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Post-Weaning Diarrhea in Piglets: Causes, Risk Factors, and Management Strategies

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Abstract

Post-weaning diarrhea (PWD) is a major health concern in pig farming, often leading to significant economic losses. This review explores the underlying causes and risk factors associated with PWD in piglets and discusses comprehensive, antibiotic-free strategies for its prevention and control. It further highlights recent findings, prevalence data, diagnostic methods, epidemiological patterns, and management models. Emphasis is placed on environmental, nutritional, and biosecurity interventions aimed at improving piglet resilience during the weaning transition. This vulnerability predisposes piglets to gastrointestinal disorders, especially post-weaning diarrhea (PWD), which remains a leading cause of morbidity, mortality, and economic loss worldwide. Enterotoxigenic *Escherichia coli* (ETEC) infection induced Postweaning diarrhea is one of the leading causes of morbidity and mortality in newly weaned piglets. The main virulence attributes of ETEC are adhesins and enterotoxins, which are mostly regulated on large plasmids. Prevalence found is less than 50% in Australia, Danish, Korea, South Ontario but no prevalence in Africa. According to antibiotic resistance, the researchers experimented, discussed and concluded that the effect of diet transitions, reducing protein levels, digestible fiber support, using probiotics, association organic acid with monogycerid or fatt could contribute or reduce the effects of post weaning diarrhea in piglet. Future research should refine diagnostics, monitor pathogen strains,

and quantify long-term impacts of combined interventions via meta-analyses and longitudinal studies.

Keywords

Post-Weaning Diarrhea, Piglet, Non-Antibiotic Strategies, Epidemiology, Diagnostic Methods

1. Introduction

Weaning is a critical and stressful period for piglets, marked by sudden changes in diet, environment, and social grouping, which severely challenge gut health and immune function [1] [2]. This vulnerability predisposes piglets to gastrointestinal disorders, especially post-weaning diarrhea (PWD), which remains a leading cause of morbidity, mortality, and economic loss worldwide [3] [4]. Traditional reliance on antibiotics to control PWD has raised concerns due to antimicrobial resistance, driving research toward sustainable, non-antibiotic interventions. Understanding PWD's multifactorial etiology, epidemiology, and effective management strategies is essential for improving piglet welfare and productivity. [5] reviewed that a post weaning diarrhea (PWD) is one of the most serious threats for the swine industry worldwide. It is commonly associated with the proliferation of enterotoxigenic *Escherichia coli* in the piglet's intestine. *E. colistin*, a cationic antibiotic, is widely used in swine for the oral treatment of intestinal infections caused by *E. coli*, and particularly of PWD.

2. Causes and Pathophysiology of PWD

[6] reported the most recent findings on the piglet gut microbiome shifts as influenced by weaning, and how these microbiome changes brought about by various factors that have been shown to affect the development of microbiota in piglets. [7] provided the details of the pig gut microbial community. These authors reported that there exist unstability and variability of microbiota. It depends of ecology and animal diversity. [8] has revealed that environmental and host factors, particularly diet, drive significant variations in microbial composition, which in turn shape host epigenetics through microbial components and metabolites. [9] reviewed that Enterotoxigenic *Escherichia coli* (ETEC) infection induced post-weaning diarrhea is one of the leading causes of morbidity and mortality in newly weaned piglets. The main virulence attributes of ETEC are adhesins and enterotoxins, which are mostly regulated on large plasmids. Almost all ETEC bacteria are known to adhere to receptors on the small intestinal epithelium by their proteinaceous surface appendages (fimbriae, pili) or by afimbrial proteins without inducing significant morphological changes [10]. ETEC attachment to the small intestine initiates ETEC colonization and infection.

The secretion of enterotoxins further disrupts intestinal barrier function and

induces intestinal inflammation in weaned pigs. However, [11] reviewed that a weaning stress often causes changes in the morphology and function of the small intestine of piglets, disrupts digestion and absorption capacity, destroys intestinal barrier function, and ultimately leads to reduced feed intake, increased diarrhea rate, and growth retardation.

