RELATIONSHIP BETWEEN HISTOPATHOLOGICAL LESIONS AND MAGNETIC RESONANCE IMAGING (MRI) IN DISEASES OF THE FOOT OF THE HORSE

RELATIONS ENTRE LES LÉSIONS ANATOMOPATHOLOGIQUES ET LES IMAGES DE RÉSONANCE MAGNÉTIQUE (IRM) DANS LES MALADIES DU PIED CHEZ LE CHEVAL

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SUMMARY

The increasing number of available pathological studies on the various tissues of the foot has helped validate the significance of Magnetic Resonance Imaging (MRI) signal variations documented with various clinical MRI systems used in equine practice. In particular MRI has helped elucidate the different grades of histological abnormalities and possibly also different stages of disease progression in the deep digital flexor tendon and the navicular bone. However, further work is needed to continue to improve our understanding of the different causes of foot lameness and their pathogenesis.

Key words: MRI, deep digital flexor tendon, navicular bone.

RÉSUMÉ

Le nombre croissant d'études histopathologiques portant sur les tissus du pied a permis d'expliquer et de valider les variations des signaux obtenus par l'imagerie de résonance magnétique (IRM) dans la pratique équine clinique. En particulier, l'IRM a permis d'élucider les différents grades des anomalies histologiques et peut-être aussi les différentes étapes de la progression de la dégénérescence du tendon fléchisseur profond et de l'os naviculaire. Cependant, des travaux supplémentaires sont nécessaires pour continuer d'améliorer nos connaissances sur les différentes causes de boiterie du pied et leur pathogénie.

Mot clés : IRM, tendon fléchisseur profond, os naviculaire.

Diseases of the horse's foot remain an important concern to the clinician, taking into account that they constitute a major cause of lameness in the horse. A detailed cinical examination and current imaging techniques have long been impeded by the presence of the hoof capsule, leading to the use of vague diagnostic terminology like "podotrochlear syndrome" and "navicular syndrome", to describe the presence of chronic lameness in the palmar aspect of the foot but without specific anatomopathological definition or localization. More recently the advent of magnetic resonance imaging has filled this void by allowing us a detailed anatomical window into all anatomical structures of the foot. Magnetic resonance imaging (MRI) is now widely used in the diagnosis of equine foot lameness ***(table 1)***.

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It has been able to provide diagnostic information not available from other imaging techniques. Understanding of the significance of alterations in signal intensity and patterns relies on the study of pathological changes that occur in the various disease processes that affect different tissues in the foot. The purpose of this paper is to review the current histopathological knowledge on various anatomical structures of the foot and the suitability of MR imaging for their identification.

Sequence	T2	T1	Proton Density	Inversion Recovery
Cortical bone	black	black	black	black
Cancellous bone	light grey	light grey	light grey	light grey
Cartilage	dark grey	light grey	grey	grey
Tendon	black	black	black	black
Ligament	black	grey to black	black	black
Fat	light grey	white	white	black
Fluid	white	dark grey	light grey	white

Table 1 : *The signal intensity of different tissues in different magnetic resonance contrast weightings.*

NAVICULAR BONE

The most common type of MR signal abnormality seen in the navicular bones of horses with lameness associated with navicular disease is short tau inversion recovery (STIR) signal hyperintensity in the medullary cavity of the navicular bone with or without additional areas of T2 and PD signal hypointensity ***(figure 1)***. Medullary STIR hyperintensity may be focal near the distal border of the navicular bone, or extend from the distal border in a vertical band along the palmar cortex to the proximal border of the bone, or spread diffusely throughout the medullary cavity (Sampson *et al.* 2009). In horses with chronic navicular disease, abnormal signal hyperintensities at the level of the palmar surface of the navicular bone are equally common as those in the medullary cavity. These can be areas of subtle, focal increase, caused by synovial fluid pooling at a site of early fibrocartilage loss ***(figure 2a)*** (Schramme *et al.* 2009), or more extensive signal increase extending deeper within the cortical bone of the flexor cortex (Sherlock *et al.* 2008).

