INTRODUCTION

Maximizing reproductive performance depends on the adequacy of genetics, nutrition, environment, management, and disease control. Each of these factors should be systematically evaluated whenever a decrease in reproductive efficiency occurs.

This paper reviews the major infectious diseases capable of reducing reproductive performance in swine. The etiology, clinical signs, diagnosis, and control methods for the major diseases are discussed. This review also includes infectious agents that cause only sporadic reproductive problems.

VIRAL AGENTS

SMEDI Syndrome

The term SMEDI is an acronym referring to stillbirth, mummification, embryonal death, and infertility in swine. In this review, reproductive failure resulting from porcine enteroviruses, a porcine parvovirus, a reovirus, and an adenovirus are discussed as part of the SMEDI syndrome.

Eight sero-groups of porcine enteroviruses can cause the SMEDI syndrome (8). Infection of gilts at or shortly before breeding can cause infertility (6). Inoculation of gilts with these viruses during early gestation can cause embryonal death and can result in pseudopregnancy. Pseudopregnancy is not entirely understood, but embryonal death at Day 30 of gestation can result in complete embryonal absorption without corpora luteal regression. The female, therefore, remains endocrinologically pregnant but fails to return to estrus until the time of normal parturition (18). Pseudopregnancies are a common cause of reproductive failure under field conditions, and presumably result from viral infection that causes embryonal death.

Enteroviral infections from approximately Days 30-50 of gestation until shortly before birth are usually manifested as fetal mummifications (7). Sunken eyes and darkened skin are the most obvious indications that fetal death has occurred during gestation and that the mummification process has begun. Placental degeneration is also associated with enteroviral infection, but whether placental changes are primary or secondary is not known. Enteroviruses have a definite affinity for the fetus, and occasionally will produce polioencephalomyelitis in the newborn pig. Enteroviral infection seldom causes obvious diseases in adult animals, except for reproductive failure.
A porcine parovirus has been associated repeatedly with infertility, abortions, and neonatal losses of British pigs (2). Fetuses inoculated with a porcine parovirus before Day 72 of gestation died 5 to 22 days post-infection, whereas all fetuses infected after 72 days of gestation survived (1). In Illinois, the authors have studied a naturally occurring parovirus infection that caused a 50% reduction in reproductive performance as a result of increased embryonal death and infertility.

Inoculation of a reovirus isolated from aborted swine fetuses into sows on Day 40, 60 or 85 of gestation resulted in fetal mummification, stillbirth and weak pigs (13). There is limited evidence indicating that an adenovirus can also cause reproductive failure.

Enteroviral infections appear to be ubiquitous. In nearly every herd studied by Dunne, antibodies to two or more enteroviruses were detectable. Our own studies on the epidemiology of the porcine parovirus revealed that it is extremely widespread. Gilts seem to be more commonly affected by the SMEDI syndrome than sows. The SMEDI syndrome often follows the introduction of new boars into a previously closed breeding herd, indicating that carrier boars may be responsible for introducing pathogenic viruses into the herd. Well-managed commercial herds that have a closed population of females, and only periodically introduce new boars from other farms, seem to have more SMEDI syndrome outbreaks than purebred herds containing animals of multiple origin. Apparently herds containing animals originating from many farms are more likely to be exposed to and immunized against the viruses capable of causing the SMEDI syndrome.

The viruses causing the SMEDI syndrome are probably passed venereally or by direct contact. The porcine parovirus has been isolated from semen, and the experimental addition of the parovirus to semen resulted in decreased litter size when compared to the non-infected controls (2).

Methods of diagnosing the SMEDI syndrome include virus isolation, antibody changes in paired serum samples, and detection of antibodies in the serum or body fluids of stillborn or colostrum-deprived pigs. Virus recovery, under natural or experimental conditions, is less than 10% effective (7). Because the enteroviruses and the parovirus are widespread in nature, detection of viral antibodies in the serum of a gilt or sow exhibiting reproductive failure is meaningless; the female may have been infected prior to breeding. Therefore, serologic tests are of diagnostic value only when paired samples are available. The first sample should be collected just prior to breeding and the second collected after the occurrence of reproductive failure. Enteroviral antibodies in the serum and body fluids of stillborn or colostrum-deprived pigs are presumptive evidence of an in utero viral infection. Serum samples from newborn
pigs may be collected from fully developed stillborn or colostrum-deprived live pigs taken from litters which have one or more mummified fetuses, one or more stillborn pigs, or a very small litter. Pigs selected must not have suckled the dam because any absorbed colostrum could reflect the dam's antibodies, not the pig's. Because of the difficulty in confirming a diagnosis, most of the SMEDI syndrome diagnoses made by veterinary practitioners and diagnostic laboratories are made after other likely causes of reproductive failure have been eliminated.

