

**proBDNF Protein (Mut-mouse)**  
**proBrain-Derived Neurotrophic Factor (Mutated-mouse), Recombinant, E. coli**  
**Catalog # PG10002**

## Specification

### proBDNF Protein (Mut-mouse) - Product Information

### proBDNF Protein (Mut-mouse) - Additional Information

**Storage**  
-20°C

#### Precautions

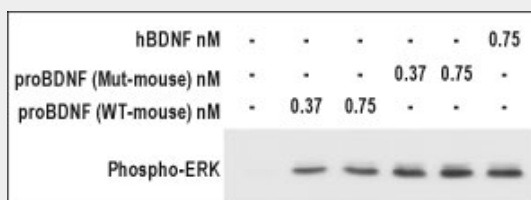
proBDNF Protein (Mut-mouse) is for research use only and not for use in diagnostic or therapeutic procedures.

### proBDNF Protein (Mut-mouse) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### proBDNF Protein (Mut-mouse) - Images



proBDNF (Mut-mouse) - Absent proBDNF (Mut-mouse) mediates ERK1/2 activation in TrkB transfected HEK-293 cells. One day post transfection, the cells were stimulated with 0.2 nM hBDNF, 0.2 nM proBDNF (WT-mouse) and 0.2 nM proBDNF (Mut-mouse). Development of neurites was visualized by fluorescence microscopy after 4 days.

### proBDNF Protein (Mut-mouse) - Background

BDNF is a neurotrophic factor produced by proteolytic cleavage of its precursor, proBDNF. The biologically relevant form of the protein was thought to be the mature form, BDNF, which has been shown to affect the development of motoneurons, modulate synaptic transmission and affect

axonal branching, dendrite growth and synapse number.<sup>1</sup> These actions are mediated via the binding of BDNF to TrkB. Different actions are mediated by the binding of BDNF to p75 such as neuronal process retraction<sup>2</sup> and neuronal apoptosis.<sup>3</sup> The precursor form was thought to be important for the correct folding, secretion and trafficking of the mature protein. A single-nucleotide polymorphism (Val66 to Met) in the pro-domain of the human BDNF gene impairs intracellular trafficking and regulated secretion of BDNF in primary cortical neurons and neurosecretory cells but not in endothelial and vascular cells.<sup>4</sup> This has been shown to affect memory and lead to abnormal hippocampal function in humans.<sup>5</sup> The finding that proBDNF and not mature BDNF is the preferred ligand for p75<sup>6</sup> has ushered in a new era which reexamines the biological roles of the two forms. Contradictory biological roles for proBDNF have been proposed. It has been shown to be a pro-apoptotic ligand for sympathetic neurons<sup>7</sup> expressing both p75 and sortlin, and to be involved in LTD<sup>8</sup>. On the other hand it has also been shown to elicit prototypical TrkB responses in biological assays, such as TrkB tyrosine phosphorylation, and activation of ERK1/2.<sup>9</sup> Binding of both proBDNF and mature BDNF to TrkB has been proposed to be via the R103 residue in the mature portion.<sup>9</sup> In addition, the question of the relative abundance of the precursor vs the mature form has been investigated. In brain homogenates a mixture of proBDNF and mature BDNF has been found.<sup>10,11</sup> The secretion of proBDNF from cortical neurons has been shown.<sup>7</sup> Most of the work showing secretion of proBDNF was done in cell lines overexpressing full length BDNF constructs.<sup>4, 6,12,13</sup> It has been argued<sup>14</sup> that this model misrepresents the authentic situation in vivo, since in the transfected systems the secretion of proBDNF results from overloading the limited capacity of the processing machinery. These authors argue that in fact proBDNF is a transient intermediate and is rapidly converted intracellularly to mature BDNF.

#### **proBDNF Protein (Mut-mouse) - References**

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