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Download Machine. 2017 Hindi 720p HDRip x264 AAC - Downloadhub Torrent in HD.Cloned human c-abl proto-oncogene protein tyrosine kinase: modulation by chemically induced transformation and by aryl hydrocarbon-receptor interactions. The human c-abl proto-oncogene encodes a 170,000 molecular weight protein tyrosine kinase (PTK) that is the product of a reciprocal translocation between chromosomes 9 and 22 t(9;22)(g34;g11). A human glioma cell line transfected with the functional c-abl PTK was treated with 2-acetylaminofluorene (2-AAF) to evaluate whether the 2-AAFinduced transformation of these cells could be correlated with any expression of the c-abl PTK at the protein level. The transformed cells expressed the c-abl PTK, as shown by immunoprecipitation and autoradiography using a specific anti-PTK antibody. PTK activity was assayed by the ability of the cells to phosphorylate exogenous tyrosine-containing peptide substrates. The PTK activity level in 2-AAF-treated transformed cells was about one-half that of the normal cells. The level of PTK activity in both transformed and normal cells was markedly increased by a tryptic peptide containing a kinase-inducible tyrosine site (pY) in a rabbit reticulocyte lysate. An added advantage of the assay is that the pY peptide competes with exogenous substrates for the PTK. Competitive inhibition of the PTK by pY was used as a monitor of modulation of the PTK by the transformation process and by the presence of aryl hydrocarbon-receptor (AhR) ligands, dioxin and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). A 31,000 molecular weight protein, p31C, was also identified as being phosphorylated on its pY site by the PTK. The AhR was found to inhibit the PTK activity by a more than 70% in all transformed cells, including p31C, in the presence of TCDD. These results demonstrate the regulatable PTK activity, particularly in the transformed cells, and demonstrate the ability of AhR ligands to modulate the PTK activity in both transformed and nontrans



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