

## SMAD5

**Reactivity:** Human Mouse Rat

**Tested applications:** WB IHC IF FC

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

**Calculated MW:** 52kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human SMAD5

**Storage Buffer:**

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

**Concentration:**

1 mg/ml

**Synonym:**

DKFZp781C1895; DKFZp781O1323; Dwfc; JV5-1; MADH5;

**Catalog #:** A1947

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 4090

**Isotype:** IgG

**Swiss Prot:** Q99717

**Purity:** Affinity purification

For research use only.

**Background:**

Bone morphogenetic proteins (BMPs) constitute a large family of signaling molecules that regulate a wide range of critical processes including morphogenesis, cell-fate determination, proliferation, differentiation, and apoptosis (1,2). BMP receptors are members of the TGF- family of Ser/Thr kinase receptors. Ligand binding induces multimerization, autophosphorylation, and activation of these receptors (3-5). They subsequently phosphorylate Smad1 at Ser463 and Ser465 in the carboxy-terminal motif SSXS, as well as Smad5 and Smad8 at their corresponding sites. These phosphorylated Smads dimerize with the coactivating Smad4 and translocate to the nucleus, where they stimulate transcription of target genes (5). MAP kinases and CDKs 8 and 9 phosphorylate residues in the linker region of Smad1, including Ser206. The phosphorylation of Ser206 recruits Smurf1 to the linker region and leads to the degradation of Smad1 (6). Phosphorylation of this site also promotes Smad1 transcriptional action by recruiting YAP to the linker region (7).

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