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# **Product Information**

# Monoclonal Anti-Protein Tyrosine Phosphatase, PEST, clone AG25

produced in mouse, purified immunoglobulin

Catalog Number P9109

Synonym: Anti-PTP PEST

# **Product Description**

Monoclonal Anti-Protein Tyrosine Phosphatase PEST (mouse IgG1 isotype) is derived from the hybridoma produced by the fusion of mouse myeloma cells and splenocytes from a BALB/c mouse immunized with full-length recombinant PTP PEST. The antibody is purified from tissue culture supernatant using immobilized Protein G.

Monoclonal Anti-Protein Tyrosine Phosphatase PEST recognizes PTP PEST isoforms in all mammalian species tested by immunoblotting and immunoprecipitation.

Protein phosphorylation and dephosphorylation are central mechanisms that mediate signal transduction events involved in a wide range of cellular processes. Protein phosphatases are considered to play a crucial role in the regulation of protein phosphorylation by reversing the action of protein kinases. Protein phosphatases are present in all eukaryotic cells and regulate several cellular processes such as cell-cycle progression, transcription, cell growth, differentiation and apoptosis. The protein phosphatases can be divided into two main groups, protein tyrosine phosphatases (PTPs) and protein serine/threonine phosphatases (PPs), which remove phosphate from proteins/peptides containing phosphotyrosine (pTyr) or phosphoserine/phosphothreonine (pSer/pThr), respectively. An additional group consists of dual specificity pTyr and pSer/pThr phosphatases, an example of which is the MAP Kinase Phosphatase family.

Of special importance among the phosphatases is the role of the PTPs in controlling cell growth, differentiation and oncogenesis. Several of the PTPs are known to control the function of growth factor receptors, many of which are tyrosine kinases encoded by oncogenes.

PTPs can be further subdivided into receptor transmembrane-type PTPs and non-receptor, intracellular PTPs. The receptor PTPs (e.g., LAR, CD45, PTP  $\alpha$ ,  $\beta$ ,  $\delta$ ,  $\mu$ ,  $\kappa$ , etc.) contain a general structure of membrane receptor with an extracellular domain, a single transmembrane domain and one or two tandem repeats of a conserved PTP catalytic domain (250 amino acid residues). The extracellular domain may contain functional domains such as IgG-like and fibronectin type III (Fn-III) repeats. The non-receptor intracellular PTPs (e.g., PTP -PEST, PTP1B, cdc25, SH-PTP1, SH-PTP2, MEG, PTP -Bas, etc.) contain a conserved PTP catalytic domain (250 amino acid residues) and additional domains, such as SH2 domains. The phosphatases can be further subdivided on the basis of their cellular localization. requirement for Ca<sup>2+</sup> or Mg<sup>2+</sup>, and sensitivity to specific inhibitors.

PTP PEST is an 88 kDa cytosolic protein tyrosine phosphatase which is ubiquitously expressed in mammalian tissues. PTP PEST exhibits a high specific activity when assayed *in vitro* with artificial tyrosine-phosphorylated substrates. PTP PEST is subject to regulation via phosphorylation of Ser<sup>39</sup> by both protein kinase C and protein kinase A.

## Reagent

Supplied in phosphate buffered saline with 0.08% sodium azide.

#### **Precautions and Disclaimer**

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

### Storage/Stability

Store at –20 °C. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

#### **Product Profile**

For immunoblotting and immunoprecipitation, it is recommended to determine optimal working dilutions by titration test.

#### References

- 1. Cho, H. et al., *Biochemistry*, **30**, 6210 (1991).
- 2. Ng, D.H. et al., *J. Immunol. Methods*, **179**, 177 (1994).
- 3. Gjorloff-Wingren, A. et al., *Eur. J. Immunol.*, **30(8)**, 2412 (2000).

ADM, PHC 11/11-1