

Learn about laminitis

Chapter 3 – Anatomy of the equine foot

In order to understand how laminitis affects the foot, we need to start by taking a look inside a healthy horse foot. We can probably best identify with what is going on inside a horse’s foot, if we compare it to our own. We share a similar internal anatomy with the horse, with the exception of one major difference. While we have five digits per limb (five toes per foot) and use our toes to balance, most of our weight is placed on the sole of our foot and our heel. Horses have all their weight on only ONE digit per limb, which would be like us walking on the very tip of only one of our toes! Surprisingly, what we often think of as the horse’s knee is actually equivalent to our wrist, while their hock is our ankle! A comparison of human and horse anatomy is shown in Figure 1 below. The same colours are used to highlight bones that are the same between the two species.

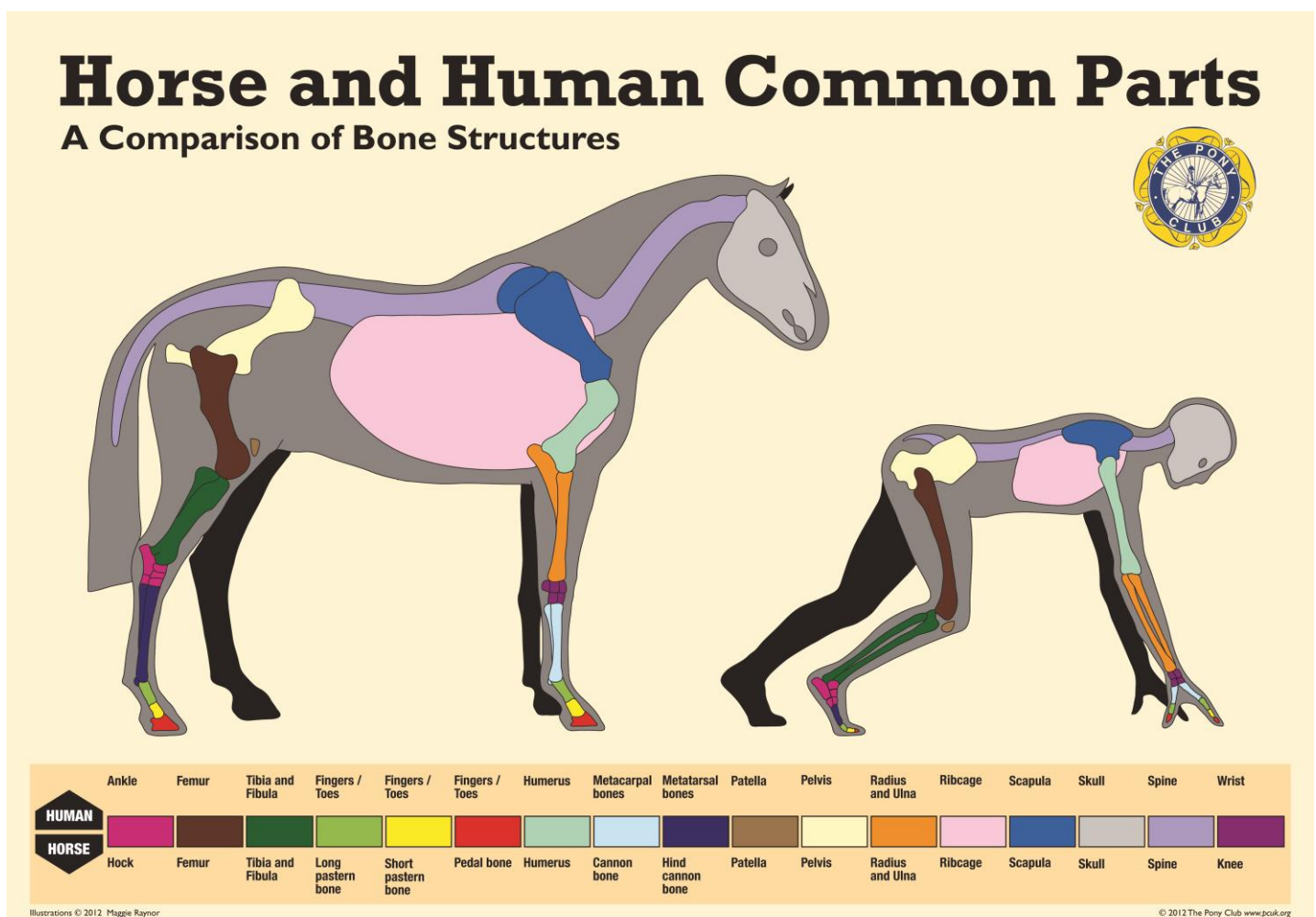


Fig. 1 Comparing the human and equine skeleton (with kind permission from The Pony Club). If you would like to purchase this handy wall chart, visit <http://shop.pcuk.org/wallcharts/horse-and-human-common-parts.html>

