Review

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Regulating Gut Microbiota in Post-Weaned Pigs: The Role of Digestive Capacity and Substrate Flow

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Abstract

In commercial pig production systems, early weaning disrupts the coordinated maturation of the gastrointestinal tract, resulting in reduced feed intake, impaired digestive capacity, altered microbial ecology and increased susceptibility to post-weaning diarrhoea (PWD). Although enterotoxigenic *Escherichia coli* (ETEC) is frequently implicated, variation in disease expression is not explained by pathogen presence alone, but reflects interactions among host physiology, nutrient flow and microbial metabolism. This review examines the regulation of the gut microbiota in post-weaned pigs through the interaction between digestive capacity and dietary substrate supply. It proposes substrate flow as the organising principle linking digestive function, diet composition and microbial metabolism. Gut microbial function is regulated primarily by substrate availability, which is determined by the alignment between diet composition and host digestive capacity. When this alignment is disrupted, undigested nutrients are redistributed to the hindgut, driving a shift from saccharolytic to proteolytic fermentation. This transition generates metabolites that impair epithelial integrity, increase luminal pH and favour proliferation of opportunistic bacteria, thereby promoting intestinal dysfunction. Within this context, nutritional strategies, including optimisation of dietary protein, provision of fermentable carbohydrates and support of gastric function, act by regulating substrate flow rather than directly modifying microbial composition. Organic acids, functional ingredients and maternal influences operate through the same mechanisms, shaping nutrient digestion, microbial exposure and metabolic outcomes. The characteristic post-weaning increase in Enterobacteriaceae and reduction in microbial diversity are therefore best understood as consequences of altered substrate flow and luminal conditions, rather than primary initiating events. This interpretation provides a mechanistic basis for the design of integrated nutritional and management strategies to improve gut health and reduce antimicrobial reliance in pig production systems.

Keywords: gut microbiota; post-weaning pigs; substrate flow; digestive capacity; saccharolytic fermentation; proteolytic fermentation; organic acids; maternal nutrition; feed intake; gut health

1. Introduction

The gastrointestinal tract of the pig is a highly adaptive system that integrates nutrient digestion, epithelial function, microbial activity and mucosal immunity. From birth to weaning, these processes develop in parallel, allowing the piglet to move gradually from milk to more complex dietary substrates. Under commercial conditions, however, weaning typically occurs at 2–4 weeks of age, before digestive and immune functions have fully matured. This creates an abrupt dietary, environmental and social transition that disrupts the relationship between digestive capacity and nutrient supply, placing pressure on gastrointestinal stability [1–3]. This review proposes substrate

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flow as the central mechanism linking digestive capacity, diet composition and microbial metabolism in the post-weaning pig.

Figure 1 shows the central concept. Gastrointestinal stability after weaning depends on how effectively nutrients are digested and absorbed in the upper gastrointestinal tract and, in turn, on the amount and type of substrate reaching the hindgut. When digestion in the upper tract is effective, fermentation is directed towards carbohydrate utilisation, supporting short-chain fatty acid (SCFA) production, lower luminal pH and a stable anaerobic microbiota. When digestion is incomplete, undigested protein and other nutrients pass to the hindgut in greater amounts, shifting microbial metabolism towards proteolytic pathways associated with increased luminal pH, impaired barrier function and a higher risk of diarrhoea.

This view contrasts with the traditional pathogen-focused explanation of post-weaning diarrhoea. Enterotoxigenic *Escherichia coli* (ETEC) is commonly associated with PWD, but its presence alone does not explain why disease develops in some pigs or herds and not others [4]. Outcomes depend on the luminal environment in which these organisms operate. Reduced feed intake, villous atrophy, impaired digestion and altered substrate flow create conditions that favour opportunistic bacteria and increase the effects of microbial metabolites on the intestinal mucosa [3,5,6].

Within this context, the gut microbiota is better understood in functional terms rather than as a list of organisms. Its activity reflects substrate supply. The key question is not only which microbes are present, but which substrates reach the hindgut and how they are metabolised. This review examines how digestive capacity develops and is disrupted at weaning, and how diet composition, maternal influences and management practices affect substrate flow and microbial function.

