Early weaning of piglets is often accompanied by a severe growth check and diarrhoea. It is well established that this process is multi-factorial and that post-weaning anorexia and undernutrition are major aetiological factors. Gastrointestinal disturbances include alterations in small intestine architecture and enzyme activities. Recent data indicate transiently-increased mucosal permeability, disturbed absorptive–secretory electrolyte balance and altered local inflammatory cytokine patterns after weaning. These responses appear to operate according to two distinct temporal patterns, an acute response followed by a long-lasting adaptation response. Pigs coexist with a diverse and dense commensal microbiota in their gastrointestinal tract. Most of these microbes are beneficial, providing necessary nutrients or protection against harmful pathogens for the host. The microbial colonisation of the porcine intestine begins at birth and follows a rapid succession during the neonatal and weaning period. Following the withdrawal of sow’s milk the young piglets are highly susceptible to enteric diseases partly as a result of the altered balance between developing beneficial microbiota and the establishment of intestinal bacterial pathogens. The intestinal immune system of the newborn piglet is poorly developed at birth and undergoes a rapid period of expansion and specialisation that is not achieved before early (commercial) weaning. Here, new insights on the interactions between feed components, the commensal microbiota and the physiology and immunology of the host gastrointestinal tract are highlighted, and some novel dietary strategies are outlined that are focused on improving gut health. Prebiotics and probiotics are clear nutritional options, while convincing evidence is still lacking for other bioactive substances of vegetable origin.

Gastrointestinal disturbances immediately post weaning cause large economic losses in the pig industry. Within the pig population total losses of all those born in the EU amount to approximately 17% and a substantial proportion of these losses can be associated with infections via mucosal surfaces. Throughout Europe it is normal commercial practice that pigs are weaned at a much earlier age than they would in the wild, which results in an increased susceptibility to infection.

The weaning transition is a complex period during which the piglets have to cope with abrupt separation from their mother, mixing with other litters in a usually new environment, and switch from highly-digestible (liquid) milk to a less-digestible more-complex solid feed. Weaning at an early age (21–35 d), as in intensive production systems, has probably exacerbated the level of general stress in these immature animals. Weaning is usually associated with low and variable feed (and water) intake resulting in a transient growth check (for review, see Pluske et al. 1997). Although 50% of weaned piglets consume their first meal within 24 h post weaning, 10% have not eaten until ≥48 h (Brooks et al. 2001). Thus, energy requirements for maintenance are only met 3 d post weaning, and it can take 8–14 d for piglets to recover their preweaning level of energy intake (Le Dividich & Sève, 2000).

Various nutritional approaches for optimising the weaning transition and minimising enteric diseases have been tested in the past decade. For example, much work has been done on organic acids, some of which, alone or in
Physiology, microbiology and immunology of the gastrointestinal tract around weaning

Physiology

The intestines display various functions including absorption of nutrients, secretion and absorption of electrolytes (and water), secretion of mucin and immunoglobulins and selective barrier protection against harmful antigens and pathogens (for review, see Lalles et al. 2004). Post-weaning changes in intestinal tissue, including changes in villus and crypt architecture and depressed activities of many brush-border digestive enzymes, have been well documented and the implication of the presence of pathogens (Escherichia coli, rotaviruses) has also been addressed (Pluske et al. 1997). Many changes in intestinal physiology occur during the 2 weeks post weaning (Boudry et al. 2004). Transient increases in net ion transport in the jejunum and colon and in glucose absorption capacity in the jejunum and decreased jejunal electric resistance have been documented in piglets fasted for 2 d after weaning (Boudry et al. 2004). Preweaning values are usually observed again at 5 d after weaning. However, long-lasting changes are also recorded up to 2 weeks after weaning. Jejunal glucose absorption and secretory responses to secretagogues decrease while ileal transmucosal electric resistance increases on day 5 post weaning and then stabilise. Permeability to macromolecules across the jejunal decreases on day 2 post weaning and remains low thereafter. Thus, weaning induces transient acute changes probably related to post-weaning fasting, followed by a period of intestinal maturation corresponding to voluntary feed intake (VFI) resumption.

