



Swine Erysipelas

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Circular 471



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COVER ILLUSTRATION

Symptoms of unthriftiness and stiffness, together with bony growths below the hock and in the region of the pastern joints, are apparent in the pigs on the cover, which are infected with chronic swine erysipelas.

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Swine Erysipelas

By G. L. DUNLAP and ROBERT GRAHAM^a

SPORADIC OUTBREAKS of swine erysipelas in various Illinois herds have been noted over a period of many years, but not until 1932 was the disease regarded as of economic importance to the swine industry of the state. The first significant outbreak coming to the attention of the Illinois Agricultural Experiment Station was in 1920. Symptoms now recognized as characteristic of swine erysipelas were observed in a herd that was fed restaurant garbage, but bacteriologic findings were not considered sufficient to warrant a positive diagnosis. Both acute and chronic forms of swine erysipelas have been reported with increasing frequency in different parts of the United States since that time. A skin form of swine erysipelas—"diamond skin" disease—was recognized as early as 1921 by Creech in apparently healthy hogs at time of slaughter, while the following year Giltner reported an outbreak of the acute and fatal malady in suckling pigs in Virginia.

In 1932 two serious outbreaks of swine erysipelas in Illinois prompted a special veterinary conference (September, 1932) at the College of Agriculture, University of Illinois, to acquaint veterinary practitioners with the clinical symptoms of the disease. At this conference naturally infected animals were used for instructional purposes and the Schoening agglutination test for the diagnosis of swine erysipelas was demonstrated. Veterinarians attending the conference were invited to confirm all clinically suspicious outbreaks of swine erysipelas coming to their attention by sending specimens to the Laboratory of Animal Pathology and Hygiene for diagnosis. Thru the cooperation of veterinarians and the Laboratory, 61 outbreaks of swine erysipelas were recognized in 15 counties in Illinois over a period of four years (1933-1936). The number of recognized outbreaks increased from 6 in 1933, to 17 in 1934, 15 in 1935, and 24 in 1936. The distribution of the 15 counties in the state in which these outbreaks occurred is shown in Fig. 1.

If the disease is disregarded, it may spread and become a serious swine problem in Illinois, the ultimate economic importance of which cannot yet be accurately predicted. This circular has been prepared in response to numerous requests received at the Illinois Agricultural Experiment Station for information regarding the cause, nature, and control of swine erysipelas.

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Fig. 1.—Counties (Shaded) From Which Hogs Infected With Swine Erysipelas Were Received at the Laboratory of Animal Pathology, University of Illinois, 1933-1936

Cause of Swine Erysipelas

Swine erysipelas is caused by a small, slender, rod-shaped microorganism (*Erysipelothrix rhusiopathiae*) (Fig. 2). In the acute type of the disease the microorganisms are found in the blood and all body tissues. In the chronic type the microorganisms become localized in the joints, skin, heart valves, gall bladder, and tonsils.

Resistance of the Organism

A knowledge of the resistant character of the swine-erysipelas organism is of practical importance in the application of measures to stamp out the disease on infected premises. Owing to its wax-like covering, drying kills this organism only gradually. Exposure to direct sunlight requires 12 days to kill. Virulent organisms survived in pieces of meat and bacon in pickle for 170 days and in smoked hams for a period of over three months. The organisms remained viable after four months in putrefying meat.

Laboratory tests show that heating to 160° F. kills the swineerysipelas organism in 5 minutes. The following disinfectants are effective in 5 to 15 minutes when applied directly on the organism: bichlorid of mercury in a dilution of 1 to 1,000; 5-percent phenol solution; 3.5 percent compound cresol solution; 2-percent formalin; 1-percent lye solution. The nature of the material in which the organisms are present greatly influences the length of time necessary to kill them.

Mode of Spread

Infection with swine erysipelas usually occurs thru the intestinal canal. Feed and drinking water contaminated thru contact with feces, urine, and other offal of diseased animals are the usual carriers of the

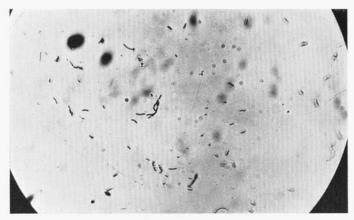


Fig. 2.—Photomicrograph of Causative Organism of Swine Erysipelas, *Erysipelothrix rhusiopathae* (Magnified × 900)

infective agent. Feeding infected pork trimmings—such as bacon rind, excess fat, and bones—in kitchen slop to susceptible animals may cause new infection. Injuries to the intestinal lining, such as those produced by the thorny-headed worm, facilitate infection. Skin wounds also serve as a portal of entry.