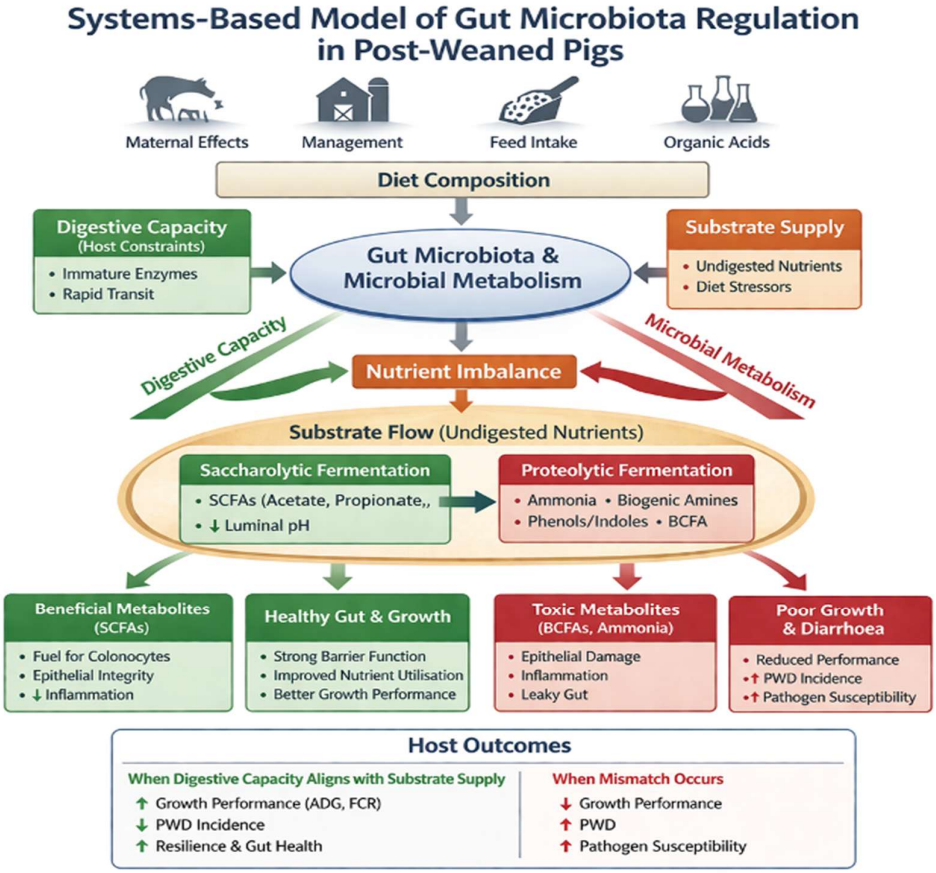

Figure 1. Conceptual model of gut microbiota regulation in post-weaned pigs. Gastrointestinal health is determined by the alignment between digestive capacity, dietary substrate supply and microbial metabolism. During the post-weaning transition, reduced feed intake and immature digestive function increase the flow of undigested nutrients to the hindgut, shifting microbial activity from saccharolytic to proteolytic fermentation.

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This results in the production of metabolites associated with impaired epithelial function, increased luminal pH and proliferation of opportunistic bacteria, contributing to dysbiosis and intestinal dysfunction. Nutritional, microbial and management strategies act by restoring alignment within this system, promoting saccharolytic fermentation, short-chain fatty acid production and microbial stability, thereby supporting gut health and growth performance.

2. Development of the Gastrointestinal Tract and Resident Microbiota

Development of the porcine gastrointestinal tract begins during gestation and continues through the suckling and post-weaning periods. Structural growth, enzyme expression and microbial colonisation occur together, and disruption of any of these processes early in life can affect later digestive function and microbial stability [7].

At birth, the gastrointestinal tract is structurally immature but highly responsive to feeding. Colostrum intake stimulates rapid intestinal growth, villous expansion and absorptive capacity, while providing immunoglobulins and bioactive compounds required for immune development [8]. During the suckling period, digestive function is adapted to a milk-based diet, with high lactase activity and limited capacity to digest starch and plant protein. Gastric acid secretion is also low due to incomplete development of parietal cell function [2,9]. This supports milk digestion but is not suited to the immediate digestion of cereal- and plant-based diets.

Microbial colonisation begins immediately after birth. Early populations are derived mainly from the sow and the environment and include facultative anaerobes that reduce luminal oxygen and allow establishment of obligate anaerobic communities [10,11]. During the suckling period, the microbiota is relatively stable and enriched in organisms adapted to milk-derived substrates, including *Lactobacillus* and *Bacteroides* species [11]. These microbes contribute to nutrient metabolism and support epithelial and immune development.

Weaning disrupts this process. The transition to plant-based diets introduces a different substrate profile and favours expansion of carbohydrate-degrading taxa such as *Prevotella*, while milk-adapted populations decline [12,13]. This shift often occurs before digestion has adapted to the new diet. As a result, the early post-weaning period is characterised by incomplete digestion, unstable fermentation patterns and reduced microbial diversity, particularly during the first one to two weeks [14].

The significance of this developmental sequence lies in the close link between digestion and microbial ecology. The digestive system and microbiota develop together, and disruption of this coordination increases the likelihood that the transition at weaning will result in excess nutrients reaching the hindgut and instability in microbial metabolism.

3. Maternal Nutrition and Early-Life Programming of the Gut Microbiota

The conditions under which piglets enter the post-weaning period are shaped in part before weaning occurs. Maternal nutrition, microbial status and the farrowing environment influence the initial establishment of the microbiota and the development of epithelial and immune function. These effects extend beyond simple transfer of bacterial taxa and include changes in substrate exposure and gastrointestinal maturation.

The sow is the primary source of microbial exposure during early life. Microorganisms are transferred through the birth canal, skin contact, colostrum, milk and ingestion of maternal faeces, establishing the first microbial communities in the piglet [10]. These early colonisers influence later succession patterns and contribute to the functional capacity of the microbiota, including its ability to utilise milk-derived and later plant-derived substrates.

Maternal diet can modify this process in several ways. It can alter the sow microbiota, change the microbial populations transferred to piglets and modify the nutritional and physicochemical characteristics of the maternal GIT and milk [15,16]. Fermented or organic acid-treated maternal diets, for example, have been reported to reduce pathogenic bacterial load and alter microbial composition,

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with downstream effects on piglet colonisation [15,17]. These effects are not limited to microbial composition; they also influence fermentation patterns and metabolite production.

Colostrum and milk contribute directly to gastrointestinal development. They supply immunoglobulins, growth factors and bioactive compounds that support mucosal maturation and immune competence [18]. Milk oligosaccharides also act as selective substrates for beneficial bacteria, promoting saccharolytic fermentation and helping establish a microbial ecosystem suited to low-pH, carbohydrate-rich conditions [19].

This early programming has clear functional consequences. Early microbial exposure contributes to maturation of the mucosal immune system and helps establish tolerance to commensals while maintaining the capacity to respond to pathogens [20,21]. Where this process is disrupted, pigs may enter weaning with a microbiota that is less stable and less capable of maintaining favourable fermentation patterns under nutritional stress.

Evidence from challenge studies supports this view. Maternal supplementation with β-glucans (laminarin) has been shown to reduce Salmonella Typhimurium shedding and improve intestinal health in piglets [22]. Other maternal dietary interventions have improved piglet growth and gastrointestinal function under ETEC challenge [23]. These findings indicate that maternal effects persist beyond lactation and influence how piglets respond to the abrupt substrate shift that occurs at weaning.

The relevance to post-weaning gut health is clear. Piglets that enter weaning with a more stable microbiota, better-developed epithelial function and greater digestive resilience are more likely to maintain saccharolytic fermentation and less likely to experience excessive protein overflow to the hindgut. Maternal nutrition therefore acts upstream of post-weaning gut stability by shaping the digestive and microbial starting point from which the piglet must adapt.

4. Weaning-Associated Gastrointestinal Dysfunction

Weaning disrupts the coordinated relationship between feed intake, digestive function and microbial activity. The abrupt withdrawal of milk and reduced consumption of solid feed in the first days after weaning are central to this process. Reduced intake leads rapidly to villous atrophy, lower digestive enzyme activity and reduced nutrient absorption, thereby reinforcing the decline in digestive capacity [3,5].

