Anti-Human Chk2

Checkpoint kinase 2 (CHK2), a serine/threonine kinase, is an integral protein in the cellular response to DNA damage. Its primary functions are regulating cell cycle progression, DNA repair, or the initiation of apoptosis if the DNA damage is severe and irreparable. CHK2 DNA damage signaling is activated in response to various types of DNA damage and acts in different DNA repair pathways, including Homologous Recombination (HR), Non-Homologous End Joining (NHRJ), and Base Excision Repair (BER) and Nucleotide Excision Repair (NER). CHK2 phosphorylates and activates proteins within the DNA within the aforementioned repair pathways, such as BRCA1, p53-binding protein 1 (53BP1), and breast cancer susceptibility gene 2 (BRCA2). By promoting the activity of these proteins, CHK2 enhances the cell's ability to repair DNA damage and maintain genomic integrity. CHK2 helps to arrest the cell cycle at checkpoints in response to DNA damage. It phosphorylates and activates several downstream targets, including checkpoint protein p53 and the CDC25 family of phosphatases. Activation of p53 leads to transcriptional activation of genes implicated in cell cycle arrest and apoptosis, while inhibition of CDC25 prevents cell cycle progression. In cases where DNA damage is severe and repair is not feasible, CHK2 triggers apoptosis by phosphorylating and activating pro-apoptotic proteins such as p53 and BCL2-associated X protein (BAX). This phosphorylation and activation lead to the activation of the apoptotic cascade and the elimination of damaged cells to prevent the propagation of genetic abnormalities.

Overall, CHK2 functions as a critical regulator of multiple DNA repair pathways, coordinating the cellular response to DNA damage, acting in multiple DNA repair pathways, regulating the cell cycle, and activating Apoptosis to maintain genomic stability. Dysregulation of CHK2-mediated DNA repair processes may lead to genomic instability and increase the risk of cancer development.

References:

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