

## SIRT1

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC IF

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

**Calculated MW:**82kDa

**Observed MW:**Refer to Figures

**Immunogen:**

A synthetic peptide of human SIRT1

**Storage Buffer:**

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

**Concentration:**

bfg

**Synonym:**

SIRT1;SIR2L1 ;

**Catalog #:**A0230

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**23411

**Isotype:**IgG

**Swiss Prot:**Q96EB6

**Purity:**Affinity purification

For research use only.

**Background:**

The Silent Information Regulator (SIR2) family of genes is a highly conserved group of genes that encode nicotinamide adenine dinucleotide (NAD)-dependent protein deacetylases, also known as class III histone deacetylases. The first discovered and best characterized of these genes is *Saccharomyces cerevisiae* SIR2, which is involved in silencing of mating type loci, telomere maintenance, DNA damage response, and cell aging (1). SirT1, the mammalian ortholog of Sir2, is a nuclear protein implicated in the regulation of many cellular processes, including apoptosis, cellular senescence, endocrine signaling, glucose homeostasis, aging, and longevity. Targets of SirT1 include acetylated p53 (2,3), p300 (4), Ku70 (5), forkhead (FoxO) transcription factors (5,6), PPAR (7), and the PPAR coactivator-1 (PGC-1) protein (8). Deacetylation of p53 and FoxO transcription factors represses apoptosis and increases cell survival (2,3,5,6). Deacetylation of PPAR and PGC-1 regulates the gluconeogenic/glycolytic pathways in the liver and fat mobilization in white adipocytes in response to fasting (7,8). SirT1 deacetylase activity is inhibited by nicotinamide and activated by resveratrol. In addition, SirT1 activity may be regulated by phosphorylation, since it is phosphorylated on Ser27 and Ser47 in vivo, however, the function of these phosphorylation sites has not yet been determined (9).

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