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ProductInformation

ANTI-PKR (ET-20)
Developed in Rabbit, IgG Fraction of Antiserum

Product Number **P 0244**

Product Description

Anti-Interferon-induced, double-stranded RNA-activated protein kinase (PKR) is developed in rabbit using as immunogen a synthetic peptide near the C-terminus of human PKR (amino acids 531-550), conjugated to KLH. The antiserum is purified by ion exchange chromatography. Anti-PKR specifically recognizes human PKR (68 kDa). It has been used in immunoblotting applications.

PKR is a ubiquitously expressed serine/threonine protein kinase (68 kDa in human) that is induced by IFN-γ and activated by dsRNA and stress signals.[1] It is a major mediator of the antiviral and anti-proliferative activities of interferons.[1-5] PKR binding to dsRNA, induces a conformational change that leads to PKR autophosphorylation and activation. Activated PKR phosphorylates its substrates, which include the α-subunit of translation initiator factor eIF-2 (eIF-2α), thereby inhibiting translation and protein synthesis.[3,6] The antiviral activity of PKR is mediated, in part, through phosphorylation of eIF-2α, which results in the sequestration of the recycling factor eIF-2B in an inactive complex with eIF-2-GDP.

In addition to its role as a regulator of translation, PKR is involved in the control of cell proliferation, differentiation, tumor suppression, apoptosis and cell cycle progression.[3,6-8] PKR has been identified as the signal transducer in cell stress signaling pathways leading to NFκB activation.[9,10] PKR mediates the activation of signal transduction pathways by pro-inflammatory stimuli, including bacterial lipopoly-saccharide (LPS), TNF-α and interleukin-1. In response to dsRNAs, PKR activates IκB kinase (IKK), leading to the degradation of the inhibitors IκBα and IκBβ and the concomitant release of NFκB. PKR activation of NFκB in cells by TNF-α and IFN-γ has been shown to be mediated by both NFκB-inducing kinase (NIK) and IKK.[11] Cells derived from PKR knockout mice or expressing dominant negative forms of PKR, display a defective induction of interferon regulatory factor 1 (IRF-1) and/or NFκB by IFN-γ or dsRNA, implicating PKR in these signaling pathways.[12] PKR interacts with p53 and phosphorylates it on Ser[392] to restrict cell proliferation, thus enhancing the transcriptional activity of this tumor suppressor protein.[13] In addition, overexpression of PKR causes induction of p53, resulting in apoptosis in mammalian cells, suggesting a role for PKR in tumor suppression. Peel et al., showed that human PKR preferentially bound mutant Huntingtin RNA transcripts suggesting a role for PKR activation in the Huntington disease process.[14]

Reagent

The Anti-PKR is provided in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

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. Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

Store at 2–8 °C. For extended storage, upon initial thawing, freeze in working aliquots. Do not store infrost-free freezers. Avoid repeated freezing andthawing to prevent denaturing the antibody. Samples at working dilution should be discarded if not used within 12 hours.

Product Profile

A recommended working dilution of 1:2,000 is determined by immunoblotting, using a whole cell extract of the human epitheloid carcinoma HeLa cell line induced with interferon-γ.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working dilutions by titration test.

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