Pili (fimbriae) of enterotoxigenic E. coli (ETEC) are contributors to the virulence of enterotoxigenic E. coli by providing the attachment of the bacteria to the receptors on the brush border of intestinal villi cells, leading to the colonization of the intestinal epithelium. It has been demonstrated that piglets nursing sows vaccinated with purified pilus are protected from a challenge with ETEC. For a review see [1]. In this experiment seven pregnant sows or gilts, aseptically (Elisa < 40) for E88 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 aseptically immunized animals served as controls. During this time weekly rectal swabs were taken to check for the presence of E88 positive E. coli. During farrowing the newborn were separated from the sow and infected orally with 5 x 10⁶ cells of ETEC (ATCC 11128) 24 hours prior to farrowing. Daily rectal swabs were taken for isolation of the challenge strain (kunmycin resistance). Isolates were identified using monospecific anti K88 ab, ac antisera. Anti-88 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 dehydration). Finally a daily diarrhea index (0–10) for vaccinated and controls was calculated. Theoretically the D1 could reach a daily maximum of 100 when all the surviving animals would score 10 (100).

Results: The weekly rectal swabs resulted in the irregular isolation of E88 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 antigens 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 (D5) was 0.4% for the vaccinated and 60.0% for the controls. The mean percenting at D5 were 36.4% for the controls and 83.6% for the vaccines.

The diarrhea index (D) as an overall assessment of the daily diarrhea was given from 81 at D1 to 2 at D5 for the vaccinees and from 187 at D1 to 71 at D5 for the controls. In the controls we observed either = 100% (3 litter) or = 50% (4 litter) mortality. The reisolation of the challenge strain, as a direct indication of elimination started at 61% at D1 and dropped to = 7% at D5 for the vaccine, while for the controls these data were 88% and 32% respectively.

Conclusions:

The isolation of E88 E. coli from rectal swabs of iguas showed a random pattern in vaccinated 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 scores of D5 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 E88 receptor on intestinal villi cells.

*ABV Vac. LT-K88 - Intravit.