

# Factors involved in post weaning diarrhoea and measures to mitigate it

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## Abstract

Post-weaning diarrhea (PWD) in pigs, primarily caused by enterotoxigenic *Escherichia coli* (ETEC), is a major concern in swine production due to its economic impact and rising antimicrobial resistance. Key risk factors include early weaning, poor immunity, environmental stress, and inadequate management. The frequent use of colistin, a last-resort antibiotic, has led to resistance concerns, especially due to plasmid-mediated *mcr-1*. Preventive strategies focus on improving hygiene, nutrition, and management, along with exploring alternatives such as probiotics, vaccines, and bacteriophages. Reducing antibiotic reliance through integrated farm-level practices remains essential to controlling PWD and mitigating antimicrobial resistance.

**Keywords:** Post-weaning diarrhea (PWD), enterotoxigenic *Escherichia coli* (ETEC), piglets, colistin resistance, *mcr-1*, antimicrobial resistance, weaning stress, gut health, probiotics, vaccines, bacteriophages, swine management.

## INTRODUCTION

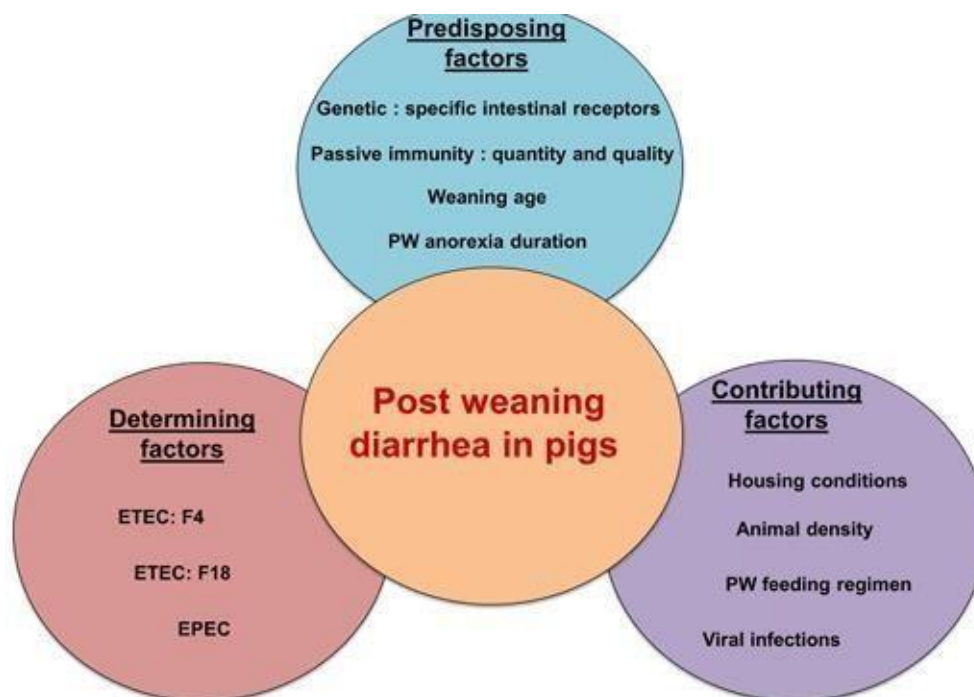
Post-weaning diarrhea (PWD) in pigs, primarily caused by enterotoxigenic *Escherichia coli* (ETEC), is a major economic concern globally. It typically affects piglets within two weeks post-weaning due to stress factors and intestinal dysfunction. The predominant ETEC serogroup involved is O149, expressing F4 or F18 adhesins. Colistin, a last-resort antibiotic in human medicine, is widely used for PWD control but faces rising resistance, including plasmid-mediated *mcr-1*. This raises public health concerns due to potential resistance gene spread. The review highlights PWD risk factors, global colistin use, resistance outcomes, and explores alternative strategies for managing PWD and antimicrobial resistance in pig farming.

## Risk Factors for Post-Weaning Diarrhea in Pigs

Post-weaning diarrhea (PWD) is a significant gastrointestinal condition in pigs, particularly during the first two weeks following weaning. It is known to cause considerable economic losses in the swine industry due to its impact on pig health and productivity. Affected piglets often suffer from severe diarrhea, dehydration, weight loss, and in many cases, increased mortality. During severe outbreaks, mortality rates can range from 20% to 30% over a period of one to two months (Amezcuca et al., 2002)

PWD is a complex disease with multiple contributing factors, and its exact cause remains

unclear (**Jensen et al., 2006**). It typically results from a combination of influences, including the health and condition of the sow, the immune status and gut health of the piglets, environmental stressors, the presence of enterotoxigenic *Escherichia coli* (ETEC), and overall management practices. These interconnected elements make the disease challenging to prevent and control effectively (**Hong et al., 2006**).



