

## CCND3

**Reactivity:** Human Mouse Rat

**Tested applications:** WB IHC

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

**Calculated MW:** 33kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human CCND3

**Storage Buffer:**

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

**Concentration:**

m

**Synonym:**

CCND3

**Catalog #:** A1084

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 896

**Isotype:** IgG

**Swiss Prot:** P30281

**Purity:** Affinity purification

For research use only.

**Background:**

Activity of the cyclin-dependent kinases CDK4 and CDK6 is regulated by T-loop phosphorylation, by the abundance of their cyclin partners (the D-type cyclins), and by association with CDK inhibitors of the Cip/Kip or INK family of proteins (1). The inactive ternary complex of cyclin D/CDK4 and p27 Kip1 requires extracellular mitogenic stimuli for the release and degradation of p27 concomitant with a rise in cyclin D levels to affect progression through the restriction point and Rb-dependent entry into S-phase (2). The active complex of cyclin D/CDK4 targets the retinoblastoma protein for phosphorylation, allowing the release of E2F transcription factors that activate G1/S-phase gene expression (3). Levels of cyclin D protein drop upon withdrawal of growth factors through downregulation of protein expression and phosphorylation-dependent degradation (4). Although the D-type cyclins are not fully redundant, cyclin D3, like D1, plays a prominent role in differentiation and proliferation, which correlates with higher expression levels of cyclin D3 in various cancers (5).

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