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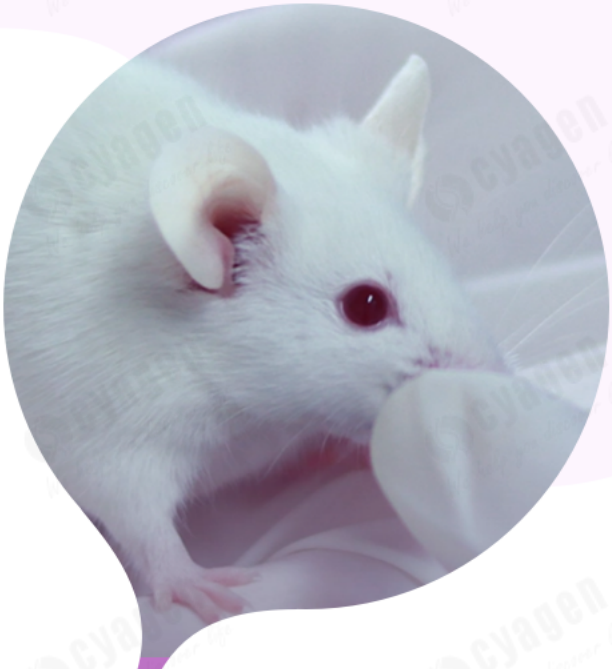
C001507

Strain Datasheet



Release Time: November 04, 2025

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B6J-Apoe KO Mouse

Product ID: C001507

Common Name: B6J-Apoe KO

Strain Name: C57BL/6JCya-*Apoe*^{em1}/Cya

Background: C57BL/6JCya

Strain Description

Apolipoprotein E (ApoE) is a lipid particle-associated polymorphic carrier protein encoded by the APOE gene. It is a core component of plasma lipoproteins, participating in the production, transport, and clearance of lipoproteins. ApoE is associated with chylomicrons, chylomicron remnants, high-density lipoprotein (HDL), very low-density lipoprotein (VLDL), and intermediate-density lipoprotein (IDL), especially showing preferential binding to HDL [1]. ApoE is the most important lipid transport protein in the body, having a profound impact on lipid metabolism. The interaction of ApoE with the low-density lipoprotein receptor (LDLR) is essential for the normal processing (catabolism) of triglyceride-rich lipoproteins [2]. In peripheral tissues, ApoE is primarily produced by the liver and macrophages and mediates cholesterol metabolism. In the central nervous system, ApoE is produced mainly by astrocytes and is the major cholesterol carrier in the brain. ApoE is essential for transporting cholesterol from astrocytes to neurons [1-4]. In addition, ApoE forms a complex with activated C1q, becoming a checkpoint inhibitor target of the classical complement pathway [5]. Polymorphisms of the APOE are associated with Alzheimer's disease and lipid accumulation, hyperlipidemia, atherosclerosis, high cholesterolemia, etc., and are related to the risk of various cardiovascular diseases.

The B6J-Apoe KO mouse is a model of ApoE deficiency. It was generated by gene editing technology to knock out the Apoe gene in mice. ApoE protein synthesis is blocked in these mice, leading to elevated cholesterol levels and spontaneous atherosclerosis. Cholesterol levels and atherosclerosis in mice fed a high-fat diet (HFD) are further exacerbated. The B6J-Apoe KO mice are viable and can be used for research in hypercholesterolemia, atherosclerosis, and Alzheimer's disease.

Application Area

① Cardiovascular disease research: Hypercholesterolemia, hyperlipidemia, atherosclerosis, etc.;

② Body metabolism mechanism research: Fat and cholesterol metabolism;

③ Neurodegenerative disease research: Alzheimer's disease.

