

C57BL/6-*ApoE*^{em1Narl}/Narl

Stock No: RMRC13302 | C57BL/6-*ApoE*^{em1Narl}/Narl

Overview

Apolipoprotein E (ApoE) plays an essential role in lipid and cholesterol homeostasis. Deletion of ApoE in mouse has been shown to induce overall elevation of serum cholesterol and atherosclerosis. This mouse model has been applied to metabolic, cardiovascular and neurobiological research. We used CRISPR/Cas9 technology to generate ApoE knockout mouse C57BL/6-*ApoE*^{em1Narl}/Narl, and validated its phenotypes including high serum cholesterol and high-fat-diet induced atherosclerosis.

Genetic Overview

Allele Symbol:	C57BL/6- <i>ApoE</i> ^{em1Narl} /Narl
Allele Name	Endonuclease-mediated mutation 1, National Applied Research Laboratories
Allele Type	Endonuclease-mediated mutation
Gene Symbol and Name	<i>ApoE</i> , apolipoprotein E
Strain of Origin	C57BL/6JNarl

Characterization

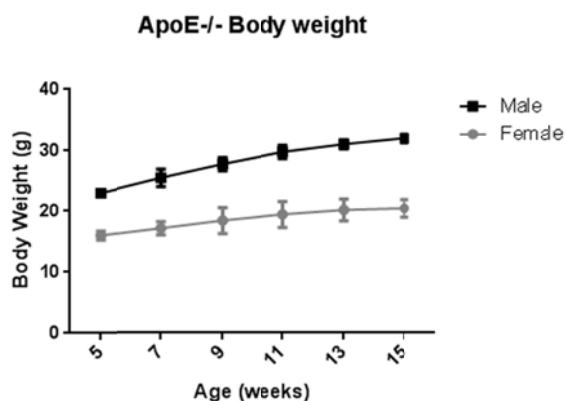


Figure 1. Body weight of ApoE homozygous knockout (ApoE^{-/-}, n = 2).

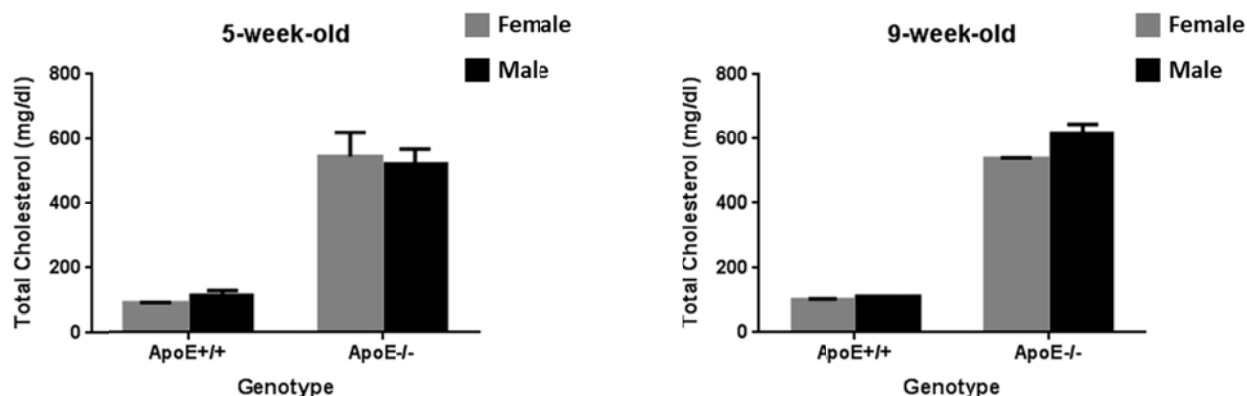


Figure 2. ApoE knockout increases serum cholesterol at early ages. Total serum cholesterol from ApoE^{-/-} and wild-type (ApoE^{+/+}) were measured at 5 and 9 week old (n = 3-7).

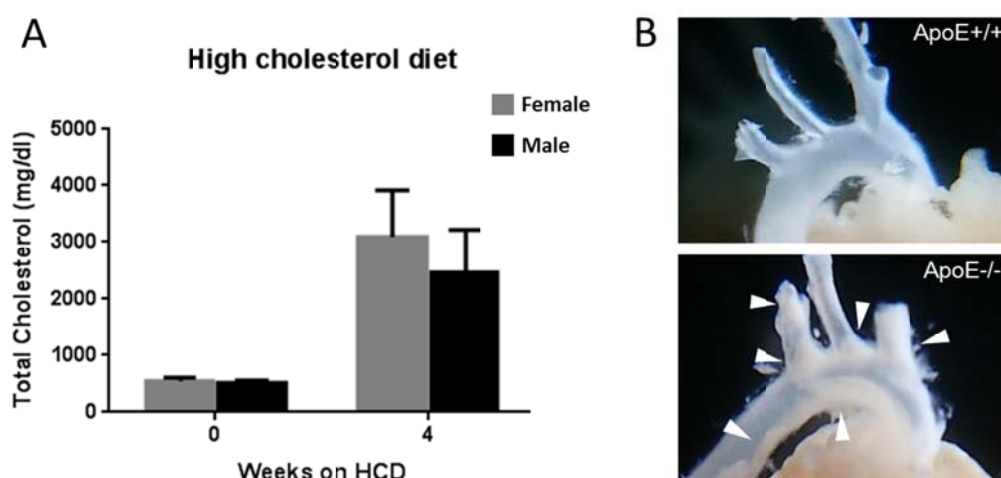


Figure 3. High cholesterol diet (HCD) further increases serum cholesterol and induces atherosclerosis. A. Total serum cholesterol. B. Representative images of thoracic aorta after 12 weeks on HCD (n = 3-8). Arrowheads indicate atherosclerotic plaques.

Research Application

ApoE knockout mouse can be used to support research areas including:

- Cholesterol and lipid metabolism
- Atherosclerosis and cardiovascular research
- Inflammation and immunology