

## PTPN22

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC

**Recommended Dilution:**WB 1:500 - 1:2000 IHC 1:50 - 1:200

**Calculated MW:**92kDa

**Observed MW:**Refer to Figures

**Immunogen:**

Recombinant protein of human PTPN22

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Concentration:**

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**Synonym:**

LYP; LYP1; LYP2; PEP; PTPN8; PTPN22;

**Catalog #:**A1406

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**26191

**Isotype:**IgG

**Swiss Prot:**Q9Y2R2

**Purity:**Affinity purification

For research use only.

**Background:**

PTPN22 (Lyp/PEP) is a cytoplasmic phosphatase expressed by hematopoietic cells (1,2). PTPN22 associates with the tyrosine kinase Csk to inhibit T cell receptor signaling through inactivation of Src kinases (3,4). Csk phosphorylates Src kinases on an inhibitory tyrosine, while PTPN22 dephosphorylates an activating site (4). PTPN22(-/-) mice have higher levels of activated Lck than wild-type, resulting in greater T cell expansion and increased serum antibody levels (5). Research studies have shown that a single-nucleotide polymorphism, 1858T of the PTPN22 gene which encodes the amino acid substitution R620W, confers increased risk for multiple autoimmune diseases including type I diabetes, rheumatoid arthritis, systemic lupus erythematosus, and Graves disease (6-9). Interestingly, although the R620W substitution disrupts the interaction between Csk and PTPN22, it is actually a gain-of-function mutation resulting in increased phosphatase activity (6,10,11). Recent evidence suggests that the autoimmune phenotype associated with the R620W variant is the result of increased calpain-mediated degradation and decreased protein levels of PTPN22 (12).

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