groups of 30 or more resulted in a high incidence of fundic erosions and ulcers. Simulated hauling of pigs also increased the incidence of gastric lesions in their experiment. Fundic ulcers were created within 24 hours by fasting and restraint in 14 of 16 pigs studied by Tournut et al.(1966). Therefore, in the present study hauling to market and 20 hours of fasting seem likely factors in fundic lesion formation. Analyses of the scores of fundic and esophagogastric lesions showed no significant phenotypic correlation between these traits, the actual value of the correlation was -0.07. Therefore, the

lesions of these two regions of the stomach were treated as separate traits.

The Effect of Litter Size on Lesions

A possible source of variation in gastric lesions was litter size at 154 days and the differing degrees of crowding and competition it could cause. The data collected in the fall of 1966 were incomplete, since the stomachs of pigs were not examined in several litters. The data from the pigs slaughtered in the spring of 1967 were analyzed using the model:

$$Y_{ijk} = \alpha + L_i + D_j + \beta X_{ijk} + E_{ijk}$$

Y_{ijk} = the observed lesion score, for either esophageal or fundic lesions, on the k^{th} pig within the l^{th} litter size, slaughtered on the j^{th} day.

α = the population mean when equal subclass frequencies exist and $X_{ijk} = 0$.

L_i = effect of the i^{th} litter size.

D_j = effect of the j^{th} slaughter date.

X_{ijk} = age at slaughter for an individual pig.

β = partial regression of lesion score on slaughter age.

E_{ijk} = random errors assumed to be $NID(0,\sigma^2)$.

The distributions of these factors by litter sizes are given in Table 4 along with the analyses of variance for esophagogastric and fundic lesions. The number of pigs in the litter at 154 days was used as the litter size. There were

Table 4. Effect of litter size on gastric lesions

Litter size	Number of litters	Number of pigs	Slaughter age	Esophago-gastric lesions	Fundic lesions
1	4	4	180	0.00	0.50
2	14	26	182	1.04	1.15
3	17	47	171	1.08	2.51
4	19	73	176	1.41	1.86
5	26	114	174	1.29	1.89
6	25	138	180	1.60	2.11
7	20	125	179	1.19	2.70
8	31	228	178	1.23	2.00
9	24	205	177	0.95	2.33
10	5	60	177	0.37	1.83
11	6	62	183	0.47	1.69
12	2	20	170	0.80	1.95
13	3	30	198	2.20	1.73
Average 6.30	198	1132	178	1.15	2.09

Analyses of variance:	Source	d.f.	Mean square
Esophagogastric lesions	Slaughter date/ breed/treatment	55	19.76 **
	Litter size	12	7.17
	Reg. on sl. age	1	0.28
	Error	1063	7.42
Fundic lesions	Slaughter date/ breed/treatment	55	34.15 **
	Litter size	12	10.76
	Reg. on sl. age	1	4.64
	Error	1063	17.15

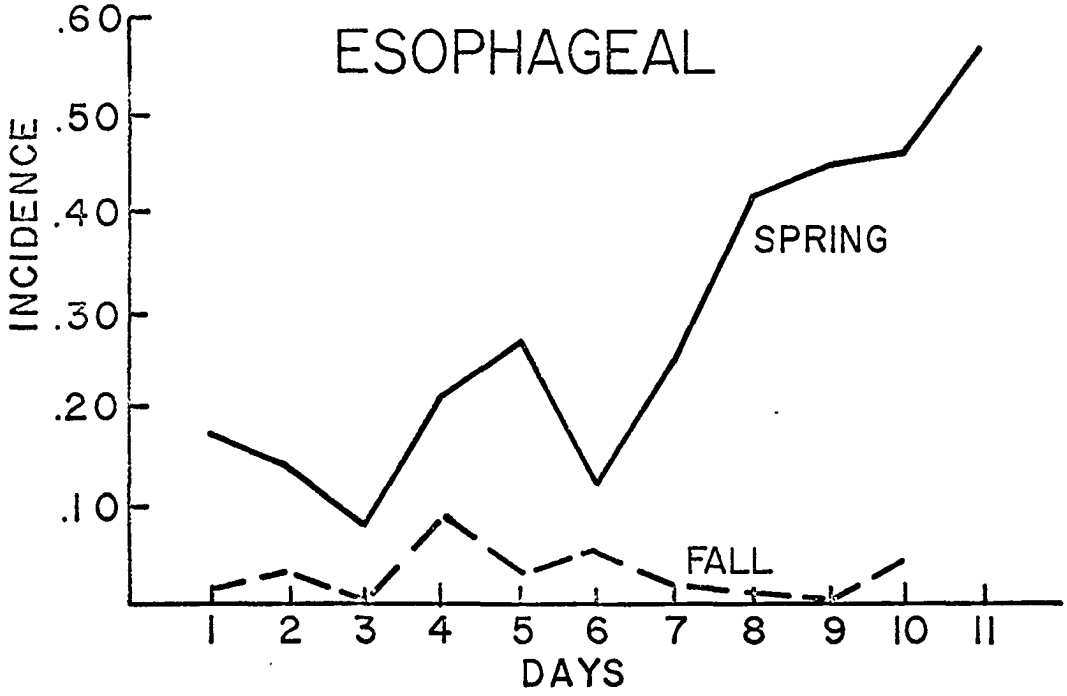
**Significant at the .01 level.

