Frovocimab

Cat. No.: HY-P99626

CAS No.: 1643672-70-1

Target: Ser/Thr Protease

Pathway: Metabolic Enzyme/Protease

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Frovocimab (LY 3015014) is a humanized IgG4 monoclonal antibody (mAb) that neutralizes PCSK9. Frovocimab inhibits PCSK9 binding to LDL receptor (LDLR) while permitting the normal proteolytic cleavage of the bound intact PCSK9[1].

In Vitro

Frovocimab (LY 3015014) binds to intact but not truncated PCSK9. The selective binding of Frovocimab to intact PCSK9 is related to its binding epitope, the linear sequence of amino acids 160-181 of the catalytic domain of human PCSK9, which is absent in truncated PCSK9. Frovocimab inhibits PCSK9 binding to LDLR while permitting the normal proteolytic cleavage of the bound intact PCSK9. Additionally, upon cleavage, truncated inactive PCSK9 is released from Frovocimab[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Frovocimab (LY 3015014; 10 mg/kg; i.v; once) causes significant PCSK9 accumulation, its duration of LDL lowering is reduced, and its clearance (CL) from serum is accelerated in mice expressing a noncleavable variant of human PCSK9[1]. Frovocimab (LY 3015014; 5 mg/kg; i.v; once) decreases LDL cholesterol in monkeys and, unlike other PCSK9 mAbs, does not cause an accumulation of intact PCSK9 in serum[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6NTac mice injected with vector containing the NC R215A/R218A variant[1]
Dosage:	10 mg/kg
Administration:	i.v; once
Result:	Caused significant PCSK9 accumulation.

Animal Model:	Cynomolgus monkeys[1]
Dosage:	5 mg/kg
Administration:	i.v; once
Result:	Decreased LDL cholesterol in monkeys.

REFERENCES

[1]. Krista M Schroeder, et al. Proteolytic cleavage of antigen extends the durability of an anti-PCSK9 monoclonal antibody. J Lipid Res. 2015 Nov;56(11):2124-32.

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