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This item is in the category "Diversions\Science & Technology\General\Science Fiction\Voyages & Travels". The seller is "godsgift" and is located in 1231 Griffin Ave. A mutation in the cytoplasmic domain of K-ras that does not affect signaling competency but blocks membrane association and is associated with the formation of liver tumors. It is well-established that the normal growth suppressing function of the K-ras oncogene is mediated by its tyrosine-specific protein kinase activity. However, the cytoplasmic domain of K-

ras, which contains a putative binding site for guanine nucleotide binding proteins, has a low level of intrinsic protein kinase activity and was thought to mediate signal transduction solely through guanine nucleotide binding protein binding. Here we show that the C-terminal region of the K-ras cytoplasmic domain contains a sequence (amino acids 186 to 189) that inhibits tyrosine-specific protein kinase activity, and that the majority of the C-terminal region of the cytoplasmic domain of K-ras (amino acids 186 to 642) is not required for K-ras signaling. In contrast, only the N-terminal region of the cytoplasmic domain of K-ras (amino acids 187 to 642) is required for K-ras signaling. The catalytically inactive K-ras (G12V) is unable to bind guanine nucleotide binding proteins. Although the K-ras (G12V) protein associates with the plasma membrane, it fails to bind to membranes in the normal environment of a quiescent cell and is unable to catalyze tyrosine phosphorylation. When K-ras (G12V) is over-expressed in a transformed

cell, a major fraction of K-ras (G12V) appears to be associated with membranes but fails to stimulate a signal transduction cascade. Interestingly, the mutant K-ras (G12V) protein is found associated with membranes in all of the liver tissues

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