

infected. Shipment of such hogs should be restricted as long as the disease exists in this country. The marketing of swine fed raw garbage should not be permitted at any time, unless the pork from them is specially heat treated. Any vehicles used to haul swine that are fed raw garbage should be cleaned and disinfected after they are used.

Swine fed cooked garbage could be sent to market if inspected by a State or Federal inspector and if all swine on the premises are free of the disease. The swine that move should be accompanied by a certificate issued by a Federal or State inspector.

All vehicles, facilities, feed, watering and rest stations, and loading and unloading points used for swine within or from the State or States involved in an outbreak should be cleaned and disinfected each time they are used.

The informational phase of an eradication program is important. The cooperation of all parts of the livestock industry is necessary to control and eradicate the disease. Therefore, a first

step is to give all of them the facts about the disease and the need to control it. Problems should be discussed with representatives of the industry as the program progresses. They should be kept informed of gains made and setbacks encountered.

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## Swine Erysipelas (Diamond Skin Disease)

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SWINE erysipelas is present in the United States wherever hogs are raised. It is worst in regions, like the Midwest, where the hog population is dense. It is serious in Europe, parts of Asia, and North America.

R. Koch, F. A. J. Löffler, L. Pasteur, and M. L. Thuillier—all of whom are famous in scientific history—are associated with the discovery of the causative organism of erysipelas in the 1880's. Previously swine erysipelas was confused with anthrax. The work of Theobald Smith, in the former Bureau of Animal Industry, showed that the

disease was present in this country before 1900. G. T. Creech, also of the Bureau of Animal Industry, in 1921 demonstrated the relationship of diamond skin disease (a clinical form of swine erysipelas) and the causative agent of erysipelas. The disease caused little trouble until the 1930's, when A. A. Fosterman, a veterinarian in South Dakota, directed attention to its acute aspect. Swine erysipelas has since become one of the most serious hazards of the swine industry.

The causative agent is a bacterium, *Erysipelothrix rhusiopathiae*. It is a

hardy organism and can live in dead animal tissue and decaying matter. It can resist the effects of pickling, smoking, freezing, and drying of meats for long periods. It is rather sensitive to heat, however, and is killed at temperatures necessary to cook the chop or roast well.

The organism is considered to live in the soil as a saprophyte and can multiply under favorable conditions of moisture and in soil rich in humus.

Disinfectants commonly available and used according to directions will destroy it.

The organisms have been found in all parts of the world—and in a wide variety of animals, birds, fish, and insects. Of major economic importance is the effect of the disease in swine, turkeys, and sheep.

Infection of people is not uncommon (erysipeloid). It is an occupational hazard of veterinarians, persons who handle fresh meat and meat products, and commercial handlers of marine fish. Home butchering and processing of pork also has been the source of infection of people.

Probably the chief source of swine erysipelas infection is the organism present in the soil. A saprophyte, the organism can live on dead and decaying organic material—it does not require a living animal to maintain itself. Perhaps it has always been present as a soil organism.

Another source is the animal carrier. The organism can exist in the tonsils and the lymphoid tissue of the intestinal tract of apparently normal swine.

Hogs that become affected with swine erysipelas may or may not show actual clinical symptoms of the disease, but in each instance it is possible for the organism to be eliminated from the animal's body through the feces and urine. Hogs that die from the disease and remain undetected in the field can contaminate the soil and can be the source of an outbreak when eaten by other hogs.

Very likely birds and other wildlife that carry the organism contaminate

the soil in the hog lots with their droppings.

Animals become infected through contact with the soil that contains the organism—by ingestion of contaminated feed and water and by infection of wounds. Fighting may lead to wounds of the lips, face, and shoulders. Sharp rocks, glass, and other objects cause wounds in and around the claws or lips while rooting. Protruding nails or broken boards and pieces of wire fence also can cause tears and breaks of the skin.

One sick hog can cause an outbreak of the disease through its elimination of the organism in its dung and urine.

Because the organism can be found also in the circulating blood of an infected animal, there is a possibility that bloodsucking insects may transmit the disease to other hogs. G. Wellmann of Germany has shown this to be possible by experimentally transmitting the disease with biting flies.

