Imiquimod

Cat. No.: HY-B0180

CAS No.: 99011-02-6

Molecular Formula: $C_{14}H_{16}N_4$

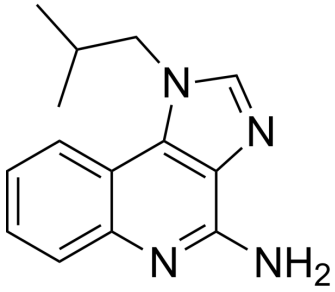
Molecular Weight: 240.3

Target: Toll-like Receptor (TLR); Autophagy; HSV; SARS-CoV

Pathway: Immunology/Inflammation; Autophagy; Anti-infection

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : 2.64 mg/mL (10.99 mM; ultrasonic and adjust pH to 2 with HCl)
DMSO : 2 mg/mL (8.32 mM; ultrasonic and warming and heat to 70°C)
Methanol : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.1615 mL	20.8073 mL	41.6146 mL
5 mM	0.8323 mL	4.1615 mL	8.3229 mL
10 mM	0.4161 mL	2.0807 mL	4.1615 mL

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

BIOLOGICAL ACTIVITY

Description

Imiquimod (R 837), an immune response modifier, is a selective toll like receptor 7 (TLR7) agonist. Imiquimod exhibits antiviral and antitumor effects in vivo. Imiquimod can be used for the research of external genital, perianal warts, cancer and COVID-19[1][2].

IC_{50} & Target		
	TLR7	HSV-1

In Vivo

Imiquimod can be used in animal modeling to construct psoriasis models. In animal models, Imiquimod stimulates the innate immune response by increasing NK cell activity, activating macrophages to secrete cytokines and nitric oxide, and inducing the proliferation and differentiation of B lymphocytes. Imiquimod stimulates the innate immune response through induction, synthesis, and release of cytokines, including interferon-a (IFN-α), interleukin (IL)-6, and tumor necrosis factor (TNF)-α[1].

Lmiquimod (IMQ) is a classic psoriasis modeling agent that induces inflamed scaly skin lesions resembling plaque-type psoriasis. IMQ induces epidermal expression of IL-23, IL-17A, and IL-17F, as well as an increase in splenic Th17 cells. Rats and mice are generally used as animal models[5][6].

Dose reference for IMQ induction[5][6]:

(1) Model animal: BALB/c mice (8-11 week)

Psoriasis Model: 3.125 mg/day, skin, 6 day

(2) Model animals: Male Wistar rats (9–12 weeks old)

Psoriasis Model: 125 mg/day, skin, 5 days IMQ cream preparation [7]:

(1) Prepare BM of lipids

STA and OA were melted at 75 °C followed by the addition of IMQ (3.5% of the BM, w/w) under continuous stirring.

Polysorbate 80 and stearoyl polyoxyl-32 glycerides at a 50:50 ratio were dispersed in water heated separately to 75 °C and added to the BM phase under continuous stirring.

(2) Prepare nanostructured lipid carriers (NLCs)

The mixture was then subjected to high-shear homogenization followed by ultrasonication. The mixture was then rapidly cooled in an ice bath to allow the formation of NLCs.

Induction of psoriasis Model[5]

- Background

  Lmiquimod (IMQ) induces inflamed scaly skin lesions resembling plaque-type psoriasis. IMQ induces epidermal expression of IL-23, IL-17A, and IL-17F, as well as an increase in splenic Th17 cells.

- Specific Mmodeling Methods

  Mice: BALB/c • male • 8-week-old

  Administration: 3.125 mg/day • skin • 6 days

  Note

  IMQ cream preparation [7]:

  (1) Prepare BM of lipids

  STA and OA were melted at 75 °C followed by the addition of IMQ (3.5% of the BM, w/w) under continuous stirring.

  Polysorbate 80 and stearoyl polyoxyl-32 glycerides at a 50:50 ratio were dispersed in water heated separately to 75 °C and added to the BM phase under continuous stirring.

  (2) Prepare nanostructured lipid carriers (NLCs)

  The mixture was then subjected to high-shear homogenization followed by ultrasonication.

  The mixture was then rapidly cooled in an ice bath to allow the formation of NLCs.

- Modeling Indicators

  Behavior Observation: Inducedthe back skin of the mice started to display signs of erythema, scaling, andthickening.

  Resulted in hyperproliferative keratinocytes and adisturbed epidermal differentiation (parakeratosis).

- Opposite Product(s):

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

Animal Model:	BALB/c mice (8-11 week) [5]
Dosage:	3.125 mg/day, 6 day
Administration:	skin
Result:	Induced the back skin of the mice started to display signs of erythema, scaling, and thickening. Resulted in hyperproliferative keratinocytes and a disturbed epidermal differentiation (parakeratosis).

CUSTOMER VALIDATION

- Nat Commun. 2022 Jul 22;13(1):4255.
- Nat Commun. 2016 May 25;7:11724.
- Nucleic Acids Res. 2021 Jan 8;49(D1):D1113-D1121.
- Biomaterials. 2022 Feb 14;282:121411.
- Biomaterials. 2021, 120724.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Leslie van der Fits, et al. Imiquimod-induced psoriasis-like skin inflammation in mice is mediated via the IL-23/IL-17 axis. J Immunol. 2009, 182, 9.

[2]. Ajla Smajlović, et al. Molecular and histopathological profiling of imiquimod induced dermatosis in Swiss Wistar rats: contribution to the rat model for novel anti-psoriasis treatments. Mol Biol Rep. 2021, 48, 5.

[3]. Sangseo Kim, et al. Development and Optimization of Imiquimod-Loaded Nanostructured Lipid Carriers Using a Hybrid Design of Experiments Approach. Int J Nanomedicine. 2023.

[4]. Athina Angelopoulou, et al. Imiquimod - A toll like receptor 7 agonist - Is an ideal option for management of COVID 19. Environ Res. 2020 Sep; 188: 109858.

[5]. Aditya K Gupta, et al. Imiquimod: a review. J Cutan Med Surg. Nov-Dec 2002;6(6):554-60.

[6]. Yuji Kan, et al. Imiquimod suppresses propagation of herpes simplex virus 1 by upregulation of cystatin A via the adenosine receptor A1 pathway. J Virol. 2012 Oct;86(19):10338-46.

[7]. Michael P Schön, et al. The small antitumoral immune response modifier imiquimod interacts with adenosine receptor signaling in a TLR7- and TLR8-independent fashion. J Invest Dermatol. 2006 Jun;126(6):1338-47.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA