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Sustainable Approaches in Pig Nutrition: A Comprehensive Review

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ABSTRACT

Feed expenditures constitute 60% to 75% of variable swine production costs, making precision nutrition essential for herd health, economic viability and environmental sustainability. This review comprehensively examines porcine digestive physiology, life-stage specific nutrient requirements, anti-nutritional factors (ANFs) and advanced feed technologies. Key findings highlight that formulating diets using the Net Energy system and Ideal Protein Concept—supplemented with synthetic amino acids—reduces environmental nitrogen excretion by over 35%. Integrating exogenous enzymes such as phytase releases bound phosphorus, decreasing manure phosphorus output by 30% to 50%. Furthermore, mitigating ANFs (e.g., trypsin inhibitors, phytate, gossypol) via thermal processing and additive supplementation restores nutrient digestibility. Automated precision feeding hardware further optimizes daily nutrient delivery, cutting feed costs by over 8%. Ultimately, integrating functional eubiotics, precision feeding and novel sustainable ingredients provides a robust framework for maximizing growth performance and welfare while minimizing the ecological footprint of modern monogastric swine production.

In modern commercial swine operations, nutrition is universally acknowledged as the single most vital factor driving biological productivity, animal welfare and microeconomic viability. Feed expenditures systematically represent approximately **60%** to **75%** of the absolute variable cost of pork production (National Research Council [NRC], 2012). Consequently, the formulation of biochemically precise, cost-effective and nutrient-dense feeding matrices is foundational to preserving operational profit margins and satisfying the steadily escalating global consumer demand for high-quality animal proteins.

Pigs are monogastric, opportunistic omnivorous animals possessing a simple, enzymatic digestive system functionally analogous to that of humans. Unlike ruminants (such as cattle or sheep), pigs lack a multi-compartmental forestomach or a continuous pre-gastric microbial fermentation vat. As a result, they are fundamentally incapable of efficiently utilizing large quantities of low-quality lignocellulosic structural fiber or coarse forages. Instead, commercial swine programs rely on highly digestible, concentrated dietary feedstuffs - predominantly consisting of highly processing cereal grains, superior oilseed meals, pure crystalline synthetic amino acids and precisely balanced macro-mineral, micro-mineral and vitamin premixes (Stein, 2012).

The absolute nutritional requirements of the pig are highly dynamic and fluctuate across the lifespan. Precision diet formulation must continuously adapt to account for specific parameters including body weight, breed cross or genetic line lines, sex (barrows, gilts or intact boars), environmental barn temperatures, current health status or systemic pathogen load and targeted market carcass criteria (NRC, 2012).

1. DIGESTIVE PHYSIOLOGY AND NUTRIENT UTILIZATION

A comprehensive understanding of the porcine gastrointestinal tract (GIT) is mandatory for evaluating ingredient biological value and tracking chemical nutrient availability. Digestion operates along a sequential pathway using mechanical breakdown, chemical adjustments and enzymatic cleavages.

The Cephalic and Oral Zone

The digestive cascade begins within the oral cavity. Mechanical mastication fractures feed particle size, expanding the surface area accessible for downstream chemical attack. This mechanical milling triggers the secretion of saliva from three primary sets of salivary glands. Beyond acting as an indispensable lubricant to prevent esophageal blockages, porcine saliva contains alpha-amylase (ptyalin). This initial enzyme breaks down complex dietary starches into simpler oligosaccharides, with thorough chewing directly correlating with enhanced downstream nutrient utilization.

The Gastric Architecture

Upon entering the stomach, the ingested feed bolus undergoes chemical acidification and primary proteolysis. Hydrochloric acid (HCl), secreted continuously by the parietal cells of the gastric mucosa, lowers internal pH, creating a highly acidic barrier that destroys a large percentage of incoming environmental pathogens. Furthermore, this low pH environment induces the autocatalytic conversion of inactive pepsinogen into active pepsin. Pepsin attacks native proteins, dismantling complex tertiary configurations into short-chain peptide fragments.

- **Post-Weaning Gastric Acidity Gaps in Neonates:** Immature, young piglets possess an underdeveloped gastric mucosa that produces insufficient levels of endogenous hydrochloric acid. When abruptly transitioned from naturally acidifying sow's milk to solid, plant-based starter diets, their gastric pH rises. This lack of appropriate acidity impairs protein digestion and permits opportunistic pathogens, such as enterotoxigenic *Escherichia coli* (ETEC), to survive the gastric barrier, proliferate in the small intestine and induce severe, often fatal post-weaning diarrheic syndromes (van Milgen & Dourmad, 2015).