Strain Strategy

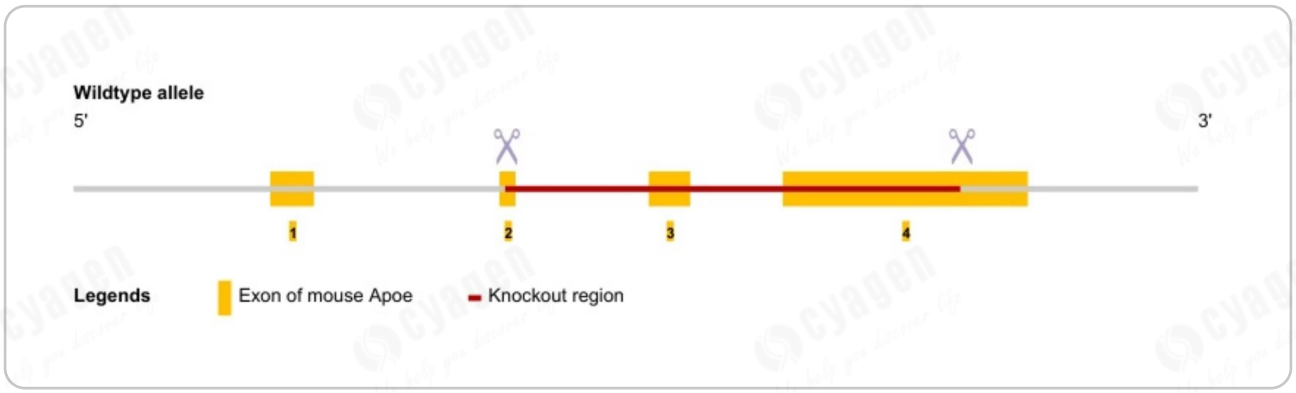

Figure 1. Gene editing strategy for B6J-Apoe KO mice. Use gene editing technology to knock out the exons 2-4 of the Apoe gene in C57BL/6JCya mice.

Validation Data

1. Expression of the APOE protein

Western blot results showed no expression of APOE protein in the livers of B6J-Apoe KO mice.

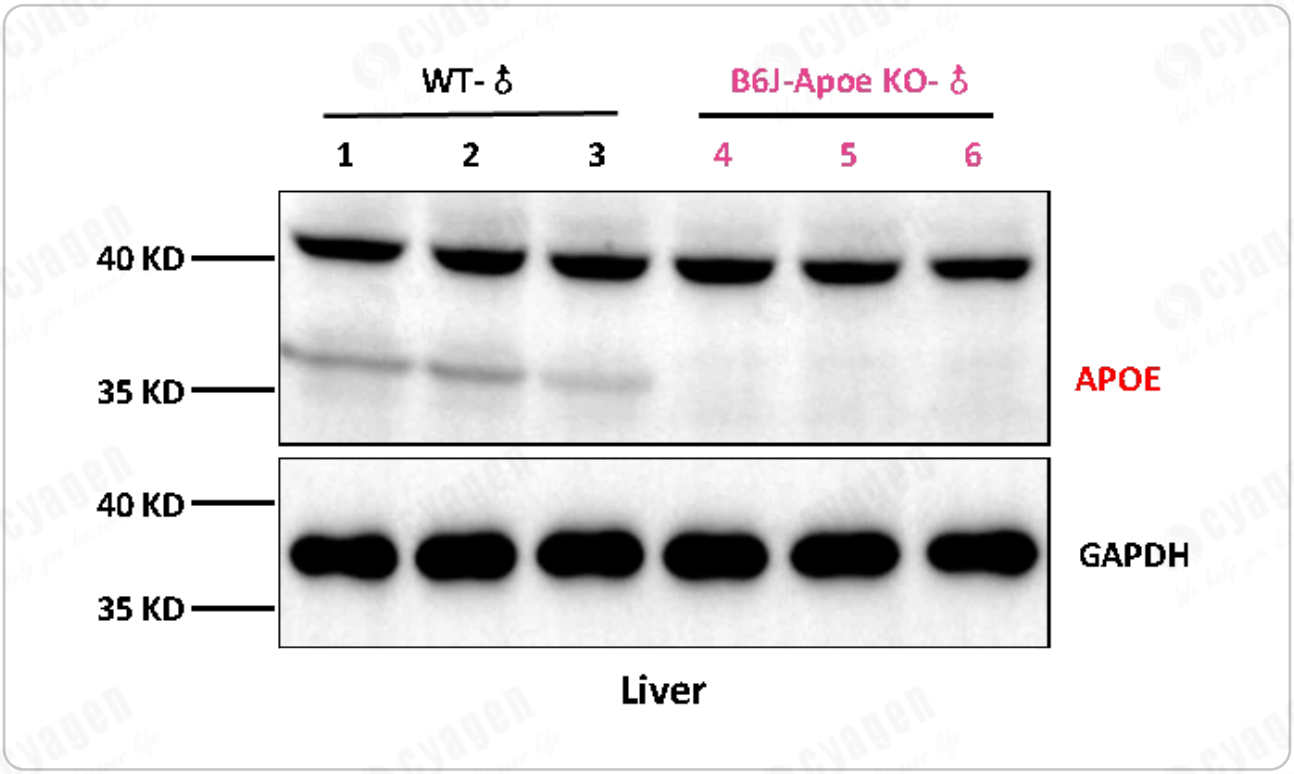

Figure 2. Expression of APOE protein in the livers of 8-week-old male B6J-Apoe KO and wild-type (WT) mice.