The selling or bartering of sick or exposed hogs at community sales may introduce the disease into healthy herds. Many chronically infected swine that appear healthy enter the channels of trade and establish new centers of the disease.

On a farm where there has been a serious outbreak of swine erysipelas, infection tends to recur in succeeding crops of pigs, either thru contact with chronically infected animals that appear healthy or thru exposure to contaminated houses, lots, and pastures.

Symptoms in Acute and Chronic Types

Swine erysipelas attacks hogs of all ages but is most serious in suckling pigs and shoats. The symptoms displayed by affected animals permit a clinical classification of acute and chronic types.

Acute type. Animals suffering from the acute type of swine erysipelas may die suddenly or in a few hours after showing symptoms. The acute form of the disease has been recognized in suckling pigs in Illinois, but the extent of pig mortality traceable to swine erysipelas is not known. In herds where young pigs die from unexplained causes tests should be made for the possible presence of this disease. In two cases coming to the attention of the Laboratory of Animal Pathology and Hygiene the acute and chronic forms of the disease occurred simultaneously in the same herds.

In the initial stages of the acute type of swine erysipelas the body temperature is elevated to 106° F. or higher, altho the infected animals may still eat and not appear to be in a serious condition. As the disease develops, the animals appear acutely ill, refuse to eat, and lie prostrate on their breasts. There usually is a thin, watery discharge from the eyes. In spite of appearing very ill, most animals affected with acute swine erysipelas are surprisingly animated when disturbed but they quickly resume a resting position when let alone.

Breathing may be accelerated and jerky. Circumscribed or diffuse reddened areas which blanch on pressure may be observed on the skin of the abdomen, and hot, tender swellings may develop on the legs. Animals may die almost as soon as these symptoms appear or may gradually improve and then develop the chronic form of the disease. Some animals make a complete recovery.

Chronic type. The chronic type of swine erysipelas occurs when the causative agent localizes in the tissues of the animal. No clinical symptoms are usually discernible when the infection localizes in the heart valves, gall bladder, or tonsils. When it localizes in the joints or skin, visible symptoms are noted. When the joints are affected, the pig walks with a stiff gait and there is gross enlargement of the bones at the joint (Figs. 3 and 4). The losses from this form of the disease are due to unthriftiness and failure to make gains.

The skin form, or urticarial form, referred to as "diamond skin disease" since the areas affected are roughly diamond-shaped, may be so mild as to go unnoticed until the hog is slaughtered, scalded, and cleaned (Fig. 5). More evident skin lesions may follow the acute type of the malady and large areas of the skin may die and slough away (Fig. 6). The denuded areas may become the site of secondary infections. Tho such animals may make a gradual recovery, they are unprofitable in the feedlot.

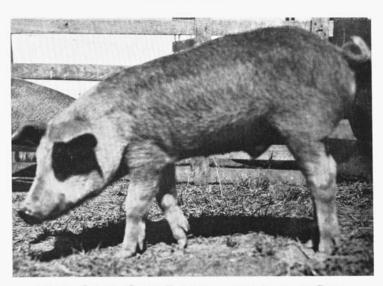


Fig. 3.—Chronic Swine Erysipelas Accompanied by Bony Enlargements on Legs, by Lameness and Stiffness