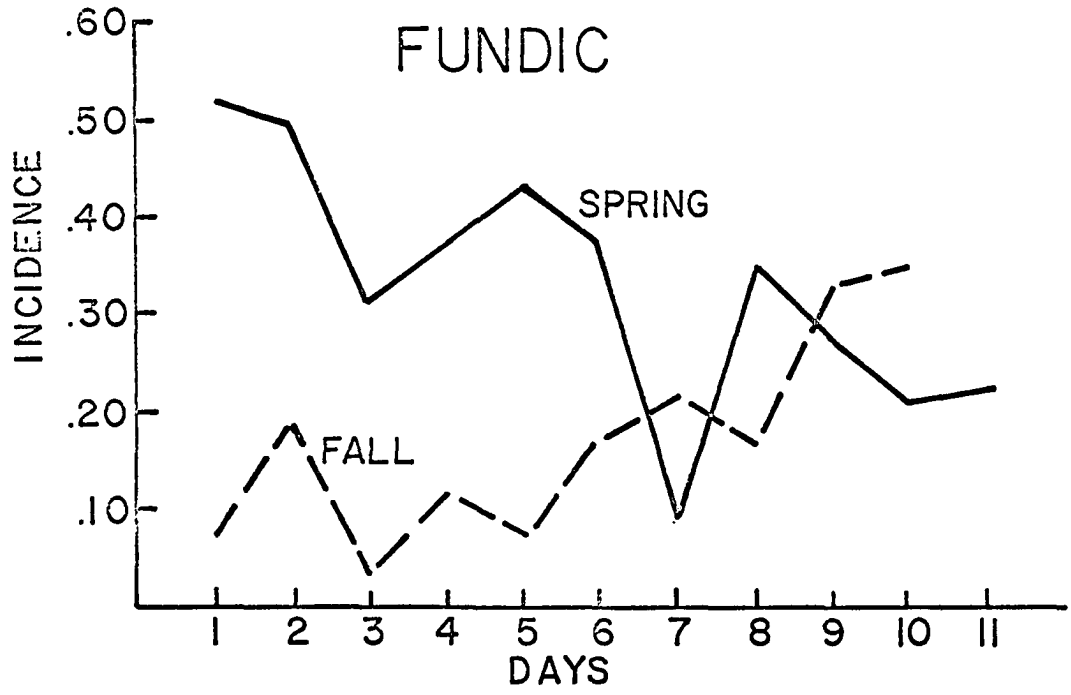
115 gilts kept for breeding purposes which were not available for the scoring of gastric lesions. Both slaughter date and age at slaughter were included in this model. The unadjusted phenotypic correlations were; +.25 between slaughter age and esophageal lesions, -.12 between slaughter age and fundic lesions, and -.07 between fundic and esophagogastric lesions. The effect due to age was not significant when slaughter date was included in the model. The size of litter was not an important factor in the formation of either esophagogastric or fundic lesions. Pigs from larger litters might have been better adapted to living in large groups and this could have influenced the apparent decrease seen in esophagogastric lesions for large litters. Table 4 shows that slaughter date contributed significantly to the variance of lesion incidence.

The Effect of Slaughter Date

Figure 3 shows the incidences of esophagogastric and fundic lesions by slaughter date. The occurrence of esophageal lesions appeared to depend upon factors peculiar to the slaughter date to a greater extent than did the occurrence of fundic lesions. Groups were slaughtered on 10 dates in the fall and 11 dates in the spring. Three of the original 14 slaughter groups with fewer than 15 pigs per group were removed from the spring data for the analysis of slaughter date. The days 1 through 11 in Figure 3 correspond to the slaughter dates shown in Table 6 for the fall and spring season. The

Figure 3. Incidences of esophagogastric and fundic lesions by slaughter dates


ESOPHAGEAL
INCIDENCE
.60
.50
.40
.30
.20
.10
SPRING
FALL
1 2 3 4 5 6 7 8 9 10 11
DAYS


FUNDIC
INCIDENCE
.60
.50
.40
.30
.20
.10
SPRING
FALL
1 2 3 4 5 6 7 8 9 10 11
DAYS

average number of pigs in a slaughter group was 72 in the fall and 103 in the spring with ranges of 42 to 102 and 73 to 177 pigs respectively. The incidence of lesions in the esophageal region was low with no observable trend in the fall but was high the following spring with a definite upward trend as the season progressed. Trends in both seasons were noted in fundic lesion incidence.

These trends within a season could be caused by temperature differences. The reverse slopes, shown in Figure 3, between fundic lesion incidences in the fall and spring indicate that perhaps day length or temperature or both were involved in the trend. The pigs slaughtered on different dates within a season were not significantly different in their average birth date and no trend for lesion incidence was noted in the averages arranged by birth date.