Humans are bipedal, upright-walking creatures. We can completely rest our two weight-bearing limbs by sitting or lying down for long periods of time without any adverse effects. In contrast, horses distribute their weight over four limbs that undergo almost constant forces of impact and weight-bearing and they cannot spend any great length of time ‘resting’ their feet. This has important implications when their feet

are compromised, crippling the animal and resulting in constant and debilitating pain. Let us examine the anatomy of the horse foot in more detail.

The hoof wall

Much as the tips of our fingers and toes are protected by our nails, the sensitive structures and soft tissues within the horse's foot are protected by a thick, hard hoof wall. Similar to our nails, the hoof wall consists of a protein called keratin, which gives it strength and rigidity, but also a certain degree of flexibility and elasticity. Like our nails grow from the nail bed, there is continual growth of the hoof wall at the coronary band. The average growth rate is 8 to 10 mm per month, meaning it takes between 9 and 12 months for the whole hoof to be replaced. Young cells, continually produced at the coronary band, are pushed towards the ground surface as they mature – resulting in the formation of long hair-like tubules running along the length of the hoof wall from the coronary band to the ground. The further away from their origin that these cells move, the more filled with keratin and "dead" they become, developing hollow centres. The same process occurs in the sole and frog regions of the foot, with continual growth of these tubules towards the ground. The space between the tubules is filled with a keratin-rich network of cells that run at right angles to the tubules and provide the bulk of the hoof wall strength. Thus, the horse foot is enclosed by a pigmented, non-living outer hoof wall. The hoof wall is most rigid on the outside and becomes more elastic and more "living" further towards the inside of the foot, where it meets the lamellar region (Fig. 2).

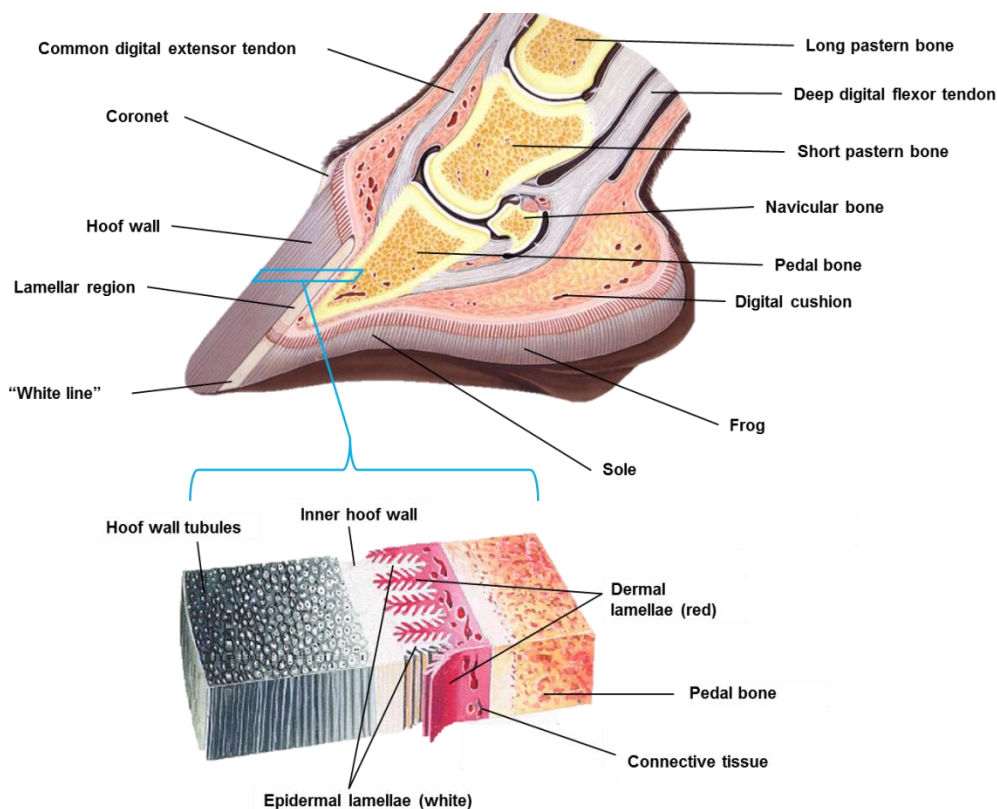


Fig. 2 The anatomy of the equine foot with an inset of the structure of the hoof wall and lamellar region (adapted from Pollitt, 2008).