Degenerative changes in the palmar fibrocartilage of the navicular bone occur in the distal half of the palmar aspect of the navicular bone, especially centered around the sagittal ridge, and may extend into the subchondral bone ***(figure 2b)***. Loss of fibrocartilage in this location is the most common lesion significantly associated with navicular disease and most likely represents the earliest pathology of classic navicular disease (Wright *et al.* 1998). Fibrocartilage loss from this location remains difficult to identify *in vivo*, even with the use of MRI (Schramme *et al.* 2009). Progression of fibrocartilage loss may result in cortical bone erosion in the flexor cortex, and even in osteonecrosis and fibroplasia extending into the spongiosa (Busoni *et al.* 2005; Schramme *et al.* 2005). Degenerative change of the spongiosa is generally only seen dorsal to extensive fibrocartilage damage. There may also be oedema, congestion and fibrosis of the marrow stroma within the medullary bone. Clinical experience with MRI in horses with foot pain from classic navicular disease provides support for the progression of lesions as outlined above.

However, there is a small number of horses with diffuse abnormalities of the medulla characterized by 'bone oedema' signal (increased signal in fat suppressed images) but no detectable abnormalities of the flexor fibrocartilage or cortex. *Postmortem* examination has revealed evidence of necrosis of medullary fat cells and active remodeling of medullary trabeculae, with both osteoclastic and osteoblastic activity along trabecular surfaces. In other horses with 'abnormal medullary fluid signal', intertrabecular edema and perivascular mononuclear cellular infiltration have been identified (Blunden *et al.* 2006 a). These lesions most likely have a different aetiopathogenesis than that of classic navicular disease and may be acutely traumatic or inflammatory in origin.

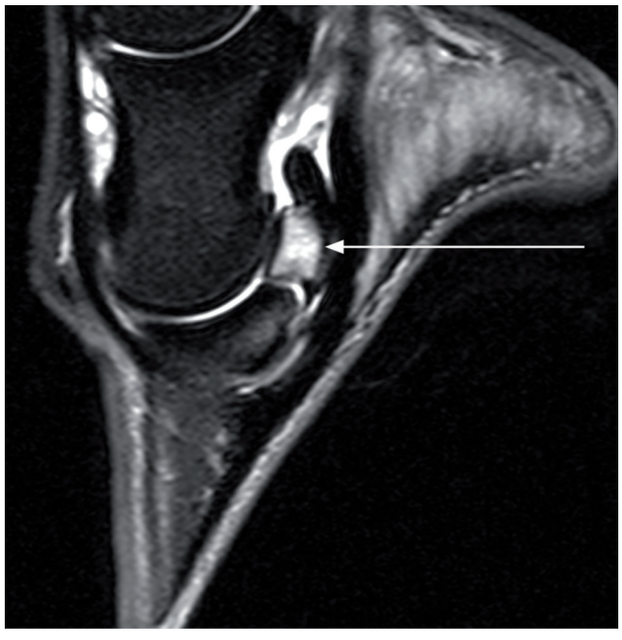

Figure 1: *Sagittal short tau inversion recovery (STIR) image of the foot of a horse with lameness that is abolished by anesthesia of the palmar digital nerves. There is marked STIR hyperintensity of cancellous bone in the medullary cavity of the navicular bone (arrow) indicating the presence of abnormal medullary fluid, medullary fibrosis or medullary fat necrosis.*

