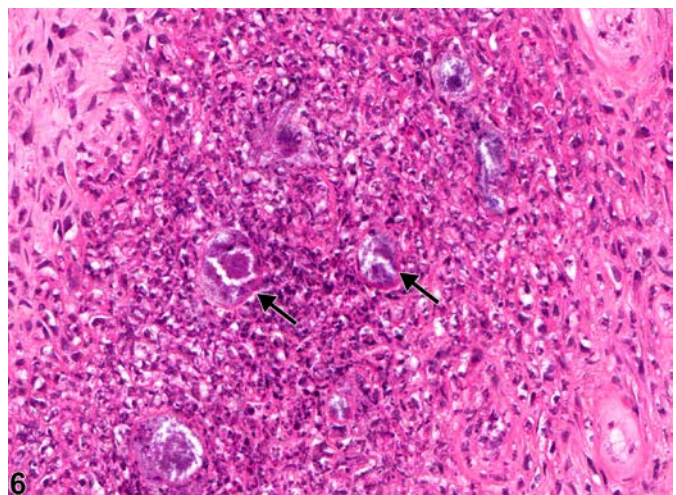
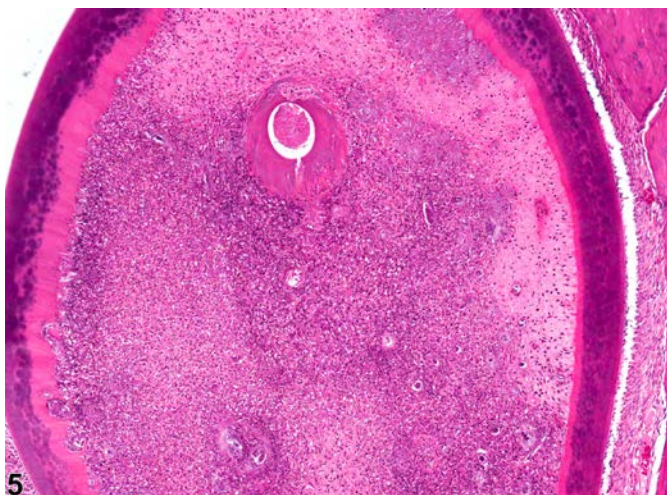
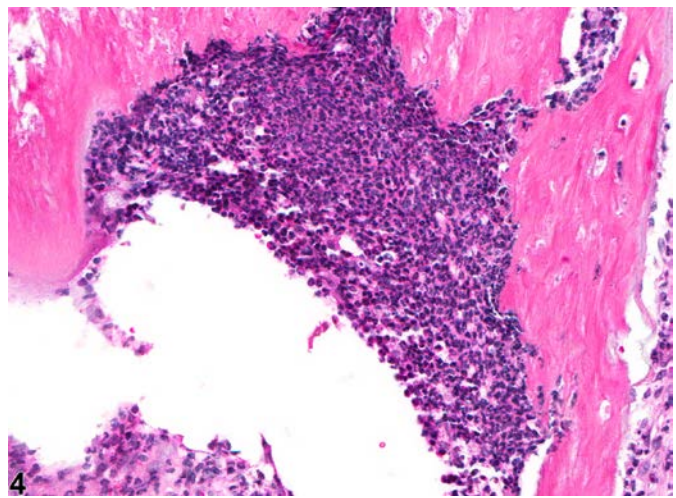
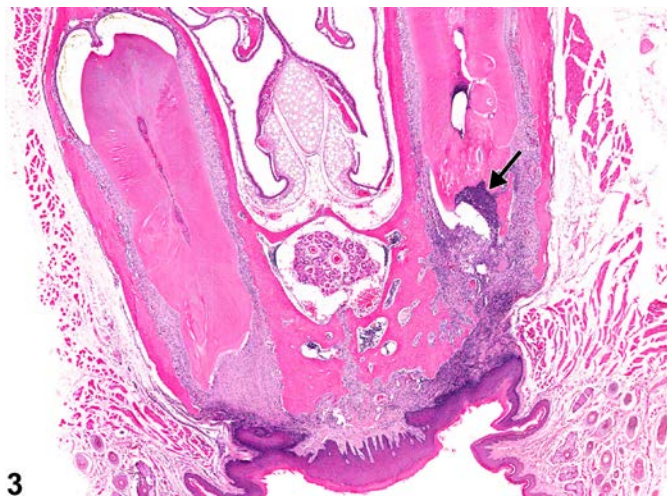
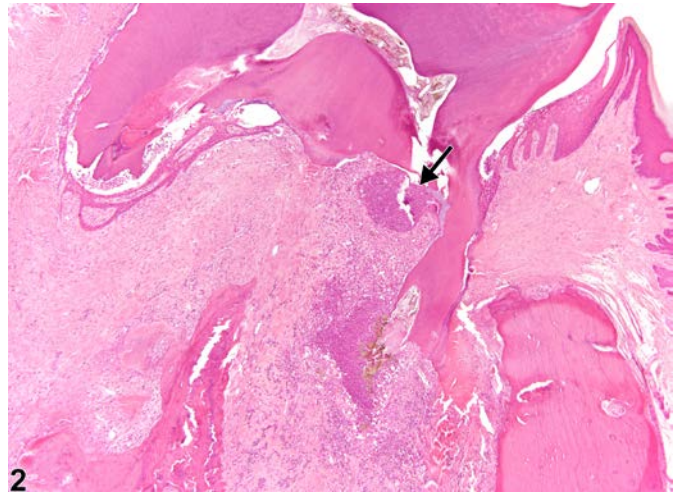
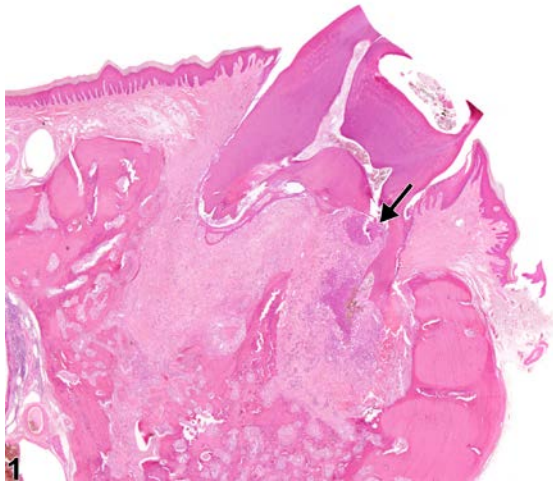
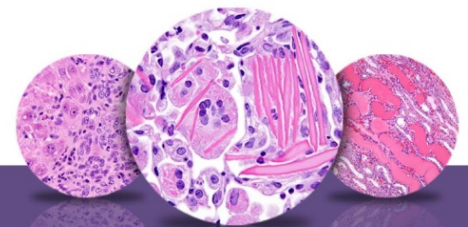




