

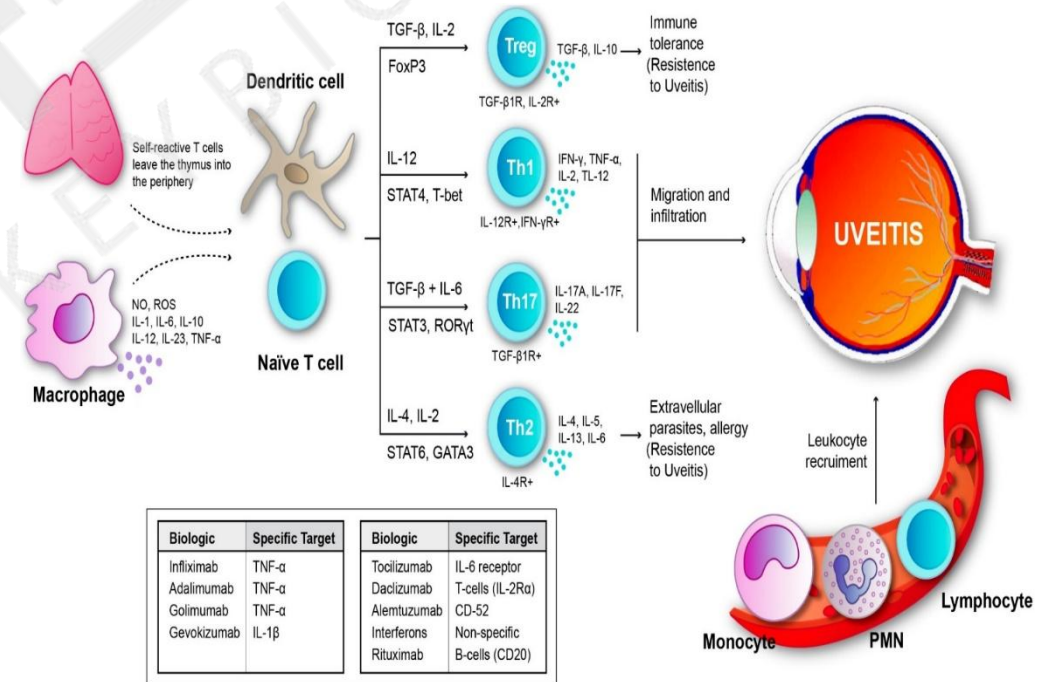


Preclinical Model of Experimental Autoimmune Uveitis(EAU)

Experimental Autoimmune Uveitis (EAU)

Symptoms and Causes

Uveitis results from imbalance between inflammatory mechanisms and regulatory mechanisms. In autoimmune uveitis, self-reactive T cells leave the thymus and when they reach the eye they come in contact with retinal antigens. Myeloid dendritic cells present a solid ability of capturing antigens, which enables them to stimulate T cells. Therefore, T-lymphocytes may differentiate into Tregs, Th1, Th17 or Th2 for precise immune response in function of the antigen encountered and cytokine presence. Th1 and Th17 cells participate in inflammatory and autoimmune uveitis. Th1 cells are crucial for the development of uveitis, whereas Th17 cells play a relevant role in the late/chronic phase of uveitis, however induced Treg cells defeat both Th1 and Th17 cell responses. Furthermore, the migration of Th1 and Th 17 to the eye, also results in the breaking down of the blood-retinal barrier and, consequently, different leukocytes from the circulation are recruited.



■ IRBP Induced C57BL/6 EAU Model

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IRBP Induced C57BL/6 EAU Model

■ Mechanism

IRBP is an interphotoreceptor vitamin A binding protein. The immunodominant epitopes of IRBP bind to important sites of MHC molecules on antigen-presenting cells and activate antigen-presenting cells, which can induce Naive T cells to differentiate into effector Th1 and Th17 cells. These cells then cause disease by producing inflammatory cytokines such as TNF- α , IFN- γ and IL-17.