Enterotoxigenic Escherichia coli (ETEC), especially strains producing fimbriae that adhere to intestinal epithelium, is the primary pathogen causing enteric infections. The infection leads to intestinal inflammation, fluid and electrolyte loss, dehydration, reduced growth, and sometimes mortality. In 2015, the authors indicated that gut microbiome in young pigs is dramatically shaped by the composition of dietary glycans, reflected by the different functional capacities of the microbiome before and after weaning [12]. [13] suggested that rotavirus A and a protoan Balantidium coli may be an etiological agent of PWD, but he estimated the association to PWD for other rotavirus groups than A.

[14] reported that factors contribute to imbalances in fluid absorption and secretion in the intestines of weaned piglets include high levels of crude protein (CP), stimulation by certain antigenic proteins, high acid-binding capacity (ABC), and contamination with deoxynivalenol (DON) in the diet.

3. Epidemiology

[15] found in all 101 case farms (42.6%) first symptoms of PWD appeared between the first and 8th day post weaning (mean = 3.8 days post weaning) in Australia. [16] determined the prevalence of the intestinal bacteria like pathogenic *Escherichia coli* (serogroups O138, O139, O141 and O149) in Danish finishing pig herds. They founded pathogenic *E. coli* in 19 herds (24.1%).

PWD is widespread in intensive pig production systems, with prevalence rates up to 50% reported in certain regions [13] [16] in Denmark. [17] reviewed that in some large-scale pig farms, the diarrhea incidence of piglets is as high as 50%, and the mortality rate is 15% to 20% and observed that ETEC infection was one of the most common factors for PWD and mortality in piglets.

[18] determined the present distribution of serogroups, hemolytic activity and virulence factor gene profiles among porcine pathogenic *E. coli* isolates in Denmark and to compare detection of these characteristics as diagnostic approaches. [18] serogrouped Five hundred and sixty-three *E. coli*.

The most prevalent serogroup was O149 accounting for 49.9% of all isolates, followed by O138 (14.9%), O139 (6.9%), O141 (4.1%) and O8 (3.7%). [19] detected hemolytic activity (87.7%) of all isolates, and considered Virulence Factor Genes such as: *F*4 (44.7%), *F*18 (39.3%), intimin (1.4%), *F*6 (0.9%), *STb* (77.6%), *EAST*1 (65.8%), *LT* (61.6%), *STa* (26.5%) and *VT*2*e* (16.4%).

[19] determined the prevalence of virulence genes in *Escherichia coli* strains recently isolated from young pigs with diarrhea in the US and isolated *K88, F18, F41, 987P and K99 fimbrial genes; LT, STa, STb, Stx2e and EAST1 toxic genes; and AIDA-I, paa and EAE adhesin genes in E. coli strains*.

[19] reported that in 304 *E. coli* isolates from diarrheic pigs, 175 (57.6%) strains possessed fimbrial genes: *K88 (64.6%), F18 (34.3%), F41 (0.57%), K99 (0.57%), 987P (0); toxin genes: LT (57.7%), STb (72.6%), STa (27.4%), STx2e (17.4%), EAST1 (35%); and adhesin genes: AIDA-I (26.9%), paa (60%), EAE (1.1%).* All toxin genes except the *EAST1* toxin gene, were almost exclusively associated with *K88+ or F18+* isolates, and most of these isolates carried multiple toxin genes. The non-fimbrial adhesin paa was found present in over half of the *K88+* isolates. [20] determined the prevalence of serogroups and virulence factors of *Escherichia coli* strains isolated from pigs with postweaning diarrhea in eastern China and isolated two hundred and fifteen *E. coli*. [20] showed that 60.5% (130/215) of the isolates only harboured the gene of *estI* (STI) while 6.0% (13/215) strains possessed the genes of *stx2e*, *estI* and *estII* and 5.6% (12/215) of strains had the genes of *estI*/*estII*. 107 (49.8%) were negative for the fimbrial antigens. The fimbria-negative isolates usually possessed genetic determinant of *estI* (78 isolates, 72.9%). [21] isolated from 1996 and 2000, 476 *E. coli* from 452 weaned pigs with diarrhea and/or neurologic sign from across Korea to the Department of Veterinary Pathology, Seoul National University. One hundred and forty-nine (31.3%) of the 476 *E. coli* isolates carried the gene for *EAST1*. Of these 149 isolates, 66 (44.3%) carried the east1 gene only. [22] in Korea, between 1995 and 1998, isolated 812 *Escherichia coli* strains from diarrheic piglets. Among the 44 isolates known to carry genes for F4, 42 (96%) isolates contained genes for F4ac, and 2 (4%) isolates contained genes for F4ab. None of the *E. Coli Strains* carried genes for F4ad. F4ac was the predominant F4 variant associated with diarrhea in piglets in Korea.

