Applying the 'Active Feeding' concept to control Edema Disease in weaned piglets

Alberto Morillo Alujas, DVM, PhD, Nutritionist, MSc Statistics

Index

Introduction

Edema disease (ED) in weaned piglets, caused by Shiga toxin-producing *Escherichia coli* (STEC) strains expressing F18 fimbriae and producing Shiga toxin 2e (Stx2e), remains a major cause of sudden mortality and economic loss. Typical outbreaks occur one to two weeks after weaning and often affect the best-growing piglets in the pen, compromising both welfare and profitability. At the same time, European and other country's regulations that restrict prophylactic antibiotic use and ban medicinal zinc oxide oblige producers to adopt new, sustainable tools to protect piglets during this vulnerable period [5].

In most pig systems, feed represents 60–75% of total production costs [1]. Nutrition is therefore not merely a cost centre, but the most powerful lever to improve health, performance and profitability when it is used strategically rather than passively. The 'Active Feeding' concept provides such a framework. It considers feed as a primary health tool designed to build resilience from within the animal and to work in synergy with vaccination, biosecurity, hygiene, housing and management. The objective is clear: maximise average daily gain (ADG), improve feed conversion ratio (FCR) and reduce mortality while minimising antibiotic use.

The 'Active Feeding' concept: from basic nutrition to proactive health

Active Feeding can be defined as a precision, science-based nutritional strategy that goes beyond simply meeting pigs' requirements for growth. It deliberately modulates gut function, metabolism, immune competence and stress responses so that pigs are intrinsically more resistant and resilient to disease challenges, especially around predictable stress points such as weaning.

Three elements are central to the concept:

• Precision: diets are tailored to physiological stage, genetic line and farm conditions. Nutrient supply is adjusted not only for growth but also for gut maturation, barrier function and immune competence. Functional amino acids (e.g. glutamine, arginine, cysteine, threonine, tryptophan among others), vitamins, trace minerals and selected functional feed raw materials and feed additives are included at levels and in combinations that target specific physiological mechanisms [2–4].

• Proactivity: nutrition is used ahead of challenges rather than as a reaction. Diets are reformulated for the post-weaning phase, seasonal disease peaks or regulatory changes, strengthening gut health and immunity before expected challenges and complementing vaccination programmes.

• Resilience: pigs are prepared to cope better with inevitable stressors such as diet changes, regrouping, co-infections and environmental fluctuations. A resilient pig maintains intake, growth and health with fewer treatments, directly improving FCR, ADG and survival and decreasing at the same time the medicine's expenditure while improving the welfare [2,3].

Importantly, Active Feeding recognises that nutrition cannot compensate for poor management. Overcrowding, poor hygiene, inadequate ventilation, unstable temperatures or poorly designed weaning strategies will limit the response to any diet. Feed must therefore be integrated with robust management, biosecurity and vaccination to unlock its full value.

Edema disease (ED) and the role of nutrition

ED is caused by F18-positive *E. coli* strains that colonise the small intestine and produce Stx2e. The toxin damages vascular endothelium, leading to oedema in eyelids, stomach wall, brain and other tissues, and often peracute death of apparently healthy piglets [5]. The window of highest risk coincides with a period of abrupt weaning, dietary change, mixing of litters and decline of maternal antibodies – exactly when gut structure, digestive capacity and microbiota are destabilised [3].

Diet composition plays a key role in ED risk. High crude-protein (CP) diets, particularly when based on poorly digestible protein sources, increase the amount of undigested protein reaching the large intestine, providing substrate for pathogenic *E. coli* proliferation and toxin production. Conversely, moderate CP supported by synthetic amino acids, and an appropriate inclusion of functional fibre and other raw materials, have been associated with lower ED incidence [3,6]. Feed presentation, particle size, acid-binding capacity, water quality and intake patterns also contribute to risk, and must therefore be addressed within an Active Feeding programme.