These structural and functional changes are accompanied by increased intestinal permeability and inflammatory activation, partly mediated by stress-related changes in immune function [6]. The consequence is a gut that is less able to digest and absorb nutrients and more exposed to the effects of microbial metabolites.

At the same time, undigested nutrients reach the distal intestine in greater quantities. Protein that escapes digestion is particularly important because it provides substrate for proteolytic fermentation. This alters the luminal environment in ways that impair epithelial integrity and favour opportunistic bacteria. Under these conditions, PWD can be understood not simply as a result of pathogen exposure, but as the outcome of a short-lived but biologically important mismatch between digestive capacity, substrate availability and microbial metabolism.

This interpretation matters because it shifts the emphasis away from treating post-weaning instability solely as an infectious problem. If the luminal environment is altered in a way that favours proteolysis and pathogen expansion, then nutritional and physiological strategies aimed at improving digestion and regulating substrate flow become central to prevention.

5. Digestive Capacity, Substrate Flow and Microbial Metabolism

The defining feature of the post-weaning period is not simply reduced digestibility, but altered substrate distribution along the GIT. Diets may be formulated to meet the nutrient requirements of the pig, yet if the digestive system cannot process those nutrients effectively, a larger fraction will

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escape proximal digestion in the duodenum/small intestine and become available for hindgut fermentation [3,7,24].

This shift in substrate flow arises from the combined effects of reduced feed intake, elevated gastric pH, limited pancreatic enzyme secretion and incomplete brush-border maturation [9,25–27]. Under efficient digestive conditions, most starch and protein are hydrolysed and absorbed in the small intestine. When digestive capacity is reduced, these nutrients are redistributed distally.

Microbial function in the hindgut is regulated primarily by the type and quantity of substrate that arrives there. This principle is well established across gut ecosystems: microbial activity reflects substrate availability more strongly than taxonomic identity alone [28–33]. In the post-weaning pig, this means that fermentation shifts according to the balance between carbohydrate-derived and protein-derived substrates.

When fermentable carbohydrates predominate, microbial metabolism is directed towards saccharolysis. SCFA are produced, luminal pH declines and epithelial cells, particularly colonocytes, receive an important energy supply [33–35]. These conditions favour obligate anaerobes and support colonisation resistance.

When protein-derived substrates predominate, metabolism shifts towards proteolysis. This produces ammonia, biogenic amines, phenols and branched-chain fatty acids, all of which are associated with epithelial irritation, increased luminal pH and reduced barrier function [36–38]. These metabolites also create conditions that favour facultative anaerobes and increase susceptibility to dysbiosis.

This balance is dynamic. Even a short reduction in feed intake can reduce digestive secretions and alter intestinal motility, increasing nutrient escape to the hindgut [39,40]. The observed post-weaning increase in Enterobacteriaceae and reduction in microbial diversity can therefore be interpreted as a consequence of altered substrate flow and luminal conditions rather than a primary initiating event [4,14,41].

The practical implication is straightforward. Gut microbial stability cannot be considered independently of digestion. Strategies that reduce nutrient overflow, particularly protein overflow, and support carbohydrate fermentation are likely to be more effective than those aimed at altering microbial composition alone.

6. Dietary Protein and the Proteolytic Fermentation Axis

Dietary crude protein is a major determinant of hindgut nitrogen supply and therefore of microbial metabolic direction after weaning. In the newly weaned pig, reduced gastric acidification and immature proteolytic capacity limit protein digestion in the upper GIT, increasing the proportion of dietary protein that reaches the large intestine [42,43].

Once in the hindgut, undigested protein supports proteolytic fermentation. The resulting metabolites, including ammonia, biogenic amines, phenols, indoles and BCFA, impair epithelial integrity, increase luminal pH and contribute to an environment favourable to facultative anaerobes such as Enterobacteriaceae [36–38,44,45].

Dietary crude protein level therefore has a direct effect on microbial metabolism. High-protein diets increase hindgut nitrogen supply and favour proteolysis. In contrast, moderate reductions in dietary crude protein, when balanced with essential amino acids, reduce substrate availability for proteolytic bacteria and shift fermentation towards more favourable patterns [46–49]. These shifts are reflected in both metabolite profiles and microbial composition. Reduced crude protein diets are associated with lower faecal ammonia and BCFA, increased abundance of Lactobacillus and butyrate-producing taxa, and reduced abundance of potentially pathogenic groups such as coliforms [50–52].

