Volume 52 | Issue 2 Article 4

1990

Salmonella choleraesuis as a cause of respiratory disease in growing and finishing swine

J. R. Kolb
Iowa State University

L. J. Hoffman
Iowa State University

Salmonella choleraesuis as a cause of respiratory disease in growing and finishing swine

J. R. Kolb, DVM*
L. J. Hoffman, BA, MS, DVM**

Introduction

Respiratory disease in swine has been increasing as production methods intensify. As the swine industry becomes more competitive and profit margins continue to narrow, the impact of respiratory disease on production and profitability will be of major importance. Veterinarians must be able to diagnose, treat, and most importantly, consult with producers to prevent these management diseases.

Respiratory disease in swine presents the practitioner with a challenging diagnostic problem. The etiology is often multifactorial and involves complex interrelationships between host, pathogen(s) and environment. The environment of intense production facilities is of major importance in respiratory disease. Such factors as temperature, ventilation and animal flow and density should be included in diagnostic investigations.

The primary pathogens associated with respiratory disease in swine are *Actinobacillus (Haemophilus) pleuropneumoniae, Salmonella choleraesuis, Mycoplasma hyopneumoniae*, pseudorabies virus, and swine influenza virus. *Ascaris suum* (visceral larval migrans) and Metastrongylus species are internal parasites which can also damage the lung and cause dyspnea. Many secondary pathogens, such as *Pasteurella multocida*, are able to infect the compromised porcine lung. This paper is a review of *S. choleraesuis* and its role as a respiratory pathogen in growing and finishing swine.

*Dr. Kolb is a 1990 graduate of the College of Veterinary Medicine.
**Dr. Hoffman is a professor in the Department of Veterinary Microbiology and Preventitive Medicine and the Veterinary Diagnostic Lab at Iowa State University.

Etiology

Salmonella species are members of the family Enterobacteriaceae, and are gram negative bacilli. They are divided into groups A through I by somatic O antigen determinants, *Salmonella choleraesuis* being a member of Group C_1. Further identification into serotypes is done with more definitive serologic evaluation. Over 2400 serotypes are currently identified .

Epidemiology

Swine are commonly exposed to low numbers of various *Salmonellae* by ingestion of feed and water contaminated with swine feces. Some of these organisms are normal inhabitants of the intestinal flora. Others may only transiently inhabit the intestinal tract. Most of the *Salmonella* species which are commonly present in a pig's environment do not cause clinical disease.

The majority of clinical isolates from cases of pneumonic salmonellosis are *S. choleraesuis* variety *kunzendorf*. This serotype is highly host adapted and is rarely isolated from other species.[1,2] Various reports indicate that *S. choleraesuis* comprises a large percentage of *Salmonella* isolates from swine.[2,3] The infected shedding pig and its contaminated environment are the sources of infection. It is known that carriers can shed organisms for greater than four months post infection.[1] Although disease caused by *Salmonellae* is most common in growing swine, especially feeder pigs less than four months old which have been commingled, it can occur at any age. Salmonellosis is rare in suckling pigs, presumably due to protective maternal antibody.[1] Other *Salmonellae* recovered from cases of clinical disease are *S. typhimurium* and *S. typhisuis*. However, pneumonia is not associated with

these organisms. In discussing pneumonic salmonellosis, only *S. choleraesuis* will be considered as an etiological agent.

Pathogenesis

Organisms are shed in feces or respiratory secretions, and ingested by infected pigs. Whether this precedes clinical disease or occurs when clinically affected animals shed bacteria is not clear. Both methods of transmission may occur. Drinking contaminated water and use of alkaline feeds are associated with increased risk of infection by increasing gastric transit rates and increasing gastric pH, respectively.[1] Clinical disease is related to infective dose, serotype virulence, and host susceptibility.

Organisms invade the mucosa and are transported to Peyer's patches in macrophages. Replication in lymphoid tissue occurs whether infection proceeds to clinical manifestation or not. The establishment of the carrier state occurs when organisms survive intracellularly in intestinal lyphoid tissues. *S. choleraesuis* is maintained in these lymphoid tissues and is shed in feces under stressful conditions. Organisms are also shed in respiratory secretions of pneumonic pigs. Antimicrobial treatment will not clear the carrier state in swine.

Septicemia often follows initial replication. Interstitial pneumonia ensues in approximately 80% of cases.[3] A cranioventral bronchopneumonia develops after the interstitial disease and can be superimposed on it. This develops either from an ascending alveolitis or an outpouring of exudates into alveoli.

Non-respiratory damage may include endotoxin induced thrombosis and necrosis of the intestinal epithelium. Ulcer formation is most prominent in the caudal small intestine, cecum, and colon. Thrombosis of the gastric fundic mucosa is also noted.