The Small Intestine: Principal Enzymatic and Absorptive Hub

The small intestine represents the principal site for complete enzymatic nutrient hydrolysis and systematic absorption. Divided into the duodenum, jejunum and ileum, its internal surface area is expanded by millions of microscopic villi covered in microvilli (the brush border). As acidic digesta exits the stomach, it is neutralized in the duodenum by bicarbonate ions within pancreatic secretions and hepatic bile, shifting the environment to a neutral-to-slightly alkaline pH. This environment supports an array of specialized enzymes:

- **Pancreatic Amylase:** Fully cleaves starches into maltose and glucose.
- **Trypsin & Chymotrypsin:** Proteases that break down peptide bonds, reducing proteins to easily absorbable free amino acids and di/tripeptides.
- **Pancreatic Lipase:** In conjunction with the emulsifying action of bile salts, hydrolyzes triacylglycerols into free fatty acids and 2-monoglycerides.

The Large Intestine and Hind-gut Fermentation

The large intestine (cecum, colon and rectum) focuses primarily on water reclamation, electrolyte balance and limited microbial fermentation. While pigs cannot process structural cellulose efficiently, a complex symbiotic microflora resides within the hindgut. These microbes slowly ferment residual, undigested carbohydrates and non-starch polysaccharides into Volatile Fatty Acids (VFAs) - primarily acetate, propionate and butyrate (NRC, 2012). These VFAs are absorbed across the colonic epithelium, contributing a small portion to the animal's basal maintenance energy requirement.

2. COMPLETE CHARACTERIZATION OF ESSENTIAL NUTRIENTS

Water: The Primary Metabolic Medium

Water is quantitatively the most critical molecule an animal consumes. It serves as the primary medium for cellular transport, waste excretion, systemic thermoregulation via evaporative cooling, cellular lubricity and the intensive synthesis of milk. A pig's daily water consumption is tightly linked to dry matter intake, environmental temperature and physiological stage (Stein, 2012).

Production Class	Body Weight Target (Kg)	Daily Water Consumption (L/Day)	Primary Physiological Driver
Nursery Piglets	5 – 20 kg	1.0 – 3.0 L/day	Hydration post-weaning, dry feed adaptation
Grower Pigs	20 – 50 kg	5.0 – 10.0 L/day	High metabolic turnover, rapid lean tissue deposition
Finisher Pigs	50 – 110+kg	8.0 – 12.0 L/day	High total dry matter intake, maintenance support
Gestating Sows	150 – 230 kg	10.0 – 15.0 L/day	Fetal tissue development, basic maternal maintenance
Lactating Sows	160 – 250 kg	20.0 – 40.0 L/day	Peak milk synthesis, high volume fluid replacement

Energy Nutrition & Thermodynamic Accounting

Energy is derived from the metabolic oxidation of carbohydrates, lipids and excess amino acids. The primary energy vehicles are cereal grains, such as yellow maize, wheat, barley and grain sorghum. To elevate energetic density, supplemental lipids (soybean oil, tallow or palm oil) are integrated into formulations, holding approximately **2.25 times** the gross energetic density of pure carbohydrates.

In contemporary swine nutrition, dietary energy is calibrated across the Net Energy (NE) system rather than older Digestible (DE) or Metabolizable (ME) frameworks (de Lange, 2013). The NE system is highly accurate because it explicitly subtracts the "heat increment of feeding"—the energy lost entirely as heat dissipation during active digestion and cellular metabolism. Formulating on an NE basis prevents the overestimation of energy values in high-fiber or high-protein ingredients, yielding superior feed efficiency and tighter control over fat-to-lean carcass ratios.

Protein Nutrition & The Ideal Protein Architecture

Pigs require a precise, bioavailable profile of specific individual amino acids to support lean muscle tissue accretion, enzyme synthesis and structural tissue repair (van Milgen & Dourmad, 2015). Swine require ten indispensable, essential amino acids that cannot be synthesized endogenously at rates matching metabolic demand, with **Lysine** operating as the first-limiting amino acid in conventional cereal-based swine diets (Boisen, 2003).

Commercial formulation relies on the **Ideal Protein Concept**. In this model, the absolute requirements for all other essential amino acids are mathematically calculated and expressed as a fixed percentage relative to Lysine (van Milgen & Dourmad, 2015). This minimizes expensive feed waste and cuts down on environmental nitrogen pollution.

