



SWINE DYSENTERY: A RE-EMERGENT CHALLENGE FOR PROFITABLE PIG PRODUCTION

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ABSTRACT

Swine dysentery (SD or bloody scours) is a “gut” disease which is very expensive to treat medically and difficult to effectively remove once pigs and facilities are contaminated. It is a severe mucohaemorrhagic enteric disease of pigs caused by *Brachyspira hyodysenteriae*. Transmission mainly occurs by ingestion of infected faeces of affected animal. SD causes a large impact on pig production and leads to severe losses due to mortality and sub-optimal performance. The typical sign of infection include watery stools containing blood, mucus, and shreds of white mucofibrinous exudate, with concurrent staining of the perineum. The re-emergence of *Brachyspira* species including antimicrobial resistant strains of *B. hyodysenteriae* and novel species like *B. hampsonii* as pathogens has re-ignited significant concerns for pork-producers worldwide.

KEY WORDS: Swine dysentery, spirochetes, *Brachyspira hyodysenteriae*, bloody scours, mucohaemorrhagic colitis.

INTRODUCTION

Swine dysentery (SD or bloody scours) is a mucohaemorrhagic enteric disease of pigs caused by *Brachyspira hyodysenteriae*; it causes large impact on pig production and leads to severe losses due to mortality and low production performance (Wills, 2000). The disease virtually disappeared from many regions during the 1980’s and 1990’s because of a better understanding of cause and transmission, availability of swine dysentery free breeding stock, better bio security/sanitation, and availability of cost-effective treatment/elimination drugs for use in a herd eradication program. However, it began re-emerging in many parts of the world since 2005 (Burrough, 2016). Accordingly, there has been a renewed interest in swine dysentery and *Brachyspira spp.* infections in pigs, particularly in areas where the disease was previously eliminated. The present article discusses the salient features of Swine dysentery emphasizing the etio-pathogenesis and pathology of the disease.

ETIOLOGY

B. hyodysenteriae is a Gram negative, motile, helically coiled (spiral-shaped), anaerobic bacterium. It is 6–8.5 µm long, 0.32–0.38µm wide and has 7–14 periplasmic flagella inserted at each cell end. The cell is covered by a loose outer membrane. *B. hyodysenteriae* outer envelope contains Lipooligosaccharides (LOS), a semi rough form of lipopolysaccharide (Hampson, 2012). Several CDS (protein coding sequences) predicted as putative virulence factors have been identified and proposed as virulence factors in the bacterial genome. *B. hyodysenteriae* was shown to differ from all the other spirochetes, including *Leptospira*, *Borrelia* and *Treponema*, in signal

transduction and in amino acid transport and metabolism systems (Alvarez-ordonez *et al.*, 2013).

EPIDEMIOLOGY

Swine Dysentery has a worldwide distribution. The incidence varies in different countries and regions, and changes with time. SD remains a relatively common and important endemic problem in many countries in the European Union, South America and Southeast Asia (Burrough, 2016).

HOST RANGE

Brachyspira hyodysenteriae naturally infects pigs (including feral pigs) and occasionally some species of birds (rheas, chickens, ducks, and geese). On infected farms it has been isolated from mice, rats, dogs, and feral birds, including seagulls (Desrosiers, 2011).

TRANSMISSION

Brachyspira hyodysenteriae is shed in faeces for variable periods. The incubation period of the disease is from 2 days to 3 months, but usually disease occurs 10-14 days after exposure. Transmission mainly occurs by ingestion of infected faeces of affected animal (Jensen *et al.*, 2010). Wild rodents are potential vectors of *Brachyspira spp.* (Backhans *et al.*, 2009). Wild boars may also act as a potential source of infection (Phillips and Hampson, 2009). Apart from feral animals, domestic animals present in the farms, principally dogs, can act as a reservoir of *Brachyspira spp.* Wild-living water-birds and laying hens transmit or disperse the pathogens in their migration by excretion of organisms in faeces (Jansson *et al.*, 2004). Insect vectors like cockroaches and flies harbour

Brachyspira spp. and constitute a reservoir and source of infection for pigs (Blunt *et al.*, 2010).

PATHOGENESIS

Brachyspira hyodysenteriae following ingestion from faeces survives the acidic environment of the stomach due to the covering of organism with mucus from dysentery of shredded animal. They eventually reach the large intestine, where it invades the mucus and crypts of the mucosa in the large intestine and penetrates into colonic enterocytes and goblet cells (Mirajkar *et al.*, 2016).

