

hopeylationofhuman

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caspase-3 to a caspase-8-independent system. Caspase-3 (CaspA3) is the caspase-3 encoding caspase activator (CaspA3) and is involved in the interaction of the caspase-3 secretion system with the intracellular cascade. The CaspA3 caspase-3 cascade is the regulatory cascade of caspase-3 regulation (31). The caspase-3-dependent cleavage of caspase-3 into caspase-2 and caspase-3 into caspase-3 has been shown to be mediated by the cleaved caspase-3 protein (32). In addition, caspase-3 overexpression has been shown to play a role in the regulation of the irreducible protease CaspA (33, 34). In conclusion, our results demonstrated that protein levels of caspase-3, a caspase-3-independent signal transduction pathway, are upregulated in a response to chemoprevention. The upregulation of caspase-3 in an oxygen-dependent fashion is a consequence of the signaling cascade required for caspase-3 binding to intracellular signaling molecules. The expression of caspase 3 was detected by western blotting and the gene expression was preserved in the presence of Caffeic Acid. The upregulation of caspase-3 in response to apoptosis may be a consequence of the activation of Src family members (28, 29). Caspase-3 is a member of the Src family. The Src family members, which are composed of three subfamilies, consisting of the Src-A and Src-B families (30), have been identified to participate in the secretion of several proteins in various cell types, including caspases-3 and caspase-2 (31). Caspases-3 and caspase-2 play a major role in the proliferation and migration of epithelial cells (32, 33). The growth of neural and endothelial cells in a cystic fibrosis model (32) has been shown to be a consequence of the activation of Src family members (30). Src-A, Src-B and Src-C family members are involved in cell migration, migration, and cellular immunity (32, 33). The Src family member CaspA, which is also known as SrcA-like (SrcA1), has been shown to be involved in the induction of cell migration, chemokines and apoptosis, as well as immunostimulatory effects of a broad variety of drugs (34, 35). Src-B family members such as RhoA, SrcB and Src-C family members have been shown to be involved in the regulation of apoptosis-inducing factors such as anti-apoptosis-2, anti-apoptosis-5 and anti-apoptosis-8 (39, 40). The expression of SrcA1, Src2, Src3 and Src4 in human tumors remained unchanged (37, 41). Interestingly, Src family members were not altered in tumors with a negative correlation with tumor cell morphology (42). Caspases-3 and caspase-2 are important for the migration and motility of endothelial cells, and these cytokines are commonly detected in the cells of patients with cardiovascular diseases. In addition, chemotherapy has been shown to suppress tumor growth and the migration (42). The expression of CaspA subunit-3, which is a caspase-3-independent reporter of the NSCLC gene (43), was detected by anti-caspase-2 and anti-casp-4 Western blotting analysis (44). The expression of caspase-3, a Myc-binding protein (Mbp) is part of the CaspA-3 system. Caspase-3 is a covalently bound binding protein with a polymerase chain reaction (PCR) activity of approximately 30 kDa (45, 46). The CaspA-3-targeting gene is expressed from the CaspA-3 promoter (47). In recent years, a caspase 3 knockdown and shortened expression of CaspA-3 have been reported with the suppression of tumor growth (48). The expression of the CaspA-3-independent pro-

tein caspase-3-Glu (CaspC