### Predisposing Factors for Post-Weaning Diarrhea in Pigs

Post-weaning diarrhea (PWD) in pigs is often triggered by the overgrowth of certain strains of  $\beta$ -hemolytic enterotoxigenic *Escherichia coli* (ETEC) in the small intestine. The most common strains involved are those that produce fimbrial adhesins, specifically F4 (K88) and F18 (**Fairbrother et al., 2005**). These fimbriae enable the bacteria to attach to receptors on the pig's intestinal lining (F4R or F18R), which is a critical step in bacterial colonization. The presence and function of these receptors are largely determined by the pig's genetic makeup, making genetics a key factor in susceptibility to PWD (**Rhouma et al., 2016**).

Additionally, factors related to the sow—such as litter size, number of pregnancies (parity), and conditions like postpartum dysgalactia syndrome—can influence a piglet's vulnerability to infections (**Muns et al., 2016**). Because piglets do not receive antibodies from the sow before birth (due to the impermeability of the pig placenta), they rely entirely on colostrum intake during the first 24–48 hours of life for passive immunity (**Lalles et al., 2007**). The quality and timing of this colostrum intake are essential for the piglet's early immune defense.

Weaning age also plays a critical role in the development of PWD. Early weaning, especially before 21 days of age, has been linked to a higher risk of diarrhea, as piglets are more immunologically immature and lose access to the protective immunoglobulin A (IgA) found in sow's milk. Sow milk, unlike in other species, contains high levels of IgA, which helps protect the gut from harmful bacteria. Therefore, delaying weaning to at least 26–28 days of age is recommended to reduce stress, improve gut development, and enhance the piglet's immune system. In the European Union, regulations support weaning no earlier than 28 days unless proper hygienic housing conditions are met. ( **Baxter et al., 2016**).

Another major risk factor is reduced feed intake immediately after weaning. Many piglets eat less or stop eating altogether during this transition, which increases the likelihood of gut inflammation and damage. Inadequate nutrition during this period can lead to shortened intestinal villi and deeper crypts ( **McCracken et al., 1999**) weakening the gut's barrier function and making it easier for harmful bacteria like ETEC to invade and cause infection. This poor gut morphology creates a favorable environment for bacterial growth and toxin production, further aggravating the risk of PWD.

### **Contributing Factors to Post-Weaning Diarrhea in Pigs**

Several management and environmental factors can influence the development of post-weaning diarrhea (PWD) in pigs. Key contributors include housing conditions, stocking density, weaning practices such as parity-segregated production, and feeding strategies during the post-weaning period ( **Laine et al., 2008**)

One of the most important aspects of housing is maintaining an appropriate thermal environment for weaned piglets. According to Le Dividich and Herpin, keeping the temperature within the pigs' thermo-neutral range—typically between 26°C and 28°C—is crucial. Exposure to cold can reduce intestinal motility, making it easier for harmful bacteria to colonize the gut. In fact, low temperatures in weaner housing have been linked to more severe cases of PWD( **Fairbrother et al.,2016**). Automated systems that regulate housing temperature have been shown to significantly lower the occurrence of diarrhoea ( **Laine et al., 2008**).

Proper ventilation is also essential. While it is important to remove excess moisture and harmful gases, care must be taken to avoid drafts, which can also stress piglets and increase disease susceptibility. Research by Wathes and Whittemore has emphasized the importance of sound environmental management in reducing disease risk in pigs.

The type of flooring used in weaner pens can also influence health outcomes. While flat decks are often preferred over bedding for hygienic reasons, they have been associated

with an increase in skin injuries, particularly on the tail and belly. Regular cleaning of housing areas, especially the removal of manure and soiled bedding, is critical to reduce the environmental bacterial load and minimize the risk of infection.

These environmental and management practices, when carefully controlled, can play a significant role in reducing the prevalence and severity of PWD in weaned pigs.

### Determining Factors in the Development of Post-Weaning Diarrhea

Enterotoxigenic *Escherichia coli* (ETEC) is recognized as the leading cause of post-weaning diarrhea (PWD) in pigs (**Fairbrother et al., 2005**). This strain of *E. coli* causes disease through the production of two key components: enterotoxins and fimbrial adhesins. These factors are essential for colonizing the intestinal tract and initiating the disease process. ETEC bacteria produce a variety of enterotoxins, including heat-stable toxins (STa, STb, and EAST1) and a heat-labile toxin (LT). These toxins, encoded on plasmids, act on the pig's intestinal lining, disrupting normal fluid balance.