Key Active Feeding strategies against edema disease

Within the Active Feeding framework, ED control in weaned piglets combines several complementary objectives:

• minimise substrate for pathogenic *E. coli*;

• protect and repair the intestinal mucosa;

• modulate the microbiota;

• bind or neutralise toxin in the gut lumen;

• support immune responses; and

• integrate nutrition with vaccination, hygiene and management. filecite turn0file0

1. Diet design: protein, fibre and feeding strategy

Lowering dietary CP to the minimum compatible with performance, while balancing indispensable amino acids with crystalline sources, reduces fermentable protein available to coliforms in the hindgut and therefore lowers ED risk [3,6]. In practice this typically means reducing CP by 1–2 percentage units in the first post-weaning diet compared with

traditional formulations, while maintaining or slightly increasing digestible lysine and other key and functional amino acids. This approach not only reduces diarrhoea and ED, but also improves nitrogen efficiency and environmental sustainability [9].

Functional fibre is used in a targeted way. Insoluble structural fibre supports gut motility and gastric function, while selected soluble, fermentable fibres (e.g. inulin, chicory fructans) promote the production of short-chain fatty acids (SCFA) that nourish colonocytes, lower luminal pH and discourage pathogen proliferation [2,3]. The aim is to stabilise gut function without unnecessarily diluting dietary energy.

Feeding strategy is equally important. Gradual transitions between nursery diets, careful control of feed presentation and particle size, and avoiding overfeeding in the most critical days after weaning all reduce ED outbreaks. Restricting feed allowance for a few days in high-risk situations, followed by a controlled step-up, can be a useful tool when implemented under close supervision [5].

Within an Active Feeding framework, exogenous enzyme cocktails are a logical complement to other nutritional tools targeting Shiga toxin-producing E. coli (STEC). Multi-enzyme blends that combine phytase with carbohydrases (e.g. xylanase, cellulase) and protease hydrolyse cell-wall polysaccharides and phytate, improving the digestibility of phosphorus, amino acids and energy while generating fermentable oligosaccharides in the distal gut. These oligosaccharides act as substrates for beneficial bacteria, increase short-chain fatty acid production and help shift the microbiota towards a more favourable profile, supporting barrier function and dampening inflammation. In practical terms, this means fewer undigested nutrients reaching the large intestine to fuel pathogenic E. coli, a more competitive beneficial flora, and a gut mucosa that is better equipped—structurally and immunologically—to limit toxin absorption during an edema disease outbreak [11-12].

A synbiotic combining xylanase with Bacillus spp. improved average daily gain and gain:feed ratio, enhanced intestinal morphology and reduced the negative impact of the F18+ strain closely related to edema disease. In herds facing recurrent STEC outbreaks, positioning well-designed enzyme cocktails (alone or as part of enzyme–probiotic synbiotics) within an Active Feeding programm therefore offers a dual benefit: more efficient nutrient use and a demonstrable improvement in intestinal resilience against F18-associated *E. coli* challenges, complementing the use of other aproaches [11-12].

2. Protecting the intestinal barrier and modulating microbiota

The intestinal epithelium is the main physical and immunological barrier against pathogens and toxins. L-glutamine, used at around 1% of the diet in the first week after weaning, is a key energy substrate for enterocytes and supports tight-junction protein synthesis, villus repair and local immune function. Trials in weaned pigs show reduced diarrhoea duration and improved feed efficiency with glutamine supplementation [3,6].

Functional animal-derived ingredients such as spray-dried porcine plasma, hydrolyzed egg-white proteins or whey proteins concentrate rich in immunoglobins provide

immunoglobulins and bioactive peptides that neutralise pathogens, down-regulate inflammation and promote mucosal healing. In high-risk farms, including these ingredients in creep and first-age diets (after the risk period) can significantly reduce post-weaning diarrhoea and ED-associated mortality [3].