2. Growth Curves

Results showed that B6J-Apoe KO mice grew similarly in normal and HFD conditions. WT mice fed HFD gained weight, and internal inspection data showed that at 20 weeks, the aortic arch and aortic vessels in WT mice were normal and no atherosclerotic plaque was formed.*

*High-fat diet was purchased from Medicience (catalog number: MD12015). High-fat feeding protocol: Mice started gradually transitioning to the high-fat diet at week 5 and began formal feeding on the high-fat diet at week 6.

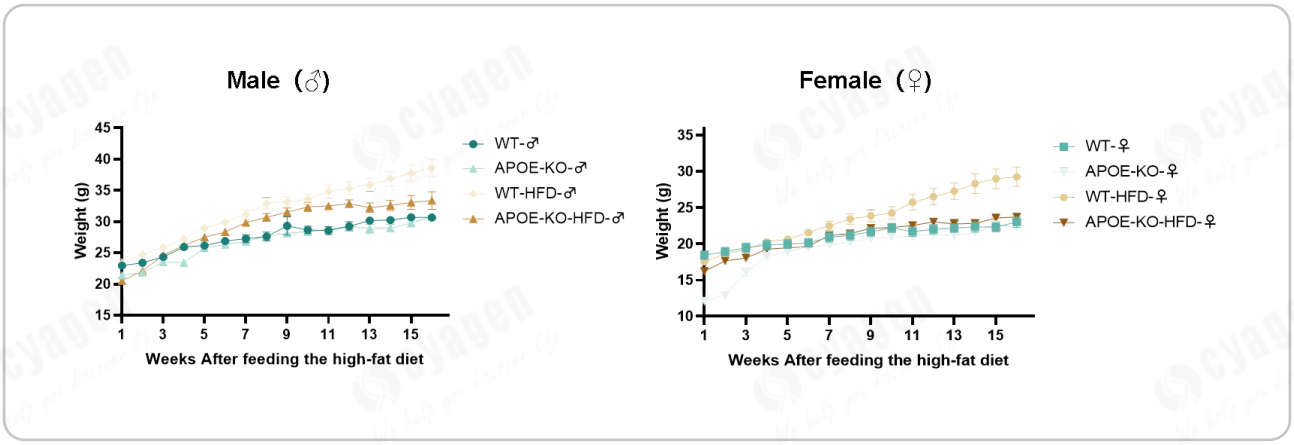

Figure 3. Body weight trajectories of B6J-Apoe KO and wild-type (WT) mice on normal diet (ND) and high-fat diet (HFD) conditions.

3. Cholesterol blood test in male mice

Data showed that under normal diet conditions, compared with wild-type mice, male B6J-Apoe KO mice had different degrees of increases in triglyceride (TRIG), cholesterol (CHOL), and low-density lipoprotein (LDL-C) levels in their bodies. High-fat diet further increased the levels of these indicators in male B6J-Apoe KO mice. The levels of high-density lipoprotein (HDL-C) in male B6J-Apoe KO and wild-type (WT) mice were not significantly different.