Minerals: Macro-minerals and the Neonatal Iron Crisis

Calcium (Ca) and Phosphorus (P) form the structural basis of skeletal bone and dental tissue. The dietary available Ca:P ratio must be strictly balanced; an uncalibrated surplus of calcium binds phosphorus in the gut, inducing rickets. Phosphorus requirements are closely evaluated using Standardized Total Tract Digestibility (STTD) values to ensure exact physiological absorption (Stein, 2012).

Among the trace minerals, Iron (Fe) occupies a unique management priority. Newborn piglets are born with low iron reserves and sow’s milk is naturally deficient in iron. In modern concrete or plastic-slatted indoor confinement facilities, piglets have zero access to iron-rich soil. Left un-supplemented, rapid neonatal growth completely exhausts internal iron stores within 72 hours, triggering severe **“Porcine Anemia” (Microcytic hypochromic anemia)**, characterized by a decline in circulating hemoglobin, extreme systemic lethargy, pale skin, rough hair coat set and high pre-weaning mortality. Administering an intramuscular injection of **200 mg of iron dextran** within the first 3 days of life is a mandatory commercial management practice (NRC, 2012).

3. LIFE-STAGE FEEDING STRATEGIES AND PHASE FEEDING

The Specialized Nursery Phase

Spanning from birth through roughly 20 kilograms of body weight, this phase marks a critical physiological transition. Diets during this nursery phase must feature high crude protein concentrations (**20% to 24%**) paired with low buffering capacity. Standard formulations incorporate highly refined ingredients: spray-dried porcine plasma protein to provide protective immunoglobulins, crystalline lactose to feed beneficial lactic-acid producing bacteria and premium low-temperature fish meal to avoid post-weaning growth depressions.

The Growing and Finishing Continuum

Spanning from 20 kg up to a final market weight of 110 to 130+ kg, this phase consumes the highest volume of total feed on the farm. The primary objective is maximizing lean muscle deposition while optimizing feed conversion efficiency. As the pig matures, its biological capacity for protein deposition plateaus, while fat deposition increases. To mirror this physiological shift, dietary crude protein is systematically stepped down from **18–20% in growers** to **15–17% in late finishers**, while the energetic profile is adjusted to avoid excessive carcass fatness.

The Breeding Herd: Gestation versus Lactation

The nutritional management of the modern highly prolific sow involves managing two completely opposing physiological states:

- **The Gestation Phase:** Sows are fed a strictly restricted, low-energy, high-fiber diet (typically 2.0 to 2.5 kg total feed per day). Overfeeding during gestation must be avoided; excessive fat accumulation induces "Fat Sow Syndrome," leading to prolonged farrowing durations, elevated stillborn rates and a collapse in feed intake during lactation.

- **The Lactation Phase:** Sows nursing large litters require an unrestricted, energy-dense, high-lysine diet. If a sow's daily feed intake is inadequate during peak lactation, she will automatically mobilize her own skeletal muscle and adipose tissue reserves to sustain milk output. This leads to excessive weight loss, structural lameness, prolonged wean-to-estrus intervals and high culling rates.

4. FORMULATION ARCHITECTURE, ANTI-NUTRITIONAL FACTORS AND ADDITIVES

Many plant ingredients contain natural anti-nutritional factors (ANFs) that impair digestion if left unmanaged. Raw soybeans contain "Trypsin Inhibitors" that bind to endogenous enzymes, neutralizing their proteolytic capacity; they are inactivated through precise thermal processing (roasting or extrusion). "Phytate" (Phytic Acid) represents the primary storage form of phosphorus in grains. Pigs lack sufficient endogenous phytase, meaning phytate-bound phosphorus is entirely unavailable to them. To bypass these barriers and eliminate subtherapeutic Antibiotic Growth Promoters (AGPs), modern swine nutrition relies on an array of specialized feed additives:

- **Exogenous Enzymes (Phytase):** The integration of supplemental Phytase cleaves the ester bonds of phytic acid, releasing bound phosphorus directly within the digestive lumen (Pomar *et al.*, 2009). This eliminates the need for expensive inorganic mineral supplements like dicalcium phosphate and reduces environmental phosphorus excretion in manure by up to 30-50%, preventing downstream aquatic eutrophication.

