INTERACTION BETWEEN A SWINE FEVER VACCINIAL VIRUS AND PASTEURELLA MULTOCIDA IN THE PRODUCTION OF PNEUMONIA IN PIGS.

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There has been abundant research published in relation to the ethiology of porcine respiratory disease, and Pasteurella multocida has been proved as an important secondary invader of the lung, frequently responsible for the advanced lesions observed in the field. However, it is difficult to experimentally reproduce pneumonia in pigs with P. multocida alone. Some workers - - - - (Smith, 1970) have demonstrated the interaction - - - between P. multocida and Mycoplasma hyopneumoniae. In cattle, an interaction between P. multocida and P1.3 Virus has also been demonstrated (in some cases such interactions have even been detected with vaccinial strains of some viruses such as Newcastle Virus). In the pig, Kasza, et al, (1969) have found the relation between porcine adenovirus and Mycoplasma hyopneumoniae. Mutyra, et al (1936) mentioned cases of "Swine Plague" in which pasteurellae seemed associated with Swine Fever field strains.

In Mexico, P. multocida is a very common pathogen - - (27.1%) in porcine pneumonic lungs, (Pijoan, Ochoa and Trigo, 1976). On the other hand, Swine Fever is - - - widespread in the country, and it is a common procedure to vaccinate against this disease with live - - - attenuated strains. Because of this, it was decided to investigate the posibility of an interaction between These vaccinial strains and P. multocida.

The first trial (Pijoan and Ochoa, 1978a) was made in vivo using 4 week old yorkshire piglets which were -- divided in 6 lots of 5 anirals each. Three lots were vaccinated with a commercial live vaccine produced with lapinized virus and challenged intratracheally with live P. multocida at 3, 5 and 15 days postvaccination. The other lots were controls. The animals were slaughtered 15 days after challenge, and the neumonic lesions were measured. The results demonstrated a statistically greater ($p < 0.05$) lesion score on - - - animals that has only received challenge. This was only true when the challenge followed vaccination at 3 or 5 days' interval, but not at 15 days. The results demonstrated that vaccination against Swine Fever results in the production of significantly more severe lesions by P. multocida

It was then decided to investigate which of the lung's defenses are most affected by the virus. Two systems were explored: a) The secretion of bactericidal substances by the tracheobronquial epithelium. b) The - - phagocytic or bactericidal activities of the alveolar macrophages.

Tracheal explants from pig embryos were prepared and maintained in culture, and their secretions were - tested for bactericidal activity against P. multocida. A strong bactericidal activity was present in the tracheal exudates, and were sensible to trypsin. It was also demonstrated that the substances involved - were not lysosyme (Pijoan and Ochoa 1978b). Later, it was proved that the secretion of these strong - - - bactericidal substances is inhibited by the lapinized vaccinial Swine Fever Virus. In effect, tracheal - - explants treated with virus at the start of the incubation period demonstrated a significantly lower capacity to kill P. multocida. This effect was more evident at 24 than at 48 hours' postinfection with the virus, suggesting that the tracheas are capable of reparing to a certain extent the damage caused by the virus. (Iglesias, 1979).

At the same time, alveolar macrophages from vaccinated and nonvaccinated piglets were tested for their - - phagocytic and bactericidal effect against P. multocida In a first experiment, 5 piglets (6 weeks old) were vaccinated with a commercial lapinized virus, and slaughtered 5 days later. Their alveolar macrophages were obtained by lung lavage and challenged with 10 bacteria/macrophage. Viable bacterial counts were performed at 30, 60 and 75 minutes. This last count was performed after disrupting the macrophages with saponin, to detect bacteria which had been phagocytozed but not killed. The results demonstrated a larger (but not statistically significant) amount of - - bacteria at 30 and 60 minutes in preparations with cells from vaccinated pigs. On the other hand, after disruption with saponin the difference became significant. The results suggest that vaccination impairs phagocytosis to some extent, but affecting mainly the microbiocidal activities of the macrophage. (Pijoan and Campos-in press). A further experiment demonstrated that the effect on phagocytosis was marked during the first two days after vaccination, not significant from days 3-9, reappearing in days 10-11 (the duration of the experiment). It is not clear at present the reasons for this delayed effect after vaccination.

Although the virus has demonstrated a capacity for affecting both the production and/or secretion of tracheal bactericidal substances, and phagocytosis of the bacteria by alveolar macrophages, the former effect would appear to be of greater significance. As has been already mentioned, when tested in vivo, the pig is affected the first 5 days but not 15 days, after vaccination. Alveolar macrophages from - - - vaccinated piglets show a significant lower phagocytosis during the first 2 days, but not at day 5. Furthermore, tha phagocytic activities of porcine alveolar macrophages against P. multocida are moderate even in non-vaccinated pigs (Pijoan and Campos-in press).

On the other hand, the trachea of pig embryos produces a strong bactericidal substance against pasteurellae, which is markedly inhibited after infection with the virus. It is therefore suggested that the main lung defense mechanism of the pig against P. multocida is the bactericidal activities of the mucociliary apparatus, and that these are the mechanisms mainly affected by vaccinial strains of Swine Fever Virus.

Conclusions:

Vaccination with live attenuated Swine Fever Virus predisposes the animal to infection by P. multocida. The main defense mechanism against P. multocida in the pig is the secretion of tracheal bactericidal substances. The secretion is affected by the virus.

Selected references: Campos, Q. M.; MVZ Thesis, 1977. Fac. Med. Vet. Zoot. UNAM (México); Iglesias, G.: MVZ. Thesis, 1979. ENEP-Cuautitlán-UNAM (México); Pijoan, C., Ochoa, G. and Trigo, F.: Tec. Pec. Mex. 1976, (29):46; Pijoan, C., and Ochoa, G.: J. Comp. Path. 1978a, 88 (2): 167; Pijoan, C., and Ochoa, G.: Rev. Lat. Microbiol. 1978b, 20 (1): 1; Smith, I.M.: Ph.D. Thesis, 1970 University of London; Kasza, F., Hodges, R.T., Bets, A.G., and Trexler, D.C.; Vet. Rec. 1969, 84:262.

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