Effects of Different Dietary Energy Sources on Intestinal Mucosal Function in Weaned Piglets: Postprint

Authors: Li Xue, Tian Changxin, Wang Xiaocheng, Xiong Xia, Yin Yulong

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Abstract

Early weaning of piglets frequently leads to insufficient energy intake, placing intestinal mucosal cells in a "starvation state" that impairs normal intestinal function and consequently exacerbates weaning stress; therefore, meeting the energy requirements of intestinal mucosal cells is crucial. This article separately discusses the effects of three major energy-providing substances for intestinal mucosal cells—glutamine (Gln), glucose (Glu), and lipid—on the intestinal mucosal function of weaned piglets.

Full Text

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LI Xue[1],[2], TIAN Changxin[2], WANG Xiaocheng[2], XIONG Xia[2], *YIN Yulong[1],[2]*

[1]College of Animal Science and Technology, Hunan Agricultural University, Changsha 410128, China
[2]Key Laboratory of Subtropical Agricultural Ecological Engineering, Institute of Subtropical Agriculture, Chinese Academy of Sciences, Changsha 410125, China

Corresponding authors: XIONG Xia, associate professor, E-mail: xx@isa.ac.cn; YIN Yulong, academician, PhD supervisor, E-mail: yinyulong@isa.ac.cn

Abstract

Early weaning of piglets often leads to insufficient energy intake, placing intestinal mucosal cells in a "starvation state" that impairs normal intestinal function

and exacerbates weaning stress. Therefore, ensuring adequate energy supply to intestinal mucosal cells is crucial. This paper examines the effects of three primary energy substrates for intestinal mucosal cells—glutamine (Gln), glucose (Glu), and fat—on intestinal mucosa function in weaned piglets.

Keywords: weaned piglets; glutamine; glucose; fat; intestinal mucosa function

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In commercial production, early weaning (3–4 weeks of age) is commonly practiced to increase economic efficiency and prevent disease transmission. However, early-weaned piglets frequently develop "post-weaning syndrome," characterized by feed refusal, diarrhea, edema, digestive disorders, low feed utilization, and growth retardation [1]. Morphological analysis of piglet intestines 11 days post-weaning reveals reduced villus height, increased crypt depth, and significantly decreased activities of sucrase and lactase secreted by epithelial cells—all of which impair nutrient absorption [2].

Intestinal nutrition, particularly energy metabolism of small intestinal mucosal cells, plays a vital role in animal nutrition. It not only supports normal intestinal development but also serves as the energy source for all physiological activities. Research indicates that endogenous starvation of intestinal mucosal cells represents the fundamental cause of many intestinal diseases, and weaning stress often places these cells in an energy-deficient state. Thus, ensuring adequate nutrition for intestinal mucosal cells is the most basic guarantee for alleviating weaning stress and reducing various intestinal diseases during the weaning period [3]. Dietary energy sources for the intestine primarily include amino acids, glucose (Glu), and fats. Studies show that porcine portal-drained viscera (PDV) extensively utilize dietary glutamine, glutamate, and aspartate, with Gln being the predominant oxidative amino acid in the intestine [4]. Therefore, Gln, Glu, and fat serve as the main oxidative energy substrates for the intestine, capable of nourishing intestinal mucosal cells and maintaining intestinal mucosal function in weaned piglets.

1.1 Structure and Function of Small Intestinal Mucosa

The small intestinal mucosa serves as the primary site for substance exchange between the internal and external environment. It consists of three layers: the epithelium, lamina propria, and muscularis mucosae, composed mainly of small intestinal epithelial cells interspersed with goblet cells [5]. Small intestinal epithelial cells perform multiple physiological functions, including nutrient absorption, transport of various active molecules, secretion of digestion-related enzymes, and important immune defense functions [6].

1.2 Relationship Between Small Intestinal Mucosa and Energy

While performing various physiological functions, the small intestinal mucosa must maintain structural integrity and undergo continuous renewal, all of which

require energy expenditure. When energy supply to mucosal cells is insufficient, epithelial cells become damaged, leading to functional disorders and digestive diseases that compromise health [7]. Recent decades of research reveal that small intestinal epithelial cells are polarized cells whose polarity maintenance demands high energy consumption. When energy metabolism disorders result in ATP deficiency, intestinal mucosal function becomes abnormal [8], manifesting in several aspects:

1.2.1 Increased Intestinal Permeability and Inflammatory Response Cell junctions are critical structures ensuring normal intestinal barrier function [9]. Studies on small intestinal epithelial cells demonstrate that ATP deficiency is the primary cause of increased paracellular permeability and redistribution of tight junctions [10]. ATP insufficiency widens intercellular spaces and increases colonic barrier permeability, facilitating bacterial and antigen translocation into the submucosa and triggering inflammatory responses [11].

