

Importance of Minerals and Protein for Bone Formation in Horses

The formation of functional bone in young horses requires the delivery of the right material in the right proportions. If any factor is disturbed, the possibility of poorly formed or malformed bone exists.

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The three major components of bone—calcium, phosphorus, and protein—must be made available at the site of bone formation. A severe or prolonged deficiency of calcium or phosphorus can limit the availability of these mineral substrates of bone. Bone protein (osteoid) is manufactured at the site of bone formation, so unless deprivation is severe, it is rarely a cause of depression of osteoid formation. A more frequent cause of malformation of bone is an interruption of the delivery of these nutrients due to disruption of the blood supply. This interruption may result from traumatic, infectious, and possibly even metabolic causes. It is usually localized and therefore creates a localized disruption of bone formation.

Any obstruction of the vascular supply to the growing bone will result in the lack of transformation of the degenerating cartilage to calcified cartilage and eventually to bone. Vascular obstruction can result in the interruption of bone formation locally even though the body-wide supplies of nutrients may be plentiful. The most common localized vascular obstruction is fibrous tissue left behind after resolution of an infectious process. A hematogenous shower of bacteria, as is frequent in a foal, can create multiple potential sites of disturbed bone formation simultaneously. Trauma can also strike at any site or in multiple sites.

The delivery of minerals in the right proportions is necessary for the hydroxyapatite crystal to be formed and deposited within the osteoid matrix. The delivery of calcium and phosphorus at about 1.7 to 1 is required to form bone. The body can fine-tune needs as long as reasonable proportions and quantities are fed. The most well-known disparity in calcium-phosphorus balance is secondary nutritional hyperparathyroidism caused by feeding excess phosphorus and deficiencies of calcium. The resultant robbing of the skeleton of the calcium necessary for the more critical metabolic needs results in an inability to reform the hydroxyapatite crystal after routine bone remodeling. This gradually reduces skeletal content of hydroxyapatite leaving the osteoid without mineral to stiffen its structure. The more rapid remodeling in the flat bones make the clinical signs apparent in the jaw first, but in the growing animal, osteochondritis dissecans (OCD) can also result.

Due to current owner awareness, nutritional secondary hyperparathyroidism is unusual. What is occasionally seen in the current management systems is absolute calcium deficiency due to a relatively low level of both nutrients. Absolute deficiencies in calcium in the presence of normal phosphorus in foals is most common and will result in poor bone strength seen as an increase in fractures of the more trabecular sesamoid and third phalangeal bones as well as physitis at the most active, most vulnerable physes at the time. Therefore, clinical signs referable to deficiencies in structural integrity of cancellous bone should suggest an examination of the available levels of calcium and phosphorus.

The correct cofactors for bone formation are still largely speculative. Copper, zinc, and vitamins A, C, and D are proven cofactors, but manganese and other nutrients may be needed in small amounts.

Horsemen are very aware of the need for [vitamins](#), but are unaware, in most instances, of the horse's ability to provide these vitamins when supplied with sunshine and pigmented roughage. Copper and zinc appear to help protect the quality of newly formed bone in the foal. Copper supplementation is most effective when trying to prevent the formation of poor-quality bone such as with phytitis and less effective with retarded cartilage mineralization.