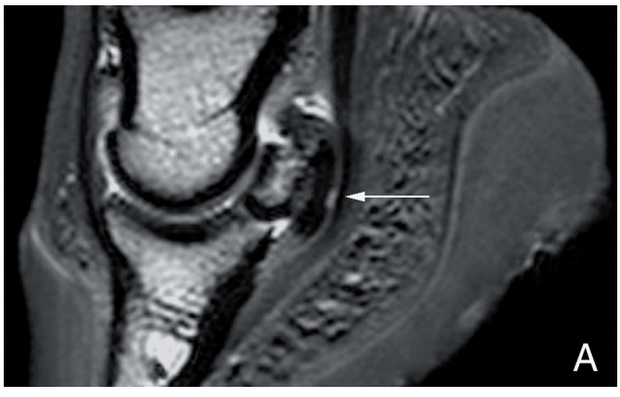

Figure 2a: *Sagittal 3D T2* GRE image of the navicular bone from a lame limb, with a focal accumulation of synovial fluid indicating a depression in the flexor surface (white arrow).*

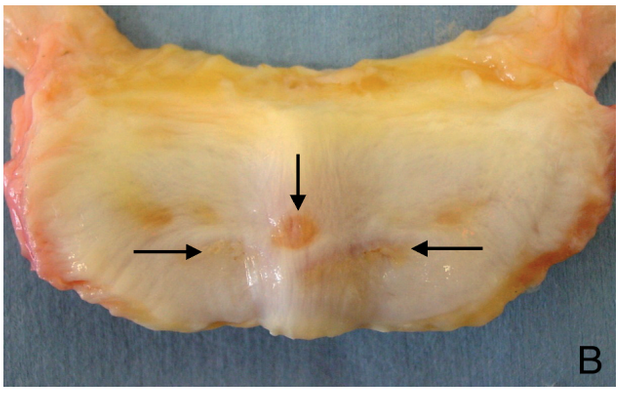

figure 2b: *Palmar view of the same navicular bone as 2a with a mid-ridge depression and 2 areas of fibrocartilage loss on both sides of the sagittal ridge (black arrows).*

In some other horses, fluid-filled osseous cyst-like lesions have been seen in the distal aspect of the bone, apparently separate from synovial invaginations, and not associated with any detectable abnormality of the flexor aspect of the bone. Such lesions have not yet been characterised histologically and their etiology remains speculative, but recent evidence suggest that their presence is associated with degenerative changes in the impar ligament (Dyson *et al.* 2010). In recent *postmortem* studies, osseous fragments associated with a defect in the distal margin of the navicular bone were more common in horses with navicular disease than in age-matched controls ***(figures 3a and 3b)*** (Busoni *et al.* 2005; Schramme *et al.* 2005; Blunden *et al.* 2006; Wright *et al.* 1998). Histologically, distal border fragments have variously been described as avulsion fractures, separate centers of ossification, osseous metaplasia of the impar ligament or synovial osteoma but pathological evidence elucidating their pathogenesis remains elusive. More recently it was shown that the presence of these fragments was associated with varying degrees of histopathological damage of collagen fibers and fibroblasts in the axial third of the impar ligament (Dyson *et al.* 2010).

DEEP DIGITAL FLEXOR TENDON (DDFT)

Tendinopathy of the DDFT in the foot is almost exclusively an MRI diagnosis. Tendon damage is seen as focal signal increase within the normal contour of the hypointense tendon lobes, on both T1- and T2-weighted sequences, variably accompanied by enlargement of the affected lobe. There is a good correlation between the MRI appearance of DDFT lesions and their pathological classification into core lesions, sagittal plane splits, dorsal plane tears, insertional lesions and dorsal abrasions (Busoni *et al.* 2005; Schramme *et al.* 2005; Murray *et al.* 2006; Blunden *et al.* 2006 b; Blunden *et al.* 2009).

Core lesions result in focal, circular areas of signal increase in the center or near the dorsal border of the affected lobe, but are completely surrounded by normal 'black' tendon signal ***(figure 4)***. Histologically they consist of various amounts of collagen necrosis, fibroplasia and fibrocartilagenous metaplasia resulting in loss of normal fascicular architecture. Core necrosis was seen more frequently in horses lame for less than 6 months. In horses with lameness of more than six months' duration, core lesions consisted predominantly of fibroplasia and/or fibrocartilaginous metaplasia (Blunden *et al.* 2006 b; Blunden *et al.* 2009).