Fig. 4.—Bony Enlargements on Legs Below Hock on Pig Shown in Fig. 3



Fig. 5.—Lesions Caused by Diamond-Skin Disease

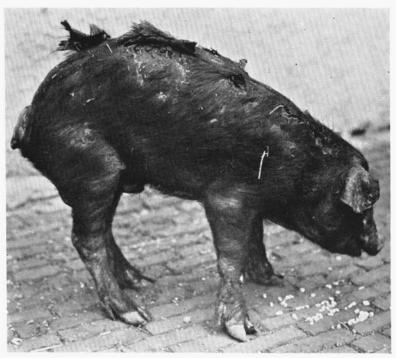


Fig. 6.—Sloughing of the Skin Following an Acute Attack of Swine Erysipelas

Postmortem Lesions

Only slight changes in the internal organs of animals which have succumbed to the acute form of swine erysipelas are apparent at postmortem examination. The lining of the stomach, particularly at the intestinal outlet, shows an inflammatory swelling, reddening, and numerous small hemorrhages. The surface is covered with a sticky, glassy mucous. The visceral and body lymph glands show varying degrees of swelling, edema, and congestion. The spleen is usually swollen and the pulp very dark in color (Fig. 7). Small hemorrhages also occur on and under the lining membrane of the heart chambers. In the advanced stages of the acute disease, as well as following death, the skin over the abdomen may show red, purplish blotches. Some animals recovering from the acute type may show sloughing of the skin over the back and shoulders.



Fig. 7.—Enlarged Spleen of Hog Infected With Swine Erysipelas, Showing White Necrotic Areas Due to Plugging of Small Arteries

In the chronic form cauliflower-like growths may develop on the heart valves. These growths sometimes constrict the valve opening to such an extent that it is hardly possible to insert in them an object the size of a lead pencil (Fig. 8). These lesions are not necessarily confined to the valves but may also occur on the walls of the heart, as shown in Fig. 9. The internal organs manifest the secondary changes which are usually produced by valvular lesions. These changes include an increased amount of fluid in the thoracic and abdominal cavities, and passive congestion of lungs, liver, and spleen. Small fragments of the cauliflower-like growths break away and are carried in the circulation to remote parts of the body, where they plug the small arteries. The small, light-colored nodules at the border of the spleen in Fig. 7 are the result of the plugging of small arteries by fragments from the heart-valve lesions.

Lesions of the joints are accompanied by definite bone changes which are easily visible at autopsy. The ends of the long bones and the small bones of the knee, hock, and pastern joints show excessive bony growth (Fig. 10). The surfaces of the joints may be eroded and there may be an increased amount of cloudy, flocculent joint fluid.

When the disease occurs in the form of "diamond-skin," a mild inflammation of the skin, sharply circumscribed, round, quadrangular, or rhomboidal spots develop, especially on the skin of the chest, back, thighs, and neck. As a rule the spots or plaques are small, but they coalesce and form areas as large as the palm of the hand.



Fig. 8.—Cauliflower-like Lesions on the Heart Valves of Hog Infected With Swine Erysipelas



Fig. 9.—Growths on Lining of the Heart as a Result of Local Infection With Swine Erysipelas



Fig. 10.—Excess Bone Formation in Region of Hock and Pastern Joints Resulting From Chronic Swine Erysipelas

Course of the Disease

Animals that survive an acute attack of swine erysipelas for four days or longer often recover. In poorly nourished young animals complete recovery is sometimes very slow, requiring several weeks. In most cases, however, the convalescent period lasts only a few days.

The possibility of the chronic form of the disease developing in animals that survive the acute form always exists. Chronic swine erysipelas cases in which there are growths on the heart valves usually terminate fatally. Animals showing the skin lesions of erysipelas following recovery from the acute type gradually recover, but prove unprofitable in the feedlot.

Diagnosis

A diagnosis of swine erysipelas based on history, symptoms, and autopsy findings should be confirmed by laboratory tests. The direct isolation of the causative organism in pure culture from enlarged joints, spleen, and heart blood is positive evidence of swine erysipelas. The rapid plate agglutination test, as described by Schoening, is also a valuable aid in the diagnosis of swine erysipelas. This test, however,

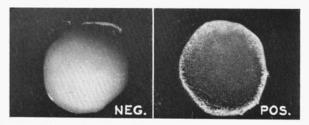


Fig. 11.—Slides Showing Negative and Positive Results in Rapid Agglutination Test for Swine Erysipelas

In negative agglutination tests (tests showing the absence of swine erysipelas) the bacterial suspension remains uniform in appearance, while in a positive test the bacteria are clumped or agglutinated.

must be skilfully employed and properly controlled to avoid misleading or false findings.