The variance components were calculated using the method given by Harvey (1963). The model for the analysis of variance in Table 5 was:

$$Y_{ijk} = \alpha + S_i + D_j + (SD)_{ij} + E_{ijk}.$$

Y_{ijk} = the presence or absence of a lesion, in the k^{th} progeny, of the i^{th} sire, slaughtered on the j^{th} day.

α = the population mean if there were equal subclass numbers.

S_i = effect of the i^{th} sire.

D_j = effect of the j^{th} slaughter date.

$(SD)_{ij}$ = effect of the ij^{th} sire-slaughter date subclass after the average effects of sire and slaughter date have been removed.

E_{ijk} = random deviation associated with the individual and assumed to be NID$(0,\sigma^2)$.

Table 5. Variance components of lesion incidences for sire, slaughter date and their interaction

Source	Component	Percent of total variance
Esophagogastric:		
Sires	.0023	1.2
Slaughter dates	.0125	6.6
Sires x slaughter dates	.0056	2.9
Within	.1686	89.3
Total	.1890	
Fundic:		
Sires	.0036	2.6
Slaughter dates	.0139	10.3
Sires x slaughter dates	.0062	4.5
Within	.1118	82.6
Total	.1355	

The data were highly unbalanced with regard to the presence of observations in all expected subclasses formed by the cross-classification of sire x slaughter date and litter x slaughter date. If each sire had had offspring in every slaughter date there would have been 480 subclasses in the fall data and 605 in the spring data. There were only 299

sire x slaughter date subclasses which contained an observation in the fall data and 317 in the spring. This left 469 of the 1085 possible subclasses empty. Missing subclasses can create a covariance between the two classifications involved which would contribute to the interaction terms shown in Table 5. The analyses indicated the variance components for the interaction effects represented less than 4.5 percent of the total variance. Therefore, the slaughter date constants were obtained from the simpler model, $Y_{ijk} = \alpha + S_i + D_j + E_{ijk}$. The observation, Y_{ijk}, is a linear function of the k^{th} pig, from the i^{th} sire subclass, which was slaughtered on the j^{th} date. The $\alpha + S_i$ equations were absorbed into the remaining equations for the computation of the slaughter date constants. The overall mean of lesion incidence with equal subclass numbers was represented by α, with D_j representing the effect due to slaughter date. The random deviations, E_{ijk}, of the individuals from their subclass means were assumed to be normally and independently distributed with a mean of zero and a variance of σ^2. The slaughter date constants and their standard errors are given in Table 6. Corrections for slaughter dates were made within each season.

Table 6. Least square constants for slaughter date

Slaughter date Year Day	Number of pigs	Esophagogastric constant	Esophagogastric standard error	Fundic constant	Fundic standard error
66-250	102	.006	.025	-.115	.052
66-257	81	.041	.032	.067	.066
66-263	68	-.027	.027	-.196	.056
66-279	82	.052	.025	-.018	.048
66-280	65	-.009	.029	-.182	.060
66-284	77	.038	.023	.018	.047
66-291	72	-.017	.025	-.021	.052
66-301	86	-.009	.022	.037	.046
66-306	42	-.047	.030	.161	.062
66-334	46	-.028	.029	.249	.058
67-060	75	-.114	.080	.322	.089
67-062	73	-.072	.100	-.021	.112
67-066	92	-.300	.092	-.158	.103
67-067	148	.147	.069	.037	.077
67-074	88	-.129	.083	.115	.092
67-081	90	-.304	.080	.151	.089
67-100	74	.156	.070	-.114	.078
67-109	98	.162	.052	-.016	.058
67-116	177	.115	.043	-.039	.047
67-126	94	.074	.052	-.113	.058
67-137	81	.265	.063	-.164	.066

Genetic Analyses

For the analyses of genetic variance, animals were classified into families. These family groups were classified by sires used in a season, dams mated to the same sire, and progeny from a single dam. Within a season the progeny of a dam were littermates and would be similar not only because of their genetic relationship but also because of common environment.

There were 103 sires in the two seasons with an average of 3.45 litters and 18 pigs per sire group. The range in fundic lesion incidence for sires with 18 or more pigs was .04 to .56 and the range in esophagogastric lesion incidence was .00 to .64. The sire groups are listed in Table 7 along with their esophagogastric and fundic lesion averages.

The incidence of ulcers was analyzed as a two valued variable with the values zero or one, which indicated respectively the absence or presence of a lesion.

Table 7. Gastric lesion incidences for sires

Identification of sires	Number of litters	Number of offspring	Fundic	Esophago-gastric
Fall 1966: Duroc				
10284	2	10	.10	.10
10285	3	21	.29	.05
10286	3	21	.24	.00
10287	4	19	.11	.00
10288	2	10	.20	.00
10289	2	4	.00	.25
10290	4	18	.17	.00
10292	4	14	.21	.00
10294	2	9	.11	.00
10295	3	12	.25	.00
10296	2	13	.08	.00
10297	4	21	.24	.09
13284	2	16	.06	.00
13285	4	21	.19	.00
13286	3	12	.08	.08
13287	3	19	.32	.05
13288	3	19	.21	.10
13289	4	18	.11	.05
13290	4	19	.16	.00
13292	2	7	.14	.00
13294	3	14	.14	.00
13295	3	12	.08	.00
13296	2	15	.13	.00
13297	5	23	.04	.00
Average:	3.04	15.3	.16	.03
Fall 1966: Hampshire				
20300	4	17	.29	.06
20301	4	16	.19	.06
20302	4	19	.10	.05
20303	2	7	.00	.00
20305	4	13	.08	.00
20306	4	14	.07	.00
20307	6	29	.07	.03
20308	2	10	.10	.00
20309	3	8	.25	.00
20310	4	17	.18	.00
20311	3	10	.00	.00
20314	5	21	.29	.00