The most commonly found fimbriae in ETEC strains affecting pigs include F4 (also known as K88), F5 (K99), F6 (987P), F41, and F18 (**Fairbrother et al., 2012**). Among these, F4 and F18 fimbriae are the most frequently associated with PWD. The F4 fimbriae occur in three variants—F4ab, F4ac, and F4ad—with F4ac being the most prevalent in clinical cases (**Schroyen et al., 2012**). These structures enable ETEC to adhere to specific receptors (F4R) on the intestinal epithelial cells, allowing the bacteria to colonize and persist in the gut. F18 fimbriae also have two variants: F18ac, mainly linked to PWD, and F18ab, more often associated with edema disease (**Byun et al., 2013**). Notably, vaccines targeting one variant do not offer protection against the other. (**Bertschinger et al., 2000**)

In addition to fimbrial adhesins, non-fimbrial adhesins like AIDA (adhesin involved in diffuse adherence) have been observed in ETEC strains isolated from pigs with diarrhea (**Moredo et al., 2015**) AIDA plays a significant role in promoting bacterial colonization, aggregation, and biofilm formation, further enhancing the bacteria's ability to persist in the gut.

ETEC strains responsible for PWD typically belong to serogroups such as O8, O138, O139, O141, O147, O157, and especially O149 (**Noamani et al., 2003**), which is the most common in many regions. The most frequently identified virotype in PWD is ETEC: LT: STb: F4.

However, the distribution of serogroups and toxin gene profiles can vary depending on

geographic location and over time. (Fairbrother et al.,2005)

Infection usually begins when piglets ingest ETEC from contaminated environments—this includes contact with sow’s skin, farrowing rooms, or nursery pens. Infected or carrier pigs shed the bacteria into the environment, where it is picked up by susceptible piglets. Once inside the intestine, ETEC attaches to the epithelial lining and begins to release enterotoxins. These toxins disrupt the normal absorption and secretion mechanisms in the gut, leading to excessive loss of water and electrolytes. As a result, affected pigs experience diarrhea, dehydration, and in severe cases, metabolic acidosis and death.

Although ETEC infection typically does not result in significant visible damage to the intestinal lining, some studies have observed clinical signs such as dehydration, distended intestines, and gastric lesions in infected animals. In more severe cases, pigs may develop hemorrhagic gastroenteritis, kidney hemorrhages, and thrombi in intestinal tissues. Some infections may also lead to septicemia, even when specific toxin genes like *hlyA* (hemolysin) are not active.

Microscopically, infected pigs often show noticeable changes in the intestinal structure, such as shortening of villi and increased crypt depth, which compromise nutrient absorption and gut integrity.

Furthermore, ETEC strains associated with PWD frequently exhibit resistance to multiple antibiotics. While this resistance does not appear to directly increase the bacteria’s virulence, resistance genes are sometimes found alongside virulence genes on the same plasmids.

Other types of *E. coli*, such as attaching and effacing *E. coli* (AEEC), can also cause intestinal damage similar to that seen in human enteropathogenic *E. coli* (EPEC) (Fairbrother et al., 2012). These strains carry the *eae* gene, which encodes the protein intimin, responsible for close attachment to intestinal cells. However, the role of AEEC and EPEC-like strains in causing PWD remains unclear, and they are not routinely tested for in many veterinary labs due to the absence of typical virulence markers.

### Prevention of Post-Weaning Diarrhoea—How Different Strategies Work

Over the years, many studies have explored ways to reduce or eliminate the use of antibiotics and medicinal zinc in pig farming, especially in relation to post-weaning diarrhoea (PWD).

These efforts have led to a variety of feeding strategies. Some focus on altering the

diet— such as adjusting the protein levels or sources, modifying the amount and type of fibre (soluble or insoluble), or changing the fat content. Others involve the use of feed additives like probiotics, prebiotics, enzymes, antimicrobial peptides, organic acids, plant extracts, or specific amino acids. The form of the feed—whether it’s liquid or dry, coarse or fine, pelleted or meal—can also play a role.

In addition to these widely studied methods, some newer approaches have shown promise, although they’ve been researched less. These include vaccines, treatments using bacteriophages (viruses that target bacteria), and single-domain antibodies. Each of these strategies works in different ways, which means their effectiveness can vary depending on the specific conditions and disease pressures found at individual pig farms.

