Demcizumab

Cat. No.: HY-P99261

CAS No.: 1243262-17-0

Target: Notch

Pathway: Neuronal Signaling; Stem Cell/Wnt

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

BIOLOGICAL ACTIVITY

Description

Demcizumab (OMP 21M18) is an anti-DLL4 monoclonal antibody. Demcizumab is a potent inhibitor of the Notch pathway. Demcizumab alone or in combination with chemotherapy is effective in various cancer models[1][2][3].

IC_{50} & Target

DLL4[1]

In Vitro

Demcizumab (0-100 μg/mL) binds to human DLL4 but not murine DLL4, and blocks DLL4 binding to Notch1 receptor in a FACS-binding assay[3].

Demcizumab (20 μg/mL, 48 h) reduces HES1 and DTX1 mRNA expression in PDTALL cells[4].

Demcizumab (0-80 μg/mL, 1 or 2 or 3 days) promotes cell death and early apoptosis in PDTALL13 cells[4].

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

Cell Viability Assay[4]

Cell Line:	PDTALL13 (patient-derived T-ALL 13) cell
Concentration:	0, 0.5, 1, 5, 10, 20, 40, 80 μg/mL
Incubation Time:	1 or 2 or 3 days
Result:	Dose-dependently inhibited cell viability.

In Vivo

Demcizumab (10 mg/kg, i.p., once a week) together with Irinotecan (7.5 mg/kg) show a significant antitumor effect in $KRAS^{WT}$ and $KRAS^{MT}$ CRC xenografts[2].

Demcizumab is efficacious alone or in combination with Irinotecan (7.5 mg/kg) in OMP-C8 colon tumors[3].

Demcizumab (20 mg/kg/week, i.p.) increases mice survival in irradiated NRG mice injected PDTALL13 cells[4].

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

Animal Model:	$KRAS^{WT}$ and $KRAS^{MT}$ CRC xenografts[2]
Dosage:	10 mg/kg, together with Irinotecan (HY-16562) (7.5 mg/kg)
Administration:	Intraperitoneal injection (i.p.), once a week
Result:	Resulted in tumor regression at day 20.

REFERENCES

[1]. Smith DC, et al. A phase I dose escalation and expansion study of the anticancer stem cell agent demcizumab (anti-DLL4) in patients with previously treated solid tumors. Clin Cancer Res. 2014 Dec 15;20(24):6295-303.

[2]. Fischer M, et al. Anti-DLL4 inhibits growth and reduces tumor-initiating cell frequency in colorectal tumors with oncogenic KRAS mutations. Cancer Res 2011;71:1520-5.

[3]. Hoey T, et al. DLL4 blockade inhibits tumor growth and reduces tumor-initiating cell frequency. Cell Stem Cell 2009;5:168–77.

[4]. Xiong H, et al. Spleen plays a major role in DLL4-driven acute T-cell lymphoblastic leukemia. Theranostics. 2021 Jan 1;11(4):1594-1608.

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