Table 7 (Continued)

Identification of sires	Number of litters	Number of offspring	Fundic	Esophago-gastric
23300	3	13	.31	.00
23301	2	8	.00	.00
23302	2	8	.13	.00
23303	4	21	.14	.09
23304	5	23	.17	.09
23307	4	16	.06	.06
23308	2	6	.00	.00
23309	2	18	.11	.05
23310	3	10	.00	.00
23311	3	10	.10	.00
23312	5	23	.48	.04
23314	4	17	.12	.00
Average:	3.50	14.8	.15	.03
Spring 1967: Duroc				
10316	4	30	.30	.37
10317	4	26	.38	.08
10318	2	12	.25	.08
10319	4	17	.18	.29
10320	4	27	.33	.33
10321	4	15	.27	.33
10322	6	29	.38	.14
10323	2	13	.62	.31
10324	2	12	.33	.25
10325	3	25	.32	.32
10326	3	21	.29	.33
10327	1	5	.60	.00
10328	1	12	.50	.08
10329	1	6	.33	.00
10330	5	29	.34	.31
13317	4	29	.45	.14
13320	4	17	.47	.18
13321	6	46	.26	.13
13322	6	40	.40	.10
13323	5	26	.38	.46
13324	5	24	.50	.25
13325	1	5	.20	.00
13328	4	26	.35	.15
13329	2	11	.18	.36
13330	4	22	.23	.23
Average	3.48	21	.35	.23

Table 7 (Continued)

Identification of sires	Number of litters	Number of offspring	Fundic	Esophago-gastric
Spring 1967: Hampshire				
20331	3	12	.33	.25
20332	2	11	.64	.54
20333	4	17	.47	.18
20334	5	30	.33	.37
20335	5	22	.18	.64
20336	3	13	.15	.31
20337	4	29	.24	.31
20338	3	19	.53	.58
20339	4	12	.25	.42
20340	5	32	.56	.25
20341	5	21	.10	.57
20342	1	7	.71	.00
20343	3	22	.41	.41
20344	3	19	.15	.05
20345	2	5	.20	.20
23331	2	13	.31	.69
23332	4	17	.18	.41
23333	4	24	.25	.21
23334	5	35	.14	.29
23335	3	19	.16	.21
23336	5	31	.45	.42
23337	4	22	.14	.27
23338	3	10	.40	.40
23339	3	14	.43	.00
23340	6	40	.38	.37
23341	4	21	.14	.33
23342	3	15	.40	.20
23343	4	26	.27	.38
23344	5	27	.33	.18
23345	4	22	.27	.32
Average:	3.7	20.2	.31	.30

The form of the analysis of variance shown in Table 8 is for a general model, $Y_{ijkl} = \alpha + G_i + S_{ij} + L_{ijk} + P_{ijkl}$. Where the Y_{ijkl} = a linear function of the l^{th} pig from the k^{th} litter by the j^{th} sire within subclasses defined by breed,

Table 8. Expected mean squares for use in calculating heritability

Source	d.f.	Expected mean square
Sires	no. of sires - 1	$\sigma_e^2 + k_2 \sigma_l^2 + k_3 \sigma_s^2$
Litters within sires	no. of litters - no. of sires	$\sigma_e^2 + k_1 \sigma_l^2$
Pigs within litters	no. of pigs - no. of litters	σ_e^2

$\sigma_s^2 = 1/4\ \sigma_A^2$

$\sigma_l^2 = 1/4\ \sigma_A^2 + 1/4\ \sigma_D^2 + \sigma_C^2$

$\sigma_e^2 = 1/2\ \sigma_A^2 + 3/4\ \sigma_D^2 + \sigma_W^2$

$h^2 = 4\ \sigma_s^2/(\sigma_s^2 + \sigma_l^2 + \sigma_e^2)$

σ_A^2 = additive genetic variance.

σ_D^2 = dominance variance.

σ_C^2 = variance common to members of a full sib family.

σ_W^2 = environmental variance within full sib families.

h^2 = heritability; the ratio of additive genetic variance to total variance.

treatment (control or irradiated) and season.

α = the theoretical population mean if there had been equal subclass frequencies.

G_i = effect of the i^{th} season, treatment, breed.

S_{ij} = effect of the j^{th} sire within the i^{th} season, treatment, breed group.

L_{ijk} = effect of the k^{th} litter within the ij^{th} season, treatment, breed, sire group.

P_{ijkl} = effect of the l^{th} progeny within the ijk^{th} season, treatment, breed, sire, litter subclass.