There are no therapeutic agents or commercially available vaccines for the control of the SMEDI syndrome. Because the epizootiology of these viruses is not understood entirely, methods of natural immunization are empirical. The critical period for controlling the SMEDI syndrome is the three weeks prior to breeding and the entire gestation period (6). During this period, producers should avoid exposing the breeding herd to new animals that may be carriers of viruses that cause the SMEDI syndrome. Because many suspected SMEDI syndromes occur after the introduction of new boars, and because enteroviruses are shed in the feces, some veterinarians recommend that the fecal material of new boars should be mixed in the feed of gilts and sows prior to breeding. Alternatively, producers can mix sows and gilts and rotate them into pens containing boar fecal material on several occasions prior to breeding. Studies concerning the epidemiology of SMEDI viruses suggest that contact through adjacent pens is not sufficient to establish a common enteroviral infection and subsequent immunity. Whenever the SMEDI syndrome is diagnosed, the affected gilts and sows should be retained in the breeding herd because they will presumably be immunized to the causative virus.

**Pseudorabies**

Pseudorabies, formerly called Aujeszky's disease, is of increasing importance in the United States. The disease can cause death in all ages of swine, and abortions and mummified fetuses in pregnant swine. The greatest losses have occurred in garbage-fed swine and feeder pig operations, but more recently have been occurring in all types of production systems.

Pregnant animals usually will exhibit coughing, fever, and anorexia followed by constipation and depression. Infections early in gestation often result in abortions, whereas infections later in pregnancy usually produce fetal death and mummification. Natural infection in baby pigs can occur by inhalation or ingestion of the virus. The primary site of viral replication is the upper respiratory tract. Within 36 hours after birth the affected pigs exhibit depression, incoordination, and trembling. Some pigs can only move backwards or in circles and some exhibit spasms involving...
opisthotonus and prostration. The major lesions are marked inflam-
mation of the meninges and intranuclear inclusions bodies in the
brain. Temperatures of infected pigs rarely exceed 105°F. Very
few newborn pigs survive the infections; however, litters three to
four weeks old at the time of the initial exposure seldom suffer
greater than 50% mortality. Serum neutralization tests detect the
presence of pseudorabies antibodies, but do not confirm the
presence of the active disease. Confirmation of an epizootic out-
break requires virus isolation (15).

Live vaccines are being used in European herds but because of the
sporadic occurrence of pseudorabies in the United States, vaccina-
tion is not recommended. Control in this country is limited to
the use of specific antiserum and the isolation of sick animals
(9). Because antiserum probably does not eliminate the excretion
of the infective virus, physical separation of diseased and healthy
animals is imperative (14). Other methods of controlling the
disease include removal of sheep and cattle from direct contact with
the infected swine and cooking of all garbage fed to swine. Be-
cause dogs can become infected with pseudorabies, their movement in
and around the swine herd should be controlled.

Surveys have shown a 5-10% prevalence of pseudorabies (10). The
disease may be endemic in some herds suggesting that movement of
carrier animals into a susceptible herd is potentially dangerous.
On most farms where pseudorabies has occurred, the period of
disease signs usually last about two weeks, after which the disease
seems to disappear, perhaps as the result of immunity.

Swine Influenza

Veterinary clinicians and diagnosticians speculate that influenza
is a major cause of swine reproductive failure. Woods and Mansfield
(20) have reported mummification, abortion and weak pigs following
experimental inoculation of the influenza virus into a limited
number of gilts. Other researchers have failed to show an associa-
tion between swine influenza and reproductive failure. A serologic
survey undertaken to determine the prevalence of antibody titers to
swine influenza revealed positive titers in 12.3% of the animals
tested (19).