The lamellar region - 'an in-depth look'

The lamellar region can be divided into the *epidermal* and *dermal* lamellae, referred to as the epidermal and dermal layers (Fig. 2). The epidermal layer is located nearest the hoof wall tubules and is part of the inner hoof wall. Much as the hoof wall next to it, this epidermal layer is 'insensitive' in that it does not have a blood supply or nerve system and relies on the next-door 'sensitive' dermal layer for supply of nutrients and oxygen. Thus the 'insensitive' layer protects and covers the neighbouring 'sensitive' layer, found nearer the pedal bone. The epidermal layer is made up of folded, leaf-like structures called epidermal lamellae.

These epidermal lamellae include parallel rows of **primary** epidermal lamellae, each of which has many finger-like extensions. These extensions are called **secondary** epidermal lamellae (Fig. 3).

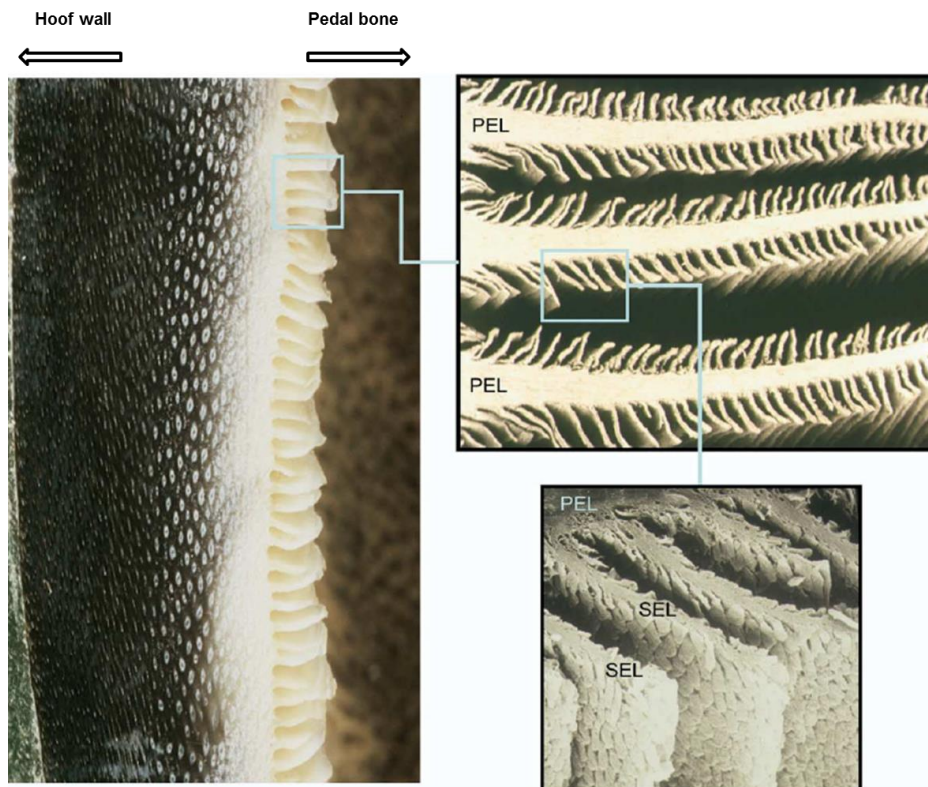


Fig. 3 A microscopic image showing the folded leaf-like epidermal lamellae and how they consist of rows of parallel primary lamellae (PEL) with finger-like secondary lamellae (SEL) extending from them (below) (Pollitt, 2008; 2004).

The **secondary** epidermal lamellae greatly increase surface area, allowing for greater exchange of nutrients and oxygen from the dermal to the epidermal layers, and increase the strength of the lamellar region. The spaces between them are snugly packed by a complementary 'mirror-image' layer of **secondary** dermal lamellae, which are finger-like projections that extend off the **primary** dermal lamellae. The **secondary** dermal lamellae and **primary** dermal lamellae together make up the dermal layer. The epidermal and dermal layers interlock at the important interface called the basement membrane (BM). This thin but tough sheet of tissue is what connects the structures of the inner hoof wall to the connective tissue on the surface of the pedal bone via specialised connections, or 'anchorage sites' called hemidesmosomes. This mechanism is what suspends the pedal bone in the foot within the hoof capsule (Fig. 4).

