

## **Technical Data Sheet**

## Flogen® Recombinant Human Noggin (rHuNoggin)

Catalog Number: PGR0108-009
Source: Escherichia coli.

**Molecular Weight:** Approximately 46.2 kDa non-disulfide-linked homodimer consisting of

two 206 amino acid polypeptide chains.

**Quantity:**  $5\mu g/20\mu g/1mg$ 

AA Sequence: MQHYLHIRPA PSDNLPLVDL IEHPDPIFDP KEKDLNETLL

RSLLGGHYDP GFMATSPPED RPGGGGGAAG GAEDLAELDQ LLRQRPSGAM PSEIKGLEFS EGLAQGKKQR LSKKLRRKLQ

MWLWSQTFCP VLYAWNDLGS RFWPRYVKVG

SCFSKRSCSV PEGMVCKPSK SVHLTVLRWR CQRRGGQRCG

WIPIQYPIIS ECKCSC

**Purity:** >95% by SDS-PAGE and HPLC analyses.

**Biological Activity:** Fully biologically active when compared to standard. The ED<sub>50</sub>

determined by inhibiting BMP-4-induced alkaline phosphatase production of murine ATDC5 cells is less than 80 ng/ml, corresponding to a specific

activity of  $> 1.3 \times 10^4$  IU/mg in the presence of 5ng/ml BMP-4

**Appearance:** Sterile Filtered White lyophilized (freeze-dried) powder.

Formulation: Lyophilized from a 0.2µm filtered concentrated solution in 30%

acetonitrile, 0.1% TFA.

**Endotoxin:** Less than 1EU/μg of rHuNoggin as determined by LAL method.

**Reconstitution:** We recommend that this vial be briefly centrifuged prior to opening to

bring the contents to the bottom. Reconstitute in 10mM HAc to a concentration of 0.1-1.0 mg/mL. Stock solutions should be apportioned into working aliquots and stored at <-20°C. Further dilutions should be

made in appropriate buffered solutions.

Storage: This lyophilized preparation is stable at 2-8°C, but should be kept at

-20°C for long term storage, preferably desiccated. Upon reconstitution, the preparation is stable for up to one week at 2-8°C. For maximal stability, apportion the reconstituted preparation into working aliquots and

store at -20°C to -70°C. Avoid repeated freeze/thaw cycles.

**Usage:** This material is for research, laboratory or further evaluation purposes.

NOT FOR HUMAN USE.

## Human Noggin

Noggin belongs to a group of diffusible proteins which bind to ligands of the TGF- $\beta$  family and regulate their activity by inhibiting their access to signaling receptors. Noggin was originally identified as a BMP-4 antagonist whose action is critical for proper formation of the head and other dorsal structures. Consequently, Noggin has been shown to modulate the activities of other BMPs including BMP-2,-7,-13, and -14. Targeted deletion of Noggin in mice results in prenatal death and recessive phenotype displaying a severely malformed skeletal system. Conversely, transgenic mice over-expressing Noggin in mature osteoblasts display impaired osteoblastic differentiation, reduced bone formation, and severe osteoporosis.