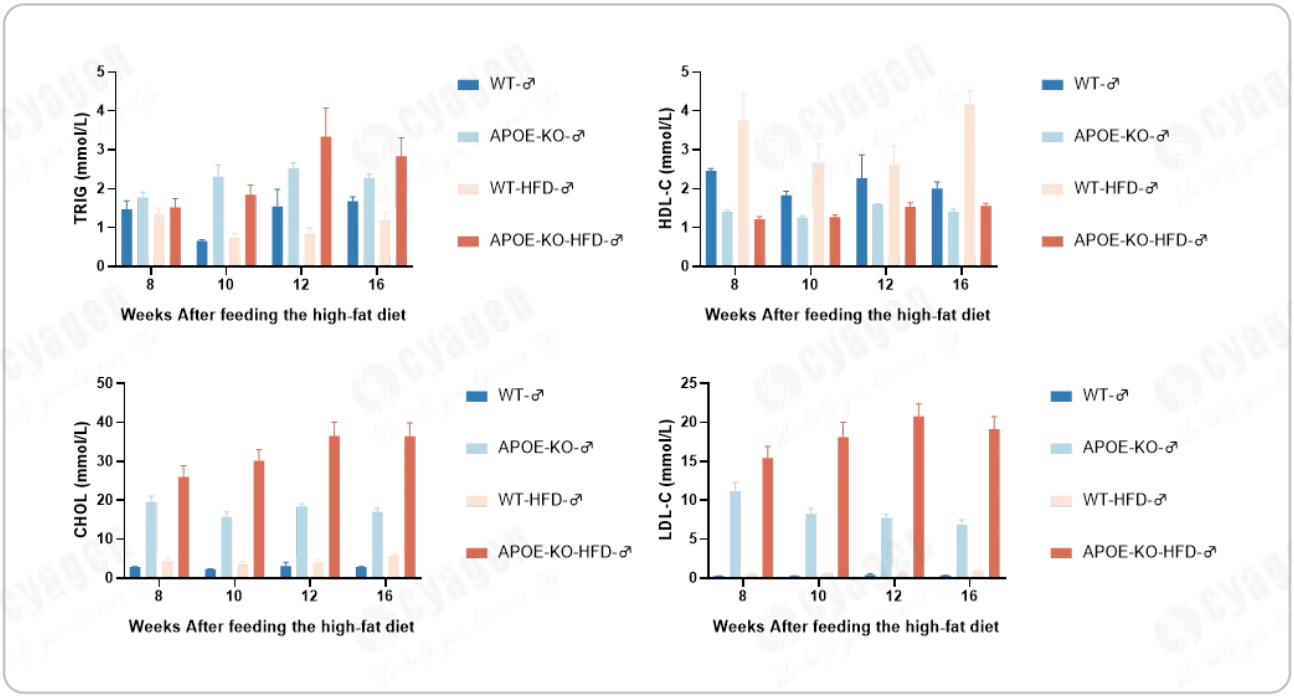

Figure 4. Serum lipid biochemical indicators of male B6J-Apoe KO and wild-type (WT) mice on normal and high-fat diet (HFD) conditions.

4. Cholesterol blood test in female mice

Under normal diet conditions, compared with wild-type mice, female B6J-Apoe KO mice had significantly elevated levels of cholesterol (CHOL) and low-density lipoprotein (LDL-C). High-fat diet further increased the levels of cholesterol (CHOL) and low-density lipoprotein (LDL-C) in female B6J-Apoe KO mice. The levels of high-density lipoprotein (HDL-C) and triglyceride (TRIG) in female B6J-Apoe KO mice and wild-type (WT) mice were not significantly different.