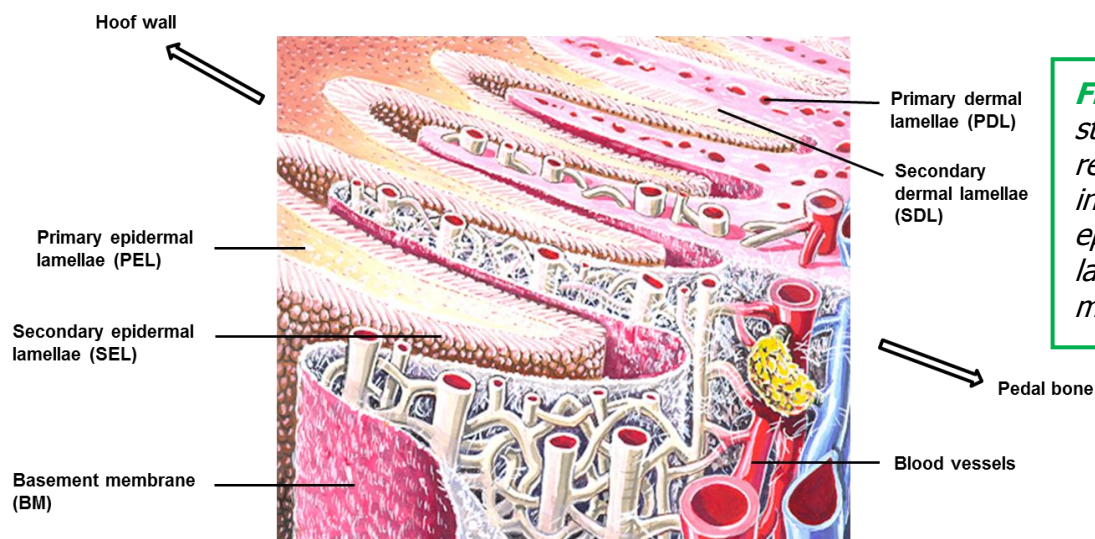


Fig. 4 A diagram of the structure of the lamellar region, showing the interface between the epidermal and dermal layers at the basement membrane (above).

How does the hoof wall grow?

As discussed earlier, the hoof wall grows continuously at the coronary band to replace the hoof lost at the ground surface. As the parallel layers of **primary** epidermal lamellae are connected to the inner hoof wall and undergo the same renewal of cells near the coronary band, they are believed to undergo the same downward growth. However, the finger-like extensions of the **secondary** epidermal lamellae are firmly anchored to the BM via the specialised anchorage-sites (hemidesmosomes) and must remain in the same place in order to keep supporting the pedal bone within the hoof capsule. So how do the **primary** epidermal lamellae move, while the **secondary** don't... and how do they stay attached?

Closely studying hoof wall growth has revealed that new cell growth is almost entirely absent from the middle region of the epidermal layer and mainly occurs at two growth zones – one being the coronary band and a second growth zone at the white line and sole (nearest to the ground). Therefore, it is thought that the downward growth of the hoof wall and **primary** epidermal lamellae, past the stationary **secondary** epidermal lamellae, occurs via a 'sliding' action where connections between cells of the two are continuously broken and reformed. Think of two pieces of Velcro detaching and reattaching a few hooks at a time – one remaining in the same place while the other moves. This allows the hoof wall to grow in length, while at the same time ensuring the constant suspension of the pedal bone within the hoof capsule. This detachment and reattachment event is termed 'remodelling' and is under the control of a number of specialised molecules whose production is usually under strict regulation. If produced in uncontrolled quantities, these molecules have the potential to interrupt this important remodelling process leading to failure in normal lamellar function. Any changes to the structure of the BM and the lamellar layers affect the strength and normal function of the foot and have been proposed in the pathophysiology of laminitis. Exactly how this process is triggered is as yet not fully understood but is thought to be influenced by factors including hormonal imbalances (particularly elevated insulin levels in blood), systemic (body-wide) inflammation, changes in circulation and mechanical trauma or overloading.

Next time we will take a look at the different 'types' of laminitis that are currently recognised.



This chapter is dedicated to the memory of Dr Simon Collins, who very sadly passed away. The CARE team is very grateful for his help with this project as well as his work with the initial RVC-AHT study into equine laminitis frequency and risk factors. His insight into the anatomy of the equine foot and laminitis will be greatly missed.

References

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