- **Eubiotics (Gut Health Modulators):** Probiotics (live beneficial cultures like *Bacillus subtilis*) and Prebiotics (non-digestible oligosaccharides such as MOS and FOS) promote competitive exclusion, preventing pathogenic bacteria from colonizing the intestinal walls.
- **Organic Acids:** Short-chain fatty acids, including formic, propionic, citric and lactic acids, lower the baseline buffering capacity of the feed and reduce internal gastric pH, supporting optimal pepsin activation and suppressing pathogens.

5. CLINICAL DIAGNOSES OF NUTRITIONAL DEFICIENCIES & ANTI-NUTRITIONAL FACTORS

When a diet fails to meet basic nutritional specifications, specific clinical pathology manifests rapidly across the herd. The table below serves as a diagnostic reference matrix for identifying major nutritional deficiencies.

Nutrient Focus	Primary Biological Function	Classic Clinical Signs & Pathology
Protein / Amino Acids	Muscle protein synthesis, enzymes	Severe growth depression, muscle wasting, poor feed conversion.
Iron (Fe)	Hemoglobin synthesis, oxygen transport	Microcytic hypochromic anemia, pale skin, dyspnea ("thumps"), high mortality.
Calcium / Phosphorus	Skeletal mineralization, structural integrity	Rickets in growing pigs (bent, painful bones), osteomalacia in mature sows.
Vitamin E / Selenium	Cellular membrane antioxidant defense	**Mulberry Heart Disease**, sudden death in nursery pigs, muscular dystrophy.
Zinc (Zn)	Epidermal integrity, enzyme cofactor	**Parakeratosis** (severe, crusty, deeply fissured skin lesions across legs and abdomen).
Iodine (I)	Thyroid hormone synthesis (T3/T4)	Goiter, piglets born completely hairless, weak neonates, high stillborn rates.

Comprehensive Review of Anti-Nutritional Factors (ANFs) in Swine Diets

Anti-Nutritional Factors (ANFs) are naturally occurring chemical compounds synthesized by plants as defensive mechanisms against herbivores, pests and environmental pathogens. When plant-derived feed ingredients are incorporated into swine rations without appropriate thermal, physical, or enzymatic processing, these ANFs interfere with nutrient digestion, reduce feed intake, cause tissue pathology and compromise immune status (Woyengo & Nyachoti, 2013). The following detailed analysis characterizes all major ANFs impacting pig production:

i. **Protease Inhibitors (Trypsin and Chymotrypsin Inhibitors):** Predominantly concentrated in raw legumes — most notably raw soybeans (Kunitz and Bowman-Birk inhibitors)—these heat-labile proteins form stable, inactive complexes with pancreatic proteases (trypsin and chymotrypsin) within the lumen of the small intestine (Liener, 1994). This inactivation directly suppresses dietary protein hydrolysis, resulting in severe amino acid malabsorption. In response to diminished luminal protease activity, the pig's pancreas hyper-secretes endogenous digestive enzymes via a negative feedback mechanism mediated by cholecystokinin (CCK), leading to pancreatic hypertrophy, visceral tissue hyperplasia and substantial endogenous amino acid loss (Woyengo & Nyachoti, 2013). Thermal processing (e.g., proper roasting, jet-cooking, or extrusion) denatures these tertiary protein structures, rendering soybean meal safe for monogastric consumption.

ii. **Phytate (Phytic Acid / Myo-inositol Hexakisphosphate):** Phytate constitutes the primary storage structure for phosphorus in cereal grains (corn, wheat, barley) and oilseed meals (soybean meal, canola meal). Because monogastrics produce negligible amounts of endogenous phytase, phytate-bound phosphorus remains biologically unabsorbable, passing directly into swine manure (Stein *et al*., 2016). Beyond restricting phosphorus utilization, the negatively charged phosphate groups on the phytate molecule act as potent chelating agents at physiological digestive pH, binding essential polyvalent cations (Ca^{2+}, Zn^{2+}, Fe^{2+}, Mg^{2+}) and forming insoluble mineral-phytate complexes. Furthermore, phytate binds basic amino acids (e.g., Lysine, Histidine) and digestive enzymes (pepsin, amylase), significantly reducing total dry matter and amino acid digestibility (Woyengo & Nyachoti, 2013). Supplemental exogenous microbial phytase is standard practice to cleave these ester bonds and mitigate phytate toxicity.

