

NTP Nonneoplastic Lesion Atlas

Stomach, Glandular Stomach – Amyloid

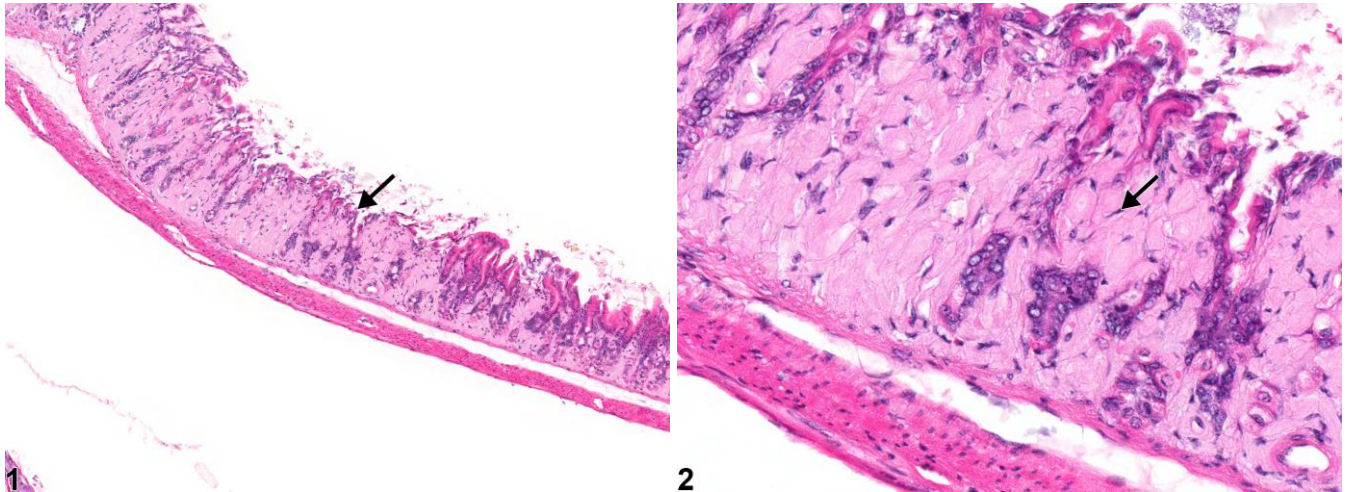
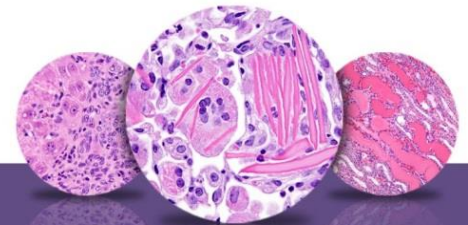


Figure Legend: **Figure 1** Stomach, Glandular stomach - Amyloid in a female Swiss Webster mouse from a chronic study. Amyloid (arrow) is deposited in the lamina propria. **Figure 2** Stomach, Glandular stomach - Amyloid (arrow) in a female Swiss Webster mouse from a chronic study (higher magnification of Figure 1). The deposition of amyloid in the lamina propria results in the loss of glandular epithelial cells.

Comment: Amyloid deposition (amyloidosis) is a systemic disease that is rare in B6C3F1 BALB/c, and C3H mice but common in CD-1, A, Swiss Webster, SJL, and B6 mice and can be a cause of death. The incidence can be increased in association with fighting among group-housed males and with ectoparasitism. Amyloid appears as an amorphous, eosinophilic, hyaline, extracellular substance that, with progressive accumulation, results in pressure atrophy of adjacent cells and tissue. Generalized (systemic) amyloid deposition can be seen in many tissues, including gastrointestinal tract, spleen, liver, kidney, tongue, and mesenteric lymph node. Amyloid deposition in the glandular stomach (Figure 1 and Figure 2) generally occurs in the lamina propria and submucosa. Amyloid deposits in the glomeruli of the kidney are usually the cause of death in animals that die with amyloidosis. Congo red stains amyloid orange to orange red and under polarized light imparts a light green, so-called apple green fluorescence.

Recommendation: Whenever present, amyloid deposits should be diagnosed as “amyloid” and graded based on the extent of the amyloid deposits. The loss of cells secondary to amyloid deposition should not be diagnosed separately but should be described in the narrative.



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References:

Bucher JR, Shackelford CC, Haseman JK, Johnson JD, Kurtz PJ, Persing RL. 1994. Carcinogenicity studies of oxazepam in mice. *Fundam Appl Toxicol* 23:280-297.

Abstract: <http://www.sciencedirect.com/science/article/pii/S0272059084711067>

Engelhardt JA, Gries CL, Long GG. 1993. Incidence of spontaneous neoplastic and nonneoplastic lesions in Charles River CD-1 mice varies with the breeding origin. *Toxicol Pathol* 21:538-541.

Full-text: <http://tpx.sagepub.com/content/21/6/538.full.pdf>

Frith CH, Chandra M. 1991. Incidence, distribution, and morphology of amyloidosis in Charles Rivers CD-1 mice. *Toxicol Pathol* 19:123-127.

Full-text: <http://tpx.sagepub.com/content/19/2/123.full.pdf>

Frith CH, Goodman DG, Boysen BG. 2007. The mouse. In: *Animal Models in Toxicology*, 2nd ed (Gad SC, ed). CRC Press, Boca Raton, FL, 19-146.

Abstract: <http://www.crcnetbase.com/isbn/9781420014204>

Greaves P. 2007. Digestive system. In: *Histopathology of Preclinical Toxicity Studies*, 3rd ed. Academic Press, London, 334-456.

Abstract: <http://www.sciencedirect.com/science/book/9780444527714>

Myers RK, McGavin MD. 2007. Cellular and tissue responses to injury. In: *Pathologic Basis of Veterinary Disease*, 4th ed (McGavin MD, Zachary JF, eds). Mosby, St Louis, MO, 3-62.

Percy DH, Barthold SW. 2001. Mouse. In: *Pathology of Laboratory Rodents and Rabbits*, 2nd ed. Iowa State Press, Ames, 3-106.

Ward JM, Mann PC, Morishima H, Frith CH. 1999. Thymus, spleen, and lymph nodes. In: *Pathology of the Mouse* (Maronpot RR, ed). Cache River Press, St Louis, MO, 333-456.

Abstract: <http://www.cacheriverpress.com/books/pathmouse.htm>

Authors:

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

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