Host responses follow the same pattern. Lower proteolytic fermentation is associated with reduced expression of pro-inflammatory cytokines, improved tight junction integrity and better barrier function [25,53]. However, protein cannot simply be minimised. Excessive reduction in

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dietary crude protein can compromise growth performance and intestinal development by limiting nitrogen supply for protein deposition and synthesis of non-essential amino acids [54,55].

The practical objective is therefore not low protein per se, but protein supply aligned with digestive capacity. This requires aligning ingredient digestibility and amino acid balance with the developmental stage and digestive capacity of the pig.

7. Carbohydrate Fermentation, SCFA Production and Microbial Stability

Fermentable carbohydrates play a central role in regulating saccharolytic fermentation in the hindgut and, in turn, microbial metabolic activity during the post-weaning period. Their importance lies not simply in providing substrate for fermentation, but in influencing the balance between saccharolytic and proteolytic pathways through their effect on substrate availability. When supplied in appropriate forms and amounts, fermentable carbohydrates promote saccharolysis, increasing SCFA production, lowering luminal pH and supporting a stable anaerobic microbiota [33,34,56].

SCFA are the main functional outputs of this process and link microbial metabolism to host physiology. Butyrate, in particular, serves as a key energy source for colonocytes and supports epithelial integrity by promoting cell proliferation, differentiation and repair. SCFA also influence immune function by reducing inflammatory signalling, including suppression of NF-κB activation and pro-inflammatory cytokine production, while supporting regulatory immune responses [57–60]. Together, these effects support a luminal environment that favours microbial stability and limits the expansion of opportunistic bacteria.

The effects of fermentable carbohydrates depend on their physicochemical characteristics, which influence both the rate and site of fermentation. Rapidly fermentable substrates such as lactose are utilised earlier in the gastrointestinal tract, while more resistant fractions, including resistant starch and certain oligosaccharides, reach the hindgut and support sustained fermentation. These differences shape SCFA production and the distribution of microbial activity along the gut. Inclusion of such substrates in post-weaning diets is associated with increased abundance of Lactobacillus, Bifidobacterium and butyrate-producing genera such as *Faecalibacterium* and *Roseburia* [61–66]. However, these changes in microbial composition are more likely a result of altered fermentation conditions than direct selective effects.

The response to fermentable carbohydrates also depends on their alignment with digestive capacity and overall nutrient flow. In the immediate post-weaning period, when digestion is limited, excessive inclusion may reduce dietary energy density, impair nutrient digestibility or alter digesta characteristics such as viscosity and transit [67,68]. Under these conditions, increased substrate supply does not necessarily improve fermentation and may contribute to further imbalance.

From this perspective, fermentable carbohydrates influence microbial metabolism by altering the availability of fermentable carbon. They therefore regulate metabolic pathways rather than directly determining microbial composition. This favours saccharolytic pathways and reduces reliance on proteolytic fermentation, limiting the production of ammonia, phenols and BCFA associated with epithelial dysfunction. Their effectiveness therefore depends on interactions with dietary protein level, gastric function and feed intake, all of which determine the substrates reaching the hindgut.

β-glucans provide a clear example. As fermentable polysaccharides, they contribute to SCFA production and can influence microbial fermentation patterns, while also affecting immune responses [69,70]. Their effects are context-dependent and reflect interactions with diet composition and digestive capacity rather than inherent properties alone.

Overall, fermentable carbohydrates should be considered as regulators of microbial metabolism through their influence on substrate availability. Their role in post-weaning nutrition depends on how well they are matched to digestive capacity and integrated with the wider diet to support stable fermentation and gut function.

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8. Gastric Function, Organic Acids and the Control of Microbial Exposure

The stomach is a major control point for both nutrient digestion and microbial exposure. Its role in post-weaning gut health is often underestimated. Effective gastric acidification is required for protein denaturation and pepsin activation, as well as limiting the survival of ingested microorganisms [2,43,71].

