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GENETIC AND IMMUNE FACTORS IN THE SUSCEPTIBILITY OF PIGLETS TO *ESCHERICHIA COLI* DIARRHŒA

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Colonization of the intestinal tract by enteropathogenic strains of *Escherichia coli* is a feature of enteric infection in many species (Smith and Linggood, 1971; Bertschinger *et al.*, 1972; Sack, 1975). It is well known that proliferation of these organisms in the intestinal tract only occurs if they can counteract removal by peristalsis, an important mechanism regulating bacterial flora (Dixon, 1960). Bacterial adhesins have evolved which permit attachment of the bacteria to the mucosal surface of the intestine thereby overcoming this elimination process (Jones and Rutter, 1972; Evans *et al.*, 1975; Burrows *et al.*, 1976) and an important adhesin with regard to *E. coli* neonatal diarrhœa in pigs is K88 (Sojka, 1965). This fimbrial antigen attaches the organism to the pig intestinal mucosa at specific receptor sites (Sellwood and Lees, 1981). These receptors allow proliferation of the bacteria in close proximity to the epithelial cell surface and an enterotoxin that is elaborated by the organisms induces fluid secretion. The absence of the intestinal receptors in some pigs was observed when using the *in vitro* brush border adhesion test (Sellwood *et al.*, 1975) and these pigs were resistant to infection by $K88^+$ *E. coli* (Rutter *et al.*, 1975). Resistance (absence of receptor) was inherited and was the product of an autosomal recessive gene (Gibbons *et al.*, 1977). The homozygous dominant (SS) and heterozygote (Ss) were susceptible (receptor positive) whereas the homozygous recessive (ss) was resistant (receptor negative).

Variation in the susceptibility of piglets to infection with enterotoxigenic *E. coli* has been observed on many occasions both in the natural disease (Sweeney, 1968; Wijeratne *et al.*, 1970) and in experimental infection studies (Smith and Halls, 1968; Rutter and Anderson, 1972). This has also been observed in other species e.g. calves (Smith and Halls, 1967). The absence of intestinal receptors for the adhesins may, therefore, have contributed to observed variation in susceptibility.

The presence of other pig phenotypes have also been observed. Sellwood (1980a) described a pig phenotype as weak-adhesive (±), (probably coded for by a third allele Sq) since very few $K88^+$ bacteria adhered to the intestinal brush borders *in vitro* when compared with the numbers that attached to the normal adhesive pig. This inherited character was carried by four boars (table 1).

Piglets that were weak-adhesive have been bred by mating one of these boars (n°. 1) to a homozygous recessive sow. Infection of the piglets with an enterotoxigenic *E. coli* strain W1 (O149:K91 (B), K88ac (L); H10) resulted in poor colonization of the intestinal tract in comparison with normal adhesive, homozygous dominant (SS) piglets (table 2). Colonization was similar to that in homozygous recessive piglets 48 h after infection and there was no diarrhœa. It is possible that the weak-adhesive receptor does not function *in vivo* or that if it does, relatively low affinity or low numbers of receptors on the intestinal epithelial cells leads only to very poor colonization.

Other phenotypes of pig have been observed in the brush border adhesion test when using three serological variants of K88 (Bijlsma *et al.*, 1982). The K88 antigen exists in three forms, K88 ab, K88 ac and K88 ad (Ørskov *et al.*, 1964; Guinée and Jansen, 1979) and phenotypes of pig exist that are (a) susceptible to adherence of all three variants, (b) susceptible to two only variants, (c)

susceptible to one variant and (d) not susceptible to attachment by any K88 variant. This latter phenotype is probably identical to the homozygous recessive (ss) genotype of Sellwood *et al.* (1975) since all three variants do not attach to the intestinal brush borders from these pigs. The homozygous dominant (SS) and heterozygote (Ss) also probably correspond to the first of the above phenotypes since all variants attach to these genotypes (Sellwood, unpublished observations). It is not known if the weak-adhesive phenotype fits into any of the above groups.

Another important factor, the mammary secretion of the dam, can moderate enteric infection of the neonate. The piglet is born devoid of any circulating antibody (Sterzl *et al.*, 1960) and is probably protected from systemic infection by absorbing immunoglobulins from the colostrum of the dam in the same way as that shown for the protection of calves (Logan and Penhale, 1970a,b). Continual protection from enteric infection, until the piglet is able to mount its own immune response is brought about by the bathing of the intestinal mucosal surfaces with colostrum and milk immunoglobulins and probably other anti-bacterial agents such as lactoferrin and transferrin (Reiter, 1978), also present in milk.

