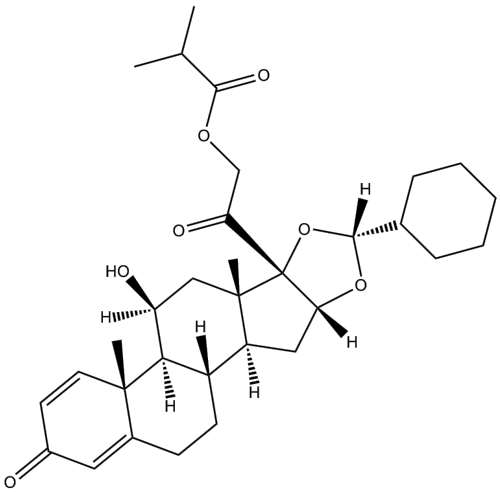
Product Data Sheet

Product Name: Ciclesonide
Cat. No.: GC17571

Chemical Properties

Cas. No.	126544-47-6		
Chemical Name	2-((6aR,6bS,7S,8aS,8bS,10R,11aR,12aS,12bS)-10-cyclohexyl-7-hydroxy-6a,8a-dimethyl-4-oxo-2,4,6a,6b,7,8,8a,8b,11a,12,12a,12b-dodecahydro-1H-naphtho[2',1':4,5]indeno[1,2-d][1,3]dioxol-8b-yl)-2-oxoethyl isobutyrate		
SMILES	CC(C(OCC([C@@]12[C@@](O[C@@](O2)([H])C3CCCCC3)([H])C[C@@]4([H])[C@]([C@]5([H])[C@](O)([H])C[C@@]41C)([H])CCC6=CC(C=C[C@@]65C)=O)=O)=O)C		
Formula	$C_{32}H_{44}O_7$	M.Wt	540.69
Solubility	≥ 15.8mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C 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



Protocol

Product Data Sheet

Cell experiment: Peripheral blood mononuclear cells are isolated from non atopic and atopic asthmatic children sensitized to Phleum pratense (PhlP5). Proliferation toward Candida albicans or PhlP5 in the presence of Ciclesonide or Desisobutyryl-ciclesonide (0.003-3.0 μM) is evaluated as [3H]thymidine incorporation. Modulation of PhlP5-specific T-cell blasts proliferation and PhlP5-induced interleukin 4 expression by Ciclesonide and Desisobutyryl-ciclesonide are measured[1].

References:

[1]. Silvestri M, et al. Ciclesonide modulates in vitro allergen-driven activation of blood mononuclear cells and allergen-specific T-cell blasts. Immunol Lett. 2012 Jan 30;141(2):190-6.

Background

Ciclesonide(RPR251526) is a glucocorticoid used to treat obstructive airway diseases.Target: Glucocorticoid ReceptorCiclesonide (CIC) is an inhaled glucocorticosteroid. CIC (5 microM) was rapidly hydrolyzed by NHBE cells (approximately 30% conversion at 4h), with almost complete conversion by 24h. In liver and NHBE cells, major involvement of cytosolic carboxylesterases, with some contribution by cholinesterases, was indicated. The highest level of conversion was found in the liver, the site of inactivation of des-CIC through rapid oxidation by cytochrome P450. Carboxylesterases in bronchial epithelial cells probably contribute significantly to the conversion to des-CIC in the target organ, whereas low systemic levels of des-CIC are a result of the high metabolic clearance by the liver following CIC inhalation [1]. Ciclesonide may have some advantage although it is not as yet licensed in all countries for paediatric use [2].

References:

[1]. Mutch, E., et al., The role of esterases in the metabolism of ciclesonide to desisobutyryl-ciclesonide in human tissue. Biochem Pharmacol, 2007. 73(10): p. 1657-64.
[2]. Barnes, N.C., The properties of inhaled corticosteroids: similarities and differences. Prim Care Respir J, 2007. 16(3): p. 149-54.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA