Laquinimod

Cat. No.: HY-13010

CAS No.: 248281-84-7

Molecular Formula: $C_{19}H_{17}ClN_2O_3$

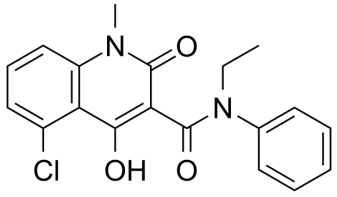
Molecular Weight: 356.8

Target: NF-κB; Apoptosis

Pathway: NF-κB; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (70.07 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8027 mL	14.0135 mL	28.0269 mL
	5 mM	0.5605 mL	2.8027 mL	5.6054 mL
	10 mM	0.2803 mL	1.4013 mL	2.8027 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Laquinimod (ABR-215062), an orally available carboxamide derivative, is a potent immunomodulator which prevents neurodegeneration and inflammation in the central nervous system. Laquinimod reduces astrocytic NF-κB activation to protect from Cuprizone-induced demyelination. Laquinimod has the potential for relapsing remitting (RR) and chronic progressive (CP) forms of multiple sclerosis (MS; RRMS or CPMS) as well as neurodegenerative diseases research[1][2][3][4].

IC_{50} & Target

NF-κB

In Vitro

Laquinimod reverses EAE and inhibits pathogenic T cell immune responses. Laquinimod reverses RR-EAE and inhibits inflammatory T cell responses via a direct effect on myeloid APC. Laquinimod alters myeloid APC subsets and inhibits Th1 and Th17 polarization of myelin-specific T cells. Laquinimod-induced type II (M2) monocytes reverse established EAE[1]. Laquinimod modulates the phenotype of B cells of healthy donors. Laquinimod modulates expression of markers related to regulatory capacity in B cells of RRMS patients. Laquinimod reduces IFNγ cytokine expression in $CD4^+$ T cells[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Laquinimod treatment inhibits donor myelin-specific T cells from transferring EAE to naive recipient mice. In vivo laquinimod treatment alters subpopulations of myeloid antigen presenting cells (APC) that include a decrease in $CD11c^+$ $CD11b^+CD4^+$ dendritic cells (DC) and an elevation of $CD11b^{hi}Gr1^{hi}$ monocytes[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Purified $CD11b^+$ cells from laquinimod- or vehicle-treated mice are cultured with naive $CD4^+$ cells isolated from laquinimod- or vehicle-treated 2D2 mice and antigen (MOG p35-55, 20 μg/mL). Cells are cultured in 96-well microtitre plates at a concentration of 0.25×10^6 cells/mL. Culture medium consisted of RPMI 1640 supplemented with L-glutamine (2 mM), sodium pyruvate (1 mM), penicillin (100 U/mL), streptomycin (0.1 mg/mL), 2-mercaptoethanol (5×10^{-5} M) and 10% (v/v) fetal bovine serum. Cells are incubated for 48 h and pulsed for 18 h with 1 μCi per well of [^{3}H]-thymidine before harvesting. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Seven to 10-week-old female C57BL/6, DBA/1 or SJL/J mice are injected subcutaneously with 50 μg MOG p35-55, 50 μg rMOG or 100 μg PLP p139-151, respectively, in complete Freund's adjuvant. After immunization and 2 days later, mice receive 200 ng (C57BL/6) or 100 ng (SJL/J) pertussis toxin intraperitoneally (i.p.). For adoptive transfer, donor SJL/J mice are immunized as described above and treated daily with laquinimod or vehicle. 10 days later, cells from draining lymph nodes and spleen are isolated, re-stimulated for 48 h (20 μg/mL PLP p139-151), and injected i.p. into naive SJL/J recipients (10^7 cells per mouse). Animals are observed daily and clinical scores are assessed as follows: 0, no signs; 1, decreased tail tone; 2, mild monoparesis or paraparesis; 3, severe paraparesis; 4, paraplegia and/or quadraparesis; and 5, moribund or death. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Neuroimmunol. 2020 Feb 20;342:577195.

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REFERENCES

[1]. Schulze-Topphoff, Ulf., et al. Laquinimod, a quinoline-3-carboxamide, induces type II myeloid cells that modulate central nervous system autoimmunity. PLoS One (2012), 7(3), e33797.

[2]. Toubi E, et al. Laquinimod modulates B cells and their regulatory effects on T cells in Multiple Sclerosis. J Neuroimmunol. 2012 Oct 15;251(1-2):45-54.

[3]. Brück W, et al. Reduced astrocytic NF-κB activation by laquinimod protects from cuprizone-induced demyelination. Acta Neuropathol. 2012 Sep;124(3):411-24.

[4]. Jan Thöne, et al. Laquinimod in the treatment of multiple sclerosis: a review of the data so far. Drug Des Devel Ther. 2016 Mar 14;10:1111-8.

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