In the newly weaned pig under commercial conditions, gastric acid secretion remains developmentally limited, resulting in elevated gastric pH [9,72]. This reduces protein digestion and allows greater numbers of viable microorganisms, including opportunistic pathogens, to pass distally [73]. Both effects increase the risk of hindgut instability as more protein reaches the hindgut and microbial challenge is increased.

Organic acids have been used extensively to address these limitations. By reducing dietary acid-binding capacity and supporting gastric acidification, they can improve gastric conditions for digestion and reduce microbial survival in the upper GIT [71,74–76]. Their antimicrobial action depends on their undissociated form, which can cross bacterial cell membranes and disrupt intracellular pH.

Organic acids may also improve protein and mineral digestibility, possibly through effects on enzyme activity and gastric emptying, although responses depend on the broader dietary context [77–79]. Their effects extend beyond antimicrobial activity to include improved nutrient utilisation and altered substrate flow to the hindgut. High dietary buffering capacity, particularly from calcium carbonate and some protein-rich ingredients, can limit their efficacy by counteracting acidification [80]. This is why organic acids are most effective when used in diets designed to minimise buffering capacity [81].

The preservation of grain with organic acids has added another layer to this discussion. Beyond reducing fungal growth and mycotoxin risk, preserved grain has an altered nutrient availability profile and microbial ecology. Improvements in digestible energy, growth performance and abundance of butyrate-producing taxa such as *Faecalibacterium* have been reported in some studies [82,83]. These findings highlight the importance of feed hygiene and pre-ingestion processing as upstream influences on gut stability.

Overall, gastric function should be viewed as a regulator of both nutrient flow and microbial exposure. Supporting gastric acidification can therefore have disproportionately large effects on post-weaning gut health.

9. Probiotics, Prebiotics and Synbiotics in the Context of Substrate Flow

Probiotics, prebiotics and synbiotics are frequently used to support gut health after weaning, but their effects vary widely across studies [84–87]. That inconsistency is not surprising when these interventions are considered in the context of substrate flow.

Probiotic organisms have numerous potential modes of action including pathogen exclusion, production of antimicrobial compounds and influencing epithelial and immune function [88–90]. Prebiotics supply substrates that favour saccharolytic fermentation and SCFA production [61,65,66]. Synbiotics attempt to combine both approaches, matching microbial strains with complementary substrates [91–93].

However, none of these interventions operates independently of the luminal environment. Their efficacy is conditional on the nutritional and physiological environment into which they are introduced. If substantial amounts of undigested protein reach the hindgut, proteolytic fermentation will dominate regardless of probiotic inclusion. Conversely, when diets support saccharolytic fermentation and limit protein overflow, microbial interventions are more likely to produce consistent effects.

This is the most useful way to interpret the probiotic and prebiotic literature. Positive responses are more likely under conditions that already favour saccharolytic metabolism; inconsistent responses often reflect unresolved nutritional and digestive constraints. Integrated strategies that

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combine dietary protein optimisation, support of gastric function and provision of suitable fermentable substrates are therefore more biologically coherent than additive-only approaches. Recent work using combinations of low crude protein diets, organic acid-preserved grain, resistant starch and butyrate-based strategies supports this interpretation, but these studies are best viewed as examples of integrated nutritional control of substrate flow rather than as evidence that any one additive class acts independently [94–99].

10. Management, Environment and Feeding Practices

It is important to note that management conditions influence feed intake, digestive activity, microbial exposure and stress physiology, and therefore alter the same substrate-flow relationships described above.

Voluntary feed intake remains the dominant factor. Reduced intake after weaning lowers gastric and pancreatic secretion, reduces nutrient digestion and increases the amount of undigested substrate entering the hindgut [3,5,39,40]. Measures that encourage early and consistent intake therefore have direct consequences for gut stability. Pre-weaning exposure to creep feed improves familiarity with solid feed and can reduce the decline in post-weaning intake [2,100,101]. Feeder design and feeding frequency also influence meal patterns and digestive consistency. Water intake is closely linked to feed intake and supports nutrient absorption, digesta flow and electrolyte balance. Poor water availability or poor-quality water can exacerbate gastrointestinal dysfunction and increase microbial challenge [102].

