

The Splay Leg Syndrome in Piglets: A Review

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Abstract: Problem statement: The splay leg syndrome is the major congenital cause of lameness in suckling piglets. It is characterized by a temporarily impaired functionality of the hind leg muscles immediately after birth, resulting in an inability to stand and walk. Etiology and pathogenesis is complex and remain still poorly understood. **Approach:** The aim of the present study is to perform the update information about the etiology, clinical signs and control strategies of the syndrome. **Results:** A sex-affected inheritance of the splay leg syndrome is assumed since higher frequencies have been observed in male piglets. Several biochemical and histomorphological investigations indicate an immaturity of the skeletal muscle in the affected piglets at birth. Splay leg is caused by a reduction of the axonal diameter and myelin sheath thickness of the fiber that innervate the hindlimb adductors. The existence of one or more major genes for congenital splay leg seems possible. Among the fragments strongly displayed in the splay leg muscle, are identified the porcine *CDKN3* gene. Various management and genetic factors have been connected with the etiology, such as the farrowing induction, low birth weight, short gestation lengths, slippery floors and breeds (e.g., Large White and Landrace). Moreover, nutrition can play a role to pathogenesis, as choline or methionine deficiency in sow diets and the fusarium toxicity. Furthermore, Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) are also involved in etiological factors. Piglets suffering from splay leg should be kept in a warm place and be helped to take colostrums or artificial milk for 2-3 days. **Conclusion:** Management practices should be applied in order to be avoided the occurrence of splay leg syndrome, such as non-slip floors, use of anti-mycotoxins products in feed, avoiding the farrowing induction before day 113. Finally, a herd health management programme should be applied in order to prevent and control PRRSV infection.

Key words: Porcine Reproductive and Respiratory Syndrome Virus (PRRSV), skeletal muscle, piglet, splay leg

INTRODUCTION

The splay leg syndrome was first recognized as a hereditary disorder by Thurley *et al.* (1967). It is the major congenital cause of lameness in suckling piglets and it is the most frequent observation amongst hereditary disorders in swine (Partlow *et al.*, 1993). The congenital splay leg syndrome is characterized by a temporarily impaired functionality of the hind leg muscles immediately after birth, resulting in an inability to stand and walk (Thurley *et al.* 1967). It affects approximately 0.4% of live births (Ward, 1978) and is a widespread problem for the swine industry, affecting typically only 1-4 pigs per litter and just a few litters at a time. Usually individual herd prevalence is less than 1%; however, sporadic increases occur when the prevalence reaches 8% or higher (Ward and Bradley, 1980). Losses among affected piglets can amount to 50%, making congenital splay leg to a source of considerable economic losses in pig production

(Dobson, 1968). The purpose of the present study is to perform the update information about the etiology, clinical signs and control strategies of the syndrome.

Etiology/pathogenesis: Etiology and pathogenesis of splay leg syndrome is complex and remain still poorly understood. However, a sex-affected inheritance of the splay leg syndrome is assumed since higher frequencies have been observed in male piglets than in female piglets (Lax, 1971; Sellier and Ollivier, 1982).

Several biochemical and histomorphological investigations indicate an immaturity of the skeletal muscle in the affected piglets at birth (Bergmann, 1976; Hillert *et al.*, 1987; Prange *et al.*, 2001). Some researchers reported muscle hypoplasia as the cause of the clinical signs, but myofibrillar hypoplasia is normal in all newborn pigs. Splay leg is caused by a reduction of the axonal diameter and myelin sheath thickness of the fiber that innervate the hindlimb adductors (Szalay *et al.*, 2001).

The existence of one or more major genes for congenital splay leg seems possible (Maak *et al.*, 1999, 2001a ; 2001b). Among the fragments strongly displayed in the splay leg muscle, are identified the porcine *CDKN3* gene (cyclin-dependent kinase inhibitor 3) (Maak *et al.*, 2002). *CDKN3* belongs to a family of dual-specificity protein phosphatases and is involved in cell cycling (Gyuris *et al.*, 1993; Hannon *et al.*, 1994; Maak *et al.* 2003). Recently, Maak *et al.* (2009), comparing genome wide gene expression of three hind leg muscles (*M. adductores*, *M. gracilis* and *M. sartorius*) between splaylegged piglets and their healthy littermates, found that four genes with different expression levels in at least two muscles are assigned to transcriptional cascades related to cell death. However, histomorphological investigations, analysis of biochemical criteria as well as investigations on putative candidate genes lead to contradictory results (Ward and Bradley, 1980; Jirmanova, 1983; Ooi *et al.*, 2006; Boettcher *et al.*, 2007; 2008). Ooi *et al.* (2006) described congenital splay leg as muscle fiber atrophy characterized by an increased expression of the atrophy marker *FBXO32* (atrogin, *MAFbx*) and histological signs of a generalized muscle fiber hypoplasia in skeletal muscles of splay leg piglets (Ooi *et al.*, 2006). In addition, other researchers reported a large individual variability in *FBXO32* expression as well as in histological characteristics of hind limb muscles, which has previously been described (Björklund *et al.*, 1987; Curvers *et al.*, 1989). A congenital, impaired functionality of skeletal hind limb muscles due to immaturity and/or atrophic properties is likely to be the major pathomorphological feature in splay leg syndrome.