SWINE ERYSIPELAS has a peculiar behavior: Why did the disease apparently lie dormant for many years and then become a menace in the early 1930's? Why does it show itself on a farm for several seasons and then seemingly disappear? Why do some areas remain known as erysipelas areas? Some outbreaks years ago could have been confused with hog cholera (they can occur at the same time), but the real reason is not known.

The other peculiarities can be explained on the basis of what is known to be possible. We have known for a long time that the causative agent can be one of relatively low virulence or one of great virulence: Because it is not always the same, its ability to produce disease can vary a lot. It is possible that we may even be dealing with different strains or variants of the same organism. At any rate, since the infectiveness of an organism can be increased or lowered by artificial means in the laboratory, it is very likely that such external factors as climate and soil conditions also can alter its disease-pro-

ducing ability. This could then be demonstrated on the farm by either a high incidence of disease, with many dead animals, or a low incidence, with just a few sick ones in the herd.

Swine erysipelas also can be present in a herd in such a mild form that it remains unnoticed. Many animals therefore become immune through natural infection. On a farm where this condition occurs, one could easily conclude that erysipelas died out or disappeared, although actually it was still there. All that would be needed to change this situation into an outbreak would be an increased virulence of the organism and the presence of susceptible hogs.

THE CLINICAL SYMPTOMS of swine erysipelas are classed as acute, subacute, and chronic.

Acute swine erysipelas is characterized by its sudden onset. Many animals in the herd may be affected at once. Only a few may be visibly sick, but a number of others may run temperatures of over  $104^{\circ}$ . Sick pigs tend to stay where they are bedded, yet at the same time their eyes give an impression of alertness. If stirred up and forced to move, they will protest with squeals as though it were painful to put weight on their feet. Sometimes the animals may show a peculiar stilty and stiff gait, or they may stand with their feet under them, and head hanging down, so that the back line has an arched appearance. The attitude becomes one of dejection or depression. Left alone, they quickly make themselves as comfortable as possible in the bedding again. Most animals show little desire for food or go off feed altogether. A not uncommon symptom is nausea and vomiting. Some animals breathe with difficulty and have a husky cough. If there is any change in the feces, it is more likely to be dry and reduced in quantity. Several of the pigs may die suddenly; others will be obviously sick, as we described, and a few may die in 7 or 10 days.

During the acute phase, many hogs may develop what is called diamond

skin disease, or the urticarial form of swine erysipelas. This phase is marked by light-pink to dark-purple areas on the skin that usually become raised and firm to the touch and in most instances have clearly defined borders. The shape is quite characteristic when seen or felt, because the outline of the skin lesion assumes a square, a rectangular, or a rhomboidal geometric pattern. The lesions might join and cover quite a large area of the skin. Although the lesions are best seen on a light-skinned animal, they can be felt (but are easily overlooked in either case, particularly if they are not too numerous) on animals with darker skins. The number of "diamond skin" lesions can vary from one to so many that it would be hard to count them.

The skin lesions may disappear in several days, or they may become areas of dry, firm, darkened, scabby, dead skin and under tissue. These areas of dead skin will eventually fall off, leaving an ugly scarred area. It is here that one will also see loss of tips of the ears and tail. Secondary infection usually takes place during this stage and prolongs the healing process. Although the mortality rate of hogs showing diamond skin disease may not be particularly high, animals that have it will probably be affected with arthritis and the owner will be left with a number of pigs, which are unprofitable to feed out.

It is easier to think of the subacute form as being less severe than the acute form. The animals seem to be less sick. The temperature may not be so high. The hogs may show more interest in food. A few diamond-skin lesions may appear. The animals do not stay sick for so long a time.

Many hogs that live through the acute and subacute forms do not always recover completely, because an infection of the joints commonly occurs during the attack and can progress to a chronic or arthritic form. Infection of the joints also can occur without the animal's being noticeably sick. Infection of the knee,

hock, and the toes is most frequent. Besides the noticeable lameness, shifting of weight, and other signs of pain in the legs, there is a gradual enlargement of the affected joint, which will also feel hot to the touch. Sometimes the symptoms disappear altogether, but may show up again. Many, however, get progressively worse, and the arthritis leads to bony enlargements and thickening of the soft parts, resulting in permanent deformity of the affected joint. This condition in hogs is similar to rheumatoid arthritis in man. Involvement of the heart is not uncommon. It is referred to as endocarditis and brings about a cauliflower-like growth at the site of the inflammation.

