

## RAF1

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC IF

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

**Calculated MW:**73kDa

**Observed MW:**Refer to Figures

**Immunogen:**

A synthetic peptide of human RAF1

**Storage Buffer:**

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

**Synonym:**

RAF1;CRAF;NS5;Raf-1;c-Raf ;

**Catalog #:**A11198

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**5894

**Isotype:**IgG

**Swiss Prot:**P04049

**Purity:**Affinity purification

For research use only.

**Background:**

A-Raf, B-Raf and RAF1 (Raf-1) are the main effectors recruited by GTP-bound Ras to activate the MEK-MAP kinase pathway (1). Activation of RAF1 is the best understood and involves phosphorylation at multiple activating sites including Ser338, Tyr341, Thr491, Ser494, Ser497 and Ser499 (2). p21-activated protein kinase (PAK) has been shown to phosphorylate RAF1 at Ser338 and the Src family phosphorylates Tyr341 to induce RAF1 activity (3,4). Ser338 of RAF1 corresponds to similar sites in A-Raf (Ser299) and B-Raf (Ser445), although this site is constitutively phosphorylated in B-Raf (5). Inhibitory 14-3-3 binding sites on RAF1 (Ser259 and Ser621) can be phosphorylated by Akt and AMPK, respectively (6,7). While A-Raf, B-Raf and RAF1 are similar in sequence and function, differential regulation has been observed (8). Of particular interest, B-Raf contains three consensus Akt phosphorylation sites (Ser364, Ser428 and Thr439) and lacks a site equivalent to Tyr341 of RAF1 (8,9). The B-Raf mutation V600E results in elevated kinase activity and is commonly found in malignant melanoma (10). Six residues of RAF1 (Ser29, Ser43, Ser289, Ser296, Ser301 and Ser642) become hyperphosphorylated in a manner consistent with RAF1 inactivation. The hyperphosphorylation of these six sites is dependent on downstream MEK signaling and renders RAF1 unresponsive to subsequent activation events (11).

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