Anti-adhesive activities of immunoglobulins have been reported as protective against K88$^+$ *E. coli* infection. The production of anti-adhesive K88 antibodies has been induced in the colostrum of sows using parenterally administered (a) whole, killed *E. coli* with added K88 antigen (Nagy *et al.*, 1976; 1978) or (b) purified K88 antigen (Jones and Rutter, 1974; Rutter *et al.*, 1976).

Other studies using oral vaccination of lactating sows with live K88$^+$ *E. coli* have also resulted in anti-adhesive antibodies in milk (Evans *et al.*, 1980). The subject of this paper is the interplay of these genetic and immune factors in the incidence of diarrhœa in piglets and the anti-bacterial properties of colostrum in a herd in which both susceptible and resistant pigs have been bred.

Table 1. — Phenotypes of progeny of homozygous recessive dams and boars possessing the weak-adhesive allele (Sq.).

Boar	Phenotype of progeny		
	+	±	−
1	0	9	0
2	5	2	1
3	4	1	0
4	0	3	4

+ : Adhesive phenotype.
: Non-adhesive phenotype.
± : Weak-adhesive phenotype. (Sellwood, 1980).

Incidence of diarrhœa

For several years the pig herd at Compton has been bred to provide both homozygous dominant (susceptible) and homozygous recessive (resistant) animals. Heterozygotes are not normally produced. During an 8-10 week period there was a major disease outbreak which was associated in particular with a K88$^+$ enterotoxigenic *E. coli* strain G205 (O8, K87 (B); K88ac (L)) (Sellwood, 1979). Before and after this period there was little or no neonatal diarrhœa in the herd. The results of an investigation of the diarrhœa episode are shown in table 3.

There was no K88$^+$ *E. coli* associated diarrhœa in the susceptible progeny of homozygous dominant sows by either homozygous dominant or homozygous recessive boars or in the resistant progeny of homozygous recessive sows. There was however some diarrhœa in both these groups attributed to an *E. coli* strain (O9, K103, 987P$^+$) another adhesive strain that was able to adhere to the intestinal mucosa of both homozygous dominant and homozygous recessive pigs.

The litters of the homozygous recessive sows, by either homozygous dominant or heterozygote boars, were severely affected. All the progeny sired by homozygous dominant boars and 44 % of the

Table 2. — Colonization of the intestinal tract of a homozygous dominant, a homozygous recessive and a weak-adhesive phenotype of piglet by a K88$^+$ enterotoxigenic *E. coli* strain W1, O149, K91, K88ac (L); H10.

Pig Type	No. of organisms in contents ($\log_{10}$ per g)				
	Stomach	Small Intestine			Colon
		1	2	3	
Homozygous dominant (SS)	7.30	9.49	9.20	9.45	9.51
Homozygous recessive (ss)	< 2.0	< 2.0	3.21	3.12	3.52
Weak-adhesive (Sq.s.?)	< 2.0	< 2.0	< 2.0	2.64	4.56

progeny of heterozygote boars had diarrhœa and 74 % of these animals died. The surviving but affected piglets of these boars were subsequently phenotyped as adhesive (i.e. receptor positive) by the brush border adhesion test and those progeny sired by the heterozygous boars that did not have diarrhœa were typed as non-adhesive (i.e. receptor negative). These results supported the earlier experimental evidence that non-adhesive piglets are resistant to infection by K88$^+$ *E. coli* even in the natural disease situation. They are also consistent with the hypothesis that the susceptible gene present in the present day pig herds is due to an unstable balanced polymorphism (Gibbons *et al.*, 1977) since there is selection against the heterozygotes born of homozygous recessive dams.

Further evidence has more recently come to light in support of these observations. A commercial farming company reared both resistant and susceptible pigs and investigated the incidence of K88$^+$ *E. coli* diarrhœa in this herd. It was found that the only group of animals to suffer from diarrhœa were those born to resistant dams and sired by susceptible boars when mortality was 21 % (Walters and Sellwood, 1982). The genetically resistant piglets were unaffected as were the susceptible piglets born of susceptible dams (table 4). This study also provided direct evidence that there was no deleterious gene linked to the gene for resistance since there was no significant differences between the susceptible and resistant groups in litter size, food conversion rate; weight gain and other performance characteristics.

