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Interaction between chicken intestinal microbiota and protein digestion

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ABSTRACT

The different sections of the broiler chicken intestinal tract are inhabited by specialist microbiota adapted to the physicochemical conditions, host physiology and available nutrients of the specific habitat. The small intestine is dominated by lactic acid bacteria which have complex nutrient requirements resembling those of the chicken host itself. Lactobacilli are unable to synthesise amino acids for their anabolism and are therefore highly dependent on amino acid availability in the growth environment. Thus, in the small intestine there is competition for amino acids between the microbiota and the chicken host. According to rough estimates, lactobacilli in the small intestine may assimilate 3–6% of total dietary amino acids. If the protein is highly digestible and amino acids are largely absorbed in the upper small intestine, where bacterial growth is suppressed, the proportion captured by the host may be higher. Exogenous enzymes which promote protein digestion are also likely to provide a competitive advantage to the chicken, offering less growth potential for amino acid-dependent bacteria.

Protein escaping the ileum comprises resistant protein of dietary origin, protein assimilated to intestinal bacteria and endogenous protein synthesised and secreted by the host, the latter synthesised in host tissues from dietary amino acids and thus representing true endogenous protein. Activities of small intestinal bacteria affect the size of the microbial protein fraction and also the production of endogenous proteins originating from mucin, epithelial cells and antibodies.

Ileal bypass protein is subject to fermentation by putrefactive bacteria in the caecum. Putrefaction produces many harmful and toxic compounds, which in high concentrations may have adverse effects on chicken growth and performance. The protein fermentation products include amines, indoles, phenols, cresol and ammonia, which can all negatively affect host or cell health. All actions to reduce the amount of ileal bypass protein potentially also reduce production of toxic protein fermentation metabolites in the caecum. Enzymes which facilitate protein digestion in the upper intestine and soluble carbohydrates resistant to ileal digestion both reduce caecal putrefaction. In the distal intestine, saccharolytic fermentation is preferred and putrefaction accelerates only when utilisable carbohydrates are depleted. Soluble oligo- and polysaccharides are produced in situ by non-starch polysaccharide degrading enzymes and can also be added directly to the diet as health-promoting prebiotics.

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1. Intestinal microbiota of the broiler chicken

Like all homeothermic animals, chickens have a complex intestinal microbiota, the composition and metabolism of which vary between intestinal compartments with highly different physicochemical microenvironments. The main factors that drive the fitness and colonisation efficiency of the microbes are the availability of suitable growth substrates, prevailing pH and redox potential and the antibacterial secretions of the host in a specific intestinal section. Ingested feed has a high concentration of readily available substrates which could potentially be utilised by a wide variety of bacteria. The availability of easy growth substrates for bacteria decreases on moving down the gastrointestinal tract. That is why bacteria in the lower intestine are often specialists in utilising feed components that are resistant to the endogenous digestive system of the host, e.g. non-starch polysaccharides, resistant starch or resistant protein. The proximal gastrointestinal tract (crop, proventriculus, gizzard) is characterised by low pH, which strongly selects bacteria and limits the growth of many species (Morgan et al., 2014). Redox potential determines the type of energy metabolism that is thermodynamically possible for the bacteria living in a particular gastrointestinal habitat. The difference between the redox state of the substrate (electron donor) and the terminal electron acceptor determines whether the reaction can proceed and generate ATP under certain conditions. For example, bacteria that are only capable of gaining energy with molecular oxygen as an electron acceptor cannot grow in the low redox conditions of the distal intestine. Moreover, strictly anaerobic bacteria are often highly sensitive to reactive oxygen species, having no means of quenching the radicals. Due to the abovementioned factors, microbiota composition in e.g. the jejunum and caecum is highly different.