iii. **Non-Starch Polysaccharides (NSPs - Soluble and Insoluble):** NSPs are structural plant cell wall carbohydrates comprising arabinoxylans (predominant in wheat and rye), β-glucans (predominant in barley and oats) and pectins. Soluble NSPs dramatically elevate the physical viscosity of luminal digesta in the small intestine (Bedford & Schulze, 1998). Elevated intestinal viscosity creates a physical diffusion barrier that hinders pancreatic enzymes from accessing starch and protein substrates, while simultaneously slowing nutrient transit and enterocyte absorption pathways. Additionally, high digesta viscosity fosters an un-aerated intestinal environment conducive to opportunistic bacterial proliferation (such as *Clostridium perfringens* and pathogenic *E. coli*). Exogenous NSP-degrading enzymes (xylanases and β-glucanases) are integrated into cereal diets to hydrolyze these polymeric networks and eliminate digestive viscosity (Bedford & Schulze, 1998).

iv. **Condensed and Hydrolyzable Tannins:** Tannins are water-soluble polyphenolic compounds present in high concentrations in grain sorghum, faba beans and rapeseed meal. Tannins impart a strong, bitter, astringent taste by cross-linking with salivary proline-rich proteins, causing a precipitous decline in voluntary feed intake in growing pigs (Woyengo & Nyachoti, 2013). In the digestive tract, tannins bind directly to dietary proteins, structural digestive enzymes and intestinal mucosal membranes, forming indigestible protein-tannin complexes that markedly reduce apparent ileal amino acid digestibility and nitrogen retention. Low-tannin genetic cultivars or chemical inactivation strategies are employed to prevent tannin toxicity.

v. **Glucosinolates:** Glucosinolates are secondary sulfur-containing plant metabolites found abundantly in brassica species, such as rapeseed meal and mustard cake. While intact glucosinolates are relatively inert, their enzymatic hydrolysis by endogenous seed myrosinase (or intestinal microflora) generates bioactive toxic breakdown products, including isothiocyanates, nitriles, thiocyanates and oxazolidine-2-thiones (goitrin) (Tripathi & Mishra, 2007). These metabolites actively inhibit thyroidal iodine uptake and suppress thyroid hormone synthesis (T_3 and T_4), inducing thyroid gland hyperplasia (goiter), metabolic suppression, severe liver and kidney hypertrophy and reduced daily live weight gain. Modern double-zero ("00") canola meal cultivars have been genetically bred to contain minimal glucosinolate concentrations (< 30 μmol/g).

vi. **Lectins (Hemagglutinins):** Lectins are carbohydrate-binding glycoproteins resistant to gastric acid and proteolytic degradation, occurring prominently in raw field beans, peas and raw soybean meal (Liener, 1994). Upon ingestion, lectins bind specifically to the oligosaccharide chains of brush-border enterocytes lining the small intestine. This binding induces extensive structural disruption of intestinal villi, leads to villous atrophy, increases intestinal permeability ("leaky gut syndrome") and triggers local systemic inflammatory responses. These structural changes severely diminish nutrient absorptive capacity and predispose piglets to enteric colibacillosis. Heat treatment during seed processing effectively denatures lectin proteins.

vii. **Gossypol:** Gossypol is a polyphenolic pigment found within the pigment glands of cottonseed meal. It exists in both "bound" and "free" chemical states; free gossypol is the active toxic moiety in monogastrics (Woyengo & Nyachoti, 2013). Free gossypol binds to cellular proteins and divalent iron (Fe^{2+}), causing functional iron deficiency anemia, dyspnea, acute cardiac toxicity (myocardial necrosis), hepatic degeneration and severe reproductive impairment in boars and sows. Dietary supplementation with ferrous sulfate ($FeSO_4$) at a 1:1 iron-to-free-gossypol molar ratio binds free gossypol in the digestive tract, forming an insoluble, non-toxic iron-gossypol complex excreted harmlessly in feces.

viii. **Mycotoxins (Fungal Anti-Nutrients):** While secondary fungal metabolites rather than plant-synthesized compounds, mycotoxins (e.g., Deoxynivalenol [DON/Vomitoxin], Zearalenone [ZEA], Aflatoxins and Fumonisins) represent major feed contaminants that exert severe anti-nutritional effects on swine. DON binds to 60S ribosomal subunits, inducing severe feed refusal, vomiting and intestinal mucosal erosion. ZEA mimics 17β-estradiol, binding estrogen receptors to cause hyperestrogenism, rectal prolapse, embryonic mortality and severe reproductive failure in gilts (NRC, 2012). Dietary inclusion of inorganic aluminosilicate clays or organic yeast-cell wall binders is essential to adsorb mycotoxins within the gut.

