Product Data Sheet

Product Name: MOG (35-55)
Cat. No.: GC17193

Chemical Properties

Cas. No.	149635-73-4		
Chemical Name	(S)-2-((Z)-((2S,3R)-3-amino-1,2-dihydroxy-4-phenylbutylidene)amino)-4-methylpentanoic acid compound with 2,2,2-trifluoroacetic acid (1:1)		
SMILES	CC(C[C@@](/N=C(O)/[C@](O)([H])[C@@](N)([H])CC1=CC=CC=C1)([H])C(O)=O)C.FC(F)(F)C(O)=O		
Formula	$C_{118}H_{177}N_{35}O_{29}S$	M.Wt	2581.97
Solubility	≥ 32.25mg/mL in Water, ≥ 86 mg/mL in DMSO	Storage	Desiccate at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	MOG (35-55)		

Protocol

Cell experiment [1]:

Cell lines

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Preparation Method	To prepare T cell lines specific for MOG (35-55) peptide, C57BL/6 mice were immunized s.c. in the flanks with 0.2 ml of an emulsion containing 200 μg of MOG (35-55) in saline and an equal volume of CFA containing 400 μg Mycobacterium tuberculosis H37RA. Ten days after immunization, lymph node cells were cultured with MOG (35-55) (20 μg/ml) at 8 × 106 cells/ml in stimulation medium (RPMI 1640 medium supplemented with nonessential amino acids, sodium pyruvate, 2-ME, and 10% FBS) for 48 h. The T cells were expanded in medium containing IL-2 (100 U/ml).
Reaction Conditions	20 μg/ml, 5-10 days
Applications	After 5-10 days in culture, the T cell lines responded specifically to MOG (35-55) peptide.

Animal experiment [2]:

Animal models	6-8 weeks old female C57BL/6 mice

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Preparation Method	Mice were immunized subcutaneously at the base of the tail with 100 µl of mouse MOG (35-55) peptide emulsified in complete Freund's adjuvant (CFA). Peptide was dissolved in phosphate-buffered saline (PBS) at 2 mg/ml, mixed at a 1:1 ratio with complete adjuvant (8 mg/ml heat-killed Mycobacterium tuberculosis (H37 RA) in incomplete Freund's adjuvant (IFA)), and emulsified by the syringe-extrusion method with two rubber-free Luer-Lock syringes (Air-Tite) connected by a 3-way stopcock until a stable emulsion was formed (approx. 10 min). Each mouse received 100 µg of peptide (and 400 µg of M. tuberculosis). On the day of immunization and two days later, 250 ng of pertussis toxin in 100 µl PBS was administered intravenously.
Dosage form	Subcutaneous injection, 100 µg in 100 µl
Applications	Immunization of C57BL/6 mice with MOG (35-55) caused inflammation and axon loss in the spinal cord but resulted in only minimal demyelination

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References:

[1]: Ito A, Matejuk A, Hopke C, et al. Transfer of severe experimental autoimmune encephalomyelitis by IL-12-and IL-18-potentiated T cells is estrogen sensitive[J]. The Journal of Immunology, 2003, 170(9): 4802-4809.
[2]: Jones M V, Nguyen T T, Deboy C A, et al. Behavioral and pathological outcomes in MOG 35-55 experimental autoimmune encephalomyelitis[J]. Journal of neuroimmunology, 2008, 199(1-2): 83-93.

Background

MOG (35-55) is a 35-55 fragment of myelin oligodendrocyte glycoprotein[1]. Immunizing mice with MOG (35-55) peptide to induce experimental autoimmune encephalomyelitis (EAE), it causes inflammation (macrophages and CD3+ T lymphocytes), demyelination, and axonal loss [1]. MOG (35-55) is often used to model EAE in mice for evaluating

various drug treatments [2].

Axon loss in the medial dorsal column (fasciculus gracilis), which is often damaged in this model of EAE, was detected as early as 7 days post-immunization (p.i.), with a loss of about 11% of axons in the defined counting area. A further 10% loss was observed by day 12 p.i., at which time behavioral disease was just beginning to become apparent [1]. T lymphocytes were detected infiltrating the spinal cord as early as day 7 p.i. CD3-immunoreactivity increased 5-fold [1]. MOG (35-55) immunized mice developed severe parenchymal infiltration in the spinal cords on day 9, day 13 and day 16, and mice showed heavy infiltration of the cerebral meninges, which prevailed in the region of the hippocampus involving the lateral and the third ventricle [3]. MOG (35-55)-induced EAE showed the cerebellar white matter infiltrates in all mice on days 13 and 16 [3].

References:
[1]. Jones M V, Nguyen T T, Deboy C A, et al. Behavioral and pathological outcomes in MOG 35-55 experimental autoimmune encephalomyelitis[J]. Journal of neuroimmunology, 2008, 199(1-2): 83-93.
[2]. Steinman L, Zamvil S S. Virtues and pitfalls of EAE for the development of therapies for multiple sclerosis[J]. Trends in immunology, 2005, 26(11): 565-571.
[3]. Kuerten S, Kostova-Bales D A, Frenzel L P, et al. MP4-and MOG: 35-55-induced EAE in C57BL/6 mice differentially targets brain, spinal cord and cerebellum[J]. Journal of neuroimmunology, 2007, 189(1-2): 31-40.