The crop and small intestine of broiler chickens is dominated by lactic acid-producing bacteria, mainly *Lactobacillus* spp., *Enterococcus* spp. and *Streptococcus* spp. (Barnes et al., 1972; Salanitro et al., 1978; Lu et al., 2003; Apajalahti and Kettunen, 2006a; Bjerrum et al., 2006; Abbas Hilmi et al., 2007). These bacteria have purely fermentative metabolism; they do not need molecular oxygen, but most species are not harmed by its presence. In various studies, up to 95% of the total small intestinal bacteria have been found to represent the genera of lactic acid bacteria. This intestinal habitat, characterised by middle range redox potential (~180 mV; Apajalahti et al., unpublished results) with some trickling oxygen from proximal digesta and host epithelium, also supports growth of facultative anaerobic bacteria which can switch between aerobic and anaerobic metabolism. The best known representatives of this bacterial group are *Escherichia coli* and *Salmonella enterica*. Over the past years, the Alimetrics Ltd laboratory has analysed intestinal samples representing 10 European commercial broiler chicken farms. Fig. 1 shows the average microbiota composition on these farms. It is worth noting that healthy broiler chickens have few strict anaerobes in their small intestine. In some cases, obligate anaerobes of the caecum were found in the small intestine. However, we cannot exclude the possibility that reverse peristalsis or careless sampling techniques introduced minor caecal digesta contamination, which would have dramatically affected the results since the bacterial density of caecal digesta exceeds that of ileal digesta by a factor of 100–1000.

The caecum of broiler chickens is dominated by strictly anaerobic bacteria, many of which cannot be assigned to a known bacterial genus. However, more than half of these bacteria belong to the order Clostridiales (families *Lachnospiraceae* and *Ruminococcaceae*, also referred to as Clostridial clusters XIVa and IV, respectively) (Lu et al., 2003; Apajalahti and Kettunen, 2006a; Bjerrum et al., 2006). One characteristic of these bacterial families is their ability to utilise complex plant-derived carbohydrates and to produce butyrate. The known representatives of the family *Ruminococcaceae* are able to attack cellulose and other highly recalcitrant polysaccharides more efficiently than the members of *Lachnospiraceae*, but the *Lachnospiraceae* are able to readily degrade less recalcitrant non-starch polysaccharides and starch (Biddle et al., 2013). A significant proportion of the caecal bacteria also belong to the families *Bifidobacteriaceae* and *Coriobacteriaceae* (Fig. 1). Bifidobacteria have been linked to degradation of simple carbohydrates and oligosaccharides and production of lactic and acetic acid. The role of coriobacteria is poorly known, but there are some reports suggesting that they are connected to lipid and cholesterol metabolism (Martínez et al., 2013).

It has long been of interest to link certain bacterial groups to the health and performance of broiler chickens. In general, the characteristics of health-promoting bacteria could include upregulation of host immune defence, stimulation of mucin production and proliferation of epithelial cells. Indeed, bacteria have been reported to catalyse such functions (Deplancke and Gaskins, 2001; Hörmann et al., 2014). While these physiological functions undoubtedly promote intestinal health in the host, they all use energy and can be expected to reduce the feed conversion efficiency of production animals. In order to improve feed conversion efficiency, bacteria should suppress the abovementioned protective functions, thus minimising the endogenous losses. The practical targets of health- and performance-promoting activities appear to be mutually exclusive (Lochmiller and Deerenberg, 2000).

Bacteria capable of utilising feed components unavailable to the digestive system of the host would expand the nutritional diversity of the superorganism (animal + its microbiota) and thus improve the overall energy capture from the diet. The extent to which e.g. the non-starch polysaccharide-utilising, organic acid-producing members of the *Lachnospiraceae* and *Ruminococcaceae* contribute to final feed conversion efficiency is currently unknown. However, a positive correlation has been found between caecal *Lachnospiraceae* spp. and feed conversion efficiency in commercial broiler chicken farms (Torok et al., 2008; Rinttilä and Apajalahti, 2013). Unexpectedly, increased abundance of lactobacilli and bifidobacteria in the caecum has been found to indicate poor performance. This may be due to digestive and nutrient uptake disorders in the small intestine, which increase nutrient bypass to the caecum and consequent activation of microbial groups that are dependent on simple substrates.