Protective mechanisms of colostrum

With a view to understanding the nature of the protective functions of the colostrum and milk of sows, an investigation was undertaken of the antibacterial properties of the colostrum of the susceptible and resistant sows of the Compton herd, collected during the outbreak. Antibody titres for the *E. coli* strain, G205, K88 and O8 antigens were compared (Sellwood, 1982).

The agglutinin titres of the colostrum of susceptible sows were significantly different ($P < 0.02$) from those of resistant sows and most activity was directed against the K88 antigen. Tanned erythrocyte haemagglutination and the passive haemagglutination titres for the K88 and O8 antigens respectively also showed small but significant differences between the two groups (K88 $P < 0.01$; O8 $P < 0.1$).

The bactericidal power of the colostra was investigated. At a concentration of 1/2 the killing of *E. coli* strain, G205, by the colostral whey of both susceptible and resistant groups was similar (fig. 1) and at 1/10 dilution there was no killing of bacteria. However, when complement was added (pre-colostral piglet serum) there was efficient killing in some cases but again there was no significant difference between the two groups.

The opsonic activity of the colostral whey samples diluted 1/10 was also investigated. *E. coli* strain G205 was mixed with the 1/10 dilution of colostrum and polymorphonuclear leucocytes (PMNs) prepared from the blood of pigs. After

Table 3. Incidence of diarrhoea and mortality in progeny of different mating groups.

Sire	Dam	No. Litters	No. Born	Diarrhoea	Deaths from Diarrhoea
SS	SS	8	64	7[a]	0
ss	SS	4	45	0	0
ss	ss	15	153	19[a]	3
SS	ss	4	32	32[b]	22
Ss	ss	7	68	30[b]	24

a : Diarrhoea attributed to *E. coli* strain O9; K103; 987P$^+$.
b : Diarrhoea attributed to *E. coli* strain O8; K87 (B), K88ac (L).
S : Dominant allele.
s : Recessive allele.

Table 4. — Piglet mortalities of different mating groups.

Sire	Dam	Mortality (%)	Total Deaths	Crushed	Weak	Scour	Other
ss	ss	13.5	21	5	12	0	4
SS	SS	9.4	15	8	5	0	2
SS	ss	21.0	45	13	14	10	8
ss	SS	15.7	18	6	10	0	2

(Walters and Sellwood, 1982).

incubation the bacterial numbers were estimated to determine the degree of killing. The results are shown in figure 2. There was a highly significant difference between the colostral whey of the susceptible sows and resistant sows ($P < 0.001$). There was efficient killing of the organisms by PMNs when colostrum from the susceptible dams was used.

These antibacterial activities are extremely interesting in view of the remarkable difference in the protective ability of the colostrum from susceptible and resistant sows. It would appear from these results that a direct bactericidal action by colostrum, with or without the presence of complement was probably not an important mechanism within the piglet intestinal tract. This view has already been taken by other investigators in vaccination studies (Jones and Rutter, 1972; Rutter *et al.*, 1976) although in some studies a broad correlation between bactericidal activity and protection occurred in vaccinated animals (Nagy *et al.*, 1978). On the other hand the highly significant difference in the opsonic activities of the colostrum samples suggests an important role for opsonophagocytosis as a major defence mechanism.

Opsonophagocytosis of bacteria within the intestinal tract has on several occasions been the subject of investigation. The gastro-intestinal (GI) tract is a major site of elimination of granulocytes (Ambrus and Ambrus, 1959; Teir and Rytöma, 1966), but there is evidence that the appearance of PMNs within the GI tract is not simply due to the disposal of degenerating neutrophils. PMNs, encountered in the intestine often have ingested bacteria (Moon, 1980) and they can also be induced to enter the intestinal lumen under certain conditions (Bellamy and Nielson, 1974). Phagocytosis of *E. coli* by these PMNs has also been observed (Bellamy, 1978).

