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Golimumab

Cat. No.:	HY-P99111
CAS No.:	476181-74-5
Target:	TNF Receptor; Apoptosis; Caspase; Interleukin Related
Pathway:	Apoptosis; Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Golimumab (CNTO-148) is a potent human IgG1 TNFα antagonist monoclonal antibody. Golimumab has anti-inflammation activitity and inhibits IL-6 and IL-1β production. Golimumab acts via targeting and neutralizing TNF to prevent inflammation and destruction of cartilage and bone. Golimumab has the anticancer activity and induces cell apoptosis. Golimumab can be used for rheumatoid arthritis, Crohn's disease and cancer research[1][2][3].

IC_{50} & Target

CD40

In Vitro

Golimumab (CNTO-148) (0.1-10 μg /mL; 24-72 hours; transmembrane TNFα–transfected Jurkat cells) induces reductions in the viability of transmembrane TNFα–expressing cells[1].

Golimumab (CNTO-148) (10 μg /mL; 48 hours; transmembrane TNFα–transfected Jurkat cells) induces apoptosis, increase the levels of active caspase-3 and induces apoptotic DNA fragmentation[1].

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

Cell Viability Assay[1]

Cell Line:	Transmembrane TNFα–transfected Jurkat cells
Concentration:	0.1, 1 and 10 μg/mL
Incubation Time:	24, 48 and 72 hours
Result:	Reduced cell viability in a dose- and time-dependent manner.

Apoptosis Analysis[1]

Cell Line:	Transmembrane TNFα–transfected Jurkat cells
Concentration:	10 μg /mL
Incubation Time:	48 hours
Result:	Had the percentage of apoptotic cells for 30.29%.

Western Blot Analysis[1]

Cell Line:	Transmembrane TNFα–transfected Jurkat cells

Concentration:	10 μg /mL
Incubation Time:	48 hours
Result:	Increased the levels of active caspase-3 and induces apoptotic DNA fragmentation.

In Vivo

Golimumab (CNTO-148) (24 mg/kg; i.h.; daily, for 7 days; swiss-albino healthy male mice) inhibits oxidative stress, apoptotic cell death inflammatory response, thus improving renal function. Golimumab reduces all markers of kidney injury and attenuates cell death[2].

Golimumab (CNTO-148) (1-10 mg/kg; i.p.;daily; for 4 weeks; Tg197 transgenic mouse model) relieves TNFα-induced arthritis in a mouse model of human[3].

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

Animal Model:	Swiss-albino healthy male mice[2]
Dosage:	24 mg/kg
Administration:	Subcutaneous injection; daily, for 7 days
Result:	Reduced serum parameters like BUN, NGAL creatinine, cystatin C, and urinary parameters like KIM-1, NAG, albumin clusterin.

Animal Model:	Swiss-albino healthy male mice[2]
Dosage:	24 mg/kg
Administration:	Subcutaneous injection; daily, for 7 days
Result:	Inhibited oxidative stress, reduces MDA concentrations and increases the GSH and catalase levels.

Animal Model:	Swiss-albino healthy male mice[2]
Dosage:	24 mg/kg
Administration:	Subcutaneous injection; daily, for 7 days
Result:	Inhibitd cisplatin-induced inflammation, decreased TNF-α, including IL-6, IL-1β, MCP-1, ICAM-1, and TGF-β1 levels and increases IL-10 concentrations, reduced caspase 3 in cisplatin injected mice kidneys.

Animal Model:	Tg197 transgenic mouse model[3]
Dosage:	1 and 10 mg/kg
Administration:	Intraperitoneal injection;for 4 weeks
Result:	Reduced the arthritic index.

REFERENCES

[1]. Ueda N, et, al. The cytotoxic effects of certolizumab pegol and golimumab mediated by transmembrane tumor necrosis factor α. Inflamm Bowel Dis. 2013

May;19(6):1224-31.

[2]. Pavitrakar V, et, al. Amelioration of Cisplatin-induced Renal Inflammation by Recombinant Human Golimumab in Mice. Curr Pharm Biotechnol. 2022;23(7):970-977.

[3]. Shealy DJ, et, al. Characterization of golimumab, a human monoclonal antibody specific for human tumor necrosis factor α. MAbs. 2010 Jul-Aug;2(4):428-39.

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