IBL Data Sheet

Code No. 18646

**Anti-Human
14-3-3 σProtein (N) Rabbit IgG Affinity Purify**

Volume   : 100 μg

Introduction   : The cell cycle checkpoint plays an important role in maintaining the integrity of cells. The G1 arrest is due to p53-mediated induction of the cyclin-dependent kinase inhibitor p21WAF1/CIP1/SDI1, but the basis for the G2 arrest is unknown. Recently, one of the 14-3-3 protein family members, 14-3-3sigma, was shown to be regulated by p53 and to play a role in the G2-M-phase checkpoint.
14-3-3 sigma was cloned by expression cloning through cyclin-dependent kinase 2 (CDK2) association. 14-3-3 sigma shares cyclin-CDK2 binding motifs with different cell cycle regulators, including p107, p130, p21 (CIP1), p27 (KIP1), and p57 (KIP2), and is associated with cyclin.

Antigen   : Synthetic peptide of N-terminus portion for human 14-3-3 Sigma protein

Purification : Affinity purified with synthetic peptide

Form   : Lyophilized product from 1% BSA in PBS containing 0.05%NaN$_3$

How to use   : 1.0 mL deionized water will be added to the product

Dilution   : PBS (pH7.4) containing 1% BSA, 0.05%NaN$_3$

Stability   : Lyophilized product, 5 years at 2 – 8 ℃
                 : Solution, 2 years at –20 ℃

Application : This antibody can be stained in formalin fixed paraffin embedded tissues after microwave treatment (10 Min, 10mM Citrate Buffer, pH 6.0) by several Immunohistochemical techniques such as Avidin Bition Complex (ABC) Method. The optimal dilution is about 5 μg/mL, however, the dilution rate should be optimized by each laboratories.

                 : This antibody can be used for western blotting in concentration of 1 - 3 μg/mL.

Specificity : Human 14-3-3 Sigma specific. Non-cross react with 14-3-3 Beta, Gamma, Epsilon, Zeta, Eta, and Tau. (Confirmed by western blotting)

Reference   : 1. Hermeking H., et al. 14-3-3 sigma is a p53-regulated inhibitor of G2/M progression. Mol. Cell. 1 (1), 3-11, 1997
                 2. Chan T. A., et al. 14-3-3 σis required to prevent mitotic catastophe after DNA damage. Nature **401** (7), 616- 620, 1999