Various management and genetic factors have been connected with the etiology of splay leg syndrome, such as the farrowing induction, low birth weight, short gestation lengths, slippery floors and breeds (e.g., Large White and Landrace pigs) (Ward, 1978). Moreover, nutrition can play a role to pathogenesis, as choline or methionine deficiency in sow diets are correlated with the presence of splay leg syndrome. Some researchers supported that one cause of splayleg was a deficiency in the sow's diet of choline and methionine which are essential for normal myelin production (Kornegay and Meacham, 1973). In contrast, the addition of 3 g choline and 5 g methionine to the sows' daily ration feed had no effect on the occurrence of splayleg (Dobson, 1971). Finally, nutrition also is involved in etiological factors and especially the fusarium toxicity. The contamination of feed in sows with more than 4 ppm zearalenone can result in increase in the number of piglets born with splay-leg from sows (Kanora and Maes, 2009).



Fig. 1: A 6-days-old piglet at with splay leg syndrome

During the entire gestation, the same contamination leads to a reduced weight of the fetuses and a larger weight variation between piglets of the same litter (Etienne and Jemmali, 1982). It also leads to found a negative association between the plasma level of zearalenone and average birth weight of the piglets (Young and King, 1986).

Furthermore, infectious factors might be also involved in etiology. Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) cause late term reproductive failure in sows, which is characterized by increased number of stillbirth, weak, light weight and splay-legged piglets (Papatsiros *et al.*, 2006; Zimmerman *et al.*, 2006).

Clinical signs: Affected piglets are characterized by an obvious weakness of the hind limbs immediately after birth leading to impaired mobility (Bergmann, 1976). Consequently, losses among splay leg piglets amount to 15-50% (Hörügel and Lorenz, 1979; Wrathall, 1988; Svendsen *et al.*, 1991).

Clinically, pigs show extreme abduction of the limbs with an inability to stand (Fig. 1). Splay leg affects the hindlimb adductors and in severe cases the forelegs are also involved (Szalay *et al.*, 2001). Usually, 50% of affected piglets die due to starvation and overlying, because the pigs have difficulties in reaching the udder, retaining hold of the nipple, competing with their littermates and moving out of the sows lying space (Dewey *et al.*, 2006). Affected pigs can be kept alive if they are fed artificially including colostrums, nursed well and have their limbs taped in a natural standing pose. By 6 days of age, the splay legged pigs do not differ from normal piglets clinically or in the histology of the muscles. Piglets that live past the first week of life will recover completely (Dewey *et al.*, 2006).

Treatment and control: First of all, piglets suffering from splay leg should be kept in warm conditions. They also should be helped to take colostrums or artificial milk for 2-3 days. The most common practice is the taping the hind limbs together or the use of elastic bands, improving piglets' ability to stand, suckle and move around until the legs strengthen. In addition, the repeated massage of the hind limbs seems to improve their survival.

Management practices should be applied in order to be avoided the occurrence of splay leg syndrome. For example, non-slip floors can improve footing and viability of piglets. Moreover, the use of anti-mycotoxins products in feed in order to prevent the zearalenone toxicity can be beneficial. In cases of feed contamination by zearalenone, the contaminated sow feed should be removed, but this takes up to 14 days to reduce the levels of zearalenone in milk and thus fostering or artificial feeding may be required to reduce mortality and aid recovery. The farrowing induction using prostaglandines should be also avoided before day 113.

Finally, a herd health management programme should be applied in order to prevent and control PRRSV infection. The main target of such programme is mainly to minimize the virus transmission and circulation in breeding stock, as well as to maximize the herd immunity against PRRSV. Key points of this programme should be the establishment of strict biosecurity measures, acclimatization of replacement gilts by exposure to the specific PRRSV circulating on farm, as well as the application of appropriate vaccination programme (Papatsiros, 2011; 2012).

CONCLUSION

Management practices should be applied in order to be avoided the occurrence of splay leg syndrome, such as non-slip floors, use of anti-mycotoxins products in feed, avoiding the farrowing induction before day 113. Finally, a herd health management programme should be applied in order to prevent and control PRRSV infection.

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