1.2.2 Impaired Epithelial Cell Functions Colonic epithelial cells perform numerous essential physiological functions, including electrolyte exchange, mucin glycoprotein synthesis, membrane lipid synthesis, structural protein synthesis, and detoxification—all energy-dependent processes [12]. Energy deficiency impairs these functions: short-term shortage causes epithelial atrophy, while prolonged deficiency disrupts colonic barrier function and may progress to inflammatory bowel disease [13]. Weaning stress easily places intestinal epithelial cells in a "starvation state," severely affecting normal physiological functions, reducing production performance, and predisposing piglets to intestinal diseases [14]. Therefore, ensuring adequate energy supply is critical for maintaining intestinal structure and function and safeguarding weaned piglet health.

2 Effects of Different Energy Sources on Intestinal Mucosa Function in Weaned Piglets

Carbohydrates, proteins, and fats constitute the primary energy sources for organism metabolism and life activities. Similarly, the main energy substrates for intestinal mucosa are these three categories, specifically Gln, Glu, and fat—all vital for maintaining intestinal mucosal structure and function.

2.1 Effects of Gln on Intestinal Mucosa Function in Weaned Piglets

Gln is the most abundant free amino acid in mammalian blood and tissues, providing nitrogen for synthesis of other amino acids, proteins, and nucleic acids while serving as an oxidative fuel like Glu [15]. Both Gln and Glu are important energy sources for intestinal mucosal cells, but Gln is better suited for intestinal metabolism and represents the primary energy substrate [16].

2.1.1 Prevention of Intestinal Cell Apoptosis and Maintenance of Mucosal Structure and Function The primary mechanism of apoptosis is oxidative damage, and weaning stress often disrupts the antioxidant defense system [17]. Glutathione (GSH) protects the intestine from bacterial and toxin damage, maintaining stable mucosal metabolism, structure, and function. The ratio of oxidized glutathione (GSSG) to GSH serves as an indicator of oxidative stress [18]. Weaning stress significantly reduces jejunal GSH content while increasing GSSG, elevating the GSSG/GSH ratio [19]. Dietary supplementation with 1.2% Gln increases intestinal GSH content and catalase activity, eliminating free radicals through Gln metabolism [20]. Additionally, Gln reduces production of the pro-inflammatory cytokine interleukin-8 (IL-8), mitigating mucosal cell damage [21].

2.1.2 Promotion of Intestinal Mucosal Cell Proliferation and Renewal The intestinal mucosal system maintains dynamic balance between cell proliferation and apoptosis, with complete renewal occurring every 3–8 days [22]. Gln deficiency accelerates apoptosis of small intestinal epithelial cells and impairs mitogen-induced proliferation [23] by selectively activating caspases, key enzymes in apoptosis, making Gln deficiency an important trigger for accelerated epithelial cell death [24]. Gln promotes proliferation through two mechanisms: providing essential energy for cell metabolism and generating necessary precursors for cell proliferation, such as polyamines and arginine [25].

2.1.3 Effects on Intestinal Mucosal Morphology Research demonstrates that dietary supplementation with 1% Gln in 21-day-old weaned piglets significantly increases jejunal villus height and lamina propria thickness, effectively alleviating weaning stress-induced mucosal atrophy [26]. Gln protects mucosal integrity through several mechanisms: First, Gln serves as the primary energy and nitrogen source for epithelial cell renewal [27] and is the most utilized amino acid by intestinal epithelial cells, especially during stress and disease [28]. Second, maintaining intestinal structure and function requires certain gastrointestinal hormones, which Gln indirectly stimulates [29]. Third, Gln acts as a substrate for synthesizing biomolecules like polyamines, glycine, and arginine that alleviate weaning stress symptoms, promote microvilli growth and digestive enzyme secretion, enhance nutrient and electrolyte absorption, and improve feed digestibility [30].

