

Recombinant Human Sonic Hedgehog/Shh

Accession #	Q15465
Alternate Names	Shh;HHG1; HHG-1; HLP3; HPE3; MCOPCB5;Sonic Hedgehog; TPT; TPTPS
Source	Human embryonic kidney cell, HEK293-derived human Sonic Hedgehog/Shh protein
Protein sequence	Cys24-Gly197
M.Wt	19.6 kDa
Appearance	Solution protein.
Stability & Storage	Avoid repeated freeze-thaw cycles. It is recommended that the protein be aliquoted for optimal storage. 12 months from date of receipt, -20 to -70 °C as supplied.
Concentration	0.2 mg/mL
Formulation	Dissolved in sterile PBS buffer.
Reconstitution	We recommend that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. This solution can be diluted into other aqueous buffers.
Biological Activity	Measured by its ability to induce alkaline phosphatase production by C3H10T1/2 mouse embryonic fibroblast cells. The EC50 for this effect is typically 5-15 ng/mL.
Shipping Condition	Shipping with dry ice.
Handling	Centrifuge the vial prior to opening.
Usage	For Research Use Only! Not to be used in humans.

Quality Control

Purity	> 95%, determined by SDS-PAGE.
Endotoxin	<0.010 EU per 1 ug of the protein by the LAL method.

Description

Sonic Hedgehog (Shh) is expressed in embryonic tissues that are critical for the patterning of the developing central nervous system, somite, and limb. It is also involved in whisker, hair, foregut, tooth, and bone development. Shh regulates neural and hematopoietic stem cell fate and is important for thymocyte differentiation and proliferation as well as T cell determination. In adult tissue Shh is associated with cancer development and tissue remodeling following injury ^[1-3]. Human Shh encodes a 462 amino acid (aa) precursor protein that is autocatalytically processed to yield a non-glycosylated 19 kDa N-terminal fragment (Shh-N) and a glycosylated 25 kDa C-terminal protein (Shh-C) ^[4]. Shh-C, which is responsible for the intramolecular processing of Shh, is rapidly degraded following Shh proteolysis ^[5]. Shh-N is highly conserved, sharing >98% aa identity between mouse, human, rat, canine, porcine, and chicken Shh-N. Shh-N can be palmitoylated at its N-terminal cysteine and

modified by cholesterol addition at its C-terminus [6]. These modifications contribute to the membrane tethering of Shh as well as its assembly into various sized multimers [6-9]. Lipid modification and multimerization greatly increase Shh-N receptor binding affinity and signaling potency [5, 6, 8, 9]. Monomeric and multimeric Shh can be released from the plasma membrane by the cooperative action of DISP1, SCUBE2, and TACE/ADAM17 [10-12]. Modifications also extend the effective range of Shh functionality and are required for the development of protein gradients important in tissue morphogenesis [9, 13]. Canonical signaling of Shh is mediated by a multicomponent receptor complex that includes Patched (PTCH1, PTCH2) and Smoothened (SMO) [14].

Reference

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