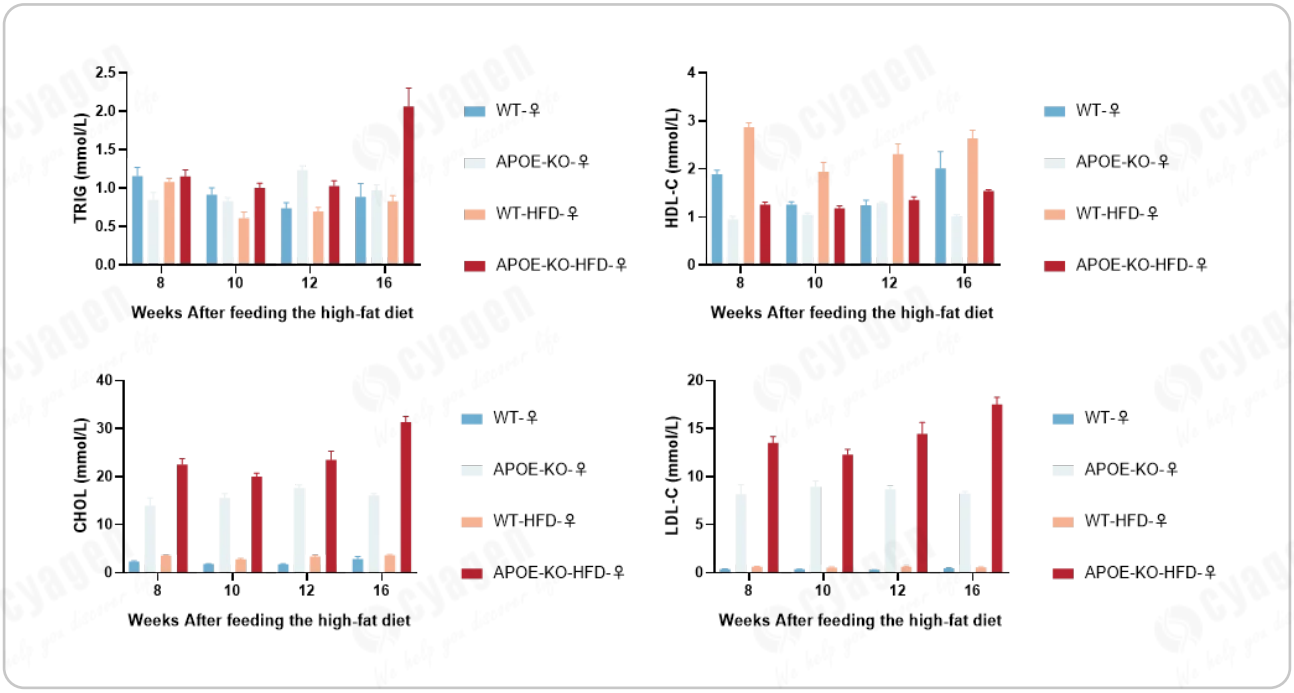

Figure 5. Serum lipid biochemical indicators of female B6J-Apoe KO and wild-type (WT) mice on normal and high-fat diet (HFD) conditions.

5. Aortic pathology in male B6J-Apoe KO mice

Male B6J-Apoe KO mice can spontaneously form atherosclerotic plaques in the aorta after 10 weeks on a normal diet, with mild symptoms. After induction by high-fat diet feeding, the atherosclerotic plaques in the aorta were exacerbated, and the symptoms became more obvious after week 12, continued to worsen at week 14, and reached the late stage of pathological phenotype at week 16.

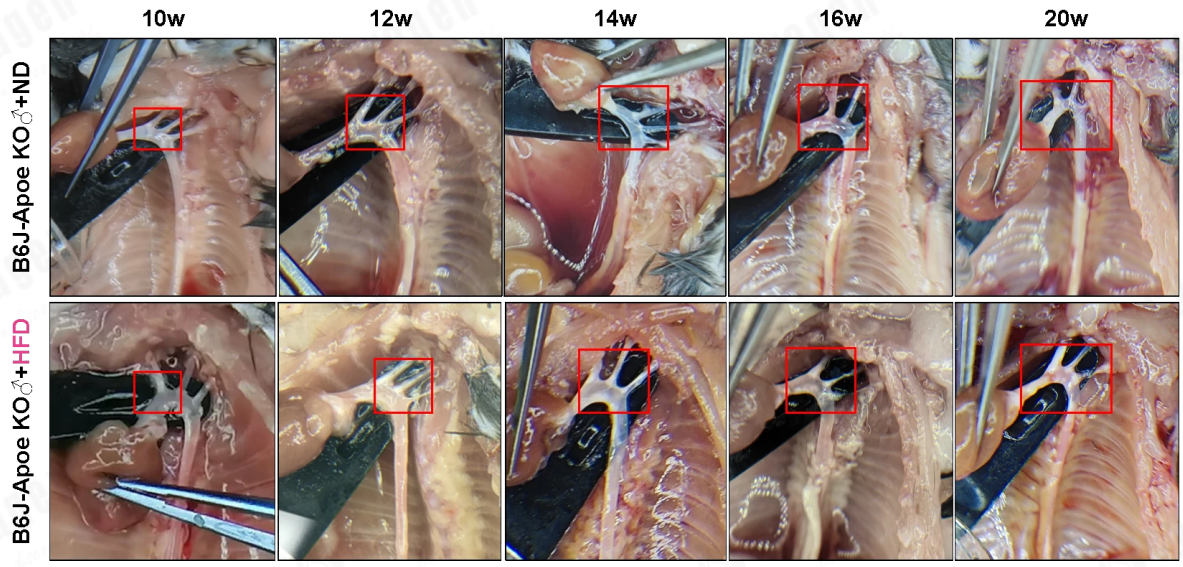

Figure 6. Detection of atherosclerotic plaques in the aorta of male B6J-Apoe KO mice on normal diet (ND) and high-fat diet (HFD) conditions.

6. Aortic pathology in female B6J-Apoe KO mice

Female B6J-Apoe KO mice can spontaneously form atherosclerotic plaques in the aorta after 12 weeks on a normal diet, with mild symptoms. After induction by high-fat diet feeding, the atherosclerotic plaques in the aorta were exacerbated, and the symptoms became more obvious after week 14 and continued to worsen at week 16.