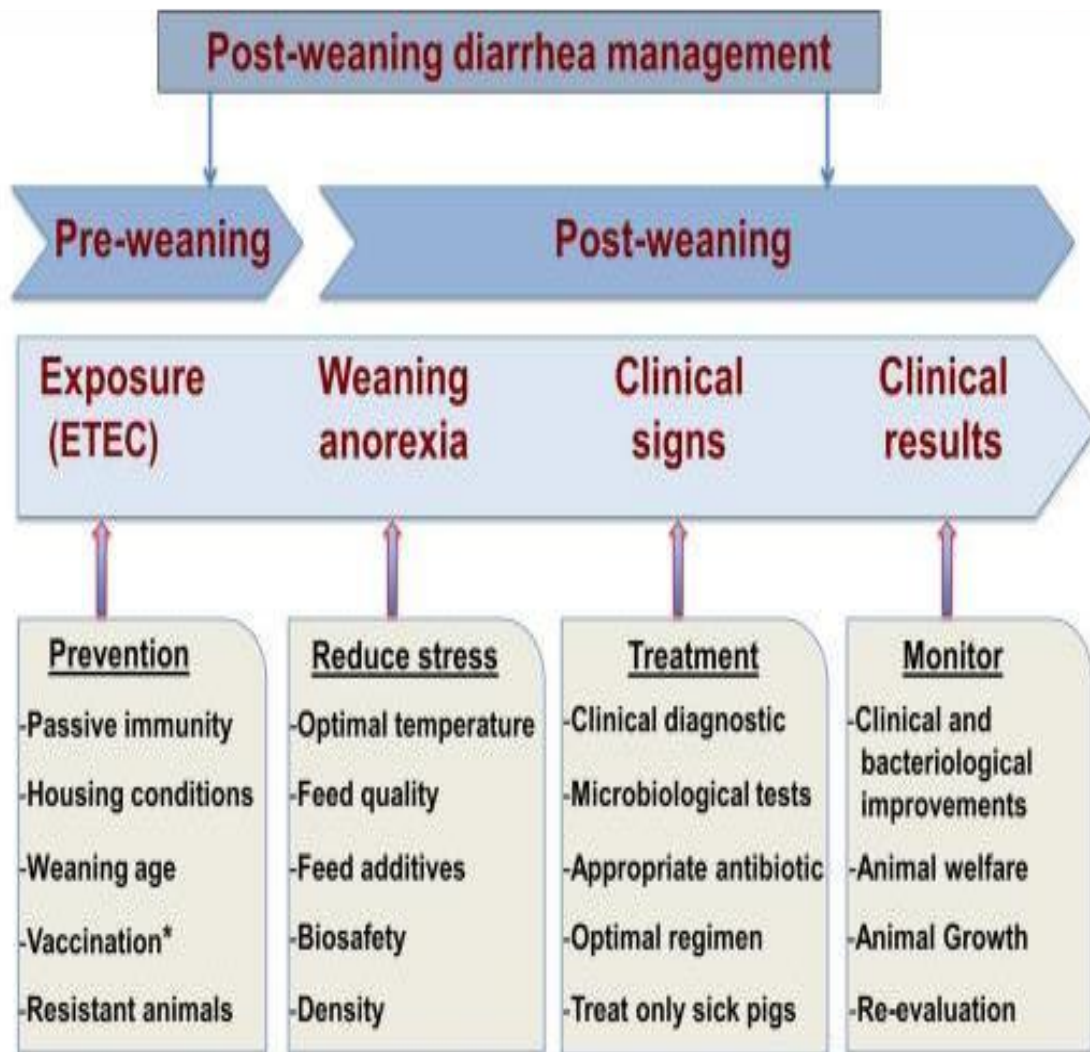


Fig. 3 Illustrative interventions for the management of post-weaning diarrhea in pig farms. Asterisk Vaccination just prior to or at weaning (Inspired from [159])

## Preventive Measures

To reduce the need for antibiotics like colistin in pig farming, the best approach is to minimize antimicrobial use at the farm level. Research shows that better housing and management practices can significantly lower the risk of post-weaning diarrhoea (PWD). For example, simply improving living conditions—such as reducing overcrowding—can help prevent PWD and other diseases. Good weaning management is also key. This includes reducing stress, controlling infections, ensuring proper temperature, and providing all pigs with suitable, accessible feed. Overall, improving the environment and care of pigs makes them less prone to infections and reduces reliance on antibiotics.

## Conclusions

In recent years, pig farms have made notable progress in preventing disease and improving animal health, yet post-weaning diarrhea (PWD) remains a significant issue causing economic losses. While antibiotics have played a major role in controlling PWD, rising antibiotic resistance and growing consumer concerns about drug residues have prompted a shift toward alternative treatments. As a result, many new therapies have been researched and introduced. However, their effectiveness often varies depending on specific farm conditions and livestock management practices.

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