Thermal conditions affect both intake and physiological stress. Newly weaned pigs are sensitive to temperatures outside the thermoneutral range, and thermal stress reduces feed intake while activating pathways associated with increased intestinal permeability and altered immune responses [6,103,104]. Maintaining appropriate environmental temperature is therefore central to preserving digestive stability.

Environmental hygiene affects pathogen pressure, but the relationship is not simply linear. Heavy contamination increases the likelihood that transient dysbiosis progresses to clinical disease [4,105]. At the same time, piglets require exposure to a commensal microbial environment for immune maturation. The aim is not sterility, but control of pathogen challenge.

Social stress and feeding competition have similar effects. High stocking density, frequent mixing and inadequate feeder access reduce intake and increase variability in nutrient delivery, further destabilising microbial activity [106]. Feed structure also matters. Particle size and pellet characteristics influence gastric retention and digestion; coarser diets can improve gastric acidification and reduce nutrient escape, whereas finely ground diets may increase the risk of rapid passage and hindgut overflow [24].

Management should therefore be viewed as part of the same biological system as diet, as it alters digestion and microbial fermentation primarily through effects on intake, stress and substrate flow.

11. A Systems-Based Model of Gut Microbiota Regulation in Post-Weaned Pigs

Taken together, the evidence supports a model in which post-weaning gut health is determined by the interaction between digestive capacity, substrate flow and microbial metabolism. This model is summarised in Figure 1. When gastric function, enzyme activity and feed intake are sufficient to support efficient digestion, most dietary nutrients are absorbed before they reach the hindgut. Hindgut fermentation is then directed primarily towards carbohydrate substrates, supporting SCFA production, lower luminal pH and epithelial stability.

When digestive capacity is reduced, larger amounts of undigested protein and other nutrients enter the hindgut. Fermentation shifts towards proteolysis, luminal pH increases and microbial metabolites compromise epithelial integrity. Under these conditions, microbial composition changes in predictable ways, but those taxonomic shifts should be interpreted as consequences of the metabolic environment rather than primary causes of dysfunction [28,29,107].

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This interpretation has practical consequences. Dietary protein level, fermentable carbohydrate supply, gastric support, maternal influences and management practices all act through a common mechanism: they alter the amount and type of substrate reaching the hindgut. Microbial composition changes should therefore be interpreted as outcomes of this metabolic environment rather than primary drivers of dysfunction.

Limitations and Application to Commercial Systems

A limitation of the current evidence base is that much of the mechanistic work underpinning this interpretation derives from controlled experimental studies. Such studies are essential for identifying causal relationships, but they do not fully represent the variability of commercial systems, where health status, housing, intake patterns and environmental conditions differ markedly between herds. In addition, many studies characterise microbial composition more readily than microbial metabolic activity, making it easier to describe taxonomic shifts than to define functional consequences. For that reason, the framework proposed here should be regarded as a biologically grounded model that requires continued validation under commercial conditions.

Future work should place greater emphasis on functional outputs, including microbial metabolites, nutrient flow and host responses, rather than relying on taxonomic description alone. Progress in post-weaning gut health will depend on integrating nutritional, microbial and management strategies within this more explicit physiological framework.

12. Conclusion

Gut health in the post-weaned pig depends on the relationship between digestive capacity and substrate flow. When digestion in the upper GIT is sufficient, substrate availability in the hindgut remains limited and fermentation supports epithelial function and microbial stability. When digestion is inadequate, nutrient overflow, particularly protein overflow, shifts fermentation towards proteolytic pathways and increases susceptibility to intestinal dysfunction.

This interpretation provides a consistent explanation for the variability observed in microbial composition, disease expression and responses to nutritional additives. Effective strategies should therefore focus on improving digestion and controlling substrate delivery through diet formulation, support of gastric function, appropriate use of fermentable substrates and management practices that maintain feed intake and reduce stress. The microbiota is best understood not as an isolated therapeutic target, but as a metabolic system shaped by the flow of nutrients through the gastrointestinal tract.

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Abbreviations

The following abbreviations are used in this manuscript:

SCFA	Short chain fatty acids
PWD	Post-weaning diarrhoea
GIT	Gastrointestinal tract
BCFA	Branched chain fatty acids

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