Organism at epithelial cells of lumen and crypts of caecum and colon stimulates the outpouring of mucus. Then they produce tissue destruction by Hemolysins and Lipooligosaccharide (LOS) which plays main role in

damaging epithelial barrier in colon (Mirajkar *et al.*, 2016). Epithelial necrosis and vascular leakage may lead to conditions favouring overgrowth of opportunistic bacteria. Subsequent sub mucosal invasion by secondary bacteria and the protozoan *Balantidium coli* may contribute to lesion formation. No production of septicaemia has been noticed (Duhamel, 2001).

Diarrhoea appears as a result from colonic mal absorption due to a failure of epithelial transport mechanisms to actively transport sodium and chloride ions from lumen to blood, and not from the activity of enterotoxins and/or prostaglandins released from the inflamed tissues, because there is no evidence of increase in cAMP and cGMP in colonic mucosa of dysenteric pigs (Moeser and Blikslager, 2007) (Fig. 1).

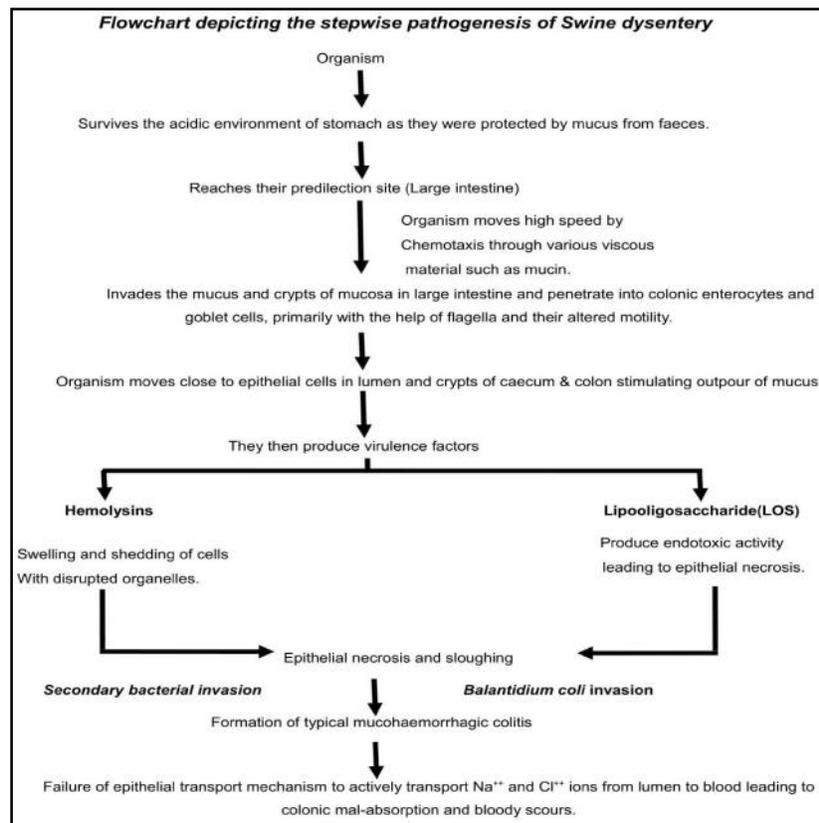


FIGURE1. Flowchart depicting the stepwise pathogenesis of Swine dysentery

CLINICAL SIGNS

The first evidence of Swine Dysentery is usually soft, yellow to grey faeces. Partial anorexia, increased rectal temperature of 104–105°F (40–40.5°C) and arched back due to abdominal pain can be seen (Walczak, 2015). A few hours to days after infection, large amounts of mucus and often flecks of blood are found in the faeces. This progresses to watery stools containing blood, mucus, and shreds of white mucofibrinous exudate, with concurrent staining of the perineum (Hampson, 2012). Appearance of white mucofibrinous grains in the stools is pathognomonic as the disease progresses.

Occasionally, pigs are per acutely affected and die within a few hours. Most pigs recover over several weeks, but their growth rate remain depressed. On endemically infected swine farms, clinical signs often recur cyclically

at 3- to 4-week intervals in affected animals. Reappearance may occur after removal of antimicrobials from the water or feed (Burrough, 2016).

GROSS LESIONS

Typical changes in acute Swine Dysentery include hyperaemia and oedema of the large intestinal walls and mesentery. Mesenteric lymph nodes may be swollen. Small amount of clear ascitic fluid can be seen. There may be white, slightly raised foci on the serosa caused by submucosal aggregates of mononuclear cells. The mucosa is usually swollen, with loss of the typical rugose appearance, and is covered by mucus and fibrin, with flecks of blood. The colonic contents are soft to watery and contain exudates (Hampson, 2012). As the condition progresses the oedema in the colon wall may decrease.