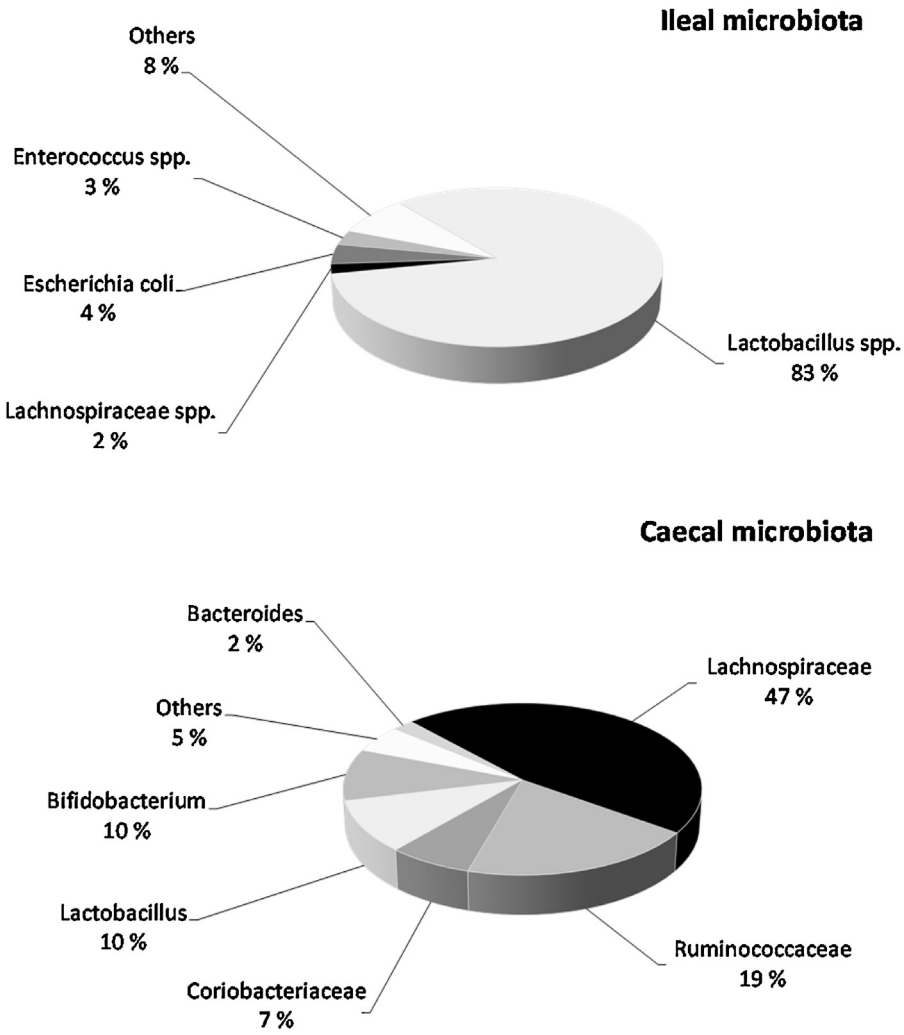

Fig. 1. *Average microbiota composition on commercial broiler chicken farms.* The diagrams show the average microbiota composition (%) in the ileum (upper panel) and caecum (lower panel) for 10 European farms (Alimetrics Ltd laboratory, unpublished results).

2. Microbial competition for dietary protein in chicken small intestine

The gastrointestinal tract of an adult broiler chicken supports the growth of up to 10^{13} bacteria. Million-fold differences in bacterial density exist between different intestinal sections, the densest populations being found in the caecum and the least dense in the proximal small intestine. The composition and digestibility of the diet has a major impact on the intestinal microbiota, since compounds of dietary origin are the most important growth substrates for microbes. Diets are formulated to match the nutritional requirements of the broiler chicken, although management of the metabolically highly active microbiota through diet may also play an important role in the health and performance of the host.