Clinical signs of influenza, include a rapid onset of coughing,
dyspnea, anorexia, and inappetence. Veterinarians also suspect that
swine influenza can cause stillbirths, mummified fetuses and early
embryonal mortality resulting in decreased conception rates. Diag-
nosis is usually based on the herd history and clinical signs.
Paired serum samples, the first taken in the acute phase of the
disease and the second 2-3 weeks later, can be used to measure in-
creasing antibody levels to swine influenza. By the time repro-
ductive failure is apparent, however, the acute influenza is no
longer apparent.

One method of controlling influenza-induced reproductive loss is to use a higher percentage of sows for autumn breeding and winter farrowing, the time when influenza is most prevalent. Sows, as compared to gilts, are more likely to be previously exposed and, therefore, immunized to the influenza virus. Influenza vaccines have not been successful in the control of the disease in the United States (20).

**Hog Cholera**

Whereas the United States has almost completely eradicated hog cholera, the disease is still prevalent in many countries, and it could reappear in the United States. Hog cholera is a viral disease affecting swine of all ages. Clinical signs of hog cholera include fever, depressed appetite, and slow, painful movement. Cholera-induced fetal mummification often occurs in pregnant animals. A tentative diagnosis is confirmed by fluorescent antibody evaluation of splenic and tonsillar tissue. Because the basis of control is eradication, treatment of the affected animals and the use of vaccines is prohibited. When hog cholera is suspected, state and federal regulatory veterinarians should be contacted immediately.

**BACTERIAL AGENTS**

**Leptospirosis**

Porcine leptospirosis usually causes abortions, stillbirths, and perinatal losses of baby pigs and should always be suspected whenever reproductive failure is being investigated. In the United States, four major serotypes have been reported to affect pigs. They are *Leptospira pomona*, *L. grippotyphosa*, *L. canicola*, *L. icterohaemorrhagiae*.

*Leptospira pomona* is the most important cause of swine leptospirosis in the world, but within specific areas, other serotypes may be more prevalent. During a 10-year period, the proportion of swine serum containing antibodies in Illinois was *L. grippotyphosa*, 3.3%, *L. pomona*, 2.4%, *L. icterohaemorrhagiae*, 0.8%, and *L. canicola*, 0.5% (12). While the incidence of *L. pomona* has decreased, the incidence of the other serotypes have remained quite constant. Table I summarizes the major carriers of the various serotypes in Illinois.
**Table I - Leptospirosis in Illinois**

<table>
<thead>
<tr>
<th>Serotype</th>
<th>Major Carrier</th>
</tr>
</thead>
<tbody>
<tr>
<td>L. pomona</td>
<td>skunk, ferral cat</td>
</tr>
<tr>
<td>L. grippotyphosa</td>
<td>raccoon, opossum</td>
</tr>
<tr>
<td>L. canicola</td>
<td>dog</td>
</tr>
<tr>
<td>L. icterohaemorrhagiae</td>
<td>rat, raccoon</td>
</tr>
</tbody>
</table>

Non-pathogenic serotypes detected in swine in the United States include L. autumnalis, L. australis, L. bataviae, L. hardjo, and L. ballum (11). Because L. autumnalis has antigenic similarities with L. pomona and L. grippotyphosa, the possibility exists for cross reactions during routine serologic testing, and this can result in an inaccurate diagnosis.

The incubation period for leptospirosis is usually one to two weeks. The acute illness includes only a moderate fever and anorexia, but leptospires are present in the urine 10 days after the onset of acute illness and are sometimes detectable up to 12 months. Reproductive failure is not manifested until approximately 10 days after the acute infection.

In the pregnant sow or gilt, the stage of pregnancy is an important factor in determining the pathogenesis of leptospirosis. There are no gross effects on the embryos when infection occurs during the first month of pregnancy. Infection during the second month of pregnancy may cause fetal death and resorption. During the third month of gestation abortion occurring 10-38 days following infection is a common sequela of leptospiral infections. Leptospires can penetrate the placental barrier, and within several days they can be isolated from the blood and organs of the affected fetuses.