Microbiology

The gut is sterile at birth and is then colonised by microbes from the mother and the environment, starting with lactic acid bacteria, enterobacteria and streptococci. After the introduction of solid feed obligate anaerobes increase in number and diversity until an adult-type pattern is achieved (Inoue et al. 2005; Konstantinov et al. 2006a). The commensal microbiota salvage energy from otherwise indigestible carbohydrates and protect the host from pathogens by forming a front line of mucosal defence (Zoetendal et al. 2004). In contrast to adults the neonatal and weaning piglet is highly susceptible to enteric diseases (Hopwood & Hampson, 2003). In the immediate post-weaning period the balance between the development of so-called healthy commensal microbiota and the establishment of a bacterial intestinal disease can be easily tipped towards disease expression (Hopwood & Hampson, 2003). Piglets weaned within a ‘production’ environment experience major changes in intestinal microbiota composition that are influenced by diet, environmental factors and the host (Konstantinov et al. 2004b). In a short period of time the intestinal microbiota must ultimately develop from a simple unstable community into a complex and stable community, thus generating a tight ‘colonisation resistance’ or ‘competitive exclusion’.

It is recognised that microbial fermentation within the GIT is very important for the pig (Williams et al. 2001). The main products of fermentation include volatile fatty acids (VFA), which are known to play an important role in water (and Na\(^{+}\)) absorption, pH control and the inhibition of pathogens. In relation to host GIT health, fermentation (i.e. microbial activity) is also important for gut motility, improvement of energy yield, vitamin production and the stimulation of gut immunity. It is also involved in the prevention of diarrhoea and defence against pathogens (colonisation resistance; van der Waa\j et al. 1971). The composition of the diet is crucial in determining the composition and activity of the intestinal microbiota, and thus the production of the VFA mixture and other end products that will be optimal to gut health. For example, there is an important difference between the fermentation of carbohydrates and protein. Fermentation of carbohydrates leads to the production of mainly straight-chain VFA (e.g. acetic, propionic and butyric acids) and the use of NH\(_3\) and other nitrogenous compounds, which are required for microbial growth. When carbohydrates are in short supply relative to the available protein of non-degradable and endogenous origin, protein will be used as an energy source for fermentation, resulting in the end products NH\(_3\), branched-chain VFA and potentially toxic end products (Williams et al. 2001). These compounds include amines, volatile phenols and indoles (Vis\ek, 1978; Yokoyama et al. 1982; Russell et al. 1983; MacFarlane et al. 1992).

Immunology

The piglet is immunodeficient at birth and is highly dependent on a supply of specific and non-specific immune factors present in maternal colostrum and milk for immune protection, development and survival. The functional immaturity of the immune systems is such that newborn pigs are only able to generate limited T- and B-cell responses when challenged with pathogens, thus contributing to their immuno-compromised state (Butler et al. 2000). Clearly, development of immuno-competence is an absolute requirement for optimum growth and performance. However, in the context of exposure to a wide range of antigens associated with pathogens and with commensal bacteria and food, immuno-competence can be defined as the ability to mount appropriate responses to antigens, including the ability to generate tolerance to food and commensal bacterial antigens as well as active immune responses to pathogens (Bailey et al. 2001). Unlike the mature pig (Vega-Lopez et al. 1993; Wilson et al. 1996), which has a high extent of organisation, the cells and structures involved in mucosal immune
responses are initially absent at birth, and preliminary studies have indicated that they populate the intestine of the young pig in a highly-programmed sequence (Bailey et al. 2005). These phenotypic studies strongly suggest that the mucosal immune system remains relatively immature throughout the 'normal commercial weaning' period. If, as postulated, the intestinal lamina propria is critically involved in determining active immune responses and tolerance in mature pigs, then it is of importance to determine the mechanisms by which this microenvironment is established and maintained in neonatal pigs and the impact of weaning and rearing environment on immunological homeostasis. The young piglet is capable of active immune responses to live virus and to dietary components by 3 weeks old, but quantitatively and qualitatively these responses differ markedly from that in older animals (Bailey et al. 2004). Early weaning at 3 weeks of age is associated with a transient reduction in the ability of intraepithelial lymphocytes to respond to mitogens and to splenic T-cells to secrete IL-2 (Bailey et al. 2005). Furthermore, tolerance to fed proteins introduced at weaning is not fully achieved until 8 weeks of age (Miller et al. 1994).