The mechanisms by which the microbiota affects broiler chicken performance vary between intestinal sections. In the small intestine, the host absorbs most of the nutrients, such as carbohydrates, amino acids and vitamins. Therefore, any disorders in the small intestine are likely to relate directly to performance. In the proximal small intestine (duodenum, jejunum), the total number of bacteria is lower than in the distal small intestine (ileum). Accordingly, there is a negative correlation between the nutrient content of small intestine digesta and its microbial density. The most obvious reasons for this are the digestive physiology and chemistry of the gastrointestinal tract. When passing through the gizzard, digesta and bacteria from the crop undergo harsh treatment with low pH and mechanical grinding, which is likely to kill a significant proportion of bacteria present and those still alive can be expected to be seriously damaged. Furthermore, many but not all bacteria are sensitive to the bile salts (Lin et al., 2007) secreted to the proximal duodenum, which weakens their metabolic activity. These mechanisms, together with the rapid passage of digesta from the low to high bacterial density region, maintain the steep bacterial density gradient in the small intestine. Towards the distal ileum, bacterial density and metabolic activity

Table 1
Amino acid requirements of some small intestine bacteria.

	Lactobacillus spp.[a]	*Clostridium perfringens*[b]	*Escherichia coli*
Alanine	±	−	−
Arginine	+	+	−
Aspartic acid	+	−	−
Cysteine	+	−	−
Glutamic acid	+	+	−
Glycine	±	−	−
Histidine	±	+	−
Iso-leucine	+	+	−
Leucine	+	+	−
Lysine	±	−	−
Methionine	+	+	−
Phenylalanine	+	+	−
Proline	±	−	−
Serine	±	−	−
Threonine	+	+	−
Tryptophan	+	+	−
Tyrosine	+	+	−

+growth of the tested strains was dependent on the amino acid
±growth of some of the tested strains was dependent on the amino acid
−growth of the tested strains was not dependent on the amino acid
[a] Morishita et al., 1981.
[b] Sebald and Costilow, 1975.

increase in parallel with the decreasing rate of digesta passage and weakening of the antibacterial potency of the digestive fluids.

It is difficult to make any general statements about the absolute bacterial density in different parts of the chicken small intestine because it is dependent on multiple different factors. One of the most important variables is undoubtedly the composition and digestibility of the diet. Unfortunately, the methods used for bacterial quantification also play an important role. The methods applied over decades are so non-comparable that between-studies comparison is not meaningful. Even the results of studies using different culture-independent tools can provide contradictory results due to varying efficiency of bacterial lysis, DNA recovery and the selection of primers (Apajalahti and Kettunen, 2006a).

The nutritional requirements of bacteria are species-dependent. Those with the simplest requirements can synthesise all biomolecules, from sugars and minerals, while bacteria with the most complex requirements are unable to grow if amino acids, vitamins and many other compounds are not available in the growth environment. As discussed in the previous paragraph, the small intestine of broiler chickens is dominated by representatives of the genus *Lactobacillus*. The members of this bacterial genus are among the most fastidious bacteria, with highly complex nutrient requirements (Morishita et al., 1981). In contrast, *E. coli*, also present in the small intestine, is independent of any exogenous amino acids, requiring only an utilisable sugar and minerals (Table 1). The fact that lactobacilli are present in the small intestine in high numbers implies that they assimilate a significant amount of amino acids, vitamins and simple carbohydrates from the intestinal lumen. It is possible to make a rough approximation of the proportion of dietary amino acids captured by small intestinal lactobacilli. In the present case, the following assumptions were made: i) The distal half of the small intestine has average bacterial counts of 1×10^{10}/g digesta (based on quantitative PCR analysis in the Alimetrics Ltd laboratory using the eubacterial primers with the highest coverage; Apajalahti and Kettunen, 2006a), ii) the average fresh weight of one cell of intestinal lactobacillus is 3–6 picograms (calculation based on microscopic examination of bacterial size), iii) the dry matter content of bacterial cells is 30% and the protein content is 55% of bacterial dry matter (microbiology textbooks), iv) 1 g ileal digesta corresponds to 4 g ingested feed (dry matter), and v) the protein content of the diet is 20%. Based on these assumptions, the small intestinal lactobacilli assimilate an estimated 3–6% of total dietary protein. However, it is worth noting that this estimate is highly sensitive to absolute bacterial density and the physical size of bacterial cells, both of which may vary significantly.