The analyses of genetic variance were computed within treatments, season, and breeds and pooled. The treatment classifications were included in the model, to separate the sires into unrelated groups, even though preliminary analysis had shown treatment to be an unimportant factor in lesion formation. This model was used for the analyses shown in Table 9 and Table 10.

Table 8 gives the expectations of mean squares used in the calculation of variance components of gastric lesion incidence. The coefficients of the variance components, represented by k_1, k_2 and k_3 in Table 8, are calculated by equating the mean squares to their expectations. The equation used for the calculation of heritability is also shown.

The data used in the analyses in Table 9 were first corrected by subtracting the least squares constants shown in Table 6. There were three slaughter date groups each with

Table 9. Analyses of lesion incidence data, corrected using least squares constants for slaughter date

Source	d.f.	Mean square	Component	Percent of variance
		Esophagogastric incidence		
Season/trt/breed	7		.024	18.8
Sires/breed	95	.287	.004	2.7
Dams/sires	253	.190	.017	10.6
Pigs/dams	1455	.109	.109	67.7
		Fundic incidence		
Season/trt/breed	7			6.4
Sires/breed	95	.243	.000	0.0
Dams/sires	253	.233	.014	7.0
Pigs/dams	1455	.168	.168	86.5

Table 10. Analyses of lesion incidence data, calculated within slaughter date, season, breed and treatment subclasses

Source	d.f.	Mean square	Component	Percent of variance
		Esophagogastric incidence		
Subclass	95	29.649	.045	27.30
Sire/subclass	519	.138	.003	1.80
Dams/sire	240	.125	.009	5.64
Pigs/dam	998	.109	.109	65.26
		Fundic incidence		
Subclass	95	13.853	.022	11.78
Sire/subclass	519	.190	.005	2.43
Dams/sire	240	.178	.007	3.39
Pigs/dam	998	.166	.166	82.50

less than 12 observations that were not included in the analyses shown in Table 9.

An alternative method for removing the effect of slaughter date from the data would be to compute the analyses within slaughter dates. Each slaughter date was considered a separate random sample, taken with replacement of sires, from the population of sires in the herd. Analyses of the data with sires grouped within slaughter date were computed and are shown in Table 10. For this analysis the term G_i in the model included the effect of the i^{th} breed/treatment/slaughter date/season subclass.

For the sire components of variance the results in Table 10 were not greatly different from those given in Table 9. The change of magnitude in the litter component was the most noticeable difference in the two methods used for removing the effect of slaughter date. Both of these methods indicated additive genetic variance, as estimated from the sire component, was an insignificant part of the total variance of lesion incidence. The partial confounding of sires with slaughter date could have biased both of these estimates of the sire component of variance. The possible bias should have been small as the interaction itself accounted for only a small portion of the variance.

The heritability estimates from the two analyses used are presented in Table 11 along with their standard errors. These estimates of heritability were small when compared to their standard errors. The two estimates for fundic lesions estimated the same population parameter and it was likely, from the estimates, that this parameter was close to zero. The two estimates for the heritability of esophagogastric lesions were larger than the comparable estimates for fundic lesions. The heritability of esophagogastric lesions was likely to be in the range .00 to .14.

Table 11. Estimates of heritability

Lesion type	Heritability	Standard error
Esophagogastric:	.141[a]	.080
	.100[b]	.161
Fundic:	-.007[a]	.052
	.116[b]	.160

[a]Estimate computed from the components of variance in Table 9.

[b]Estimate computed from the components of variance in Table 10.

Covariances between esophagogastric and fundic lesion incidence were calculated using the analyses in Table 10 along with an analysis of the same model using the sum of the two variables. Covariances were calculated using the relationship $\text{Cov}(E,F) = \frac{1}{2}[\text{Var}(E+F) - \text{Var}(E) - \text{Var}(F)]$. The sum of the

sire, dam and pig components of variance were used to calculate the phenotypic covariances shown in Table 12. The genetic covariances were calculated from the sire components of variance. The environmental variances and covariances were calculated by removing the additive genetic variance or covariance from the variance or covariance of pigs within dams. The correlations were calculated using the method described by Hazel et al.(1943).

Table 12. Variances, covariances and correlations for lesion incidence

	Phenotypic	Additive[a] genetic	Environmental
Variances:			
Esophagogastric	.120	.014	.102
Fundic	.178	.020	.156
Covariance:			
Esophagogastric, Fundic	.005	.016	-.008
Correlation:			
Esophagogastric, Fundic	.03	.97	-.06

[a]Estimated from sire components of variance and covariance.

The phenotypic correlation estimate was positive but small while the environmental correlation was small and negative. The genetic correlation was estimated to be .97. An

approximate standard error for the genetic correlation using the method presented by Robertson (1959) was .71. This large but poorly estimated genetic correlation implied that the genes which controlled the susceptibility of a pig to gastric lesions were the same for both the esophageal and fundic regions. The two regions have different cell types which could have been one of the reasons for the negative environmental correlation. The cells of the esophageal region are not as well protected by mucous as those of the glandular region. The biological significance of these estimates of correlation was questionable.