Prebiotics – such as mannan-oligosaccharides (MOS), fructo-oligosaccharides, inulin and chicory fructans – selectively stimulate beneficial bacteria and can competitively inhibit binding of F18 fimbriae to the intestinal mucosa [3,6].

Probiotics based on authorised strains of *Bacillus subtilis, Enterococcus faecium* or *Lactobacillus plantarum* further stabilise the microbiota, produce bacteriocins and increase secretory IgA, all of which help limit *E. coli* colonisation [3,6].

Organic acids (formic, lactic, propionic, butyric), used in combinations and often in protected forms, help maintain a low pH in the stomach and proximal intestine, directly inhibiting *E. coli* and supporting digestive function. Reduced acid-binding capacity of the diet (e.g. via careful selection of mineral sources) reinforces this effect [3,6].

3. Binding and neutralising Shiga toxin; supporting immunity

Adsorbent clay minerals such as bentonite, montmorillonite, kaolin and clinoptilolite can bind Stx2e and other bacterial toxins, forming inert complexes that are eliminated in the faeces. They also adsorb bacteria and contribute to firmer faeces and better villus morphology [5]. When these clays are used at 0.5–1.0% of the diet, ideally in combination with yeast cell wall fractions rich in mannan and β-glucans, they constitute a first line of defence at the luminal level.

Plant polyphenols and tannins (e.g. chestnut and quebracho extracts, grape polyphenols, cranberry proanthocyanidins) precipitate bacterial proteins, interfere with fimbrial adhesion and reduce secretory diarrhoea. When included at moderate levels, they complement clays and organic acids without penalising intake. [3,6] Oral antibodies (e.g. IgY from hyper-immunised hens or IgG-rich plasma) directed against F18 fimbriae and Stx2e toxin can bind the pathogen and toxin in the gut lumen, block adhesion and neutralise toxicity, especially in the first 10–14 days post-weaning [5,10].

Active Feeding also targets systemic immune competence. Functional amino acids such as arginine, cysteine and tryptophan, along with vitamins A, D, and E and highly bioavailable trace minerals (zinc, copper, selenium), are adjusted to support antioxidant defences, cytokine responses and antibody production [2–4,6]. Yeast β-glucans, nucleotides and selected phytogenics act as immunomodulators, priming innate immunity and enhancing the response to vaccination. Antimicrobial peptides (e.g. lactoferrin, colicins, cationic peptide hybrids) and enzymes with antibacterial activity (egg lysozyme, bacterial muramidase) can further reduce pathogen burden and inflammation while improving nutrient digestibility [6,10].

Integration with vaccination, management and hygiene

A key message of the Active Feeding concept is that diets and additives must be integrated with, not substituted for, sound vaccination and management. For ED, oral F4/F18 vaccines and parenteral Stx2e toxoid vaccines have demonstrated substantial reductions in diarrhoea and mortality when correctly applied [5]. Active Feeding improves the consistency and magnitude of this protection by stabilising the gut, ensuring adequate nutrient and micronutrient supply for the immune system and reducing secondary stressors.

Equally important are hygiene and management: strict all-in/all-out flows, thorough cleaning and disinfection, dry and warm pens at weaning, sufficient feeder and drinker space, correct water quality and flow, and avoiding overcrowding and large mixing of litters. These measures reduce infectious pressure and stress, allowing nutritional tools to express their full potential. Without them, the return on investment of even sophisticated diets will be disappointing.

Economic impact and practical objective

Because feed is the largest single cost in pig production, every percentage improvement in FCR, ADG or survival and every reduction in mortality and medicines usage achieved through Active Feeding has a disproportionate impact on profitability [1,7,9]. Combinations of functional amino acids, prebiotics, probiotics, organic acids and toxin-binding agents can reduce post-weaning diarrhoea and ED-associated mortality, increase ADG and improve FCR to levels comparable with, or better than, traditional antibiotic-based programmes [3,6–8]. From a practical standpoint, the aim is to maximise the benefit of the nutritional investment – higher growth and lower mortality – while minimising the use of antibiotics and meeting regulatory and market demands.