As shown in Table 1, some intestinal bacteria are highly dependent on amino acids in easily digestible form. Therefore, it is obvious that the resident microbiota competes with the host for dietary amino acids. The more the physiological functions of the host (digesta passage, secretion of acids, bile salts and antibodies) limit the growth of microbiota, the larger the proportion of amino acids available for growth of the host. In practice, this means that there is less competition for nutrients in the proximal part of the small intestine, where the density of microbiota is low. The further down the intestine the amino acids move before being absorbed by the host, the more likely it is that they will be captured by intestinal bacteria and support their growth. Unlike in ruminants, in monogastric animals all protein (amino acids) assimilated to microbial biomass will most likely be permanently unavailable for the host. Thus, rapid digestion of dietary proteins and uptake of amino acids as early as possible in the small intestine can be expected to reduce the proportion of amino acids assimilated by intestinal bacteria. A low concentration of amino acids in the distal small intestine can be expected to improve the competitiveness of bacteria that are not dependent on externally available amino acids (e.g. *E. coli*) and reduce the proportion of bacteria dependent on amino acids (e.g. lactobacilli; see Table 1).

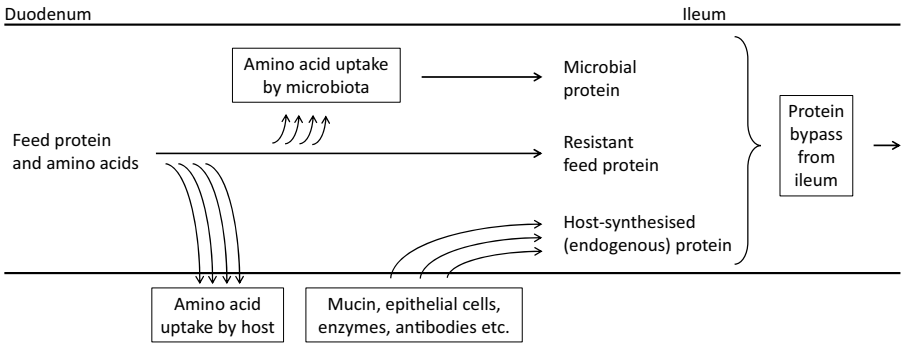

Fig. 2. *Schematic presentation of protein flow in the small intestine.* The protein escaping the ileum consists of undigested protein of feed origin, microbial protein and protein synthesised by the host (true endogenous protein).

Like lactobacilli, *Clostridium perfringens*, the causative agent of necrotic enteritis, is also dependent on several amino acids in the habitat in which it resides (Fuchs and Bonde, 1957; Sebald and Costilow, 1975; Wilkie et al., 2005) (Table 1). Indeed, this may be a reason why excess dietary protein (e.g. fish meal) is commonly used when the objective is to reproduce necrotic enteritis in challenge trials (Shojadoost et al., 2012). The tissue damage caused by *Eimeria* parasites predisposes the small intestine to necrotic enteritis (Shojadoost et al., 2012). It is possible that in this case the tissue fluids leaking into the gut from the damaged epithelium provide *C. perfringens* with the amino acids it requires for growth.

3. Impact of microbiota on ileal bypass of amino acids

Even with the highest quality diet, not all dietary protein is captured by the host in the small intestine. As illustrated in Fig. 2, the protein bypassing the small intestine represents i) resistant protein of dietary origin, ii) microbial protein or iii) protein synthesised by the host. The fraction of dietary protein which cannot be digested by either host proteases or small-intestine bacteria constitutes part of the protein escaping the small intestine, the amount of which is directly dependent on the quality of the dietary protein (raw material or feed processing).