The variation due to pH and backfat probe were not removed in the analysis of the genetic variance. The pH was considered to be related to the biological processes involved in ulceration. Thus removal or adjustment of this factor would have removed some of the genetic variance of lesions. Backfat probe is a heritable trait and adjustment for it would remove some genetic variance if there was a genetic correlation between backfat and the trait measured.

Temperature and Backfat Analyses

An experiment by Riker et al.(1967) demonstrated that the alternating of pigs between different temperatures increased esophagogastric ulcers and lesions in pigs. Riker's results, and the differences observed between average incidences in the two seasons in the present data prompted an analysis of

the relation of ambient temperatures and the formation of gastric lesions. The range in temperature was taken as the difference between the maximum and minimum temperature for a day. The ranges for the ten days preceding slaughter were averaged for use in this analysis. The variance of these ten values was also included in the model as a measure of the temperature variability to which the pigs were exposed. The average backfat probe at 154 days was included in the model to evaluate its contribution to differences caused by temperature changes. Also if the leaner pigs had more esophagogastric lesions this could be a partial explanation for the increased incidence of esophagogastric lesions observed in the last decade. This premise assumes that the trend in swine has been toward less backfat. The model used in this analysis was:

$$Y_{ijkl} = \alpha + L_i + T_j + S_k + \beta_1 R_{ijkl} + \beta_2 V_{ijkl} + \beta_3 A_{ijkl} + \beta_4 W_{ijkl} + \beta_5 F_{ijkl} + E_{ijkl}$$

Y_{ijkl} = the occurrence or absence of a lesion in the l^{th} pig, within the i-j-k^{th} litter, treatment, sex subclass.

α = the population mean when equal subclass frequencies exist and $R_{ijkl} = V_{ijkl} = A_{ijkl} = W_{ijkl} = F_{ijkl} = 0$.

L_i = effect of i^{th} litter subclass.

T_j = effect of the j^{th} treatment class.

S_k = effect of the k^{th} sex.

R_{ijkl} = average range of temperature for the ten days preceding slaughter for a given pig.

β_1 = partial regression of lesion occurrence on the average

temperature range.

V_{ijkl} = variance of the range in temperature for the ten days preceding slaughter for a given pig.

β_2 = partial regression of lesion occurrence on the variance of the temperature range for the ten days preceding slaughter.

A_{ijkl} = slaughter age for an individual pig.

β_3 = partial regression of lesion occurrence on slaughter age.

W_{ijkl} = 154 day weight for an individual pig.

β_4 = partial regression of lesion occurrence on 154 day weight.

F_{ijkl} = the average backfat probe for an individual pig.

β_5 = partial regression of lesion occurrence on backfat probe.

E_{ijkl} = random deviation of the q^{th} pig from his adjusted subclass mean, assumed to be NID$(0,\sigma^2)$.

The analyses are presented in Table 13 along with the subclass means. The variance of the temperature range was significant in esophagogastric but not in fundic lesion formation. The regression of esophagogastric lesions on the variance of the temperature range indicated that a variable range in temperature decreased lesion formation. The average range in temperature for the preceding ten days had no significant effect in esophagogastric or fundic lesion development. The regressions of lesion formation on 154 day weight

Table 13. Effects of backfat and daily range in temperature on gastric lesions

Subclass	Number of pigs	Mean backfat averages	Mean range in temperature
Control	903	1.06	25.91
Treated	950	1.05	25.93
Season 66-2	721	.97	28.31
Season 67-1	1132	1.11	24.33
Duroc	892	1.20	25.60
Hampshire	961	.92	26.14
Males	1022	1.11	25.76
Females	831	.99	26.12
Combined	1853	1.05	25.92

Analyses of variance	Source	d.f.	Mean square	$\hat{\beta}$
Esophagogastric incidence				
	Treatment	1	.056	
	Sex	1	.127	
	Reg. on temp. range	1	.283	.0062
	Reg. on var. of range	1	.619 *	-.0004
	Reg. on sl. age	1	4.932 **	.0052
	Reg. on 154 day wt.	1	.916 **	.0020
	Reg. on backfat	1	.081	.0589
	Error	1497	.113	
Fundic incidence				
	Treatment	1	.079	
	Sex	1	.109	
	Reg. on temp. range	1	.038	-.0023
	Reg. on var. of range	1	.328	.0002
	Reg. on sl. age	1	.568	-.0018
	Reg. on 154 day wt.	1	.627	-.0017
	Reg. on backfat	1	.423	.1348
	Error	1497	.175	

*Significant at .05 level.

**Significant at .01 level.

and slaughter age were significant in the analysis of esophagogastric lesions. Heavier pigs had a higher incidence of esophagogastric lesions. The weight effect was partially confounded with the slaughter date within a season and most of the regression attributed to weight may in fact have been due to that portion of the variance of lesions caused by slaughter date which was not caused by temperature differences. Slaughter date was not included in the model since the temperature measurement was the same for all pigs on each date. The partial regression of esophagogastric lesion incidence on slaughter age was highly significant. The older pigs within the litters had an increased incidence of esophagogastric lesions.

Differences in backfat did not contribute significantly to the variance of esophagogastric or fundic lesion incidence within litters. The partial regressions of esophagogastric lesions on backfat and temperature range were highly significant when litters were not considered in the model, indicating the necessity of considering this source of variation in future investigations of ulcers in pigs.