The first hypothesis put forward to explain intestinal damage shortly after weaning is adverse immune responses to dietary antigens (Dréau & Lallès, 1999). A second hypothesis is that the lack of intestinal stimulation as a result of post-weaning anorexia is a primary factor in intestinal inflammation, with responses to dietary antigens being secondary (McCracken et al. 1999). Indeed, inflammatory cytokine gene expression is transiently up-regulated soon after weaning (Pié et al. 2004).

Feed intake around weaning

The post-weaning period is characterised by a marked reduction in VFI, poor growth and development, diarrhoea and an increased risk of disease, particularly from enterotoxigenic Escherichia coli (ETEC) and Salmonella. The hypothesis is that the reduction in VFI following weaning is critical in determining subsequent gut development, growth, maturation of gut-associated lymphoid tissue and related disease susceptibility (Pluske et al. 1997; van Beers-Schreurs et al. 1998; Spreeuwenberg et al. 2001; Vente-Spreeuwenberg et al. 2003). A relationship between feed intake after weaning and villus height has been reported (Pluske et al. 1997) and it was speculated that this relationship may affect the overall efficiency of nutrient capture and utilisation. It is also recognised that feed intake in pigs is highly variable following weaning.

Bruininx et al. (2001) have reported a highly-variable individual VFI post weaning in group-housed pigs weaned at 27–28 d of age. They have suggested that this variability may be linked to weaning weight, genotype and gender. The same authors (Bruininx et al. 2002) have established a positive relationship between cumulative feed intake during days 0–3 post weaning and villus height. However, they conclude that the physiology and function of the gut is not markedly influenced by the time between weaning and the onset of feeding or by the subsequent increase in daily food intake. Bruininx et al. (2002) have reported a minimal and variable creep intake preweaning and a highly-variable individual VFI post weaning in group-housed pigs. Interestingly, they conclude that even minimal creep feed consumption during the suckling period stimulates early post weaning VFI. English (1981) has suggested that adequate creep intake before weaning confers protection against disease. This notion is supported by a recent study showing links between creep VFI before weaning and post weaning occurrence of colibacillosis (Carstensen et al. 2005). E. coli infection is lower in piglets consuming an optimal amount of creep feed as compared with those consuming either no creep feed or high amounts before weaning. This finding illustrates the importance of VFI regulation at this period. Several factors are likely to influence VFI at weaning, including preweaning environment, age at weaning, creep feeding, mixing or stress at weaning, diet, weaning environment and health status.

Managing gastrointestinal tract health of weanling piglets through the diet

Proteins and amino acids

Dairy products are known to have a beneficial effect on VFI, growth performance, feed efficiency and health in young pigs, because of the high palatability and digestibility of protein and energy (for review, see Thacker, 1999). Intestinal villus atrophy post weaning is limited with a diet based on skimmed-milk powder as compared with feather meal (Vente-Spreeuwenberg et al. 2004a). Hydrolysing protein has no favourable effect shortly after weaning (Vente-Spreeuwenberg et al. 2004b).

Spray-dried plasma (SDP) incorporated into the diet stimulates growth performance mostly through an increase in VFI (van Dijk et al. 2001); small intestinal alterations as well as the incidence and severity of post-weaning diarrhoea are often reduced. SDP has a high palatability, which is mostly associated with the IgG fraction of SDP (Pierce et al. 2005). SDP as compared with fish meal and colistin in pigs challenged with ETEC favours the growth of lactobacilli in the ileum and caecum (Torrallardona et al. 2003). Part of the positive effect of SDP may be related to the action of insulin-like growth factor 1 on growth, non-Ig glycoprotein-enhanced intestinal protection against E. coli and improved immuno-competence through the provision of Ig (van Dijk et al. 2001). A positive beneficial effect of anti-E. coli antibodies in SDP has been demonstrated (Owuasu-Asiedu et al. 2002), and SDP decreases gene expression of IL-1β, IL-6 and TNFα in many organs (Touchette et al. 2002) and lowers immune cell density in the intestinal mucosa (Jiang et al. 2000). However, Van Dijk et al. (2002) have found no improved response to an E. coli challenge in SDP-supplemented piglets, and an immune over-response with associated increased intestinal damage has even been reported in response to a lipopolysaccharide challenge (Touchette et al. 2002). SDP has shown beneficial effects on intestinal architecture in weaned pigs challenged with E. coli (Torrallardona et al. 2003; Bosì et al. 2004). The magnitude of VFI and growth improvements is lower when SDP...
is associated with vegetable ingredients as compared with dairy ingredients (van Dijk et al. 2001). Feeding pea (Pisum sativum)-protein isolates as compared with SDP is responsible for more diarrhoea and higher mortality, increased ETEC colonisation and proliferation in the small intestine and shorter villus height (Owusu-Asiedu et al. 2003a,b). All the available data support SDP as a valuable alternative to in-feed antibiotics in weaner diets.