A positive and negative reaction to the agglutination test is shown in Fig. 11. The most satisfactory bacterial antigen for the rapid plate agglutination test for swine erysipelas, as applied at the Laboratory of Animal Pathology and Hygiene, has been secured from Schoening of the United States Bureau of Animal Industry.

Inoculation of susceptible laboratory animals is often resorted to in order to arrive at a positive diagnosis. Pigeons and mice are the

experimental animals most commonly used for this purpose.

The acute form of swine erysipelas may be confused with hog cholera, hemorrhagic septicemia, or the acute stage of necrotic enteritis. The chronic form may be confused with malnutrition, rickets, chronic porcine infectious abortion (Brucellosis in swine or Traum's disease of the bones and joints), as well as streptococcic infections.

Swine Erysipelas in Other Animals

Swine erysipelas has been encountered in lambs, ducks, and turkeys, as well as in field mice. Pigeons are also susceptible. Infected animals and animals that have apparently recovered from the disease may serve as carriers of the infection, and an outbreak in swine may spread to lambs, ducks, and turkeys on the same farm or from these animals to swine. Furthermore certain precautions should be taken by anyone who handles infected animals, for germs of swine erysipelas can cause a local infection on human hands. In the treatment of infected animals, as well as in handling the carcasses of animals that have died of the disease, gloves should be used to avoid human infection.

Prevention and Control

Animals infected with swine erysipelas should be isolated and the herd inspected daily for further evidence of the disease.

Herds showing suspicious clinical symptoms and gross autopsy lesions of acute swine erysipelas may be inoculated with specific antiserum pending a definite laboratory diagnosis. Marked improvement has been noted in 48 hours following the administration of anti-swine-erysipelas serum in acutely infected herds. For chronically infected swine, inoculation with anti-swine-erysipelas serum has proved of little value.

When diagnosis is doubtful and the disease may be either hog cholera or swine erysipelas, experience to date suggests that it is always advisable to use anti-hog-cholera serum and virus to reduce the possibility of losses from hog cholera.

Healthy susceptible swine should be kept away from contaminated houses, lots, and pastures. Carcasses of diseased swine should be

deeply buried or burned. Offal from slaughter houses and pork trimmings from kitchens should not be fed to pigs.

Contaminated houses should be thoroly cleaned by being scraped, brushed, or swept, and should then be scrubbed with hot lye water, 1 pound of lye to 30 gallons of water. After the lye solution has been allowed to dry two or three days, the house should be sprayed with a 3-percent compound cresol solution (U.S.P.) or its germicidal equivalent. Lots and pastures used by swine infected with erysipelas should not be used again for hogs, lambs, ducks, or turkeys for at least a year. All manure from contaminated houses should be spread thinly on ground not used for these susceptible animals.

When purchasing replacement stock, extreme precaution should be exercised. Swine erysipelas is commonly introduced on a farm by the purchase of chronically infected animals that appear healthy. In fact the disease was introduced on many Illinois farms in 1936 thru the purchase of diseased feeder pigs from drouth-stricken areas.

Preventive measures are important as a means of avoiding future losses. Dirty hog houses and contaminated lots perpetuate infection.



A special veterinary conference on swine erysipelas, held at the University of Illinois, September, 1932

OTHER SWINE PUBLICATIONS

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Address College of Agriculture University of Illinois Urbana, Illinois Printed in furtherance of the Agricultural Extension Act approved by Congress May 8, 1914. H. W. Mumford, *Director*, Extension Service in Agriculture and Home Economics, University of Illinois. SWINE ERYSIPELAS, in both acute and chronic forms, has been recognized for many years in central and northern Illinois and has resulted in serious death losses in farm herds. Swine of all ages are susceptible.

The disease is most commonly introduced into a herd by infected animals that appear healthy or by feeding pork trimmings from infected swine. The disease organism may remain on the premises for an indefinite period and cause repeated outbreaks.

Anti-swine-erysipelas serum is effective in the prevention and control of the acute malady, which is sometimes confused with hog cholera; but it is of no value for the chronic type.

When either type of swine erysipelas is known to have been introduced on the premises, all houses should be thoroly cleaned and disinfected, and contaminated feedlots and pastures not used for hogs for at least a year.

Unless vigorous steps are taken to curb this disease, it may become a serious problem to swine growers thruout the state.