■ IRBP Induced EAU Model



Animal	C57BL/6
Evaluation	Clinical: Fundus camera, OCT
	Cytokine:
	Pathology: HE

IRBP Induced C57BL/6 EAU Model

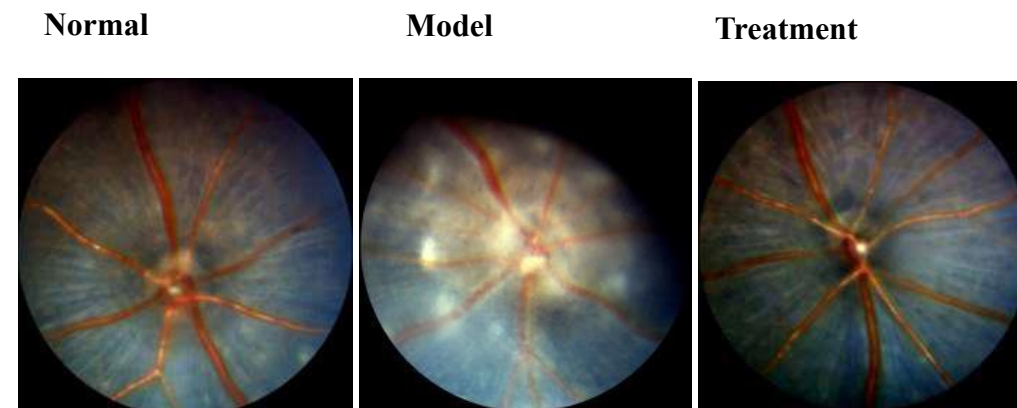
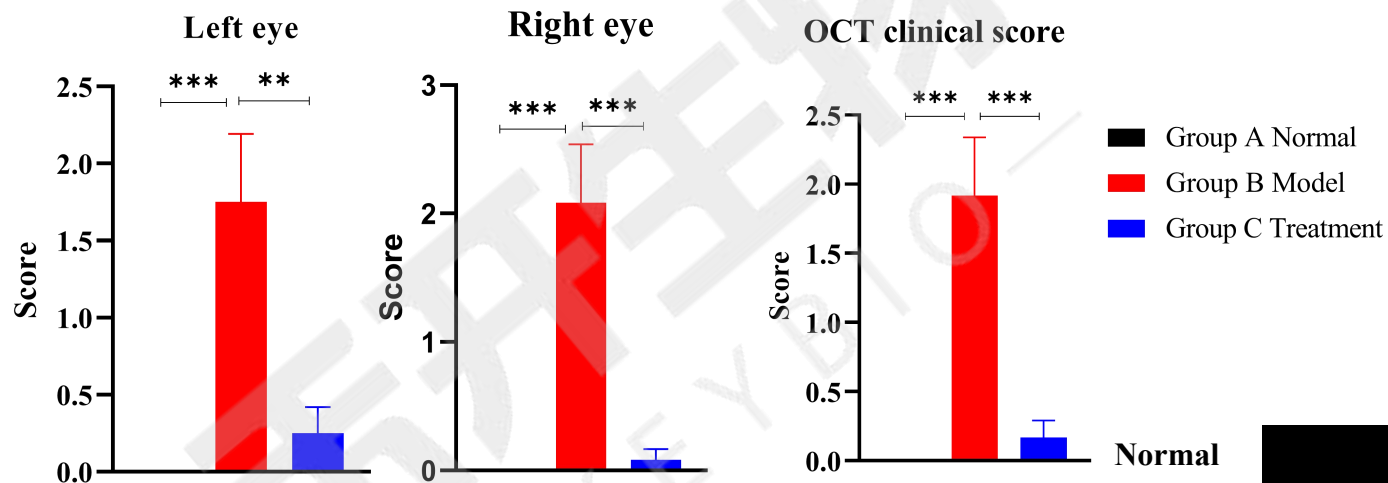