Effects of Lesions on Gain

The gain in weight from 154 days to slaughter was studied to determine the effect of lesion formation on gain. The probable components in this relationship could be: the loss in weight of pigs reacting adversely to the stresses of handling

and mixing. The secretory differences existing in pigs consuming varying amounts of feed and the buffering of gastric secretions by the food.

The lesion scores were placed into four classes for the analysis shown in Table 14. The classes used were; Class 1 - zero scores, Class 2 - scores 1 through 3, Class 3 - scores 4 through 11, and Class 4 - scores 12 through 15. The source of variation labeled "litters +" contained variation due to the differences among litter subclasses, within sire, breed, treatment and season. The model used in this analysis was:

$$Y_{ijkl} = \alpha + L_i + G_j + F_k + \beta X_{ijkl} + E_{ijkl}$$

Y_{ijkl} = gain of the l^{th} pig belonging to the k^{th} fundic lesion class, the j^{th} esophagogastric lesion class within the i^{th} litter subclass adjusted for slaughter age.

α = the population mean when equal subclass frequencies exist and $X_{ijkl} = 0$.

L_i = effect of the i^{th} litter subclass.

G_j = effect of the j^{th} esophagogastric lesion class.

F_k = effect of the k^{th} fundic lesion class.

X_{ijkl} = age at slaughter for an individual pig.

β = partial regression of gain from 154 days to slaughter on age at slaughter.

E_{ijkl} = random deviations of the individual pig from his adjusted subclass mean. Assumed to be $NID(0,\sigma^2)$.

Table 14. Effect of ulcers at slaughter on gain from 154 days to slaughter

Lesion class E^a F^b	Number of pigs	Age of pigs	Gain	Gain adjusted by least squares
E_1	1492	180	15	15.6
E_2	225	185	15	15.9
E_3	82	196	19	13.7
E_4	33	191	17	16.0
F_1	1347	183	16	15.0
F_2	253	180	15	15.0
F_3	123	180	13	16.0
F_4	109	172	10	16.0
Mean		182 ± 0.41	15.4 ± 0.53	

Analysis of variance: Source	d.f.	Mean square	F
Litters + Reg. on age at slaughter	154	1,568.4	
	1	1,243.4	6.91 **
Esophagogastric	3	444.1	2.47
Fundic	3	245.1	1.36
Error	1670	179.9	

[a]Esophagogastric lesions.

[b]Fundic lesions.

**Significant at the .01 level.

Neither fundic nor esophagogastric lesions were significant factors in influencing the gain preceding slaughter.

The age at slaughter was included in the model so that the influence of ulcers on gain would not be confused with the differences in age. The differences in average age shown in

Table 14 reflect the length of time the pigs in a lesion class were held at the farm in order to reach market weight. Pigs weighing 86 kg. or more at 154 days would have been slaughtered before 164 days of age and would have been influenced by the stresses of weighing, probing and placing of pigs in large groups. The lighter weight pigs would have had an opportunity to recover from the weighing and probing. The placing of pigs in large groups would have had adverse affects on all groups but probably to different degrees. The differences in lesion incidences on different slaughter dates would also affect the average slaughter age of the lesion classes. Groups that had been weighed at approximately the same time were marketed together. This confounded age with slaughter date and, because of the relationship between slaughter date and lesions, there was partial confounding between lesions and age regardless of any biological relationship.

Effects of Gastric pH on Lesions

Samples of the liquid gastric contents were collected fom 639 pigs in 11 slaughter groups as shown in Table 15. Samples were not obtained from all pigs in these groups since some stomachs were perforated in removal from the gastrointestinal tract. The relative acidity of the gastric contents could have been influenced by the gastric secretions, saliva secretion, water consumed, rate of gastric emptying and

Table 15. pH and lesion means by slaughter groups

Year	Day of[a] slaughter	Number of pigs	Esophag-ogastric incidence	Fundic incidence	pH ( )[b]
66	301	86	.012	.163	5.22(74)
66	334	46	.043	.348	5.13(38)
67	060	75	.173	.520	5.26(66)
67	062	73	.137	.479	4.67(63)
67	066	92	.076	.304	4.31(72)
67	067	148	.203	.378	4.24(133)
67	074	88	.261	.432	4.37(77)
67	086	8	.126	.126	3.87(8)
67	088	12	.000	.333	3.99(12)
67	095	10	.300	.200	5.29(10)
67	126	94	.426	.202	4.47(86)

[a]Represents the day of the year on which pigs were slaughtered.

[b]Number of pigs sampled.

the reflux of duodenal contents.

The incidence of lesions in pigs classified by pH are presented in Table 16. Preliminary analyses were made to determine the effects of season, breed, sex, sire, slaughter age and slaughter weight on pH and no significant effects were found. The litter component accounted for 24 percent of the variance in pH values.