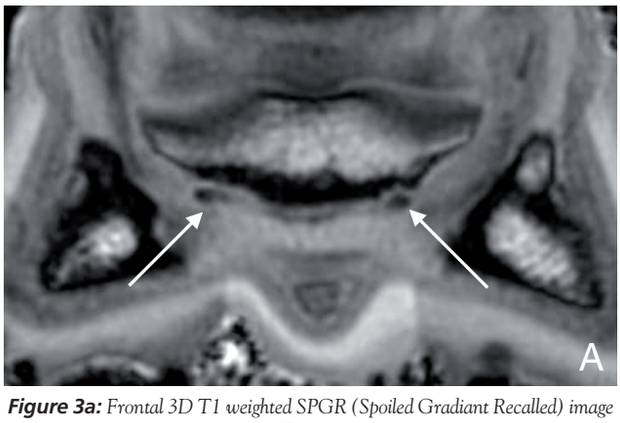

Figure 3a: *Frontal 3D T1 weighted SPGR (Spoiled Gradiant Recalled) image of the navicular bone of a lame foot. There are two focal areas of low signal intensity at the angles of the horizontal distal border with the sloping borders of the navicular bone.*

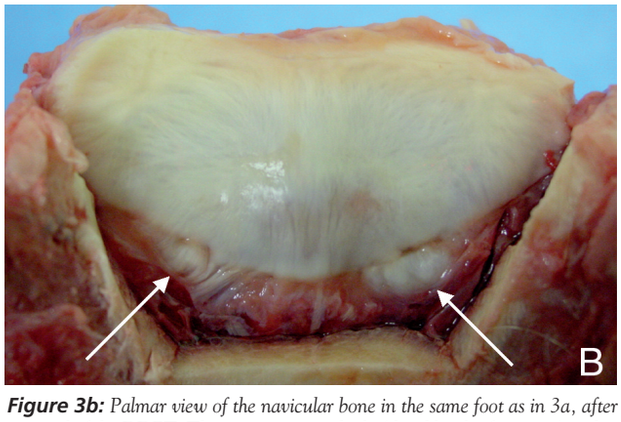

Figure 3b: *Palmar view of the navicular bone in the same foot as in 3a, after removal of the DDFT. There is an osseous body (distal border fragment) within the insertion of the DSIL at each angle of the distal border of the navicular bone with the sloping borders.*

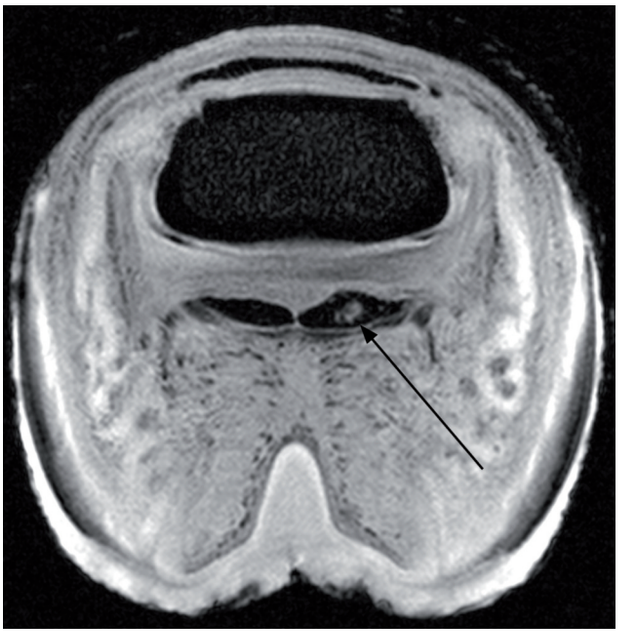

Figure 4: *Transverse T1-weighted fast low-angle shot (FLASH) image with fat saturation of the right front foot at the level of the middle phalanx of a horse with acute onset foot lameness. The lateral lobe of the DDFT is enlarged and contains a core lesion characterized by a central, circular area of signal hyperintensity (arrow) surrounded by normal, low intensity, tendon signal.*