As discussed in the previous section, the small-intestinal lactobacilli and other bacteria compete for amino acids, inevitably absorbing some dietary amino acids and utilising them in cellular anabolism. This bacterial protein constitutes the second part of the protein that bypasses the ileum. The main fraction of dietary protein is digested by the host digestive system and taken up as amino acids and small peptides by the host. The third fraction escaping the small intestine is the protein synthesised and secreted by the host and it originates from the dietary amino acids previously taken up by the host epithelium. Unlike the protein fractions discussed above, this fraction can be referred to as truly endogenous protein of the host (produced or synthesised within the host organism). In practice, this endogenous protein comes from sloughing epithelial cells, mucin produced by goblet cells, digestive enzymes, secretory antibodies etc.

The cross-talk between microbiota and host regulates the magnitude and type of host defence mechanisms and the production of endogenous proteins responding to foreign antigens (Deplancke and Gaskins, 2001; Canny and McCormick, 2008; Abraham and Medzhitov, 2011; Hörmann et al., 2014). Immunological tolerance to harmless commensal microbiota is essential when the objective is to avoid unnecessary expression of defence functions and inflammatory reactions in intestinal tissue. Indeed, well-developed homeostasis and accurate recognition of harmful antigens and pathogens helps to reduce consumption of limited amino acid and energy resources for maintaining non-justified alertness (Kelly et al., 2005). Microbiota is known to regulate the rate of mucin production and its composition (Deplancke and Gaskins, 2001). The rate of epithelial cell proliferation is also affected by the presence of microbiota (Hörmann et al., 2014). It is obvious that the active proliferation of epithelial cells and the production of mucin and antibodies help the host to defend itself against potential threats from the environment. The mode of action and the specific role of different intestinal microbes for endogenously produced proteins are not completely understood.

When the size of any protein fraction escaping the small intestine grows, the impact from the animal growth performance point of view is undesirable. In the ideal case, dietary protein should be easily digestible, microbial protein in the small intestine should be kept to a minimum and endogenous proteinous secretions used for defence should not be overexpressed due to the high costs for feed conversion efficiency.

4. Effect of protein bypass on caecal putrefaction

A high level of bypass protein indicates that dietary amino acids have escaped the host digestion and become unavailable to the host. However, it also has major impacts on distal intestinal functions and, potentially, animal health. In addition to indisputably recognised pathogens, there are many functional bacterial groups which under certain conditions release metabolic products considered harmful for the well-being of the host. Such functional bacterial groups include putrefactive or protein-fermenting bacteria, which produce toxic end-products in their amino acid metabolism. Protein entering the

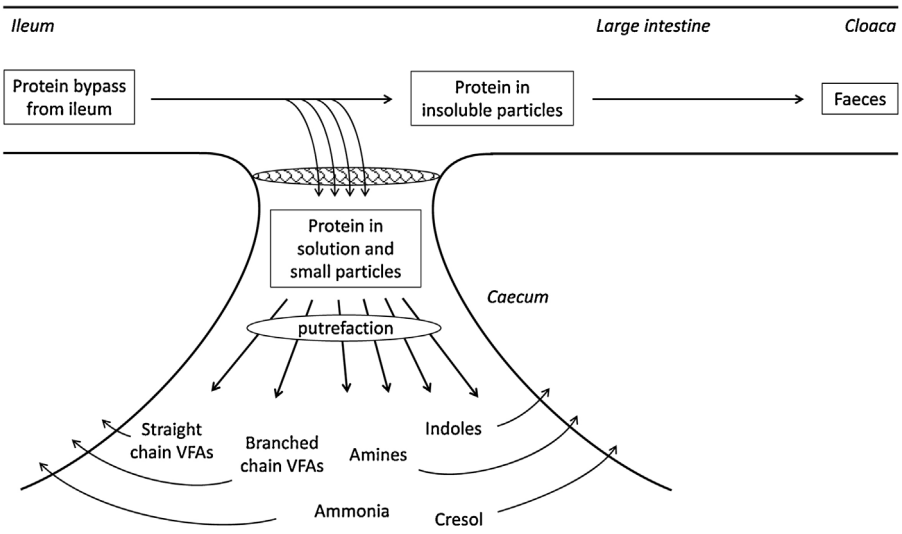

Fig. 3. Protein fermentation of ileal bypass protein in the caecum.

caecum is a potential target for protein-fermenting bacteria. From the terminal ileum, protein can either enter the caecum or pass across the ileocaecal junction to the large intestine. The caecal opening is controlled by an interdigitating meshwork of villi and musculature that acts as a filter, allowing entry to the caecum of fluid and fine particles only (Ferrando et al., 1987; Clench and Mathias, 1995). Larger insoluble particles do not enter the caecum, but are passed on to the cloaca (Fig. 3).