Table 16. Means for effect of pH, by classes, on gastric lesions

pH class	Number of pigs	Esophagogastric lesion frequency	Fundic lesion frequency
≤ 2.0	46	.304	.304
2.1-3.0	160	.256	.325
3.1-4.0	236	.161	.347
4.1-6.5	168	.083	.393
> 6.5	29	.207	.276
Means and standard errors:			
4.61 ± .045		.177 ± .015	.347 ± .019

The analyses were corrected for litter differences by using a least squares procedure in which litter subclasses were absorbed into the remaining equations. Examination of the data suggested that pH change might cause a curvilinear response in the occurrence of gastric lesions especially on those of the esophageal region. For this reason the model used for the analyses was a second degree polynomial. The analyses were computed using the model:

$$Y_{ij} = (\alpha + L_i) + \beta_1 X_{ij} + \beta_2 (X_{ij})^2 + E_{ij}$$

Y_{ij} = a score for lesions of the j^{th} pig, in the i^{th} litter subclass.

α = the population mean when equal frequencies exist in all subclasses and $X_{ij} = (X_{ij})^2 = 0$.

L_i = effect of the i^{th} litter subclass.

X_{ij} = pH reading for a given pig.

Table 17. Analyses of the effects of pH on gastric lesions

	Source	β	Standard error
Esophagogastric incidence	$\beta_1 pH$	-.005 **	.0018
	$\beta_2 pH^2$	-.000	.0001
Fundic incidence	$\beta_1 pH$	.004	.0022
	$\beta_2 pH^2$	-.000	.0001
Esophagogastric score	$\beta_1 pH$	-.033 **	.0105
	$\beta_2 pH^2$	.000	.0007
Fundic score	$\beta_1 pH$	.047 *	.0224
	$\beta_2 pH^2$	-.001	.0015

*Significant at the .05 level.

**Significant at the .01 level.

β_1 = partial regression of lesion score on pH.

$(X_{ij})^2$ = the square of the pH reading for a given pig.

β_2 = partial regression of lesion score on pH^2.

E_{ij} = random deviation of the individual pig from its adjusted subclass mean.

As can be seen in Table 17 the relation of pH to lesion incidence and score was linear with the regressions being of opposite sign for esophagogastric and fundic lesions. Whether the effect of pH was determined on lesion score or incidence made little difference, in both cases a change in pH had a greater effect on the esophageal than the fundic region. The regression of esophagogastric lesion score on pH indicated

that an acid increase in the gastric environment increased both the incidence and the total score of esophagogastric erosions. The relation of pH and fundic lesion severity was positive, meaning that lowered acidity of the gastric environment favored the formation of these lesions. The regression of fundic incidence on pH was also positive but not significant at the .10 probability level.

SUMMARY

This thesis presents the results of a study of the genetic variance in gastric lesions. Also included was an investigation of the effects of season, gastric pH, backfat probe and temperature range on the incidence and severity of gastric lesions.

The data consisted of 1853 pigs from 356 litters by 103 sires of two breeds farrowed in two seasons. Stomachs were examined after slaughter and scored for lesions occurring in three stomach regions.

The difference between the Duroc and Hampshire breeds was negligible indicating little genetic diversity for gastric lesions between these two breeds. Sex differences were also nonsignificant. The X-irradiation of sires had no significant effect on lesion formation among their progeny. Pigs which were stressed by unavoidable electric shock while being restrained showed no increase in lesions when compared to their control littermates.

There was a large increase in lesion incidence for the spring over the fall slaughter period. Most of this increase of lesions in the spring over the fall data was attributed to lesions in the esophageal region. Differences between the fall and spring data comprised 18 and 6 percent of the variance in esophagogastric and fundic lesions, respectively. The variance due to slaughter date within a slaughter period ac-

counted for seven percent of esophagogastric and ten percent of fundic lesion variances.

An increase in the acidity of the liquid gastric contents increased the frequency of esophagogastric lesions and decreased the frequency of the fundic lesions. An increase in the variability of the temperature range for the 10 days preceding slaughter decreased the frequency of esophagogastric lesions within a litter but showed no significant effect on fundic lesions. An increased average range of temperature for the 10 days preceding slaughter showed no significant effect on lesion incidences within litters. Within litters the incidences of lesions did not show a significant relationship to the average backfat probe.

Age, weight and gain from 154 days to slaughter were probably not important factors in lesion formation, except as they related to the slaughter date within a season. The data did not permit an analysis of these factors completely independent of the effect attributed to slaughter date.

Estimates of genetic variance were calculated from the variance components in hierarchical analyses of variance of lesion incidences. The additive genetic variance for fundic lesion incidence was probably less than one percent of the variance. Analyses of esophageal lesion incidence indicated that additive genetic variance accounted for between 2 and 10 percent of the total variance.

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ACKNOWLEDGMENTS

The author wishes to express his appreciation for the many helpful suggestions offered by Dr. David F. Cox, during the course of this study and in the preparation of the manuscript; also to Dr. L. N. Hazel, Dr. R. M. Melampy, and Dr. R. L. Willham for their helpful criticisms of the study.

The author is indebted to Dr. Oscar Kempthorne, for his generous counsel and assistance in the statistical analysis of the data.

The author would like to acknowledge the contributions of Dr. D. Dal Kratzer whose assistance with the data collection and computer programming of the analysis was invaluable.

The author also expresses his appreciation to his wife, Florence, for typing the manuscript.