Case # 2011-106
Dermal Xanthosis in ApoE-null Mouse

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Apolipoprotein E (ApoE) is essential for lipid metabolism.

In ApoE-null mice, the gene has been “knocked-out”.

Reduced post-prandial clearance of lipoproteins

Research applications:
- Cholesterol/ triglyceride metabolism
- Lipoprotein transport
- Atherosclerosis plaque formation
- Alzheimer’s disease
- Neurodegeneration
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- Intact male ApoE null mouse
- Approximately 6 months old
- Jackson Laboratories; 6/20/11
- Standard 12-hour light : dark cycle; Static microisolator cage with corn cob bedding
- Tekclad Adjusted Calorie Diet (42% fat) ad libitum; Bottled water

Clinical History

- Trio-housed males reported for dermatitis
- BAR, BCS 2.5/5
- Varying clinical signs: pruritus, alopecia, excoriations
- Pelage tape: negative
- Unresponsive to empirical therapy
Gross Pathology

- Euthanasia due to poor prognosis
- No other experimental manipulations
- 2.5 cm x 1.25 cm dermal lesion
- No other gross lesions found
Granulomatous inflammation

Ulcerative dermatitis (block arrows)
Cholesterol clefts (black arrow) H&E Bar = 500um

Morphologic Diagnosis

- Dermal Xanthosis
Morphologic Diagnosis

- Dermal Xanthosis

Superficial Dermis: Numerous cholesterol clefts and lipid-laden macrophages
H&E  Bar = 100um

Cholesterol clefts and lipid-laden macrophages
H&E  Bar = 50um

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Dermal Xanthosis

- Apo E serves an important role in tissue distribution of cholesterol deposition\(^1\)
- Lack of ApoE in the skin promotes a proinflammatory condition\(^6\)
- Specialized macrophages, called \textit{foam cells}, eventually die, leaving deposition of extracellular cholesterol crystals\(^2\)
- Cutaneous foam cell formation = degree of atherosclerosis in aorta\(^3\)
- Apo E null mice develop atherosclerotic lesions faster than wild type
  - Accelerated further by “Western type” diet\(^4\)
- Natural-occurring xanthomas: birds > domestic animals >> dogs and cats\(^7\)


Questions?