



Product Information

Anti-Cdk2

produced in rabbit, IgG fraction of antiserum

Catalog Number **C5223**

Product Description

Anti-Cdk2 (cyclin-dependent kinase 2) is produced in rabbit using as immunogen a synthetic peptide (QDVTKPVPHLRL) corresponding to amino acids 287–298 of human cdk2. The antibody is supplied as protein A purified rabbit IgG.

Anti-Cdk2 specifically reacts with human cdk2 (p33^{cdk2}) protein kinase and is predicted to cross-react with mouse, rat, non-human primates, and hamster. Anti-Cdk2 may be used for immunoprecipitation and immunoblotting (33 kDa) of cdk2.

During the cell cycle of most somatic cells, DNA synthesis (S-phase) and mitosis (M-phase) are separated by two gap phases (G₁ and G₂) of varying duration. Thus, a typical eukaryotic cell sequentially passes through G₁, S, G₂, and M and back into G₁ during a single cycle.¹ Regulation of cell cycle progression in eukaryotic cells depends on the expression of proteins called cyclins.² These proteins form complexes with several different cyclin-dependent kinases (CDKs). Within the complexes, the cyclin subunit serves a regulatory role, whereas, the CDKs have a catalytic protein kinase activity.³ Complexes of cyclins and CDKs play a key role in cell cycle control. The eukaryotic cell cycle is regulated by the sequential activation of CDKs. The association of members of the cyclin family with the kinase subunit forms an active kinase, which can initiate M phase of mitosis and meiosis, or function as key regulators of each step of the cell cycle by phosphorylation of several cellular targets. Two general mechanisms, protein phosphorylation and association with regulatory subunits, which include the cyclins and the CDK inhibitors (CKIs), regulate the catalytic activity of CDKs. Several mammalian CDK inhibitors have been identified including p16^{INK4a}, p15^{INK4b}, p18^{INK4c}, p19^{INK4d}, p21^{Cip1}, p27^{Kip1}, and p57^{Kip2}.

Cdk2, in mammalian cells, is responsible for catalyzing the transition from G₁ to S phase. Cyclin A binds to one side of the Cdk2 catalytic cleft, inducing large conformational changes in its PSTAIRE helix and T-loop. These changes activate the kinase by realigning active site residues and relieving the steric blockade at the entrance of the catalytic cleft. It is only after phosphorylation of cdk2 at Thr¹⁶⁰ by cdk-activating kinase (CAK) that the cyclin-cdk2 complex is fully functional.⁴ Phosphorylation of cdk2 at Thr¹⁴ and/or Tyr¹⁵ inhibits kinase activity. These phosphorylations are dominant, so that activation of cdk2 requires dephosphorylation at these sites by the phosphatase Cdc25.

Reagent

The product is supplied as 100 µg of protein A purified rabbit IgG in 100 µL of 0.1 M Tris-glycine, pH 7.4, with 20% glycerol, 0.15 M sodium chloride, and 0.05% sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Due to the sodium azide content a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at –20 °C. Aliquot to avoid repeated freezing and thawing. Do not store in frost-free freezer. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

Product Profile

2–4 µg of Anti-Cdk2 will immunoprecipitate cdk2 from 0.5–1 mg of a nuclear extract of human HeLa cells.

A working concentration of 0.5–2 µg/mL of Anti-Cdk2 is determined by immunoblotting using a nuclear extract of human HeLa cells, anti-rabbit IgG conjugated to peroxidase, and enhanced chemiluminescence.

Note: In order to obtain the best results and assay sensitivity in various techniques and preparations, we recommend determining optimal working dilutions by titration.

References

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4. Connell-Crowley, L., et al., *Mol. Biol. Cell*, **4**, 79 (1993).
5. Wang, X., et al., *J. Biol. Chem.*, **276**, 44504-44511 (2001).
6. Rani, C.S., et al., *J. Biol. Chem.*, **272**, 10777-10783 (1997).

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