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Jan 06, 2016 · Moreover, CIN tumors are distinguished by the accumulation of mutations in specific oncogenes and tumor suppressor genes [e.g., APC, KRAS, phosphatidylinositol-4,5-bisphosphate 3-kinase, catalytic subunit alpha (PIK3CA), B-Raf proto-oncogene, serine/threonine kinase (BRAF), SMAD4, and TP53], thereby activating pathways critical for carcinogenesis.

Current concepts in ameloblastoma-targeted therapies in B-raf proto-oncogene serine/threonine kinase V600E mutation: Systematic review

Rogelio González-González, Sandra López-Verdín, Jesús Lavalle-Carrasco, Nelly Molina-Frechero, Mario Isiordia-Espinoza, Ramón G Carreón-Burciaga, Ronell Bologna-Molina

Abstract

BACKGROUND

Ameloblastomas are common benign epithelial odontogenic neoplasms that present an aggressive and unpredictable behavior that may modify treatment strategies. Different signaling pathways that participate in the progression of these tumors have been identified. B-raf proto-oncogene serine/threonine kinase (BRAF) is a protein involved in the behavior of ameloblastomas, and it is related to many cell mechanisms. BRAF gene mutations have been identified in ameloblastomas, of which the BRAF V600E (valine substituted by glutamic acid at amino acid 600) mutation has been the most common and

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Raf Family Kinases - ncbi.nlm.nih.gov

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When **B-Raf** mutations were discovered in cancer, 20 a puzzling observation was that while the most frequent **mutation**, **V600E**, massively stimulated **B-Raf kinase** activity, several less frequent mutations activated **B-Raf** only mildly or not at all. 31 However, even the low-activity **B-Raf** mutants could hyperstimulate the ERK pathway. Intriguingly ...

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DNA methylation aberrancies delineate clinically distinct ...

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