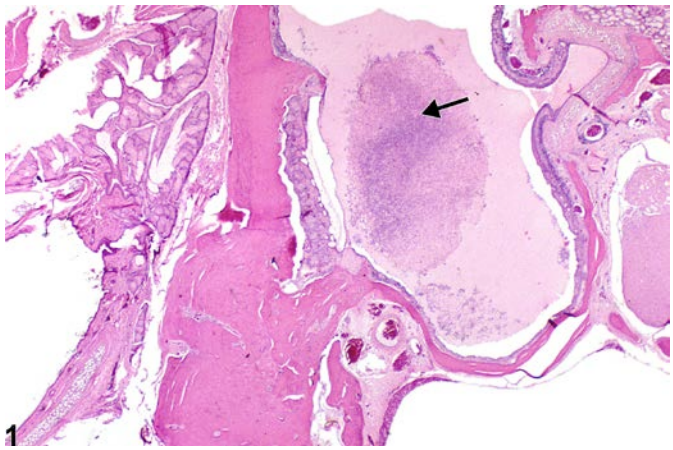
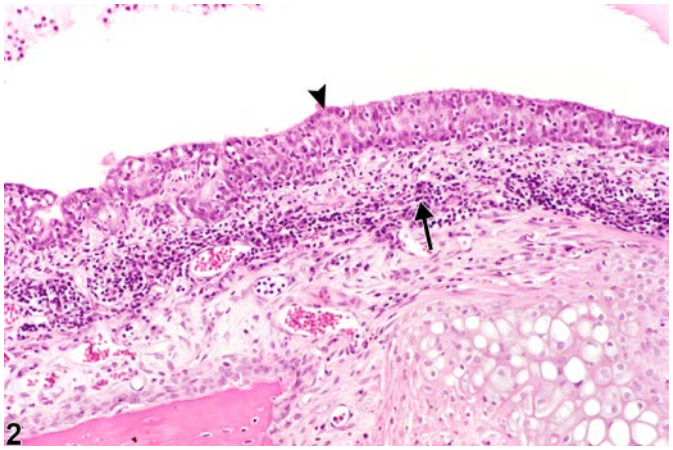
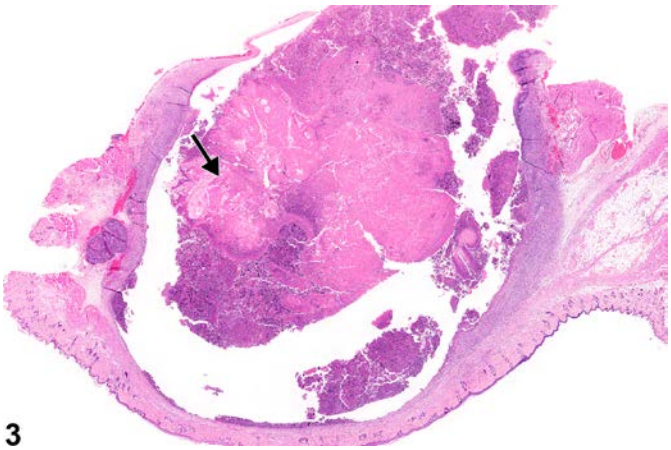
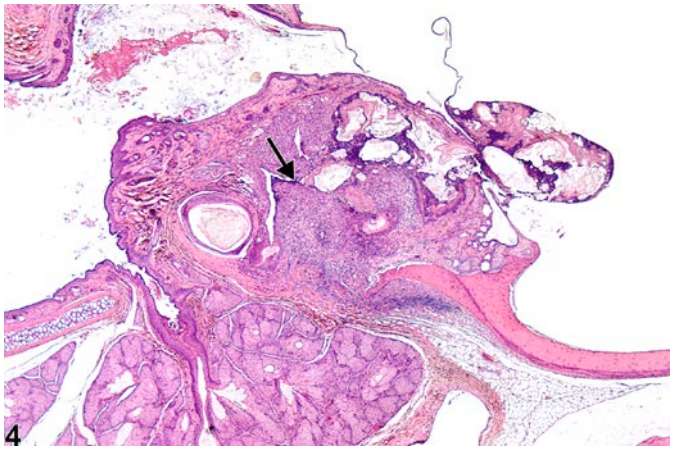
NTP Nonneoplastic Lesion Atlas