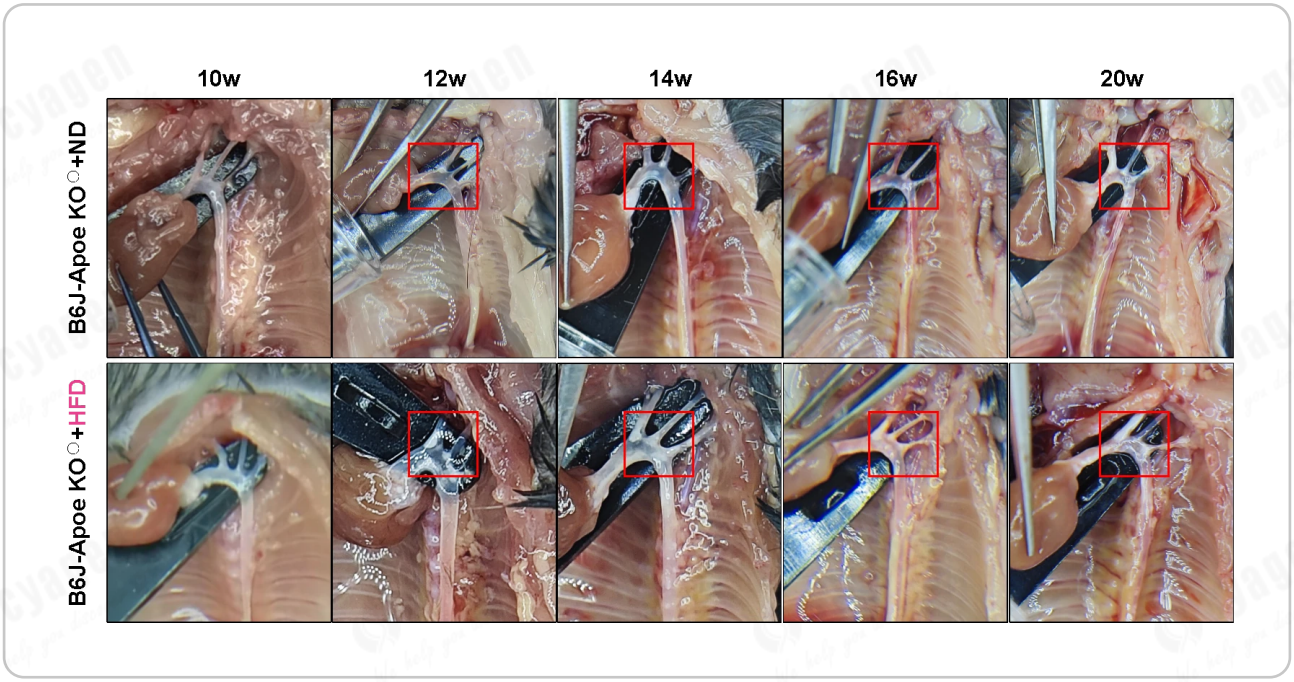

Figure 7. Detection of atherosclerotic plaques in the aorta of female B6J-Apoe KO mice on normal diet (ND) and high-fat diet (HFD) conditions.

7. Oil Red O staining of the aorta

The red color indicates positive fat staining. The results show that wild-type mice, after 20 weeks of both chow and high-fat diets, exhibited no significant arterial lesions. However, B6J-Apoe KO mice, following a high-fat diet, showed noticeable fat accumulation near the aortic arch, with a more severe phenotype after 20 weeks. Similarly, B6J-Apoe KO mice, after a chow diet, also exhibited spontaneous atherosclerosis, which worsened after 20 weeks.