Many of the metabolic products of putrefaction are specifically found as end-products of protein, and not of carbohydrate, fermentation. Straight-chain volatile fatty acids (VFAs) are produced in both fermentation types, while branched chain fatty acids (BCFAs) are only produced in protein fermentation, specifically when branched-chain amino acids are fermented. The amino acids valine, leucine and isoleucine are converted to isobutyrate, 2-methyl-butyrate and isovalerate, respectively (Smith and Macfarlane, 1998). Thus, the presence of these BCFAs indicates ongoing protein fermentation activity. Unlike many other protein fermentation products, BCFAs are not known to be toxic.

Aromatic amino acids produce toxic end-products when fermented by putrefactive bacteria. Perhaps the best known example is 3-methyl-indole (skatole), which is the compound causing boar taint in male pigs. Skatole is produced by specific intestinal bacteria from the amino acid tryptophan (Jensen et al., 1995). The toxicity of indoles is based on their function as uncouplers of the proton gradient across biological membranes and thus inhibition of oxidative phosphorylation (ATP production) already at concentrations found in the human colon (Chimerel et al., 2013). The amino acid tyrosine is converted to *p*-cresol and phenol by intestinal bacteria (Tsudji, 1919; Vanholder et al., 1999). The mechanism of toxicity is the same as for indole. When there is excess protein available in the intestine, deamination reactions produce more ammonia than can be assimilated by the growing bacteria. Ammonia has been described as having several adverse health effects on human epithelial cells, including altering epithelial morphology, metabolism and DNA synthesis (Clausen and Mortensen, 1992; Matsui et al., 1995). The concentration of ammonia in the lumen has also been shown to be negatively correlated with villus height in piglets (Nousiainen, 1991).

Biogenic amines are a range of compounds produced by host cells and by microbial decarboxylation of amino acids. All known biogenic amines are biologically active, some are toxic and some regulate the rate of cell proliferation and other cell functions (Smith et al., 2000). Putrescine is a product of ornithine decarboxylation and is a precursor for the synthesis of other polyamines, spermidine and spermine. In fact, at low, non-toxic concentrations, putrescine and spermine have been shown to have positive effects on growth in chickens (Smith, 1990; Smith et al., 1996).

Small intestine disorders in protein digestion and amino acid uptake bring a pulse of protein to the distal intestine, consequently elevating the level of protein fermentation metabolites in the caecum. Apajalahti and Bedford (2000) showed that *Eimeria maxima* challenge caused such elevation in total biogenic amine levels in the caecum, possibly by interfering with the integrity and absorptive capacity of small intestinal epithelium. Biogenic amines in the caecum peaked on day 8 after the coccidial challenge, when the measured 20 mM concentration was four-fold higher than in the unchallenged birds. Six days later the birds had recovered and the residual concentration of biogenic amines had dropped back to the normal level. The challenge did not affect the amine levels in the ileum. The bypass protein and carbohydrate also caused transient shifts in microbial community structure. A highly significant change in the microbial profile coincided with the most significant change in the metabolite profile and also returned to normal by day 14 after the challenge (Apajalahti and Bedford, 2000). The example above describes the change in caecal protein metabolism following *E. maxima* challenge. A corresponding modulation of caecal microbiota, although less drastic, could be expected when the bypass of soluble protein from the ileum changes due to other modulations, such as a shift in mucin production, epithelial cell sloughing, use of exogenous proteases etc.