2.1.4 Effects on Intestinal Barrier Function The intestinal barrier system comprises the mucus layer and cellular barriers. The mucus layer consists of mucins secreted by goblet cells and epithelial cells, plus secretory immunoglobulin A (IgA) from plasma cells [31]. The cellular barrier comprises epithelial cells and intercellular tight junctions that prevent macromolecule entry. Factors affecting barrier integrity include epithelial apoptosis and necrosis, and disruption of tight and gap junctions [32]. Gln promotes expression of tight junction proteins and mucins while stimulating mucosal cell proliferation, thereby enhancing

barrier function and reducing bacterial translocation [33].

2.2 Effects of Glu on Intestinal Mucosa Function in Weaned Piglets Glu is the most widespread and important monosaccharide in nature, serving as the primary energy source and metabolic intermediate for animal organs, tissues, and cells. It supplements nutrition, improves tissue metabolism, enhances physiological activity, and can be used as a feed attractant to increase feed intake [34]. Research shows dietary Glu protects hepatocytes from damage and enhances reticuloendothelial and liver cell activity [35]. Gluconic acid, produced by glucose oxidase action, functions similarly to acidifiers by inhibiting harmful bacteria, promoting beneficial bacteria, and improving microbial balance. Glucose oxidase also possesses antioxidant properties that reduce oxidative damage to intestinal mucosa [36].

2.2.1 Elimination of Pathogen Environment and Maintenance of Microbial Balance Under glucose oxidase catalysis, Glu produces gluconic acid and hydrogen peroxide while consuming oxygen, creating an anaerobic environment for beneficial bacteria and inhibiting pathogens like *E. coli* and *Salmonella*, thereby reducing intestinal infection risk [37]. Gluconic acid creates conditions for lactobacilli growth, enhances digestive enzyme activity, strengthens intestinal motility, and promotes nutrient absorption. Hydrogen peroxide provides strong bactericidal effects, maintaining beneficial microbial balance [38].

2.2.2 Promotion of Intestinal Development and Feed Digestibility Fermentation of gluconic acid produces volatile short-chain fatty acids (SCFAs) like acetic, propionic, and butyric acids—the primary energy substrates for intestinal cells. SCFAs significantly increase villus height, mucosal thickness, and villus area, enhancing nutrient absorption and feed digestibility [39].

2.2.3 Protection of Epithelial Integrity and Defense Against Pathogens Weaning stress triggers oxidative reactions producing abundant free radicals. When these cannot be cleared promptly, the mucosal barrier is compromised, increasing pathogen invasion risk [40]. Glucose oxidase eliminates free radicals, preserving barrier integrity and blocking pathogen entry [41].

2.2.4 Detoxification of Mycotoxins Moldy feed containing mycotoxins reduces piglet performance, immune function, and may cause disease. Glucose oxidase inhibits various molds in the intestine, particularly preventing aflatoxin toxicity [42]. Additionally, some glucose oxidase reaches the liver, promoting redox reactions and enhancing metabolism of toxic components [43].

2.3 Effects of Fat on Intestinal Mucosa Function in Weaned Piglets Weaning stress reduces feed intake and digestibility, causing energy deficiency

that triggers fat mobilization. However, neonatal piglets have minimal fat reserves, and mobilization cannot meet energy needs while reduced body fat limits subsequent growth [44]. Fatty acid structure, type, chain length, and saturation degree all affect fat digestibility [45]. Limited research exists on how different fat sources affect weaned piglet performance, making investigation of structural fat effects on growth and glucose-lipid metabolism important for rational fat utilization in piglet diets.

Fat, composed of glycerol and fatty acids (triglycerides), is an important energy source with caloric efficiency multiple times that of carbohydrates. It provides physical protection and participates in metabolic regulation of nearly all cells; long-term deficiency harms health and causes disease [46]. Decomposition products like linoleic acid, linolenic acid, and arachidonic acid are essential fatty acids that constitute cellular structures and biologically active substances—linolenic acid, for instance, forms the basis of cell membranes and enzymes [47].

2.3.1 Nutritional Roles of Fat in Weaned Piglets 2.3.1.1 Energy Provision

Fat provides approximately 2.25 times the energy of carbohydrates with a heat increment of only 5–10%, significantly lower than protein (30%) and carbohydrates (10–15%) [48]. Therefore, summer dietary fat supplementation effectively reduces heat production from feeding and digestion, alleviating heat stress.