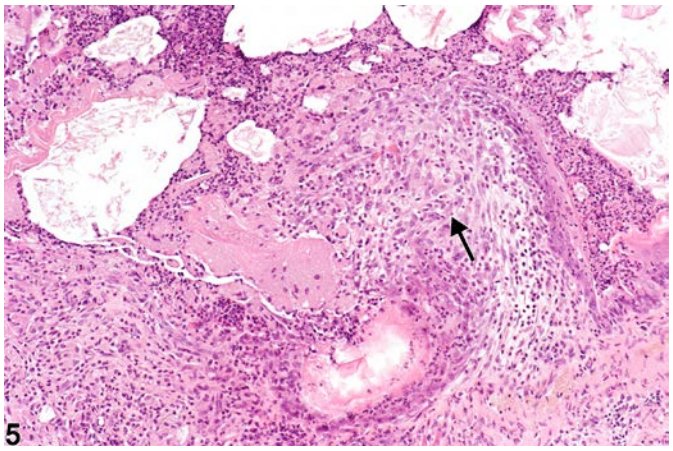
Ear – Inflammation

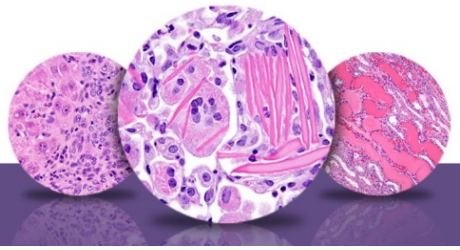

1


2


3


4


5



Ear – Inflammation

Figure Legend: **Figure 1** Ear - Inflammation, Acute in a male Fischer 344/N rat from a chronic study. There is predominantly neutrophilic infiltrates in the epithelium and lamina propria and abundant neutrophils and proteinaceous fluid in the external ear canal (arrow). **Figure 2** Ear - Inflammation, Chronic active in a male Fischer 344/N rat from a chronic study. There are neutrophils and mononuclear cells in the lamina propria (arrow) extending into the hyperplastic epithelium (arrowhead) lining the tympanic cavity. **Figure 3** Ear - Inflammation, Suppurative in a male F344/N rat from a chronic study. There is intraluminal necrotic debris and neutrophils (arrow); epithelial necrosis with denudation and neutrophil infiltration of the laminal propria is also present. **Figure 4** Ear - Inflammation, Chronic in a female Fischer/344N rat from a chronic study. There are macrophages and mononuclear cells in the lamina propria (arrow) with stromal fibrosis in the wall of the external ear canal and tympanic cavity. **Figure 5** Ear - inflammation, chronic in a female Fischer/344N rat from a chronic study (higher magnification of Figure 4). The inflammatory cells and fibrosis in the wall of the external ear canal are shown in greater detail.

Comment: Inflammation of various ear regions can result from many causes, including infectious agents; foreign bodies; topically applied irritants, and systemic toxins. Chronic inflammation of the ear with accumulation of fluid, cerumen, and cellular debris in the ear canal can result in cystic dilation of the ear canal (see Ear canal - Dilation).

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, however, in suppurative inflammation, the neutrophils are aggregated and many of them are degenerate (suppurative exudate). Cell debris, both from the resident cell populations and infiltrating leukocytes, proteinaceous fluid containing fibrin, fewer macrophages, occasional lymphocytes or plasma cells, and, possibly, an infectious agent may also be present in within the exudate. Grossly, these lesions would be characterized by the presence of pus. In the tissue surrounding the exudate, there may be fibroblasts, fibrous connective tissue, and mixed inflammatory cells, depending on the chronicity of the lesion. Lymphocytes predominate in chronic inflammation. Lymphocytes also predominate in chronic-active inflammation, but in chronic-active inflammation, 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.

Recommendation: Inflammation of the ear should be diagnosed and graded. The type of inflammation (e.g., acute or chronic active) should be indicated in the diagnosis as a qualifier. Similarly, the location of the lesion within the ear should be indicated with an appropriate site modifier (external ear or middle ear). Findings considered secondary (cyst, epithelial hyperplasia, etc.) should not be diagnosed separately unless warranted by severity. Secondary lesions not diagnosed separately should be described in the pathology narrative.