Common sources of leptospirosis include infected urine, contaminated surface water, direct contact with the urine of shedder animals, and infected tissues, such as the carcass of a rodent. The leptospires enter the body through the skin or mucous membranes. Because swine can shed a large number of organisms over an extended period of time, they are a serious threat to adjacent animals and to humans.

A diagnosis of leptospirosis is made by use of a herd history, necropsy findings, and serologic tests. The herd history should determine vaccination schedules, methods for leptospirosis testing of new stock entering the herd, and exposure to wildlife, including rodents.

Post-mortem examination of infected pigs reveals the presence of petechial hemorrhages in the cortex of the kidney and occasionally in the liver. Fetal membranes of the aborted litters show only small hemorrhages and edema. Histopathologic findings include interstitial nephritis and edema, congestion and hemorrhage of uterine tissue, and meningoencephalitis.
A definitive diagnosis of leptospirosis can usually be achieved by measuring antibody titers from a single sample of serum from infected dams. Because the acute disease precedes the observed reproductive failure by about two weeks, antibodies to leptospiral organisms are detectable at the time of the observed reproductive failure. A reliable procedure for measuring leptospiral antibody titers is the microscopic agglutination test (11). Because the test requires the use of serospecific antigens, laboratories should be requested to check for all serotypes of leptospires within the geographic location. Many laboratories have now successfully developed FA techniques to diagnose leptospirosis.

Control of leptospirosis is dependent upon a satisfactory vaccination program and the isolation of the herd from sources of leptospires. Breeding swine should be vaccinated with bacterins for each pathogenic leptospiral serotype that is prevalent in the area. To be assured of continued protection, bacterin injections should be repeated every six months or prior to every breeding. Currently available leptospirosis bacterins are: \textit{L. pomona}, \textit{L. canicola}, \textit{L. icterohaemorrhagiae}, \textit{L. grippotyphosa}, and \textit{L. hardjo}. Confinement of breeding swine will reduce exposure to wildlife and substantially reduce the probability of leptospirosis.

Treatment of leptospirosis using chemotherapy is possible, but usually impractical. Dihydrostreptomycin at the rate of 25 mg. per kg. of body weight in a single dose is usually an effective treatment (17). Chlorotetracycline fed at 400 gm. per ton for 14 days will stop losses caused by leptospirosis; however, the organisms will not be eradicated from the kidney. Oxytetracycline fed at 500 gm. per ton of feed for 14 days appears to prevent the spread of the disease within the herd. While treatment at the time of abortion or stillbirth does not affect the course of the acute infection prior to treatment, it may reduce the spread of leptospirosis throughout the herd.

\textbf{Brucellosis}

Although the incidence of swine brucellosis has decreased markedly during the past two decades, it remains as a cause of abortion and infertility. The disease, caused by \textit{Brucella suis}, is usually spread by ingestion of the organism, although it can be transmitted venereally. Sterility or infertility may be the only manifestation of brucellosis. Infertility is usually the result of early unobserved abortions or fetal resorptions (4). The cause of infertility in sows appears to be a persistent metritis. The incidence of abortion is usually less than 30% in naturally occurring brucellosis, but it can vary from 0-100%. The time of abortion ranges from 21 to 105 days of gestation, with the average being 65-72 days.
In the boar, brucellosis will cause sterility and reduced fertility as a result of orchitis and infection of the accessory genital organs. Testicular changes result in decreased spermato genesis and depressed libido.

A positive diagnosis can be made using the standard seroagglutination test (Card Test). Treatments and immunization methods have not proven effective in controlling brucellosis. The best control is eradication of the disease. Purchased breeding stock should come from validated brucellosis-free herds. If this is not possible, brucellosis testing prior to purchase of new stock is imperative.

Other Bacterial Agents

Bacteria capable of causing sporadic abortion, stillbirths, and endometritis include: Streptococcus spp., Staphylococcus aureus, Salmonella spp., Escherichia coli, Corynebacterium pyogenes, Pseudomonas spp., Mycobacterium tuberculosis, Listeria spp. and Pasteurella multocida (15). Erysipelothrix rhusiopathiae and Pfeifferella whitnori have also been incriminated as a cause of reproductive failure (5). Based upon the limited evidence available, it is impossible to judge the economic importance of the reproductive failure caused by these bacteria. Their diagnosis depends upon the continued efforts of diagnostic laboratories to identify the cause of reproductive failure.