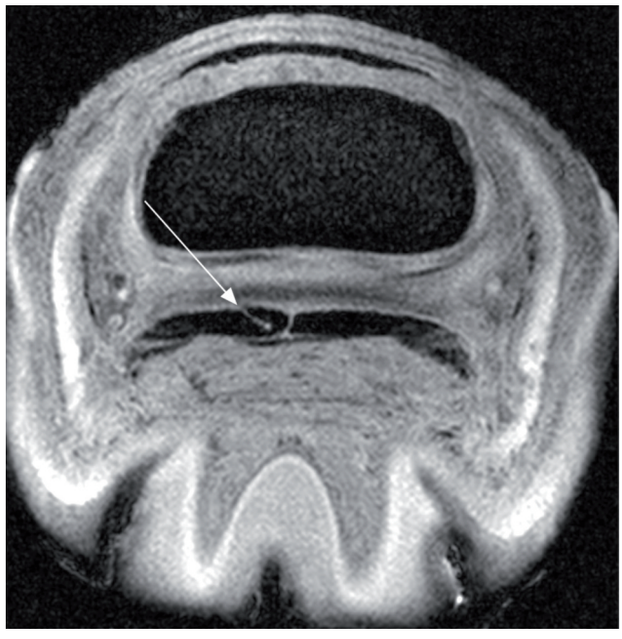

Figure 5: *Transverse 3D spoiled gradient echo fast low-angle shot (FLASH) with fat saturation of the left foot at the level of the navicular bone. There is a hyperintense parasagittal linear defect that extends from the dorsal to the palmar surface of the lateral lobe of the DDFT.*

Sagittal plane or oblique splits form linear hyperintensities of variable depth arising from the dorsal surface of the tendon and progressing palmarly ***(figure 5)***. Histologically, disruption of the deep dorsal layer of the tendon by deep splits extending from the surface can be observed (Murray *et al.* 2009). Splits propagate mostly along septal lines, with chondrones clustered around fibrillating tissue and around crevices but no evidence of inflammatory cells (Blunden *et al.* 2006 b).

Insertional lesions are limited to the distal 20 mm of the DDFT, distal to the distal border of the navicular bone, near the tendon's insertion on the distal phalanx ***(figure 6)***. They consist of small core lesions, sagittal plane splits or osseous changes of the insertion site (Blunden *et al.* 2009).

Severe dorsal border abrasions of the DDFT usually cause signal increase extending from the dorsal surface towards the center of the affected lobe ***(figure 7)***. Histopathologically, dorsal DDFT fibrillations, erosions and abrasions consist of longitudinal strips of superficial fiber damage extending the proximodistal length of the navicular bursa. Bundles of fibers torn away from the surface of the tendon have a tendency to curl up proximally in the navicular bursa. Crevices, splits and fibrillations of the dorsal border of the DDFT may be accompanied by either small, discrete or large, circumscribed foci of necrosis (Wright *et al.* 1998). Chondrocyte clusters can be seen to produce chondroid matrix in areas of degenerative change (Blunden *et al.* 2009).

Degenerative vascular changes consisting of thrombosis and occlusion of septal arteries and veins are also seen in the distal portion of the DDFT. They were initially described with equal frequency in horses with clinical navicular disease and age-matched control horses (Wright *et al.* 1998). Although it was suggested that these vascular changes could be age-related (Wright *et al.* 1998), larger and later pathological studies have found that vascular changes in the DDFT were significantly more common in the DDFT of horses with foot pain (Busoni *et al.* 2005; Murray *et al.* 2009; Blunden *et al.* 2006 b; Blunden *et al.* 2009). It was therefore proposed that vascular thrombosis and occlusion could result in matrix changes that predispose horses to injury of the distal portion of the DDFT (Blunden *et al.* 2006 b). As these changes are predominantly seen in the intratendinous septa, there is a strong possibility that they predispose to the development of sagittal splits in the dorsal surface of the tendon along these septal planes. The lack of any histological evidence of hemorrhage or inflammatory cell infiltration in core lesions, splits and abrasions of the distal portion of the DDFT adds further support to the notion that these lesions may be primarily degenerative in nature (Busoni *et al.* 2005; Blunden *et al.* 2009).