Conclusions

Edema disease remains a serious threat to the performance and welfare of weaned piglets, especially in the context of restricted antibiotic and zinc oxide usage. The Active Feeding concept offers a coherent, science-based framework to redesign nursery nutrition so that feed becomes a central health tool rather than a passive commodity. By adjusting protein and fibre, reinforcing the intestinal barrier, modulating microbiota, binding toxins and supporting immunity, Active Feeding can reduce ED incidence and severity while improving ADG, FCR and survival.

However, nutrition alone cannot control ED. The best results are obtained when Active Feeding is integrated with appropriate vaccination, rigorous hygiene, good weaning management and high standards of housing and biosecurity. When these elements are combined, producers can simultaneously minimise antibiotic use, safeguard piglet health and maximise the economic return on their feeding programmes – turning the largest cost item on the farm into a strategic investment in resilience and profitability.

References

1. Swine Nutrition Guide — General Nutrition Principles. Kansas State University, Animal Science and Industry. Last up-date: 2024

2. Fouhse JM, Zijlstra RT, Willing BP. The role of gut microbiota in the health and disease of pigs. Animals. 2023;13(8):1350.

3. Heo JM, Opapeju FO, Pluske JR, Kim JC, Hampson DJ, Nyachoti CM. Nutritional management for intestinal health in weaned pigs: a review. Anim Feed Sci Technol. 2013;173(1–2):3–16.

4. Atoo, Ashir F., Andres F. Bolivar-Sierra, Jorge Y. Perez-Palencia, et al. 2025. «Supplementation of Multienzyme with Acidifier Alleviates the Antinutritional Effects of a High Soybean Meal Diet for Nursery Pigs». Frontiers in Veterinary Science 12.

5. Edema disease in pigs. MSD Veterinary Manual. Last up-date: 2024. Acceded on November 22, 2025.

6. Rodrigues, Lucas A., Bonjin Koo, Martin Nyachoti, y Daniel A. Columbus. 2022. «Formulating Diets for Improved Health Status of Pigs: Current Knowledge and Perspectives». *Animals* 12 (20): 2877.

7. Managing Post-Weaning Diarrhea in Pigs with Natural Feed Additives. Amlan International. (https://amlan.com/blog/natural-mineral-based-neutralization-of-e-coli-toxins/). Acceded on November 22, 2025.

8. Rhouma M, Fairbrother JM, Beaudry F, Letellier A. Post weaning diarrhea in pigs: risk factors and non-colistin-based control strategies. Acta Vet Scand. 2017;59(1):31.

9. Pomar C, Remus A, Dubeau F, Andretta I. Feeding strategies to reduce nutrient losses and improve the sustainability of growing pigs. Front Vet Sci. 2021;8:742220.

10. Cutler, S. A., S. M. Lonergan, N. Cornick, A. K. Johnson, y C. H. Stahl. 2007. «Dietary Inclusion of Colicin E1 Is Effective in Preventing Postweaning Diarrhea Caused by F18-Positive Escherichia coli in Pigs». *Antimicrobial Agents and Chemotherapy* 51 (11): 3830-35.

11. Koo, Seong-Min, Jae-Hyeok Lee, Sang-Hyon Oh, y Jae-Cheol Jang. 2025. «Impacts of Bacillus-Based Biotics and an Enzyme Cocktail on Growth Performance, Immunity, and Gut Pathogenic Microorganisms of Nursery Pigs under Commercial Conditions». Frontiers in Veterinary Science 12.

12. Duarte, Marcos Elias, Yesid Garavito-Duarte, y Sung Woo Kim. 2023. «Impacts of F18+ Escherichia Coli on Intestinal Health of Nursery Pigs and Dietary Interventions». Animals 13 (17): 17.