PARASITES

Eperythrozoonosis

Eperythrozoonosis can cause intrauterine infections resulting in the production of weak pigs (16). There is speculation that eperythrozoonosis is widespread. Clinical evidence suggests that 800 gm. of tetracycline/ton of feed will eliminate the problem when fed for two weeks prior to farrowing. Diagnosis of the disease can be achieved by measuring antibody in the serum.

Toxoplasmosis

Toxoplasmosis, caused by Toxoplasma gondii has been related to stillbirths and premature births. A 50% mortality rate in baby pigs has been reported on farms with a confirmed toxoplasmosis infection. Some deaths were related to stillbirths and premature births, however the majority of pigs died between one and three weeks of age (3).
<table>
<thead>
<tr>
<th>Disease</th>
<th>Clinical Signs</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Prevention and Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parvovirus</td>
<td>-stillbirths</td>
<td>-paired serum samples from breeding stock collected at start of breeding period &amp; time of farrowing; -antibody titers in fetal fluid of colostrum-deprived pigs</td>
<td>None</td>
<td>-closed herds</td>
</tr>
<tr>
<td></td>
<td>-mummification</td>
<td>-isolate sick animals from breeding stock &amp; gilts 30 days prior to breeding</td>
<td>Antiserum</td>
<td>-transfer of fecal material between boars &amp; females 30 days prior to breeding</td>
</tr>
<tr>
<td></td>
<td>-embryonic death</td>
<td>-virus isolation</td>
<td>Supportive</td>
<td>-retain infected animals</td>
</tr>
<tr>
<td></td>
<td>-infertility</td>
<td>-serum neutralization to detect antibody levels</td>
<td></td>
<td>-isolate movement of dogs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-herd history</td>
<td></td>
<td>-high percent of sows for autumn breeding</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-clinical signs</td>
<td></td>
<td>-eradicate</td>
</tr>
<tr>
<td>Enterovirus</td>
<td>-depression</td>
<td>-Fluorescent Antibody Test (FAT)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reovirus</td>
<td>-incoordination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenovirus</td>
<td>-weak pigs at birth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SMEDI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudorabies</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swine Influenza</td>
<td>-stillbirths</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hog Cholera</td>
<td>-mummification</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### TABLE II (continued)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Clinical Signs</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Prevention &amp; Control</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Leptospirosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>L. pomona</em></td>
<td>-abortion</td>
<td>-Leptospiral antibody titers</td>
<td>Not practical</td>
<td>-vaccination with specific serotypes</td>
</tr>
<tr>
<td><em>L. grippotyphosa</em></td>
<td>-stillbirths</td>
<td></td>
<td></td>
<td>-herd isolation</td>
</tr>
<tr>
<td><em>L. canicola</em></td>
<td>-mummified fetuses</td>
<td></td>
<td></td>
<td>-closed herd</td>
</tr>
<tr>
<td><em>L. icterohemor-rhagiae</em></td>
<td>-perinatal losses</td>
<td></td>
<td></td>
<td>-add lepto-free boars</td>
</tr>
<tr>
<td><strong>Brucellosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Brucella suis</em></td>
<td>-sterility</td>
<td>-standard sero-agglutination test (Card Test)</td>
<td>None</td>
<td>-purchase breeding stock from validated brucellosis free stock</td>
</tr>
<tr>
<td></td>
<td>-infertility</td>
<td></td>
<td></td>
<td>-test all animals entering herd</td>
</tr>
<tr>
<td></td>
<td>-abortion</td>
<td></td>
<td></td>
<td>-eradication</td>
</tr>
<tr>
<td></td>
<td>-stillbirths</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-weak pigs</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
REFERENCES

1. BACHMANN, P.A.: Institut de Microbiologie, Ludwig Maximilians University, Munich, Germany: Personal communication, 1974.


McADARAGH, J.P.: Animal Disease Research and Diagnostic Laboratory, South Dakota State University, Brookings: Personal communication, 1974.


SMITH, A.R.: Department of Clinical Veterinary Medicine, College of Veterinary Medicine, University of Illinois, Urbana: Personal communication, 1974.