NTP Nonneoplastic Lesion Atlas

Tooth – Inflammation





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Figure Legend: **Figure 1** Tooth - Inflammation, Chronic active in a male Harlan Sprague-Dawley rat from a chronic study. Cementum and dentin of the root are being resorbed because of chronic active inflammation (arrow). **Figure 2** Tooth - Inflammation, Chronic active in a male Harlan Sprague-Dawley rat from a chronic study (higher magnification of Figure 1). Cementum and dentin of the root are being resorbed because of chronic active inflammation (arrow). **Figure 3** Tooth - Inflammation, Chronic active in a male B6C3F1 mouse from a chronic study. Chronic active inflammation of the tooth is present (arrow). **Figure 4** Tooth - Inflammation in a male B6C3F1 mouse from a chronic study (higher magnification of Figure 3). Chronic active inflammation of the tooth is present. **Figure 5** Tooth, Pulp - Inflammation, Suppurative in a male F344/N rat from a chronic study. Suppurative inflammation affects the majority of the tooth pulp. **Figure 6** Tooth, Pulp - Inflammation, Suppurative in a male F344/N rat from a chronic study (higher magnification of Figure 5). There is suppurative inflammation of the pulp with colonies of bacteria (arrow).

Comment: In NTP studies, there are five standard categories of inflammation: acute, suppurative, chronic, chronic active, and granulomatous. In *acute inflammation*, the predominant infiltrating cell is the neutrophil, though fewer macrophages and lymphocytes may also be present. There may also be evidence of edema or hyperemia. The neutrophil is also the predominant infiltrating cell type in *suppurative inflammation*, but the neutrophils are aggregated, and many of them are degenerate (suppurative exudate). The exudate may also contain cell debris, both from the resident cell populations and from infiltrating leukocytes; proteinaceous fluid containing fibrin; fewer macrophages; occasional lymphocytes or plasma cells; and, possibly, an infectious agent. Grossly, these lesions would be characterized by the presence of pus. Fibroblasts, fibrous connective tissue, and mixed inflammatory cells may be present in the tissue surrounding the exudate, depending on the chronicity of the lesion. Lymphocytes predominate in *chronic inflammation*. Lymphocytes also predominate in *chronic active inflammation*, but there are also a significant number of neutrophils. Both lesions may contain macrophages. *Granulomatous inflammation* is another form of chronic inflammation, but this diagnosis requires the presence of a significant number of aggregated, large, activated macrophages, epithelioid macrophages, or multinucleated giant cells.

Inflammation of the tooth can involve the pulp, the cementum, the dentin, or any combination of these and can extend to the periodontal tissues. Chronic tooth inflammation can result in tooth resorption.



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Tooth – Inflammation

Tooth inflammation is most often caused by bacteria but can be the result of foreign bodies in the oral cavity. If a bacterial infection gains access to the dentin tubules, it can spread throughout the dentin and into the tooth pulp. Because of its enclosure within the dentin, swelling of the tooth pulp due to inflammation can result in increased intrapulpal pressure, decreased blood flow, and ischemia. Caries is a gross term for inflammation of the calcified portions of the tooth. Dental caries are not common in mice or rats, but high levels of carbohydrates (e.g., sucrose or xylitol) in the diet can induce their formation. Tooth inflammation can be a background finding in animals with dental injury.

Recommendation: Whenever present, inflammation of the teeth should be diagnosed and graded based on the extent of tooth involvement, the density of the inflammatory infiltrate, and the number of teeth affected. The type of inflammation (e.g., acute, suppurative, chronic active) should be included in the diagnosis as a modifier. If the tooth pulp only is involved, the term “pulp” should be added to the diagnosis as a site modifier (i.e., “tooth, pulp - inflammation”). If the inflammation involves the dentin and/or cementum, or if the dentin and/or cementum is involved in addition to the pulp, the diagnosis should be “tooth – inflammation.” Associated necrosis should not be diagnosed separately unless it is considered to be a primary lesion (not secondary to the inflammation) or if it is severe enough to warrant a separate diagnosis.

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Authors:

Linda H. Kooistra, DVM, PhD, DACVP
Pathologist
Charles River Laboratories, Inc.
Research Triangle Park, NC



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Tooth – Inflammation

Authors:

Abraham Nyska, DVM, Diplomate ECVP, Fellow IATP
Expert in Toxicologic Pathology
Visiting Full Professor of Pathology
Sackler School of Medicine, Tel Aviv University
Timrat Israel