2.3.1.2 Essential Fatty Acids and Liposoluble Vitamin Absorption

Gastrointestinal fat digestion produces lecithin, medium-chain fatty acids, essential fatty acids, EPA, and DHA—functional fatty acids essential for weaned piglet growth that enhance immunity, improve intestinal health, and promote development [49]. Fat serves as a solvent for liposoluble nutrients; liposoluble vitamin absorption requires fat as a medium, and blood lipids facilitate vitamin transport [50]. Research shows dietary fat promotes liposoluble vitamin utilization, and low fat levels impair vitamin absorption [51].

2.3.1.3 Feed Intake Enhancement and Dietary Structure Modification

Dietary fat improves palatability and appearance, increasing early-weaned piglet feed intake [52]. It also elevates the energy-to-protein ratio, reducing protein antigenicity and putrefaction, thereby decreasing protein-induced diarrhea [53].

2.3.1.4 Anti-Stress Effects

With low water content and poor thermal conductivity, body fat reduces heat loss, maintaining body temperature and effectively relieving cold/heat stress in weaned piglets [52].

2.3.1.5 Additional Metabolic Effects

Due to immature digestive tracts, weaned piglets have low nutrient utilization efficiency. Dietary fat slows gastrointestinal transit, prolonging digestion and absorption time to improve nutrient utilization efficiency [54].

2.3.2 Effects of SCFAs on Intestinal Mucosa Function in Weaned Piglets Short-chain fatty acids (SCFAs), also called volatile fatty acids (VFAs), include formic, acetic, propionic, isobutyric, butyric, isovaleric, and valeric acids. SCFAs not only store energy and reduce osmotic pressure but also maintain colonic epithelial cell morphology and function [55].

2.3.2.1 Energy Provision for Intestinal Mucosal Cells
Butyrate and other SCFAs provide over 70% of energy required by colonic epithelial cells. Butyrate also promotes production of enzymes that repair mucosal damage, ensuring local mucosal health [56]. Research shows butyrate directly energizes intestinal epithelial cells, significantly improving mucosal morphology by reducing crypt depth and increasing the villus height-to-crypt depth ratio [57].

2.3.2.2 Intestinal Microflora Balance
Balanced intestinal microflora indicates intestinal health. Butyrate penetrates Gram-positive and Gram-negative bacterial membranes, dissociating into butyrate ions and hydrogen ions inside cells. When intracellular hydrogen ion concentration reaches a threshold, bacteria die. Thus, butyrate eliminates hydrogen-intolerant pathogens, maintaining microbial balance [58].

2.3.2.3 Inhibition of Intestinal Inflammatory Response
Butyrate sodium significantly improves inflammatory symptoms in rats with colitis by reducing xanthine oxidase activity and increasing GSH content in mucosa, thereby decreasing free radical production, inhibiting peroxidation, reducing tissue damage, and ameliorating inflammation [59].

3 Interactions Among the Three Dietary Energy Substrates

Recent research on gastrointestinal development in weaned piglets reveals that the effects of Gln, Glu, and fat are not independent.

Although intestinal Glu uptake from food is the sole source for the organism, most Glu is transported via blood for other bodily functions. Intestinal cells can utilize Gln, sparing Glu consumption—crucial for energy-deficient weaned piglets [60]. Furthermore, dietary Gln supplementation promotes post-transcriptional expression of sodium glucose cotransporter (SGLT) mRNA in the gastrointestinal tract, enhancing Glu absorption [60].

Fat and Glu digestion and absorption also interact in weaned piglet intestines. Increased Glu absorption promotes expression of carbohydrate response element binding protein (ChREBP) and sterol regulatory element binding protein (SREBP) in the liver, facilitating synthesis of body fat and essential amino acids from absorbed fatty acids [61]. Conversely, dietary fat modulates Glu absorption because fat decomposition and synthesis of essential fatty acids and body fat reduce the need for excess Glu to synthesize these substances via glucose-lipid metabolism, thereby relatively inhibiting excessive Glu absorption [62].

Weaning stress often causes insufficient energy supply to intestinal mucosal cells,

compromising structural and functional integrity. Gln, Glu, and fat serve as primary energy substrates, supplying energy and maintaining normal physiological functions through their unique mechanisms, thereby alleviating weaning stress-induced damage and ensuring nutrient digestion and absorption. Further research is needed to determine optimal combinations of these three energy substrates in piglet diets.

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