Product Data Sheet

Product Name: COG 133
Cat. No.: GP10010

Chemical Properties

Cas. No.	514200-66-9		
Formula	$C_{97}H_{181}N_{37}O_{19}$	M.Wt	2169.73
Solubility	≥ 217mg/mL in DMSO; ≤1mg/ml in Water (Need ultrasonic)	Storage	Store 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	COG 133		

Protocol

Cell experiment [1]:

Cell lines	IEC-6 cell monolayers
Preparation method	Soluble to 1 mg/ml in sterile water. General tips for obtaining a higher concentration: Please warm the tube at 37 ℃ for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below -20℃ for several months.
Reacting condition	0.02, 0.2, 2, 5, 10, and 20 μM, 24 h

Caution: Product has not been fully validated for medical applications. For research use only.
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Address: 10292 Central Ave. #205, Montclair, CA, USA

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Applications	COG 133 (0.02, 0.2, and 2.0 μM) improved cell numbers in glutamine free media. In IEC-6 cells, COG 133 (0.2-20 μM) improved cell migration following 5-FU challenge, reaching the same migration level as controls.

Animal experiment [1]:

Animal models	5-fluorouracil (5-FU)-challenged Swiss mice, C57BL6J ApoE knock-out mice
Dosage form	Intraperitoneal injection, 0.3, 1, and 3 μM, twice daily for 4 days
Application	COG 133 (3 μM) significantly increased the mitotic crypt numbers in C57BL6J wild-type animals. COG 133 treatment improved crypt architecture and reduced lamina propria inflammation. COG 133 (3 μM) significantly reduced the intestinal MPO levels. COG 133 partially decreased TNF-α level in the proximal small intestine from 5-FU-treated mice. In both wild-type and ApoE knock-out mice, COG 133 (3 μM) reverted the increase in Tunel-positive cells in the proximal intestine induced by 5-FU. COG 133 caused higher expression of the NF-κB in the improved intestinal mucosa.
Other notes	Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

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References:

[1]. Azevedo O G R, Oliveira R A C, Oliveira B C, et al. Apolipoprotein E COG 133 mimetic peptide improves 5-fluorouracil-induced intestinal mucositis[J]. BMC gastroenterology, 2012, 12(1): 35.

Background

COG 133, ($C_{97}H_{181}N_{37}O_{19}$), a peptide with the sequence Ac-Leu-Arg-Val-Arg-Leu-Ala-Ser-His-Leu-Arg-Lys-Leu-Arg-Lys-Arg-Leu-Leu-amide,MW= 2169.73.Apolipoprotein E (ApoE) is 299 amino acids long and transports lipoproteins[1], fat-soluble vitamins, and cholesterol into the lymph system and then into the blood. It is synthesized principally in the liver, but has also been found in other tissues such as the brain, kidneys, and spleen. In the nervous system, non-neuronal cell types, most notably astroglia and microglia, are the primary producers of APOE, while neurons preferentially express the receptors for APOE. There are seven currently identified mammalian receptors for APOE which belong to the evolutionarily conserved low density lipoprotein receptor gene family.APOE was initially recognized for its importance in lipoprotein metabolism and cardiovascular disease[2]. Defects in APOE result in familial dysbetalipoproteinemia aka type III hyperlipoproteinemia (HLP III), in which increased plasma cholesterol and triglycerides are the consequence of impaired clearance of chylomicron, VLDL and LDL remnants[*citation needed*]. More recently, it has been studied for its role in several biological processes not directly related to lipoprotein transport, including Alzheimer's disease (AD), immunoregulation, and cognition[3].

References:

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1. Singh PP, Singh M, Mastana SS (2002). "Genetic variation of apolipoproteins in North Indians". Hum. Biol. 74 (5): 673-82.
2. van den Elzen P, Garg S, LeÒn L, Brigl M, Leadbetter EA, Gumperz JE, Dascher CC, Cheng TY, Sacks FM, Illarionov PA, Besra GS, Kent SC, Moody DB, BrennerMB. (2005). "Apolipoprotein-mediated pathways of lipid antigen presentation.". Nature 437 (7060): 906-10.
3. Zhang HL, Wu J, Zhu J (2010). "The Role of Apolipoprotein E in Guillain-BarrÀ Syndrome and Experimental Autoimmune Neuritis". J. Biomed. Biotechnol. 2010: 357412.

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