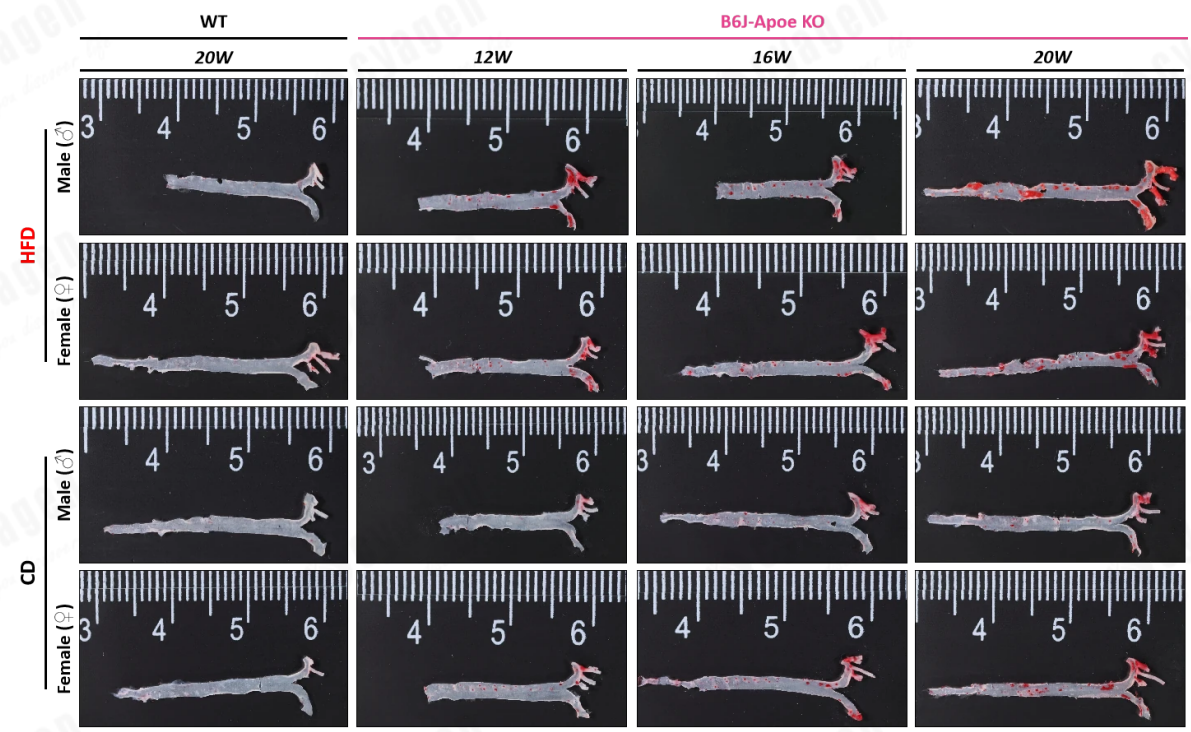

Figure 8. Oil Red O staining results for B6J-Apoe KO mice and wild-type mice (WT) under chow diet (CD) and high-fat diet (HFD) conditions.

8. Oil Red O staining of cardiac valves

The results show that wild-type mice, after 20 weeks of both chow and high-fat diets, exhibited no fat accumulation near the cardiac valves. However, B6J-Apoe KO mice, following a high-fat diet, showed noticeable fat accumulation near the cardiac valves, with a more severe phenotype after 20 weeks. Similarly, B6J-Apoe KO mice also exhibited fat accumulation under a chow diet, which worsened after 20 weeks.

