Targeting the small intestinal F4-receptor in piglets with maternal antibodies results in enhanced recall responses.

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Oral vaccination remains a challenge. We observed that oral immunization of piglets with F4 fimbriae induces a protective intestinal immune response evidenced by an F4specific serum and intestinal IgA response (Van den Broeck et al., 1999) and demonstrated that this is due to targeting of aminopeptidase N (APN) on the brush border of small intestinal enterocytes (Melkebeek et al., 2012). This results in transcytosis of the fimbriae which are subsequently taken up by SIRP $\alpha$  positive cells (Snoeck et al., 2008). Dendritic cells differentiate to a semi-mature state, which is sufficient to enhance F4 presentation to T-lymphocytes and leads to a robust F4specific IgA response (Devriendt et al., 2009). Most piglets have colostral immunity against F4 and it had not been determined if targeting APN with F4 fimbriae, could overcome this passive immunity. In 3- to 4-week-old piglets without maternal antibodies the oral immunization induced F4-specific serum IgG and IgA responses, which were not observed in piglets with maternal antibodies. However, ELIspot assays revealed F4-specific circulating antibody secreting cell responses. Enriching the IgA+ B cell fraction, demonstrated a more robust F4-specific IgA booster in pigs with than without maternal antibodies, suggesting that the maternal antibodies enhanced the secondary response. Results indicate that in the presence of colostral

immunity, ELIspot assays on enriched IgA<sup>+</sup> B-cells could be used to optimize

mucosal immunization protocols.

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