Martech Rcd Activation Management Software

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Cdc-e/DYRK1A is involved in transducing the biological processes of cell division, survival, and differentiation into a chemical signal such as cyclin A (Betts et al., 2000; Matassa et al., 1998; Massol et al., 2000). CDKs are important regulators of cell cycle control that are known to control cell cycle checkpoints that are involved in DNA damage repair and programmed cell death (Chalk et al., 1991; Li and Nurse, 1996). CDK2 is a key regulator of cell cycle G1-S transition which is inhibited by Rb and by p16 and p19 proteins (Sheaff et al., 1997). Cdc-e/DYRK1A and cdk1 both regulate transcription by phosphorylating the transcription factor E2F and, in association with PCNA, promote DNA repair (Clark et al., 2001). Therefore, one possible mechanism for Cdc-e/DYRK1A-mediated transformation may involve an interaction with cell cycle control pathways such as p53, p16/Rb and E2F/PCNA. This is not surprising since all of these pathways converge at the level of the cell cycle and control its progression through the S-G2/M phase. The p53 tumor suppressor gene plays a central role in the control of the cell cycle in response to DNA damage. Cellular p53 is a short-lived protein whose half-life is controlled by the activity of an E3 ubiquitin ligase complex (Mendoza et al., 2000). Under normal conditions, the p53 protein levels are low. However, after DNA damage, p53 is stabilized by the accumulation of a large amount of p53 protein. p53 is active and can induce cell cycle arrest, apoptosis or senescence. In addition to the p53-dependent pathway, several other pathways lead to p53 stabilization. One of these pathways involves cdk2 and cyclin E (Breslow et al., 1999; Stein et al., 1997). Cdc-e/DYRK1A might be a downstream target of p53, the most commonly mutated tumor suppressor in human cancers. p53 is known to be mutated in virtually all human tumors (Ravdin and Basehoar, 1998). Some of the most frequent mutations are in the C-terminal DNA-binding domain. The function of the mutated p53 protein is defective due to a loss of either of 82157476af