Clinical Signs

Disease is most common in stressed commingled feeder pigs, especially in the first 10-14 days after arrival. Pigs become restless, go off feed and huddle as body temperatures rise to 105-107^{0}F (40.5-41.7^{0}C) or more. Dyspnea and abdominal "thumping" respiration ensue.

When septicemia develops, the ventral abdomen becomes cyanotic, as with *Actinobacillus pleuropneumonia*. Ears and the tips of extremities also turn purple. Diarrhea may become evident 24-72 hours after the onset of clinical signs. It is typically pasty in nature, but not explosive.

Gross lesions

Lungs are diffusely congested with interlobular edema, consolidation and generalized hepatization. Petechia may be noted on the pleura, as well as the heart and kidneys.

Non-pulmonary lesions are typical of septicemic disease. There is notable splenomegaly and hepatomegaly, and subtle evidence of paratyphoid nodules. These nodules are pale and give the liver a miliary appearance. Lymph nodes are mottled and congested. Cyanosis of the ventral abdomen, ears and extremities occurs. The gastric fundic mucosa may become congested or infarcted. The caudal small intestine, the cecum and the colon may have well circumscribed "button ulcers" with a fibrinonecrotic crust.

Histopathological lesions

Respiratory lesions are commonly those of a subacute diffuse histiocytic interstitial pneumonia. Histiocytes will be seen in thickened alveolar walls. A purulent bronchiolitis may also be noted. Alveolar walls are often edematous with congested vasculature. Fibrinous exudates are also noted in some cases. Typically there is little to no exudate within alveolar lumens.

The most diagnostic lesion is the paratyphoid nodule in the liver. These nodules are clusters of histiocytes in necrotic foci which correspond to the pale foci seen grossly. These are commonly present in septicemic salmonellosis, and are reportedly only seen with this condition.[1] Hepatic sinusoids will often have an increased cellularity, which is inflammatory in nature.

Multiple fibrinoid thrombi are found in vessels throughout the body, especially cyanotic skin, glomeruli, fundic mucosa, and occasionally the lungs.[1] The mononuclear phagocytic system exhibits multifocal hyperplasia with a generalized histiocytosis.

Diagnosis

Differentials for pneumonic salmonellosis includes *A. pleuropneumoniae,* pseudorabies virus and swine influenza virus. Erysipelas should also be considered when making of differential diagnosis of acute deaths with lesions of septicemia.

Definitive diagnosis is based on isolation of *S. choleraesuis*. Tissues for isolation attempts should include lungs, especially the cranioventral portion of the caudal lobe, mesenteric lymph nodes, spleen, liver, and kidneys.[4] Feces may be cultured when tissue specimens are not available.

Tetrathionate (TT) enrichment broth should be inoculated with minced tissue, fecal material, or rectal swabs and incubated at 43°C for 24-48 hours to decrease growth of contaminants.[5] A sample is then streaked on blood agar. A modification of this technique involves maintaining the TT broth at room temperature for five days, then reinoculating 0.5 ml of the original volume into a second TT broth.[6] Preliminary results have been promising with this double enrichment method, but may be too time constraining for most laboratories and clinics.

Blood agar and the selective media brilliant green, Hektoen enteric and Salmonella-Shigella agars can be used to facilitate identification. MacConkey agar and tergitol-7 may also be incorporated in the culture protocol. Any suspect colonies should then be inoculated into Kligers iron agar, SIM, and urea agar tubes. Some *Citrobacter* species may produce similar biochemical results but are contaminants. Lysine iron agar can be utilized to differentiate between *Citrobacter* and *Salmonella*. Self contained rapid identification systems such as API 20E and Micro Id can also be used for identification purposes but may be prohibitively expensive for the practitioner.

Isolates which produce typical reactions of *S. choleraesuis* should be subjected to serological grouping for final confirmation as group C_1. Complete serotyping procedures are conducted at reference laboratories.

Isolation attempts from animals with septicemia may not require extensive use of selective media. Liver, lung, kidney, and spleen often yield highly pure cultures of *S. choleraesuis*. As such, use of multiple selection media may be unnecessary for experienced personnel.

Treatment

Isolation and antimicrobial sensitivity testing are essential in treatment of salmonellosis. Sensitivities vary with isolates. Plasmid mediated transfer of antimicrobial resistance is often present. Prior knowledge of the antimicrobial sensitivity pattern of a particular *Salmonella* in a herd can facilitate effective early treatment. Clinically affected animals should be isolated in treatment pens to prevent further dissemination of the organism and facilitate individual animal treatment. Colored marking chalk or some other means of identification should be used to monitor the duration of therapy and evaluate response to the treatment used.

