



A p105-based NF-(kappa) B Super repressor

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Technology description

C04290: A p105-based NF-(kappa) B Super repressor

Technical Details:

Nuclear factor (NF)-kB is a family of transcription factors that regulate immune and inflammatory responses, programmed cell death (apoptosis), and developmental processes. In normal cells, NF-kB is latent, and is activated transiently by stimuli and stresses. However, under pathological conditions such as inflammation, oncogenesis and pathogen infections, NF-kB is persistently activated. One profound consequence of NF-kB activation is the expression of several cytokines and cellular factors that mediate cell's resistance to apoptosis. Therefore, persistent activation of NF-kB is thought to contribute to uncontrolled growth the cancer cells, and the chemo- or anti-cancer drug-resistance of the cancer cells. Because NF-kB also regulates the expression of pro-inflammatory cytokines, persistent activation of NF-kB often enhances the inflammations. Effective repression of NF-kB, hence, may curtail or relieve many pathological conditions and facilitate drug- or chemotherapy reagent-mediated killing of cancer cells. The current available NF-kB inhibitors target the prototypical NF-kB complex p50/RelA only. Since other NF-kB family members also contribute to cell regulations, and these members can be dominant in different tissues or pathological conditions, broad inhibition of NF-kB is clinically important. JHU researchers have developed a super repressor that achieves this goal. Our reagent effectively inhibits all NF-kB species in the cell. Comparing to the currently available NF-kB super repressor IkBa(sr), our reagent is more potent at facilitating tumor necrosis factor-mediated killing of skin tumor cells.

Institution

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