[23] estimated the cumulative incidences of PWD the first 14 days after insertion to the nursery units were 41.8% (CI 33.6, 50.4) and 51.1% (CI 42.3, 60.0) at the two producers, respectively. A low incidence of cases associated to ETEC and detected a substantial proportion of cases associated to rotavirus while a biphasic pattern in the assumed etiology with rotavirus occurring first, and then a shift towards cases associated to ETEC/non-ETEC hemolytic *E. Coli*.

[24] in southern Ontario, the hemolytic *E. coli* from 82% of the case herds were positive for 3 enterotoxins (STa, STb, and LT), those from 12% of the case herds were positive for STb and LT only, and those from one herd (6%) were positive for 3 enterotoxins, as well as for verotoxin and F18 pili.

In a German, study of piglets with diarrhea, Isospora suis (26.9%) was most common in suckling piglets, while rotavirus and ETEC were predominant in weaned piglets; overall, ETEC accounted for 17.6% of cases, making it a major contributor alongside rotavirus and coronavirus [25].

These models incorporate environmental variables (hygiene, stocking density), host susceptibility, and climatic factors impacting transmission [26] [27]. [28] reported that classified litters according to the sow parity numbers (1, 2 - 5, and 6 - 9), average birth weight of the piglets (0.80 - 1.29, 1.30 - 1.79, 1.80 - 2.50 kg), number of littermate pigs (5 - 7, 8 - 10, 11 - 12, and 13 - 15 piglets), and size of the herd (small, medium, and large). He found that on average, piglet pre-weaning mortal-

ity was 11.2% (median = 9.1%) and varied among herds from 4.8% to 19.2%. Among all the litter, 62.1, 18.1, and 19.8% of the litter had a piglet pre-weaning mortality rate of 0 - 10, 11 - 20, and greater than 20%, respectively. As the number of littermate pigs increased, piglet pre-weaning mortality also increased ($r = 0.390$, $P < 0.001$). Litter with 13 - 16 littermate pigs had a higher piglet pre-weaning mortality than litter with 5 - 7, 8 - 10, and 11 - 12 littermate pigs (20.8%, 7.8%, 7.2%, and 11.2%, respectively; $P < 0.001$).

[5] reviewed that mortality associated with PWD may reach 20% - 30% over a 1- to 2-month time span among infected weaned pigs during acute outbreaks of PWD.

Although PWD has been extensively reported in Europe, Asia, and North America, there is a notable scarcity of published epidemiological data from Africa, which likely reflects limited surveillance studies rather than absence of the disease.

4. Diagnostic Methods

[13] reported that he assumed no errors in diarrhea diagnoses assigned by fecal dry matter content estimation. Evidence indicates some uncertainty when assigning a diarrhea diagnosis based on a clinician's evaluation of a fecal sample. he assumed a sensitivity of 74.6% (95% CI 70.2; 78.7) (beta distribution: $\alpha = 302$, $\beta = 103$) and a specificity of 83.3% (95% CI 78.8; 87.2) (beta distribution: $\alpha = 248$, $\beta = 50$) when using the presence of diarrheic soiling of the hind-part as a diagnostic criterion.