MR signal intensity varies between different echo sequences not only with severity but also with duration of a tendon lesion. Acute lesions generally have higher T2 signal intensity due to the presence of fluid and increased cellularity, whereas fibrous scar tissue in chronic lesions produces a more intermediate to low T2 signal intensity. It may therefore be possible to use T1-to-T2 signal differences to estimate the age or stage of healing of a tendon lesion (Schramme *et al.* 2010). In the chronic stages of healing by fibroplasia, signal intensity in core lesions generally decreases in T2

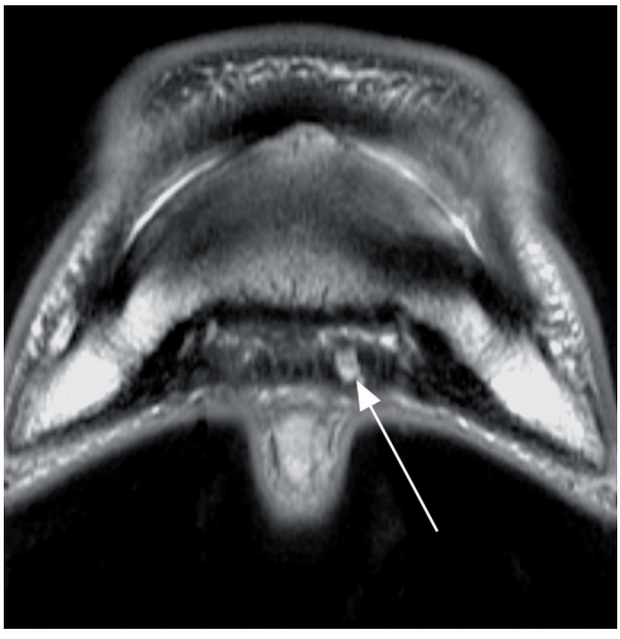

Figure 6: *Sagittal 2D proton density echo of the left foot of an 8-year-old Quarterhorse with acute onset left forelimb lameness. There is enlargement of the distal part of the DDFT and a hyperintense core lesion near the insertion (white arrow) to the distal phalanx.*

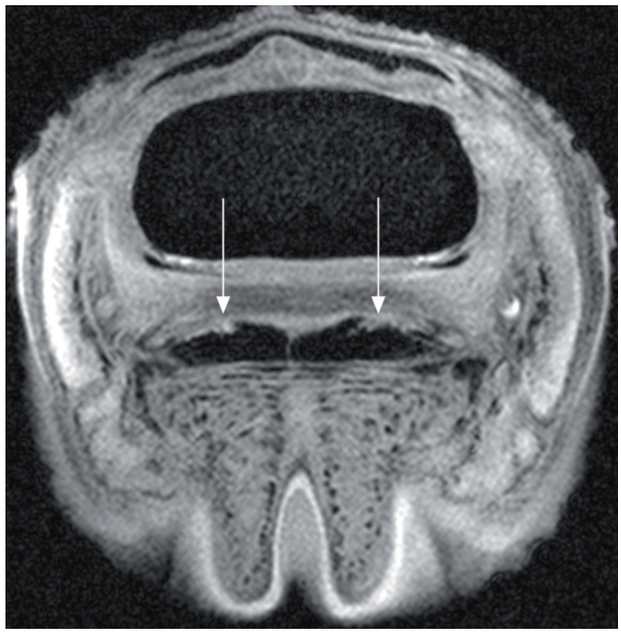

Figure 7: *Transverse 3D spoiled gradient echo fast low angle shot (Flash) with fat saturation of a left foot at the level of the navicular bursa. Two marginal erosions are present in the dorsal surface of the DDFT (white arrows) corresponding with superificial areas of fibrillation and fiber loss.*

and STIR images but can remain high in T1-weighted sequences. It was suggested that T2-weighted imaging may be more sensitive for detecting the transition of blood-rich immature granulation tissue to fibrous scar tissue (Fujikawa *et al.* 2007).

