

## EEF2

**Reactivity:** Human Mouse Rat

**Tested applications:** WB IHC IF

**Recommended Dilution:** WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:20 - 1:100

**Calculated MW:** 95kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human EEF2

**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:**

EEF-2; EF2;

**Catalog #:** A2068

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 1938

**Isotype:** IgG

**Swiss Prot:** P13639

**Purity:** Affinity purification

For research use only.

**Background:**

Eukaryotic elongation factor 2 (eEF2) catalyzes the translocation of peptidyl-tRNA from the A site to the P site on the ribosome. It has been shown that phosphorylation of eEF2 at threonine 56 by eEF2 kinase inhibits its activity (1-4). eEF2 kinase is normally dependent on Ca<sup>2+</sup> ions and calmodulin (5,6). eEF2 kinase can also be activated by PKA in response to elevated cAMP levels (7-9), which are generally increased in stress- or starvation-related conditions. A variety of treatments known to raise intracellular Ca<sup>2+</sup> or cAMP levels have been shown to result in increased phosphorylation of eEF2, and thus to inhibit peptide-chain elongation. The inactive phosphorylated eEF2 can be converted to its active nonphosphorylated form by a protein phosphatase, most likely a form of protein phosphatase-2A (PP-2A). Insulin, which activates protein synthesis in a wide range of cell types, induces rapid dephosphorylation of eEF2 through mTOR signaling and may involve modulation of the activity of the PP-2A or the eEF2 kinase or both (10).

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