Diagnosis of PWD involves clinical signs such as watery diarrhea, dehydration, and growth retardation. [28] isolated *Escherichia coli* from fecal samples and identified ETEC out of the *E. coli* colonies, referring to specific primers targeting LT and ST sequences. In this study, a total of 19 ETEC isolates were confirmed out of 412 *E. coli* strains isolated from pig fecal samples, resulting in a recovery rate of 4.61%.

[29] investigated the presence of pathogenic *E. coli*, *Clostridium perfringens* types A and C toxins (Cpα, Cpβ and Cpβ2), *Clostridioides difficile* toxins (TcdA and TcdB), Rotavirus A, B and C, porcine epidemic diarrhoea virus (PEDV) and transmissible gastroenteritis virus (TGEV) by using molecular methods and bacteriology. Rotavirus type A was the only agent that could be statistically correlated with diarrhea.

Also [30] used PCR for examination of 283 enterotoxigenic strains and detected K88 in 237 strains, among them 232 strains possessed the K88 variant. He found the genotype K88ab in two strain and K88ad of the serogroup O8 respectively from one herd and other herd. He concluded that the K88ac was predominant in ETEC strains with colonisation factors K88 in pig herds in the Czech Republic.

Multiplex polymerase chain reaction (PCR) assays allow simultaneous detection of multiple virulence genes, such as fimbrial (F4, F18) and toxin genes (*STa, STb, LT*) of enterotoxigenic Escherichia coli (ETEC) [18] [31] [32]. Also [2] introduced a test that uses a dual-electrode electrochemical chip (DEE-Chip) and a

barcode-releasing electroactive aptamer for rapid on-farm detection of porcine epidemic diarrhea viruses (PEDv).

[13] added that the researchers have recommended a pen-side test to differentiate etiologies in cases of porcine and human diarrhea because the different pathophysiological processes are expected to cause certain alterations of the fecal pH. However, he found no empirical evidence supporting the practice. For that he used culture-based methods supplemented by matrix-assisted laser desorption-ionization time-of-flight (MALDI-TOF) and PCR genotyping to detect *E. coli* (ETEC and non-ETEC). A high-throughput real-time PCR platform, BioMark HD (Fluidigm, San Francisco, California), and a 192.24 dynamic array integrated fluidic circuit system (Fluidigm) were used to quantify *E. coli* fimbrial types F4 and F18.

Therefore, the O and H serotypes were obtained from the producer and also predicted from whole genome sequencing for six of the *E. coli* isolates from Herd C to verify that had not cultured the vaccine strains. DNA isolation, sequencing, and sequence analysis for this part were done as described elsewhere. This precluded the essential quantitative interpretation of ETEC diagnostics based on the culture results. In these instances, he made imputations of the abundance of hemolytic *E. coli* (low or high) by using the established cut-off of 17,500 F18 copies detected by real-time PCR. [18] used multiplex PCR modified or newly designed primers identified each individual fimbrial gene and four toxin genes.

A limitation worth noting is that diagnostic tools such as fecal consistency scoring, dry matter estimation, or visual assessment have variable sensitivity and specificity. For example, the cotton-swab method compared with fecal dry matter measurement shows approximately 85% sensitivity and 95% specificity in weaned pigs [33]. In addition, methods based on fecal pH or soiling scores may further reduce diagnostic accuracy. Such variability can lead to under- or overestimation of PWD prevalence and may influence the apparent effectiveness of interventions across studies.

5. Risk Factors

5.1. Nutritional

High-protein diets and abrupt feed changes favor undigested substrates, promoting pathogenic overgrowth [34] [35]. [13] reported that an increased risk of PWD was associated with the regimen of twice a day feeding and feed restriction after weaning ($P = 0.02$; compared to feeding three or more meals a day or the use of *ad libitum* feeding) and with a higher number of sows on the farm ($P = 0.02$; risk increasing with increasing number of sows).

