LAMINITIS AND HOOF HORN HEALTH

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INTRODUCTION

Aside from lameness associated with digital dermatitis, the majority of lameness in cattle is the result of the production of hoof horn of inferior quality. Subclinical laminitis has been considered to be the most important underlying cause of inferior hoof horn production. As more is learned regarding hoof horn production it has become evident that the term "subclinical laminitis" does not always describe events that lead to production of low quality hoof horn. Recently, the term "claw horn disruption" has been proposed as more accurately describing the events that occur (1). Claw horn disruption is considered to be of multifactorial etiology. Feeding management, nutrition, parturition, other diseases, cow comfort and environment are risk factors, involved in claw horn disruption.

ANATOMY AND PHYSIOLOGY

Hoof horn is modified epidermis that is arranged into tubular and intertubular horn. Both consist of cells and an intercellular substance known as membrane cementing material. Hoof horn is formed by the innermost and only living layers of epidermis. Within the horn-producing cells, keratin proteins that provide strength to horn are arranged into fibers that are bound together by disulfide bonds. As epidermal cells mature they are pushed away from their nutrient source and eventually die producing cornified horn. Hoof horn of the wall is produced in the coronary segment at a rate of approximately 5 mm per month (2). Hoof horn formed by papillae in the sole typically reaches the weight-bearing surface 2-3 months after it is produced.

The white line forms a hinge-like junction between the rigid wall and the somewhat more flexible sole. The white line consists of horn formed from 3 segments of the wall, namely cap horn, terminal horn, and laminar horn leaflets. Horn leaflets are the hardest horn of the white line and are produced in the coronary-wall transition region. Cap and terminal horn portions are produced distally and are relatively soft and pliable. They constitute a filling horn substance that occupies the space between the horn leaflets (3).

The dermis layer in the hoof is known as the corium or pododerm. The pododerm, like dermis elsewhere, contains blood vessels and nerves. Anteriorly, there is no subcutaneous space between the sole and PIII. Under the posterior aspect of PIII in the region of the flexor tuberosity there is subcutaneous tissue which permits some cushioning as the animal walks. Hoof horn produced in the coronary segment of the wall "slides" down the dermal leaflets as it grows toward the weight-bearing surface. One function of the laminae, which are in reality folds, is to increase contact area between the hoof horn and underlying pododerm. In this way the weight of the cow is transferred from the skeleton to the ground by way of the laminae (4).

PATHOPHYSIOLOGY

The pathophysiology that results in the production of inferior quality hoof horn is not completely understood. With subclinical laminitis, it is generally accepted that there occurs a vascular disturbance in the corium mediated by vasoactive substances such as histamine and endotoxins. Thus, at the cellular level there is an interruption of nutrient and oxygen supply. This in turn causes the keratin-producing cells to produce hoof horn of inferior quality. A relative deficiency of certain nutrients, eg. biotin, zinc, calcium could also lead to a similar problems in horn quality. With subclinical laminitis, episodes are thought to be repetitive and an initial bout may increase the likelihood of subsequent episodes (5). The theory of "burdening of the pododerm" or overgrowth of the sole proposes that repeated episodes result in height differences between the inner and outer claws of the hindlimb (6). The increase in height results in the outer claw bearing an even greater proportion of the weight than it does normally and thus becoming even more susceptible to pathologic changes. Others have indicated that as a result of inflammation of the pododerm there is separation between dermal and epidermal layers resulting in a positional change in PIII. This causes more pressure-induced hemorrhage and necrosis of the pododerm or corium of the sole (7).

LESIONS

Hoof horn lesions are the result of the cellular response to insults to the keratin-producing cells. Lesions observed are not necessarily specific as to cause and may include:

- Yellow discoloration of the sole – Yellow discoloration indicates intercellular material escaping from an inflamed corium and is associated with soft, poor-quality horn.

- Hemorrhages of the sole and sole ulcer – Hemorrhages of the sole represent a continuum that ranges from those that are barely perceptible to severe ulceration with exposed corium (8). The most common site for sole ulcers is the rear lateral claw under the flexor tuberosity of PIII. Research has shown that cattle that have ulcers in one lactation are predisposed to having them in subsequent lactations (9).

- Separation of the white line – Electron microscopic studies found that low-quality horn preceded the appearance of sole hemorrhages and white line disease by several weeks (4). An early indication of disease is widening of the white line. When the white line widens it becomes softer. The striated appearance that may be present in the white line is the result of softer cap and terminal horn crumbling and falling away leaving horn leaflets remaining.

- Heel horn erosion – Some believe that not only is heel erosion part of the subclinical laminitis syndrome but that heel erosion also increases the subsequent risk of sole ulcer (6). Others have proposed that although heel horn erosion results primarily from bacterial infection the subclinical laminitis process may be indirectly involved as a consequence of the production of horn of inferior quality (10).

- Other lesions – Other lesions include abscess of the sole and white line, sole overgrowth, inflamed coronary band, deformed claws, horizontal grooves in hoof horn, sunken or rotated distal phalanx, overgrown claws and double sole.

RISK FACTORS

Environment, nutrition, feeding management, parturition, other management factors, other foot diseases, and genetics have all been implicated as risk factors for hoof horn disruption. Interactions among the various risk factors have been found. There is general consensus that causation is multifactorial, with differences occurring among herds in the relative importance of a particular factor.

- Parturition – Periparturient stress, dietary changes, hormonal changes and increased risk of acute systemic disease have all been implicated in the pathogenesis of subclinical laminitis and other forms of hoof horn disruption. The first parturition appears to be one of particularly high risk.

- Nutrition – Implicated at one time or another have been excess energy, inadequate fiber, excess protein, vitamin deficiencies including that of a relative deficiency of biotin, deficiency of minerals and amino acids.

- Feeding management – Feeding diets not balanced for carbohydrates, diets with small particle size, and rapid changes to high concentrate feeding after parturition may all be important. These practices often chronically decrease rumen pH to less than 5.6. Virtually all agree that feeding cattle to maintain a stable rumen pH above about 6 is desirable and that acclimation of the rumen to feed changes is important.

- Environment – Excessive standing or inadequate resting have been implicated. Type of housing facilities, bedding materials, floor surfaces, and manure management have all been considered important.

- Other factors – Cows that had been treated for metabolic, digestive, and reproductive disorders have been shown more likely to develop sole ulcers (9). Foot and leg conformation, claw conformation, and claw size have been mentioned as possible important individual animal or genetic factors related to the occurrence of subclinical laminitis. Improper foot trimming techniques may result in traumatic laminitis (11).

CORRECTIVE STRATEGIES

- **Environment** – Cow comfort; clean and dry facilities should be stressed.

- **Reduction of disease in periparturient period** – Institute accepted practices designed to reduce metabolic and infectious diseases during this high-stress period.

- **Feeding management and nutrition** – Avoid acidosis. Research is unclear on the severity of acidosis required to cause an episode of subclinical laminitis or the degree of interaction with environment, cow-comfort, and other causative factors.

- **Hoof trimming** – Restoration and maintenance of normal anatomic relationships may disrupt the cycle of self-perpetuating changes associated with hoof horn disruption. This can best be achieved through a regular program of functional hoof trimming.

Data reported to date suggest that multiple factors are likely to be involved in causation of hoof horn disruption. The relative importance of a particular factor may be expected to differ from herd-to-herd. Therefore, although certain basic concepts should be universally adopted by producers, specific intervention strategies will be expected to vary from herd-to-herd.

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