References:

Dogru S, Haholu A, Gungor A, Kucukodaci Z, Cincik H, Ozdemir T, Sen H. 2009. Histologic analysis of the effects of three different support materials within rat middle ear. Otolaryngol Head Neck Surg 140:177-182.
Abstract: http://www.ncbi.nlm.nih.gov/pubmed/19201284

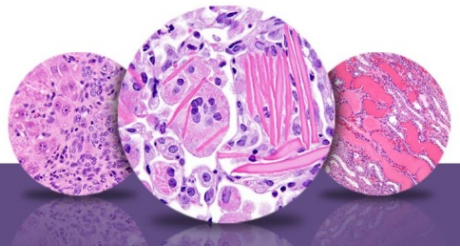
Huang C-C, Shi G-S, Yi Z-X. 1988. Experimental induction of middle ear cholesteatoma in rats. Am J Otolaryngol 9:165-172.
Abstract: http://www.ncbi.nlm.nih.gov/pubmed/3265859

MacArthur CJ, DeGagne JM, Kempton JB, Trune DR. 2009. Steroid control of acute middle ear inflammation in a mouse model. Arch Otolaryngol Head Neck Surg 135:453-457.
Full-text: http://archotol.jamanetwork.com/article.aspx?articleid=410115

MacArthur CJ, Trune DR. 2006. Mouse models of otitis media. Curr Opin Otolaryngol Head Neck Surg 14:341-346.
Abstract: http://www.ncbi.nlm.nih.gov/pubmed/16974149

Maeda K, Hirano T, Ichimiya I, Kurono Y, Suzuki M, Mogi G. 2004. Cytokine expression in experimental otitis media with effusion in mice. Laryngoscope 114:1967-1972.
Full-text: http://onlinelibrary.wiley.com/doi/10.1097/01.mlg.0000147930.29261.51/full

National Toxicology Program. 1982. NTP TR-193. Bioassay of Reserpine for Possible Carcinogenicity (CAS No. 50-55-5). NTP, Research Triangle Park, NC.
Abstract: http://ntp.niehs.nih.gov/go/10117



Ear – Inflammation

References:

National Toxicology Program. 1986. NTP TR-259. Carcinogenesis Studies of Ethyl Acrylate (CAS No. 140-88-5) in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies). NTP, Research Triangle Park, NC.
Abstract: http://ntp.niehs.nih.gov/go/13957

National Toxicology Program. 1993. NTP TR-394. Toxicology and Carcinogenesis Studies of Acetaminophen (CAS No. 103-90-2) in F344 Rats and B6C3F$_1$ Mice (Feed Studies). NTP, Research Triangle Park, NC.
Abstract: http://ntp.niehs.nih.gov/go/12239

National Toxicology Program. 1993. NTP TR-420. Toxicology and Carcinogenesis Studies of Triamterene (CAS No. 396-01-0) in F344/N Rats and B6C3F$_1$ Mice (Feed Studies). NTP, Research Triangle Park, NC.
Abstract: http://ntp.niehs.nih.gov/go/8057

Piltcher OB, Swarts JD, Magnuson K, Alper CM, Doyle WJ, Hebda PA. 2002. A rat model of otitis media with effusion caused by Eustachian tube obstruction with and without *Streptococcus pneumoniae* infection: Methods and disease course. Otolaryngol Head Neck Surg 126:490-498.
Abstract: http://www.ncbi.nlm.nih.gov/pubmed/12075222

Widemar L, Hellström, Stenfors L. 1986. Different structural changes in membrane Shrapnelli in serous and purulent otitis media: An experimental study in the rat. Acta Otolaryngol 102:266-273.
Abstract: http://www.ncbi.nlm.nih.gov/pubmed/3776521

Wright CG, Pawlowski KS, Roland PS, Meyerhoff WL. 2000. An animal model for external ear canal inflammation. Laryngoscope 110:1112-1118.
Abstract: http://www.ncbi.nlm.nih.gov/pubmed/10892679

Yoshitomi K, Brown HR. 1990. Ear and pinna. In: Pathology of the Fischer Rat: Reference and Atlas (Boorman GA, Eustis SL, Elwell MR, Montgomery CA, MacKenzie WF, eds). Academic Press, San Diego, CA, 227-238.
Abstract: http://www.ncbi.nlm.nih.gov/nlmcatalog/9002563

Author:

Margarita M. Gruebbel, DVM, PhD, DACVP
Senior Pathologist
Experimental Pathology Laboratories, Inc.
Research Triangle Park, NC