Injuries and the absence of a proper nutritional balance or adequate assimilation of the mineral elements can cause lameness. Thus it might be confused with swine erysipelas. Other recognized causes of arthritis include infection with streptococcus, staphylococcus, brucella, and salmonella organisms. S. H. McNutt, T. S. Leith, and C. K. Underbjerg, as well as W. P. Switzer, isolated a filterable agent that may be of significance in this regard. Despite all these possibilities, we know of no survey into the causes of arthritis in swine that would alter the picture presented by C. G. Grey, O. L. Osteen, and H. W. Schoening in 1941. They examined 472 joints of hogs that came from 91 counties in 14 States; they found the swine erysipelas organism in 375, or 76.5 percent, of the joints.

DEATH LOSSES can run from none to 75 percent, but swine erysipelas is more noted for its crippling effect than for the deaths it causes. The hogs cannot get around so well; for a while they show little interest in food, and therefore they become unthrifty. This systemic effect of the disease—plus the permanent damage to the joints and even the heart—leaves an animal in such a condition that it never does very well. It remains rough, undersized, or backward in growth.

Those that do reach the market may

be docked at the packinghouse. Many thousands of dollars are lost annually in this manner, as diseased tissue requires trimming out as well as the removal of visibly affected joints.

THE DIAGNOSIS of swine erysipelas at times can be quite difficult, and it is well to remember that diseases—hog cholera and salmonellosis, for example—other than erysipelas can be present in herds at the same time.

Whenever an owner experiences sudden deaths in hogs for no apparent reason, he should consult his veterinarian immediately.

Diagnostic serological tests have been developed, but there are still definite limitations on their general use. Service laboratories are invaluable in assisting the veterinarian in his diagnosis.

Early diagnosis and treatment can put hogs on the road to recovery sooner and with less overall damage to the herd. Antibiotics alone and antibiotics with a specific immune serum are useful in treating swine erysipelas.

Biologics are available to the veterinarian. Hyperimmune serum was the only tool available before 1938, and it was not always adequate. Vaccination, for which a living virulent culture of the causative organism of swine erysipelas was used along with hyperimmune serum, had been practiced in Europe for many years.

The Department of Agriculture, in cooperation with the Nebraska Bureau of Animal Industry and the University of Nebraska, set up a program for introducing that method of control on an experimental basis. In the beginning only a few States were permitted to use live-culture vaccine, but as the results from the field were quite encouraging, the program was extended under the terms of a cooperative agreement. Up to 1956, 26 States were permitted to use the vaccine. The number of pigs vaccinated has increased; in 1954 they numbered more than 5 million head. An average of more than 2 million swine has been vaccinated annually since 1943.

Other methods of vaccination have used the principle of a living but mild vaccine, or what has been called an "avirulent" vaccine. This type of vaccine does not require the use of the protective serum.

There also have been introduced bacterins that do not contain living organisms. One is called the adsorbate and the other is the lyso bacterin. The advantage of such products is that they eliminate any possibility of spreading the disease and accidentally infecting the veterinarian while vaccinating the swine. These bacterins show considerable promise as additional tools for the control of swine erysipelas.

No vaccination procedure offers complete protection in all circumstances, however, and it is a common mistake to think that they do.

AN OWNER can do much to prevent disease by following established principles: Inspect his animals regularly; learn as much as possible about the health of animals and the conditions on the premises where replacement

animals are bought; isolate newly purchased animals for at least 30 days before introducing them into the herd; keep pens and pastures clean and sanitary; use disinfectants often and according to directions; and feed a balanced ration.

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## Diseases of Baby Pigs

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NEARLY one-third of all pigs born each year die from various disorders and hazards.

Not all of the loss can be said to be due to diseases in the strict sense. The causes, which often are ill-defined and overlapping, include various management factors that involve housing, facilities, and care; various prenatal factors, which involve several variables such as genetics, physiology, disease organisms, and nutrition; and post-natal factors reflected in physiological well-being of dam and pig, nutrition,

infections, parasites, numbers of pigs per litter, and others.

The multiple nature of the causes is evident in the cataloging of deaths outwardly apparent as due to mashing or injury. Many of the deaths are entirely physical. They are caused by the sow and often indirectly by inadequate housing and lack of protective guardrails, brooder lamps, and other pen facilities, of which the undersized, weak, and sick pigs are more prone to become victims.

The reasons why they are under-