Glutamine and glutamate are important fuels for intestinal cells. They improve growth performance and feed efficiency post weaning and limit intestinal villus atrophy (Wu et al. 1996; Ewtushick et al. 2000; Domeneghini et al. 2004, 2006). Interestingly, glutamine stimulates the division of enterocytes while decreasing apoptosis of enterocytes and lymphocytes (Domeneghini et al. 2004, 2006). It also stimulates both innate and adaptive components of immunity, as shown by increased densities of macrophages and intra-epithelial lymphocytes. Alanine and glycine have been shown to stimulate the production of the so-called anti-secretory factor, improve growth performance and reduce the incidence of diarrhoea (Goransson, 1997). This anti-secretory factor provides protection against diarrhoeal diseases and intestinal inflammation, and it has been shown to be low immediately post weaning in pigs (for review, see Lange & Lonnroth, 2001). Arginine also prevents villus atrophy (Ewtushick et al. 2000). Cystine provided at 25% above requirements decreases jejunal mass, possibly because of its mucolytic properties (Harte et al. 2003). Finally, dietary supplementation with l-tryptophan can improve villus:crypt in the small intestine but it may depress VFI and growth (Koopmans et al. 2006).

**Prebiotics**

The addition of fermentable carbohydrates to the diet of weaning piglets is regarded as a comparatively straightforward way to improve microbiota composition and functionality in both the small and large intestines of piglets (Williams et al. 2001; Bauer et al. 2006).

As a simple molecule, lactose can be used as a fermentable substrate by weaned pigs, since intestinal lactase activity decreases rapidly after weaning. The magnitude of its positive effects depends on the level of incorporation of lactose and the presence of other fermentable substrates or the crude protein (N x 6.25) level in the diet (Pierce et al. 2006, 2007). Small intestinal architecture is improved when inulin is added to a diet low in lactose, but it has no effect when added to a diet high in lactose. A high dietary level of lactose favours bifidobacteria and lactobacilli while decreasing *E. coli* (Pierce et al. 2006, 2007). Higher levels of total VFA, and particularly butyric acid, and lower levels of branched VFA are produced with high dietary levels of lactose. Thus, the positive effects of lactose-containing diets may be a result of lower protein fermentation and NH3 production with a low lactose and inulin diet. Increasing protein to >185 g/kg fails to stimulate growth performance at a low level of lactose but has a stimulatory effect at a higher level of lactose (Pierce et al. 2006). This effect is related to decreased protein fermentation and *E. coli* counts and increased VFA production, including butyrate in the colon. Furthermore, there is also a stimulating effect on villus height in the proximal jejunum.

The composition of the bacterial community in the gut of weaning piglets is affected by the dietary addition of sugarbeet pulp, inulin, lactulose and wheat starch, a diet specifically designed to stimulate the fermentation along the entire GIT (Konstantinov et al. 2003, 2004). Fermentable carbohydrates could enhance colonic microbiota stability and diversity, with concomitant stimulation of the growth of *Lactobacillus sobrius*, a novel and beneficial member of the porcine commensal microbiota (Konstantinov et al. 2004a, 2006b). Similarly, it has been shown, using chaperonin-60 gene clone libraries, that *Lactobacillus amylovorus*-related populations, most probably *L. sobrius*, are highly abundant, although with considerable individual variation, in the ileum of weaned piglets fed either maize-barley- or wheat-based diets (Hill et al. 2005). Interestingly, the addition of inulin to different basal diets affects the proportion of piglets with detectable levels of bifidobacteria, while lactobacilli are unaffected (Loh et al. 2006). This study, however, did not discriminate between different species within groups, thus not allowing for the detection of stimulation of specific populations.