Fig2. Fundus pictures

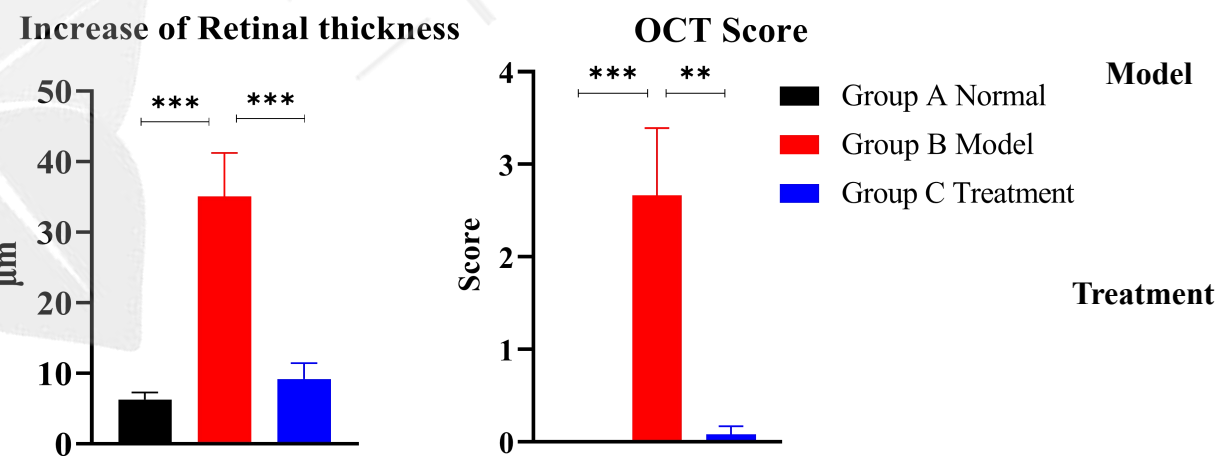


Fig3. Retinal Thickness

Fig4. OCT score

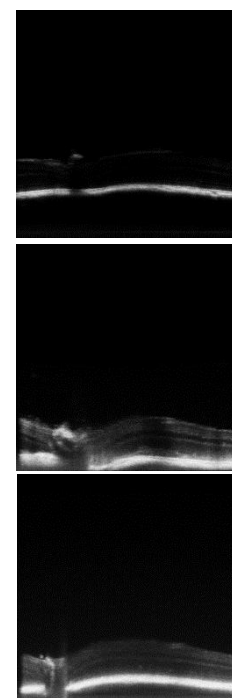


Fig5. Retinal pictures

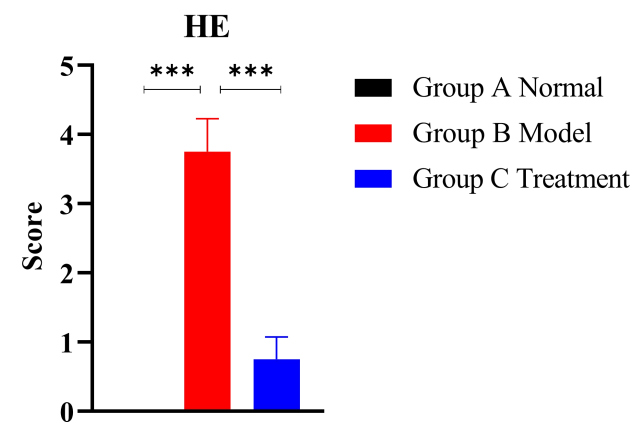
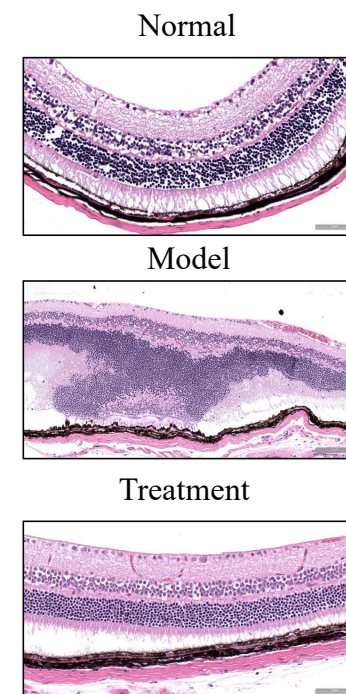


Fig6. Pathology





Our Sites

Non-human Private Center: Guangxi
Analysis Center: Suzhou
Rodent Center: Suzhou



WeChat

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