Human serum albumin [70024-90-7]

#Cat: NB-64-69053-25mg Size: 25mg
#Cat: NB-64-69053-50ml Size: 50ml
#Cat: NB-64-69053-100mg Size: 100mg
#Cat: NB-64-69053-500mg Size: 500mg

Chemical Properties

Cas No: 70024-90-7
Formula:
Molecular weight:
Appearance: no data available
Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Human serum albumin (HSA), the most abundant protein in plasma, significantly contributes to plasma oncotic pressure and exhibits antioxidant, anticoagulant, antiinflammatory, and anti-platelet aggregation activities, as well as colloid osmotic action. It can block the inhibitory effect of GML on human T cells, providing a protective function, and is associated with cardiovascular diseases. HSA partially prevents LPSinduced oxidative stress and the upregulation of NF-κB, NF-κB, and peroxynitrite (ONOO−) in the vascular wall, helping to reduce blood pressure [1] [2] [3].
In vitro	Human serum albumin (HSA) at a concentration of 5 μM for 0-90 minutes can bind with Glycerol monolaurate (GML), with a dissociation constant (K d) of 1.4 μM, and it serves to protect T cell function by preventing GML from inhibiting human T cells. HSA at concentrations of 0.01, 0.05, and 0.1 μM over 0-24 hours can mitigate the GML-induced phosphorylation of AKT at threonine 308 and serine 473, as well as the formation of LAT, PLC-γ1, and AKT microclusters, while restoring the production of IFN-γ, IL-2, IL-10, and TNF-α in GML-treated cells. Western Blot analysis in activated peripheral blood T cells at a 0.05 μM concentration with incubation times of 0, 2, 5, and 15 minutes showed that it affected GML's ability to inhibit AKT phosphorylation. RT-PCR studies, using activated peripheral blood T cells with concentrations of 0.1, 0.05, and 0.005 μM over 24 hours, demonstrated the restoration of IFN-γ, IL-2, IL-10, and TNF-α production in GML-treated cells.
In vivo	In a study on male Swiss mouse models, human serum albumin administered intravenously at a dose of 10 ml/kg once at 1 hour and once at 5 hours effectively mitigated the oxidative stress and nitric oxide overproduction induced by lipopolysaccharide (LPS, 50 mg/kg via intraperitoneal injection). The treatment successfully inhibited iNOS expression and formation of peroxynitrite (ONOO−), reduced the upregulation of NF-κB, and prevented the decline in vascular response to phenylephrine, muscle tone, and endothelial function caused by endotoxin.