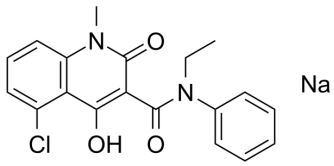
Laquinimod sodium

Cat. No.:	HY-W062904
CAS No.:	248282-07-7
Molecular Formula:	$C_{19}H_{17}ClN_2NaO_3$
Molecular Weight:	379.79
Target:	NF-κB; Apoptosis
Pathway:	NF-κB; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Laquinimod (ABR-215062) sodium, an orally available carboxamide derivative, is a potent immunomodulator which prevents neurodegeneration and inflammation in the central nervous system. Laquinimod sodium reduces astrocytic NF-κB activation to protect from Cuprizone-induced demyelination. Laquinimod sodium 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 sodium 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 sodium 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.

REFERENCES

[1]. 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.

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

[3]. 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.

[4]. 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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