NAVICULAR BURSA

Villous hypertrophy, hyperplasia of synovial cells and venous congestion have been described in the navicular bursa of horses with navicular disease (Murray *et al.* 2009; Blunden *et al.* 2006 a; Svalastoga & Neilsen, 1983). There was a positive association between histological abnormalities of the bursa and lesions of either the dorsal aspect of the DDFT or the flexor aspect of the navicular bone.

DISTAL SESAMOIDEAN IMPAR LIGAMENT (DSIL)

Ageing changes have been described in the impar ligament, characterised by change in fibroblast shape and increased proteoglycan content (Bowker *et al.* 2001). Evidence of inflammation was also seen at the intersection of the DSIL and DDFT in horses with navicular syndrome (Bowker 2003). The functional significance of these findings remains unknown. Using MRI, injury to the impar ligament is generally seen as part of an injury complex that includes other components of the navicular apparatus (Dyson & Murray, 2007). One histopathological study limited the histological grading of impar ligament abnormality to the presence and extent of fibrocartilaginous metaplasia, that was more extensive in diseased than control limbs (Blunden *et al.* 2006 a). In a more recent report, the histological changes in the mid body of the impar ligament included degeneration of collagen, loss of fibroblasts, fibrocartilage metaplasia and reduction in vascularity. These changes were well correlated with MR abnormalities at the origin and the insertion of the impar ligament, specifically the presence of a cystic structure in the distal third of the navicular bone, one or more distal border fragments, the presence of entheseous new bone, or increased signal intensity in fat suppressed images at the insertion of the impar ligament on the distal phalanx (Blunden *et al.* 2009). Abnormal MR signal in the body of the impar ligament on the other hand, was not associated with the presence of histological abnormalities.

COLLATERAL LIGAMENTS OF THE DISTAL INTERPHALANGEAL JOINT

MR descriptions of injuries to the collateral ligaments of the distal interphalangeal joint and associated osseous abnormalities are well documented (Dyson *et al.* 2004; Dakin *et al.* 2009). A good agreement has been reported between MR and histopathological findings in collateral ligaments of 25 horses with palmar foot pain (Dyson *et al.* 2008). Lesions appeared to be degenerative, characterised by extensive fibrocartilaginous metaplasia and development of multiple, intercommunicating fissures within the degenerate collagen in severe lesions.

COLLATERAL SESAMOIDEAN LIGAMENTS (CSL)

The presence of entheseous new bone on the proximal border of the navicular bone, reflecting previous insertional desmopathy of the CSL is well documented radiographically and histopathologically, in both clinically normal horses and horses with navicular disease (Pool *et al*. 1989; Verschooten *et al*. 1989). Fibrocartilaginous metaplasia has been described in the body of the CSL but there was no difference between horses with navicular disease and control horses (Blunden *et al*. 2006 a).

In conclusion, the increasing number of available pathological studies on the various tissues of the foot has helped validate the significance of MR signal variations documented with various clinical MR systems used in clinical equine practice. It has brought to light a number of injuries involving structures in the foot other than the navicular bone, that had previously not been recognized as important causes of lameness, including tendinopathy of the DDFT, desmopathy of the CSL, the impar ligament, the collateral ligaments of the distal interphalangeal joint, and bone bruising of the distal and middle phalanx. In addition MRI has helped the clinician understand the different pathological processes occurring in the navicular bone. This new understanding of various new causes of lameness in the horse's foot has resulted in changing treatment strategies in the management of foot lameness. However, further work is needed to continue to improve our understanding of the different causes of foot lameness and their pathogenesis.

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