PMNs may also reach the piglets intestinal tract from the dam's mammary secretions which are known to contain many cell types including PMNs (Evans *et al.*, 1982). Although increases in neutrophils have usually been associated with mammary infection, large numbers can also be present in apparently normal individuals (Jensen and Ebe-

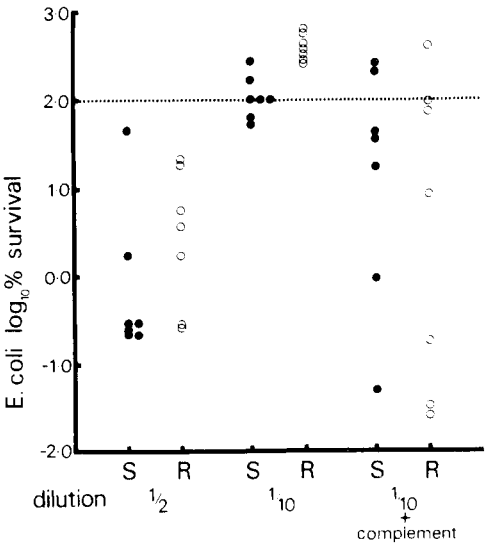

Fig. 1.— The bactericidal activity of colostrum from susceptible (S) and resistant (R) sows against *E. coli* strain G205.

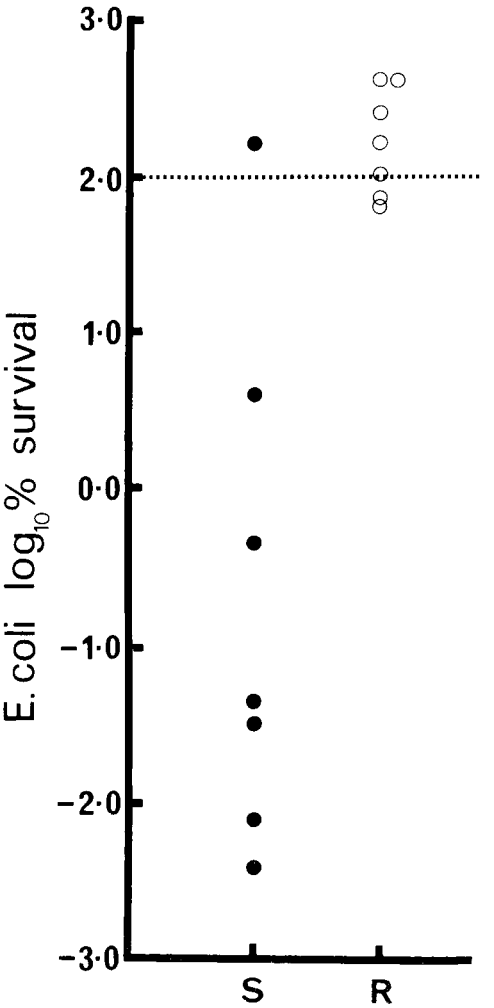

Fig. 2.— The opsonic activity of colostrum from susceptible (S) and resistant (R) sows against *E. coli* strain G205.

rhart, 1975 ; Reiter, 1978). Evidence for the importance of these cells to the suckling neonate is somewhat scanty. Suckling rats survived an oral infection with a *Klebsiella* strain whereas those rats fed milk substitute alone died. Furthermore, rats fed milk substitute and PMNs (prepared from blood or milk) also survived (Pitt, 1974). Hence the significance of colostrum and milk PMNs and the enteroluminal emigration of neutrophils still needs to be evaluated in relation to the protection of the neonate from enteric infections.

The anti-adhesive activities of the colostrum samples from both susceptible and resistant dams have been compared by inhibition of radiolabelled cell-free-K88 antigen binding to intestinal brush borders (Sellwood, 1980b) (fig. 3). A significant difference ($P < 0.001$) in the inhibition of binding of ^{125}I-K88 antigen was evident between the two groups of sows (Sellwood, 1982). Although complete inhibition of binding was not accomplished (due to the concentration of the colostrum used, 1/10) these results do support other evidence that an anti-adhesive function of colostrum and milk is important for neonate protection.

The disease episode in a herd possessing known susceptible and resistant animals also provided an opportunity to study the class of antibody important in the potentially protective mechanisms already described. Fractionation of the colostrum of susceptible sows by gel filtration and ion-exchange techniques has enabled identification of the class of immunoglobulin responsible for most of the opsonic activity (Sellwood, 1982). This activity was always associated with the fraction containing the immunoglobulin IgM. Ion-exchange chromatography of colostrum on DE52 cellulose (Whatman, Maidstone, Kent) resulted in fractions rich in IgG, IgA and IgM (fig. 4). The IgG rich fractions had opsonic activities which gave rise to 50 % *E. coli* survival, while the IgA fraction gave 85 % survival and the IgM fraction 3 % survival in *in vitro* opsonophagocytosis assays.

