IMMUNITY TO NEONATAL E. COLI ENTEROTOXICOSIS IN PIGLETS AFTER PARENTERAL VACCINATION WITH A SUBFRACTIONATE VACCINE

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Pili (fimbriae) of enterotoxic E. coli (ETEC) are contributors to the virulence of enterotoxigenic E. coli by providing the attachment of the bacteria to the receptors on the brushborder of intestinal villi cells, leading to the colonization of the intestinal epithelium. It has been demonstrated that piglets nursing sows vaccinated with purified pili are protected from a challenge with ETEC. For a review see (1). In this experiment seven pregnant sows or gilts, seronegative (Elisa $\leq$ 80) for K88 ab, K88 ac and Labile toxin (LT) were vaccinated IM with a vaccine* consisting of purified K88 ab, K88 ac and semi-purified LT in an oil emulsion, at the 8th week of pregnancy. A booster was given at two weeks before the earliest possible farrowing date. Seven seronegative animals served as control. During this time weekly rectal swabs were taken to check for the presence of K88 positive E. coli. During farrowing the newborns were separated from the mother and infected orally with $2x10^9$ cells of ETEC (H10:0157: K88ac LT^+, ST^-). After exactly 60 minutes the newborns were placed with the sow and attention was made that each piglet nursed colostrum immediately. Daily rectal swabs were taken for reisolation of the challenge strain (kanamycin resistance). Isolates were identified using monospecific anti K88 ab, ac antisera. Anti-K88 ab, ac and anti LT antibodies in serum colostrum and milk were determined by ELISA using purified K88 ac, K88 ab and LT antigens. Diarrhea was recorded daily and expressed as + (soft to liquid feces) ++ and +++ (acute diarrhea with visual deshydratation). Finally a daily diarrhea index (DI) for vaccinated and controls was calculated. Theoretically the DI could reach a daily maximum of 300 when all the surviving animals would score (+++).

Results: The weekly rectal swabs resulted in the irregular isolation of $K88^+$ E. coli in most animals. Vaccination did not seem to have any influence on this isolation. The titers obtained after vaccination and revaccination showed that all vaccinated animals had an adequate immune response to the injected antigen and with few exceptions, animals showed a typical secondary immune response. The titers of colostrum were in general considerably higher than those of serum. Milk titers at 5 days post farrowing showed a significant drop from those of colostrum.

The mortality attributable to diarrhea at the end of day 5 (D_5) was 1.4% for the vaccinates and 60.0% for the controls. The weaning percentages at D5 were 35.4% for the controls and 83.8% for the vaccinates.

The diarrhea index (DI) as an overall assessment of the daily diarrhea went from 81 at D_1 to $\simeq$ 2 at D_5 for the vaccinates and from 187 at D_1 to 21 at D_5 for the controls. In the controls we observed either $\simeq$ 100% (3 litters) or $\simeq$ 50% (4 litters) mortality. The reisolation of the challenge strain, as a direct indication of elimination started at 61% at D_1 and dropped to $\simeq$ 7% at D_5 for the vaccinates, while for the controls these data were 86% and 32% respectively.

Conclusions:

The isolation of $K88^+$ E. coli from rectal swabs of sows showed a random pattern in vaccinates and controls and seemed to reflect the natural condition in a pig population. The results of the challenge experiment showed clearly that piglets from vaccinated animals had virtually no mortality attributable to diarrhea, and were able to clear the challenge strain much faster than the controls. The drop in titer of D_5 milk gives an indication that in colostrum the antibody involved is mainly IgA. Preliminary results confirm this hypothesis. These results indicate that the mechanism of protection could be that of plasmid curing. Our finding that mortality in the controls was either 50% or 100% gives further evidence for the existence of two phenotypes with respect for the K88 receptor on intestinal villi cells.

*Nobi Vac LT-K88 - Intervet

Selected references: 1. M. AWAD-MASALMEH, et al. Infect. Immunity - 35: 305-313, 1982. Linggood, M.A. and P. Porter - Immunol. 35: 125-127, 1978. Sellwood, R. et al. J. Med. Microbiol. 8:405-411, 1975. Nagy, B. et al Infect. Immunity 16:344-352, 1977. Gibbons et al. J. Gen. Microbiol., 86:228-240, 1975. Wilson, M.R. and A.W. Hohmann. Infect. Immunity 10:476-482. 1974.

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