Bone – Increased Osteoid

**Figure Legend:** Figure 1 Bone - Increased osteoid in a male F344/N rat from a chronic study. There is deposition of unmineralized osteoid along the existing trabeculae. Figure 2 Bone - Increased osteoid in a male F344/N rat from a chronic study (higher magnification of Figure 1). Increased numbers of osteoblasts are evident along the surface of new bone formation (arrows).

**Comment:** An increase in the osteoid (previously called osteomalacia) is characterized by an increase in the surface area of bone by the accumulation of osteoid (Figure 1 and Figure 2). There may be widened seams along existing surfaces of bone. Defective mineralization results in an accumulation of homogeneous, unmineralized osteoid. Increased osteoid has been induced in rats by various compounds that inhibit mineralization of osteoid, including bisphosphonates, aluminum, cadmium, phosphorus deficiency, and vitamin D toxicity. It may also be observed as new bone is rapidly being formed; increased osteoid secondary to rapid new bone formation should be differentiated from a primary defect in mineralization or inhibition of osteoclastic bone resorption.

The term “osteomalacia” has historically been used to describe failure of newly formed osteoid to mineralize. However, when describing the above lesion, the term “increased osteoid” is preferred over “osteomalacia,” since the latter denotes a gross morphologic change of bone (softening).

Increased osteoid should not be used as a diagnosis for delayed or inadequate mineralization of physeal or epiphyseal cartilage when occurring at the growth plates. This is more appropriately recorded as physeal dysplasia (rickets) and differs from increased osteoid by the presence of thickened
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cartilaginous growth plates and retained cartilage cores within the metaphysis (see “Bone - Physeal Dysplasia”) rather than excess osteoid.

**Recommendation:** When present, increased osteoid should be diagnosed and given a severity grade. Increased osteoid should not be diagnosed when it is secondary to increased bone, but it can be described in the pathology narrative as a component of increased bone.

**References:**


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