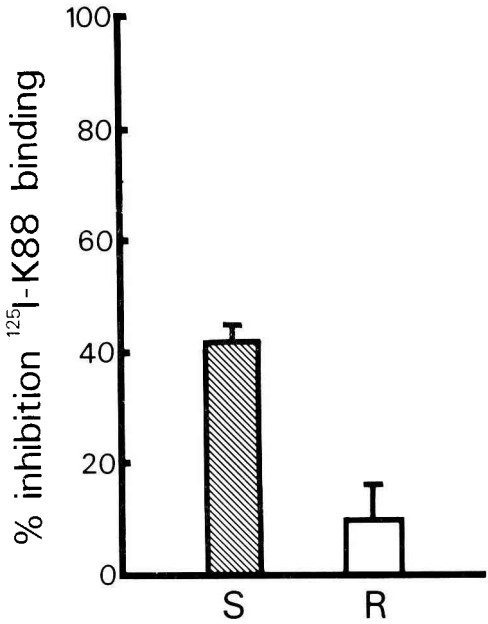

Fig. 3.— Anti-adhesive activity (± SEM) of colostrum from susceptible (S) and resistant (R) sows.

Conclusions

Many factors may contribute to the pathogenesis of enteric infection by K88$^+$ *E. coli* in piglets. However, two major host factors appear to outweigh all others in influencing the progress of infection and these are the presence or absence of intestinal receptors and the maternal protection by mammary secretions. The first of these factors also appears to influence directly the efficiency of the second but further studies of the colonization of the sow by K88$^+$ *E. coli* are necessary before any definitive conclusions can be drawn. Presumably increased colonization of the intestinal tract of the susceptible sow when compared to the colonization of the resistant sow resulted in the increased antibody titres and opsonin levels through direct stimulation of the intestinal lymphoid cells. In the resistant sow a lower degree of colonization may not have provided sufficient antigenic loading for an immune response to be invoked.

The anti-adhesive activity of the colostrum of the susceptible sows, much higher than in the resistant sows, supports earlier studies that anti-adhesion plays an important role in the protection

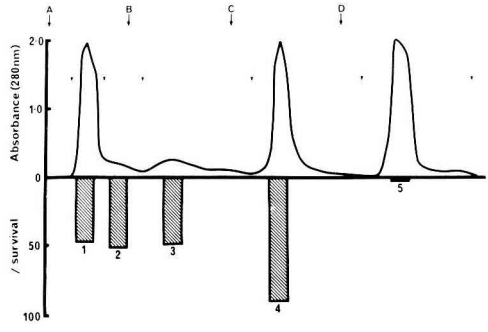

Fig. 4.— Ion-exchange chromatography of colostrum from a susceptible sow and the opsonic activities of each fraction. Predominant immunoglobulins present were : Fractions 1-3 IgG ; 4 IgA ; and 5 IgM. A-D indicate the commencement of a different buffer in elution procedure (Sellwood, 1982).

of the young piglet; but this was not the only difference observed. The significant difference in opsonic activity between susceptible and resistant sows also suggests a possible role for phagocytosis within the intestinal lumen with phagocytes either being provided by the mammary secretions or by the enteroluminal emigration of polymorphs. It is also interesting in this context that the important immunoglobulin class for opsonic activity was found to be IgM whereas anti-adhesive activity has been associated probably with either IgG (after parenteral vaccination) (Rutter *et al.*, 1976) or IgA (after oral vaccination) (Kohler *et al.*, 1975; Evans *et al.*, 1980).

Increase in our knowledge of antibacterial mechanisms operating in the intestinal tract of both resistant and susceptible piglets should clarify the situation such that more effective control measures may be devised.

EEC seminar on gastro-intestinal diseases in the young pig and calf, 1-3 December 1982, INRA CRZV de Theix 63110 Beaumont, France.

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Question

From Dr. Newtby to Dr. Sellwood.

Do sows with the weakly adhesive phenotype protect their litters ?

Answer

This has not been tested.

Question

From Dr. Pahud to Dr. Sellwood.

Can you block K88 adhesion with nutritional factors such as sugars and polysaccharides ?

Answer

I have tested many mono-, di-, polysaccharides for inhibition of adhesion of the K88 antigen to the brush borders of pigs and I have not found any to inhibit adhesion significantly. Hexosamines and other substances with a free-NH_2 group do inhibit to a degree as well or galactose containing polysaccharides such as stachyose