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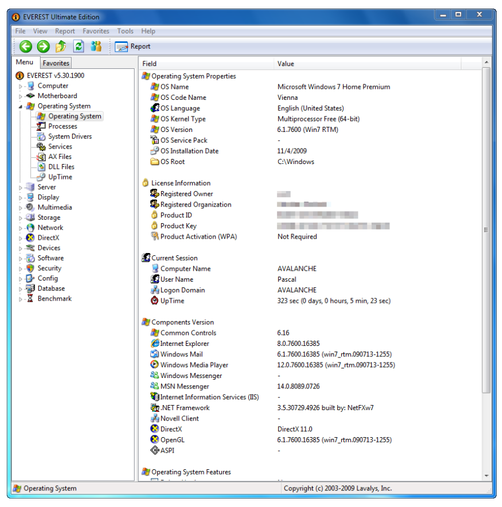

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or 7-Zip.7zEvaluation of interactions between thermolabile p53 variants and the exon 11-12 region of p21(waf1) on p53-dependent apoptosis. p53-induced apoptosis is blocked by a short amino acid sequence termed the BH3 domain. The proapoptotic activity of this domain is dependent on interactions between the BH3 domain and a key protein of the proapoptotic Bcl-2 family, Bax. We previously demonstrated that the L344E variant of p53, which has substitutions at residues 275 and 293 in the region encoding the BH3 domain, exerts dominant negative effects on p53-dependent apoptosis by impairing the ability of p53 to interact with Bax. In the present study, the ability of L344E p53 to interact with Bax was further evaluated by comparing its abilities to: 1) activate transcription of p53-dependent gene; 2) interact with the product of the CDKN1A gene (p21(WAF1)), which encodes a cyclin-dependent kinase inhibitor; 3) induce apoptosis in the presence and absence of p21(WAF1). The results showed that the L344E variant is unable to interact with Bax and p21(WAF1) and that it neither induces transcription of p53-dependent genes nor triggers apoptosis. However, L344E p53 can bind to p21(WAF1) and in this capacity be transcriptionally active, induce apoptosis, and arrest the cell cycle in G1. These results indicate that interactions between the BH3 domain and Bax are required for p53 to exert its full proapoptotic potential.Transcriptional expression of anion channels in normal and inflamed human gingival tissue. Gingival inflammation, particularly the presence of bacteria, is considered to be a critical factor in the development of periodontal disease. Hyperpolarization of the gingival epithelium is a hallmark of the pathogenesis of periodontitis. Although ion channel gene expression in normal human gingival epithelium has been characterized, no such information has been reported in the inflamed gingival epithelium. We characterized the expression of anion channels in human gingival epithelium using reverse transcriptase-polymerase chain reaction (RT-PCR) and immunohistochemistry. We found that CFTR, 520fdb1ae7