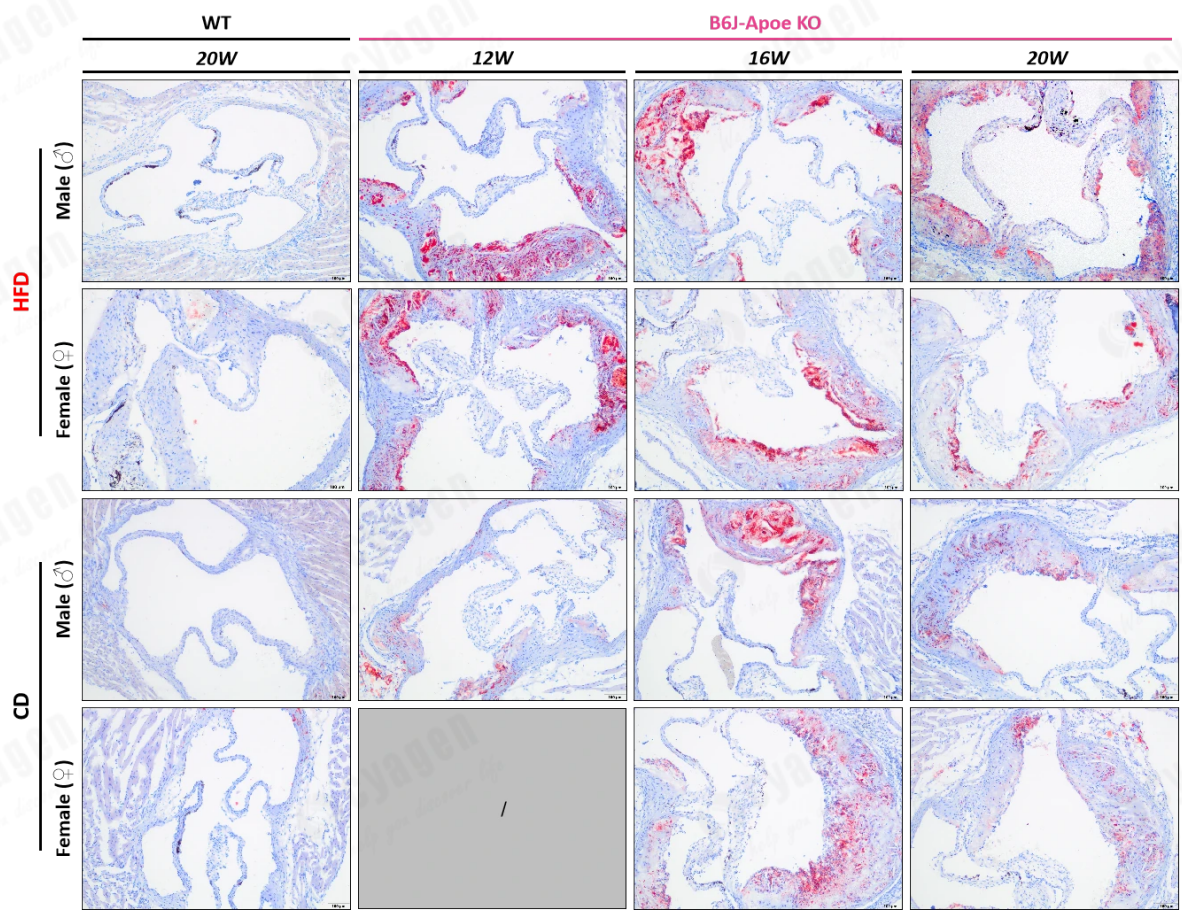

Figure 9. Oil Red O staining of cardiac valves for B6J-Apoe KO mice and wild-type mice (WT) under chow diet (CD) and high-fat diet (HFD) conditions.

Dietary Information

Temperature: 20-26℃

Humidity: 40%-70%

Bedding: Change high-pressure sterilized bedding weekly.

Normal Chow Diet: Standard Maintenance Diet (Jiangsu Xietong Pharmaceutical Bio-engineering, catalog number: SWC9101).

High-fat Diet: HFD Diet (Medicience, catalog number: MD12015).

Suggested Modeling Period: Begin high-fat feeding from 5-8 weeks of age. After 1 week of adaptive feeding with a high-fat diet (as shown in Table 1), switch to a complete high-fat diet. Tissue collection after 12-16 weeks of high-fat feeding may reveal atherosclerotic lesions in the aorta.

Day	Condition
Day 1-3	Standard: High-fat diet ratio = 2:1
Day 4-5	Standard: High-fat diet ratio = 1:1
Day 6-7	Standard: High-fat diet ratio = 1:2

Table 1: Adaptive feeding conditions for a high-fat diet. *Dietary effects on this model may vary based on environmental and feeding conditions. Data provided is for reference to help you optimize study design.

Publications

Eur Heart J 2023.12

Myocardial infarction drives trained immunity of monocytes, accelerating atherosclerosis

Journal of Molecular and Cellular Cardiology 2022.06

ICAM-1-related noncoding RNA accelerates atherosclerosis by amplifying NF-κB signaling

European Journal of Pharmacology 2023.10

DL-3-n-butylphthalide improves the endothelium-dependent vasodilation in high-fat diet-fed ApoE−/− mice via suppressing inflammation, endothelial necroptosis and apoptosis

Reference

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[2]. Mahley RW, Weisgraber KH, Huang Y. Apolipoprotein E: structure determines function, from atherosclerosis to Alzheimer's disease to AIDS. J Lipid Res. 2009 Apr;50 Suppl(Suppl): S183-8.

[3]. Wang H, Kulas JA, Wang C, Holtzman DM, Ferris HA, Hansen SB. Regulation of beta-amyloid production in neurons by astrocyte-derived cholesterol. Proc Natl Acad Sci U S A. 2021 Aug 17;118(33):e2102191118.

[4]. Serrano-Pozo A, Das S, Hyman BT. APOE and Alzheimer's disease: advances in genetics, pathophysiology, and therapeutic approaches. Lancet Neurol. 2021 Jan;20(1):68-80.

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