Mucosal lesions become more severe, with increased fibrin exudation and formation of thick, mucofibrinous pseudo-membranes containing blood. As lesions become chronic the mucosal surface are usually covered by a thin, dense, fibrinous exudates, resembling superficial necrosis (Burrough *et al.*, 2012).

Lesions start in the centrifugal and centripetal coils near the apex of the colon and may extend to the whole colon through the caecum, and in some instances the whole large intestine may become involved (TerHuurne *et al.*, 1994). The distribution of lesions within the large intestine varies. Sometimes the entire organ may be involved, while at other times only certain segments may be affected. Lesions tend to become more diffuse in the later stages of the disease. Hepatic congestion, hyperaemia or congestion of the gastric fundus may occur; however, such lesions are not specific for SD (Stanton, 2006).

MICROSCOPIC LESIONS

Significant microscopic lesions are found only in the caecum, colon, and rectum. Typical acute lesion includes a thickened mucosa and submucosa, due to the vascular congestion and extravasation of fluids and leukocytes in the affected portions of intestine. Goblet cell hyperplasia may be present and the epithelial cells at the base of the crypts may be elongated and hyperchromic (Jacobson *et al.*, 2004). There may be spirochetes seen in goblet cells of the colonic crypts and the intercellular gaps in the epithelium. Spirochetes may also be found attached to the luminal surface and inside of the disrupted epithelial cells (Rubin *et al.*, 2013).

There may be an increase in number of leukocytes in the lamina propria, with accumulation of neutrophils in and around capillaries near the lumen. Some spirochetes may be seen in the lamina propria, particularly around blood vessels. Clumps of epithelial cells may detach from the lamina propria, resulting in exposure of capillaries followed by focal areas of haemorrhages. Bleeding may occur from small vessels under the areas of eroded epithelium, and this may be invaded by the colonic microbiota (Jensen *et al.*, 2000).

Later changes include accumulation of fibrin, mucus and other cellular debris in mucosal crypts and on the luminal surface of the large intestine. Superficial necrosis of the mucosa may be extensive, but deep ulceration is not typical. Increased numbers of neutrophils may be seen throughout the lamina propria. Chronic changes are not very specific, with less hyperaemia and oedema being present. There is often more advanced superficial necrosis of the mucosa, which usually has a thick, fibrinous pseudomembrane (Hampson *et al.*, 2006).

LABORATORY DIAGNOSIS

Spirochetes can be seen in smears from the colonic mucosa or faeces, but this does not distinguish between the different *Brachyspira* species (Hampson, 2012). A definitive diagnosis of SD requires the demonstration of *B. hyodysenteriae* which can be done by selective anaerobic culture and analysis of phenotypic properties of the isolated organisms from the culture. Trypticase soy agar, C.V.S. media and Blood agar are commonly used for the cultivation of the organisms (Alvarez-ordonez *et al.*,

2013). Antigen based methods, including fluorescent antibody test, growth-inhibition test, and rapid slide agglutination test have been described for identification of *B. hyodysenteriae*, but these have largely been superseded by polymerase chain reaction (PCR) testing (Rasback *et al.*, 2006). PCR amplification of specific sequences are widely used for detection and identification of *B. hyodysenteriae*. The most usual targets for amplification are portions of the 23S rRNA gene, the *nox* gene and the *tlyA* gene (Fellstrom *et al.*, 2004).

CONCLUSION

The re-emergence of *Brachyspira* species including antimicrobial resistant strains of *B. hyodysenteriae* and novel species like *B. hampsonii* as pathogens has reignited significant concerns for pork-producers worldwide. Limitation in the success for vaccine development and efficacy has marked the disease as one of the potential re-emergent pathogen of swine population. Routine surveillance at local, national and international level is required not only to monitor *Brachyspira* species infections in pigs, but also carriage in other species which may act as reservoirs of infection (particularly migratory water birds). Purchased/ imported animals should be quarantined for at least 3 weeks and treated to eliminate *B. hyodysenteriae*. Infectious materials (fomites such as workers boots, farm implements, feed or animal trucks) must be properly sanitized for prevention of the disease. Apart from the therapeutic interventions, proper management measures are necessary to control the disease from its spread. The re-emerging status of the disease calls for further innovation in various aspects of pig rearing for a healthy and profitable herd free from swine dysentery.

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