The chart below lists some antimicrobials which have been reported to be effective in vitro against *S. choleraesuis* based on susceptibility testing at the Iowa State University Veterinary Diagnostic Lab.[4]

Antimicrobial drug	% isolates sensitive
Ampicillin	76
Gentamicin	100
Spectinomycin	80
Trimethoprim-Sulfa	97
Neomycin	80

Many of the antimicrobials listed above, if used in treatment of salmonellosis would be considered extra-label in nature. As such, a meaningful veterinarian/client relationship must exist before the drugs can be administered. Long withdrawal times are required with parenteral use of aminoglycosides. Their use should be avoided in animals within at least 60-90 days of slaughter. Gentamicin soluble powder may also be used in water medication, but is poorly absorbed. Sulfa drugs alone were virtually never effective. Nitrofurazone was not assayed in this study due to technical difficulty. It is frequently reported effective in clinical use (500 grams/ton). Ceftiofur (Naxel™) has also been efficacious (11 mg/kg). Please consult a current issue of the Feed Additive Compendium for regulations regarding medication of feeds.

Control and Prevention

General management factors can have a tremendous impact in the prevention of clinical salmonellosis. Maintenance of proper environmental conditions such as ventilation, stocking density and building temperature is essential in confined livestock production. Numerous sources may be consulted for specific criteria to evaluate environmental quality.[7,8,9] At all stages of growth, use of all in/all out production methods combined with thorough cleaning and disinfection of pens after pigs are moved is important to prevent infection between groups. Feeder pigs should be purchased from sources with a known health history, preferably from a single source. Do not mix pigs from multiple sources if they are brought into a herd simultaneously. Direct purchase from one owner carries much less risk of disease than purchase through commercial sale barns.

Prophylactic medication of feeder pigs on arrival may be effective, especially on farms where salmonellosis outbreaks occur on a regular basis. One protocol involves feeding neomycin or nitrofurazone in feed for the first 10 days after arrival. Carbadox is then used to 75 pounds.[4] Gentamicin has also been used effectively in water. Culture of infected herds is difficult, due to sporadic shedding of the organism. If salmonellosis is a consistent problem in pigs from a specific source, animals may be cultured on arrival.[4]

Vaccination against salmonellosis is controversial. Efficacy has been mixed but generally poor. The antigens involved are often high in carbohydrates, which are weakly antigenic. They are also variable between isolates. Care must be taken when using autogenous bacterins, due to endotoxin. Kramer and Wood used avirulent *Salmonella* in conjunctival, per os, and IM administrations.[10] All three groups shed on challenge but disease severity and shedding was significantly reduced in the orally exposed group. Further work is needed to develop vaccines which are routinely usable.

Aleda Straley-Cheng

Conclusion

Salmonellosis in growing and finishing swine presents the practicing veterinarian with a definite challenge. With appropriate and careful clinical and diagnostic evaluations, the local practitioner can make an accurate diagnosis and establish effective programs to control respiratory disease caused by *Salmonella choleraesuis*.

References

1. Leman AD, Shaw BE, Glock RD, Mengeling WC, Penny RHC, Scholl E: *Disease of Swine*, 6th edition. Iowa State University Press, Ames, Iowa, 50011. 1986.

2. NVSL *Salmonella* Isolation data. 10/1/88 through 9/30/89.

3. Hoffman LJ, Kramer TT. Unpublished data. 1990.

4. Schultz RA. Salmonellosis - The Problem - How Do We Handle It? *Proc Ann Conv AASP*, Des Moines, Iowa:183-188. 1989.

5. Hoffman LJ. A Review of Laboratory Methods used in the Diagnosis of Swine Respiratory Disease. *ISU Veterinary Diagnostic Laboratory Procedures Handbook*. 1989.

6. Kinyon JM. Unpublished data. 1990.

7. Schultz RA. Haemophilus, in Blue Print Series for Top Managers: Pneumonia. *National Hog Farmer*, Webb Division, Intertech Publishing Corp, St Paul, MN. Spring 1989.

8. *Structures and Environment Handbook*, Midwest Plan Service, Iowa State University, Ames, Iowa, 50011. 11th Edition 1983, Revised 1987.

9. Donham KJ. Air Quality in Swine Buildings-The Veterinarians's Role in Promoting Human Health and Swine Productivity. *Proc Ann Conv AASP*, Denver, Colorado:231. 1990.

10. Kramer TT, Wood RL. Local and Systemic Immunity to Salmonella choleraesuis. *Proc U.S. Animal Health Assoc*:497-498. 1988.