Thus, increased intake of prebiotics creates more favourable lumen conditions for gut health but direct evidence for enhanced resistance to unfavourable conditions is still lacking.

**Probiotics**

In the weaning period the most promising effects of the use of probiotics are related to the competitive exclusion of pathogenic bacteria. This effect could be a result of their positive influence on gut microbiota balance, intestinal epithelium integrity, appropriate maturation of the gut-associated tissue and function of the neuro-endocrine system (Metzler et al. 2005). In weaning pigs challenged with pathogens a strategy to select favourable commensal strains from the pig gut seems the more successful. A mix of four lactobacilli isolated from weaning pigs can reduce *E. coli* and anaerobe counts in the gut, and decrease diarrhoea (Huang et al. 2004). The probiotic effect of *L. sobrius* 001T against ETEC K88 has been examined using *in vitro* and *in vivo* approaches. Supplementation of a diet based on fermentable fibre with *L. sobrius* improves the body-weight gain of weaned pigs orally challenged with ETEC K88 and reduces ileal ETEC abundance, but it does not reduce diarrhoea (Konstantinov, 2005). Beneficial effects of this strain can also be related to its ability to counteract intestinal permeability disturbances induced by ETEC on porcine IPEC-1 intestinal cells (M Roselli, A Finamore, SR Konstantinov, MS Britti, WM de Vos, H Smidt and E Mengheri, unpublished results). However, after supplementation with *Lactobacillus rhamnosus* GG (used in human subjects) in weaning pigs orally challenged with ETEC reduced growth and a trend to more ETEC excretion in faeces has been observed (Trevisi, 2005).

A study on the interaction between intestinal physiology, dietary supplementation with the probiotic *E. coli* strain Nissle 1917 and ETEC challenge has recently been
reported (Schroeder et al. 2006). Before being weaned at 21 d of age the piglets were creep-fed a diet with or without the probiotic supplement for 10 d starting at 7 d of age, and at 4 and 24 h post weaning they were challenged with ETEC. The probiotic was found to abolish diarrhoea, reduce secretagoge-induced chloride secretion at the jejunum and suppress the decreased paracellular permeability observed after ETEC challenge in non-supplemented pigs (Schroeder et al. 2006). Many mechanisms may be involved in the intestinal epithelial cell protection by probiotics against ETEC, including competitive exclusion, reduced ETEC adhesion, maintenance of epithelial tight-junction integrity, reduced neutrophil transmigration and increased mucin gene expression, depending strongly on the probiotic strain used (Roselli et al. 2005). Administration of live yeast (Saccharomyces cerevisiae spp. boulardii) to weaned pigs for 3–4 weeks improves growth performance post weaning, villus height, epithelial cell proliferation and the numbers of macrophages at various sites of the small intestine (Baum et al. 2002; Bontempo et al. 2006). Interestingly, supplementing the sows with Escherichia faecium strongly decreases the incidence of diarrhoea in piglets in the first week post weaning (Taras et al. 2006). It also reduces the level of cytotoxic (CD8+) T-cells in the jejunal epithelium of the piglets, probably in relation with the lower frequency of β-haemolytic and O141 serovars of E. coli (Scharek et al. 2005).