Anti-Nutritional Factors (ANF)	**Primary Dietary Sources**	**Physiological Mechanism & Impact on Pig**	**Primary Mitigation Strategy**
Trypsin Inhibitors	Raw soybeans, legume seeds	Inactivates trypsin/chymotrypsin; induces pancreatic hypertrophy & endogenous amino acid loss.	Thermal processing (moist heat roasting, extrusion).
Phytate (Phytic Acid)	Maize, wheat, barley, soybean meal	Binds phosphorus, chelates Ca, Zn, Fe; decreases amino acid and energy digestibility.	Dietary inclusion of exogenous microbial Phytase.
Non-Starch Polysaccharides	Wheat, rye, barley, oats	Increases digesta viscosity; impairs enzyme access; triggers pathogen proliferation.	Exogenous enzyme addition (xylanase, β-glucanase).
Condensed Tannins	High-tannin sorghum, faba beans	Reduces feed intake (astringency); binds dietary protein and enzymes; lowers protein digestibility.	Selection of low-tannin genetic seed varieties.
Glucosinolates	Rapeseed meal, mustard cake	Toxic metabolites inhibit iodine uptake; causes goiter, liver damage and severe growth depression.	Use of double-zero ("00") Canola cultivars (<30 μmol/ g).
Lectins (Hemagglutinins)	Raw kidney beans, field peas, raw soy	Binds enterocyte villi; induces villous atrophy, leaky gut and intestinal inflammation.	Thermal denaturation via steam cooking or extrusion.
Free Gossypol	Cottonseed meal	Induces anemia, cardiac hypertrophy, pulmonary edema, liver damage and infertility.	Ferrous sulfate ($FeSO_4$) supplementation (1:1 Fe:gossypol ratio).
Mycotoxins (DON, ZEA)	Mold-contaminated maize, wheat	DON causes feed refusal and mucosal erosion; ZEA causes hyperestrogenism and abortion.	Inclusion of inorganic clays or organic yeast-wall binders.

6. MODERN FRONTIERS IN SWINE NUTRITION

The field of swine nutrition is moving beyond static feed matrices, driven by advances in molecular biology, automated engineering and global sustainability mandates.

Low-Protein, Amino Acid-Supplemented Diets: Environmental regulations regarding nitrogen excretion have catalyzed the development of ultra-low crude protein diets. By reducing reliance on raw soybean meal and utilizing synthetic, crystalline amino acids (L-lysine, DL-methionine, L-threonine, L-tryptophan and valine), nutritionists can reduce total crude protein levels by 2 to 4 percentage points. This strategy maintains growth performance while reducing environmental nitrogen excretion in waste by more than 35% (Pomar *et al*., 2021).

Precision Feeding Systems: Moving away from generic group-feeding strategies, modern swine facilities are integrating smart automated feeding stations (Pomar & Remus, 2019). Utilizing individual Radio-Frequency Identification (RFID) ear tags, these systems monitor each pig's weight and feed consumption patterns in real-time. Automated blending valves mix concentrated base feeds inside the barn, delivering a customized, nutrient-dense ration that matches the animal's exact daily genetic growth trajectory, reducing feeding costs by over 8% (Pomar & Remus, 2019).

Nutrigenomics and Circular Economy Feedstuffs: Nutrigenomics examines how dietary components interact directly with the porcine genome to alter gene expression, allowing nutritionists to select specific functional nutrients to upregulate genes responsible for intestinal tight-junction proteins, boosting natural gut barrier function. Concurrently, extensive research is validating alternative ingredients including highly digestible insect meals (such as black soldier fly larvae) and single-cell proteins derived from bacterial fermentation to safely decouple swine production from traditional resource constraints.

CONCLUSION

Swine nutrition is a dynamic, multi-faceted discipline that bridges monogastric digestive physiology, precise life-stage requirements, feed technology and sustainable management. Because feed accounts for up to 75% of total production costs, optimizing nutrient utilization is vital for both farm profitability and environmental conservation. Adopting Net Energy accounting, the Ideal Protein Concept, exogenous enzymes like phytase and targeted processing to neutralize anti-nutritional factors significantly improves feed conversion while reducing nitrogen and phosphorus waste. Furthermore, emerging frontiers — including automated precision feeding, nutrigenomics and alternative circular feedstuffs — offer promising avenues to enhance gut health, reduce reliance on antibiotics and lower the ecological footprint of swine farming. A holistic, science-driven approach to pig nutrition remains essential to achieving efficient, welfare-conscious and economically resilient pork production.

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