5.2. Environmental

Temperature fluctuations, high stocking density, and poor ventilation increase stress and gut permeability. [13] [16] reported that an automatic temperature control was associated with decreased risk of PWD ($P = 0.03$; compared to manual temperature control).

5.3. Management

[36] reported that managing hygiene alone was insufficient for reducing antimicrobial resistances in piglet rearing. He concluded that the complex factors contributing to the presence and distribution of AMR in piglet barns underscore the necessity for a comprehensive management strategy.

5.4. Host

Age at weaning, genetic predisposition (e.g., receptor expression for ETEC fimbriae), and concurrent infections influence susceptibility [3].

6. Non-Antibiotic Management Strategies

To provide a clearer overview, the main non-antibiotic interventions against post-weaning diarrhea are summarized in Table 1, highlighting their proposed mechanisms of action and reported effects on disease reduction.

Table 1. Summary of non-antibiotic interventions for post-weaning diarrhea (PWD) in piglets, their proposed modes of action, and reported effects.

Intervention	Mode of Action	Reported Effect on PWD	References
Lowering crude protein (CP) & fiber inclusion	Reduces undigested protein fermentation; supports microbial balance	↓ Diarrhea incidence, improved gut health	[37] [38]
Organic acids (OA) & medium-chain fatty acids (MCFA)	Acidify gut, inhibit pathogens, modulate microbiota	↓ Diarrhea scores, ↓ fecal *E. coli*; mixed effects on ETEC	[39]-[41]
Probiotics, colostrum management	Enhance intestinal barrier, competitive exclusion of pathogens, immune modulation	↓ PWD prevalence (27% vs. 47% in farms without probiotics)	[15] [42] [43]
Butyrate (microbial or dietary)	Strengthens epithelial integrity, promotes SCFA-mediated gut health	Improved jejunal adaptation, ↓ diarrhea	[44] [45]
Colicin E1	Antimicrobial peptide against ETEC	↓ Incidence/severity of F18-ETEC diarrhea, ↑ growth performance	[46]
Plant extracts (e.g., horseradish)	Antimicrobial and anti-inflammatory activity	Farms supplementing horseradish: 75% no PWD problems	[15] [47]-[49]
Vitamins, selenium, bioactives (e.g., glucans, mushrooms)	Enhance immunity, antioxidant function	Improved resilience against weaning stress, ↓ diarrhea risk	[25] [50] [51]
Environmental & hygiene measures	Reduce stress and pathogen exposure	>20% reduction in PWD incidence with better hygiene and lower stocking density	[36] [38] [52]

7. Review Scope and Meta-Analysis

This review synthesizes findings from more than 50 studies published between 2010 and 2024 on the causes, diagnostics, and antibiotic-free management of

PWD. Recent meta-analyses confirm the efficacy of probiotics and nutritional interventions in reducing diarrhea incidence [16] [34] [53]. Genomic and functional studies have also expanded understanding of intervention mechanisms; for example, [54] elucidated the molecular pathways through which zinc oxide alleviates PWD, while [55] demonstrated via metagenomics how antibiotic and non-antibiotic treatments shape the gut microbiome and resistome in weaned piglets. In addition, recent genomic surveillance of enteric viruses in Spanish swine farms has highlighted the evolving diversity of viral agents contributing to diarrhea outbreaks [56]. Despite these advances, holistic models that integrate environmental management, nutritional strategies, and diagnostic standardization remain underdeveloped and represent an important frontier for future research.

8. Conclusion and Future Perspectives

Combatting PWD requires integrated management prioritizing gut health through nutrition, environment, and hygiene. With antibiotic restrictions growing, sustainable alternatives like medical plant extracts, phytochemical compounds such as alkaloids, flavanoids, cummarines, lignanes, probiotics, and improved biosecurity are essential. Future research should refine diagnostics, monitor pathogen strains, and quantify long-term impacts of combined interventions via meta-analyses and longitudinal studies.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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