**Dietary fibre and colibacillosis in weaned piglets**

Australian studies (Hopwood & Hampson, 2003) have provided the basis for dietary management of important enteric infectious diseases in pigs, including swine dysentery, intestinal spirochaetosis and gastric ulceration; the main conclusion being that to reduce the proliferation of associated pathogens diets should be low in soluble NSP and/or resistant starch. However, a recent Danish study (Lindecrona et al. 2003) has failed to confirm these observations. The conclusion of the Australian studies (Hopwood & Hampson 2003, Hopwood et al. 2004) is that post-weaning colibacillosis is influenced by dietary components, especially NSP, which generates high viscosity of intestinal contents. Viscous carboxymethylcellulose has been shown to favour post-weaning colibacillosis in piglets when incorporated into a diet based on cooked white rice and animal protein, which is itself considered to be protective to the GIT (McDonald et al. 2001). However, this model substance associated with skimmed milk and maltodextrin reveals a relatively protective effect on the intestine of weaned pigs (Lallès et al. 2006; Piel et al. 2005). Thus, carboxymethylcellulose may impact positively on the gut depending on circumstances. Replacing animal proteins with plant protein sources in a cooked white rice protective diet (McDonald et al. 2001) does not increase the risk of post weaning colibacillosis (Montagne et al. 2004).

On the other hand, pearl barley rich in β-glucans increases digesta viscosity and exacerbates post-weaning colibacillosis and diarrhoea (Hopwood et al. 2004). In contrast, β-glucans from lentinan (from Lentinus edodes) stimulate jejunal villus length and villus height:crypt depth, possibly as a result of depressed bacterial load (Van Nevel et al. 2003). Other sources of non-digestible oligosaccharides include guar gum, locust bean gum (which is extracted from carob tree (Ceratonia siliqua) seeds) and carob tree seeds, of which carob tree seeds have been shown to have the more marked effects, reducing villus height and increasing crypt depth in the proximal jejunum (Van Nevel et al. 2005). Locust bean gum decreases the numbers of lactobacilli in the stomach and jejunum, while carob tree seeds increase E. coli counts in the contents and mucosa of the distal jejunum (Van Nevel et al. 2005). Phosphorylated mannan derived from yeast (Saccharomyces cerevisiae) cell wall stimulate phagocytosis of intestinal macrophages and increase the proportion of CD8+ T lymphocytes in the jejunal lamina propria, in addition to improving growth performance (Davis et al. 2004).

**Other substances**

Natural plant or herbal extracts are yet another potential viable alternative to traditional antimicrobials. Isoflavone phyto-oestrogens have been shown to affect performance, physiology and gut microbiota of farm animals (for review, see Han et al. 2006). Interestingly, administration of daidzein causes an increase in the relative abundance of lactobacilli in in vitro fermentation assays (Yao et al. 2004). A mixture of carvacrol, cinnamaldehyde and capiscum oleoresin, given at increasing doses to piglets from 12 d post weaning, linearly increases intestinal lactobacilli as well as lactobacilli:enterobacteria (Manzanilla et al. 2004). Similarly, an extract containing cinnamon (Cinnamomum zeylandica), thyme (Thymus vulgaris L.) and oregano (Origanum vulgare L.) extract reduces the growth of coliforms (Namkung et al. 2004). Curiously, little information is available on the effect of antioxidants on the GIT of weaning pigs despite the body of literature existing on the protective properties of such substances (e.g. antioxidants from tea; Asfar et al. 2003).

**Conclusions and perspectives**

The weaning period in pigs is complex and its nutritional control is not easy to achieve. However, important progress has been made in understanding this complexity at the GIT level, with reference to the physiology, microbiota and local immune system. Based on this knowledge, stimulating beneficial bacteria and (or) bacterial metabolites through the consumption of fermentable carbohydrates (prebiotics) in the diet is clearly one major option. Recent studies of the effects of some probiotics on GIT health also look promising, but the responses show strain specificity. By contrast, there is still the need for a basic knowledge of the development of mucosal immunity in order to envisage practical nutritional approaches to control the regulator and effector arms of immune responses.

Future investigations should be directed towards: understanding the intimate interactions between microbiota, mucosal physiology and immunity; systematic underscore diverse bacterial roles in the gut. The maintenance of microbial balance is crucial for a basic understanding of gastrointestinal physiology and disease.
quantitative assessment of GIT microbiota requirements for fermentable carbohydrates at various GIT sites; definition of optimal combinations of simple and complex prebiotics together with other dietary components that maximise animal performance and health; evaluation of the impact of such diets balanced for microbiota requirements on piglet susceptibility to GIT disorders and enteric infections. Finally, many other substances of plant origin have the potential to contribute to GIT health around weaning. However, more convincing data and mechanistic interpretations are needed before these substances can be considered for inclusion in commercial diets.

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