A positive correlation between ileal protein digestibility and broiler chicken performance has been reported (Cowieson and Bedford, 2009; Cowieson and Roos, 2014). When the diet becomes limited in any essential amino acids due to poor protein digestibility, it is natural for body weight to be reduced. However, it is possible that part of the growth suppression comes from the adverse effects of protein fermentation in the caecum. As a consequence of poor protein digestibility in the small intestine, protein bypass to the caecum may increase and the elevated putrefaction may increase the concentration of harmful metabolites that suppress animal growth. The extent to which this mechanism plays a role in real-life situations remains to be answered in future research.

5. Potential ways to reduce protein-microbiota interactions and the resulting negative effects on health and performance

As described above, the metabolism of dietary amino acids and protein by small intestinal and caecal microbiota has a potentially negative effect on the performance of broiler chickens. However, the mechanism is fundamentally different in these two compartments of the gastrointestinal tract. In the small intestine, the microbiota competes with the host for dietary amino acids and assimilates them into cell biomass, while the caecal bacteria exclusively utilise protein and amino acids that have already escaped the host digestion. Instead of competing for nutrients, the putrefying bacteria of the caecum cause problems for the host by producing potentially toxic metabolites. Many feed ingredients and feed processing technologies have an impact on these interactions between microbiota and dietary protein, although the mechanisms have been little studied. The most widely used product group with a significant impact are growth-promoting antibiotics. Growth promoters reduce bacterial numbers in the small intestine, in parallel with improved body weight gain (Apajalahti and Kettunen, 2006b). It is highly likely that one of the most important modes of action of antimicrobial growth promoters is to improve the capture of amino acids and other nutrients by the host epithelium by reducing microbial competition.

Exogenous proteases improve both the overall digestibility of dietary protein and the kinetics of protein hydrolysis (for review see Cowieson and Roos, 2014). By increasing the rate of protein digestion in the small intestine, a protease can be expected to increase the proportion of amino acids and peptides absorbed by the host instead of intestinal lactobacilli. The earlier in the small intestine the dietary proteins are hydrolysed and amino acids released, the lower the probability of amino acids being assimilated by bacteria. This is because bacterial growth activity in the proximal small intestine is low, due to the mechanisms discussed in previous sections. If a protease positively affected not only the kinetics of protein breakdown, but also the total ileal digestibility of the dietary protein, it could reduce the overall protein bypass from the ileum and the protein load to the caecum. This would reduce health risks caused by protein fermentation and production of toxic metabolites in the caecum. Thus, exogenous protease might extend positive microbial effects all the way from small intestine to caecum.

Hemicellulases may promote ileal digestibility of proteins by breaking up plant cell wall structures which trap proteins. This explains why in-feed xylanases have shown effects on protein digestibility similar to those reported for proteases (Cowieson and Bedford, 2009). Polysaccharide-degrading enzymes have been shown to improve broiler chicken performance in multiple studies (e.g. Choct et al., 1995). With xylanase supplementation of the diet, the carbohydrate-degrading microbiota in the caecum are stimulated (for review see Bedford and Apajalahti, 2001). This can be explained by enzymatic solubilisation of xylan with production of *xylo*-oligosaccharides which, unlike insoluble xylan, can enter the caecum. When both proteins and carbohydrates co-exist in the intestinal habitat, bacteria preferentially ferment carbohydrates. Accordingly, non-starch polysaccharide degrading enzymes which produce oligosaccharides in situ could be expected to reduce putrefaction in the caecum. There is currently little information available on the relative activities of saccharolytic and putrefactive bacteria in the caecum of broiler chickens. In other intestinal systems, the presence of digestible carbohydrates in the intestinal digesta has been shown to suppress protein fermentation (Xu et al., 2002; Sanchez et al., 2009). When carbohydrates in the distal intestine become depleted, putrefaction becomes the dominant type of fermentation. This largely accounts for the health effects of prebiotics and dietary fibre; as slowly digestible structures, they provide carbohydrates also for the distal colon, thus suppressing putrefaction.

Conflict of interest

None.

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