EXPERIMENTALLY INDUCED LAMENESS IN PIGS MULTIPLY INFECTED WITH MYCOPLASMA HYOPNEUMONIAE

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Arthritis caused by microbial infections in pigs is a problem in production units in many parts of the world. It ranks second in the United States as a cause of condemnation of whole or parts of carcasses in packing plants. Among the known causes of swine arthritis are two mycoplasma species: Mycoplasma hyosynoviae and M. hyorhinis (Ross, Switzer and Duncan, 1971; Ross and Spear, 1973; Duncan and Ross, 1973). Preliminary studies indicate that another species of mycoplasma, M. hyopneumoniae associated with enzootic pneumonia of swine (Goodwin, Pomeroy and Whittlestone, 1965; Maré and Switzer, 1965; Williams and Gallagher, 1978) may also cause an arthritic condition. Recent findings indicate that pigs intravenously administered M. hyopneumoniae become lame and develop clinical signs of arthritis (Lloyd and Etherridge, 1981a; 1981b). In addition, M. hyopneumoniae may disseminate by hematological spread from lung or brain in pigs and cause cytopathological effects not only to respiratory tissues but also to the central nervous system resulting in locomotory disturbances which may resemble an arthritic condition (Williams, 1980; Williams and Gallagher, 1982; Williams, Gallagher and Pirtle, 1982).

The purpose of this investigation was to induce arthritis in experimental animals by either cerebrospinal or intra-articular joint inoculations with M. hyopneumoniae. Strain virulence was assessed by necropsy and culture of M. hyopneumoniae from affected tissues. Evidence of infection was based on positive culture, and positive serology or pathology.

Three reference (J, 11 and JF439) and four field (2069, 29, 30, TAM-4L) strains of M. hyopneumoniae were each inoculated into separate groups of pigs varying in live-weights from 3.7 to 110 kg. Pigs under light anesthesia were either cerebrospinally inoculated ($4\text{-}9 \times 10^9$ cfu in 1.0 ml/pig), by suboccipital puncture in the midline at the level of a line that joins the anterior borders of the wings of the atlas, or into synovial fluids of leg and shoulder joints ($4\text{-}9 \times 10^8$ cfu in 0.1 ml/joint).

The intensity of clinical signs varied depending on strain of M. hyopneumoniae used, route of inoculation, and the age of the animal being tested. Following cerebrospinal inoculation, particularly with strains 2069, 30 and JF439, pigs at 21 to 55 days were lame. These pigs when first disturbed were reluctant to get up. On rising they tended to rest on their anterior surfaces of carpal regions and held their affected legs forward or picked up, indicating pain. Pigs that showed labored breathing also arched their backs, performed exaggerated stretching motions, placed their hind feet more anteriorly and splayed their toes from increased weight bearing. Following synovial fluid inoculations, pigs were slow (55 to 165 days) to show clinical signs regardless of mycoplasmal strain being tested.

Antibodies to M. hyopneumoniae were revealed in blood sera of the experimentally infected animals. Indirect hemagglutination titers were relatively low, 1:16 or 1:32, with a high of 1:128 registered 21 days postinoculation in 1 animal that had been cerebrospinally inoculated.

In affected pigs necropsied, synovial fluid from shoulder, elbow, carpal, hip, stifle and tarsal joints of lame limbs was increased in volume and was serofibrinous or serosanguineous. Severe fibrosis, synovial hypertrophy and cartilage damage characteristic of the arthritis of swine erysipelas was not observed.

Mycoplasma hyopneumoniae was frequently isolated and cultured from joints, lungs, brains, cerebrospinal fluids, spleens and lymph nodes of affected pigs but never from control pigs. Stained smears of synovial fluids revealed neutrophilic granulocytes and fibrin comparable to control (uninoculated) animals. In the chronic phase, granulocytes were sparse. By Giemsa and DNA staining techniques, mycoplasmas were not reliably demonstrated in syncvial fluid, but were in bronchial mucus.

Because of the comparatively short lifespan and high incidence of respiratory and arthritic disease in pigs raised under commercial conditions, an understanding of M. hyopneumoniae as a pathogen capable of disseminating and causing cytopathological effects in multiple sites of the body is of considerable importance. Lameness in pigs would seem to seriously affect the reproductive efficiency of breeding pigs, shorten the longevity and cause culling of breeding stock.

Conclusions: Mycoplasma hyopneumoniae was established as a pathogen causing pneumonia, lameness and clinical signs of arthritis. The organism could be isolated in the first 3 weeks of infection. Data presented suggest M. hyopneumoniae plays a greater role in the disease processes of swine by causing pneumonia and arthritis, two of the three most common condemnations of carcasses.

Selected references: Ross, R.F., Switzer, W.P., and Duncan, J.R.: Am. J. Vet. Res. 1971, 32:1743; Ross, R.F., and Spear, M.L.: Am. J. Vet. Res. 1973, 34: 373; Duncan, J.R. and Ross, R.F.: Am. J. Vet. Res. 1973, 34:363; Goodwin, R.F.W., Pomeroy, A.P., and Whittlestone, P.: Vet. Rec. 1965, 77:1247; Maré, C.J., and Switzer, W. P.: Vet. Med. Sm. Anim. Clin. 1965, 60:841; Williams, P.P., and Gallagher, J.E.: Infect. Immun. 1978, 20:495; Williams, P.P.: Proc. IPVS Cong., Copenhagen, Denmark 1980, 215; Williams, P.P., and Gallagher, J.E.: SEM 1982, (in press); Williams, P.P., Gallagher, J.E., and Pirtle, E.C.: SEM 1982, (in press); Lloyd, L.C., and Etheridge, J.R.: Res. Vet. Sci. 1981a, 30:124; Lloyd, L.C. and Etheridge, J.R.: J. Comp. Path. 1981b, 91:77.