

The Role of TP53 Gene Mutations in Oral Cancer: Implications for Early Detection, Residual Cancer Identification, and Surgical Outcomes

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Oral cancer frequently involves mutations in the TP53 gene, which encodes the p53 protein responsible for regulating cell growth and apoptosis. TP53 mutations impair the protein's ability to suppress abnormal cell proliferation and facilitate the development of malignancies. This study explores the impact of TP53 gene mutations on the progression and management of oral and oropharyngeal cancers. We review evidence suggesting that genetic testing for TP53 mutations can enhance early detection of oral cancers, identify residual tumor cells post-surgery, and predict the likelihood of tumor response to surgical interventions. By integrating these genetic insights, clinicians may improve

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TP53 gene mutations are a hallmark of oral cancer, often leading to the loss of the p53 protein's tumor suppressor function. This protein normally acts as a "guardian of the genome" by initiating DNA repair or apoptosis in response to DNA damage. Mutations in the TP53 gene, particularly in the DNA binding domain, can result in a mutant p53 protein that is unable to perform these critical functions. Consequently, cells with TP53 mutations can proliferate unchecked, leading to the development and progression of oral cancer. The presence of TP53 mutations is associated with various clinical outcomes, including a higher risk of local recurrence, distant metastasis, and poorer overall survival. This review discusses the implications of TP53 gene mutations for early detection, residual cancer identification, and surgical outcomes in oral cancer patients. It highlights the potential of TP53 testing as a prognostic tool and the importance of integrating genetic information into clinical decision-making.

Background and Significance

Oral cancer is a leading cause of cancer-related mortality worldwide. The TP53 gene, located on chromosome 17, encodes the p53 protein, which plays a central role in maintaining genomic stability and preventing the development of cancer. Mutations in the TP53 gene are found in approximately 50-90% of oral cancer cases. These mutations can lead to a loss of p53 protein function, which in turn allows for the accumulation of additional genetic alterations and the progression of the disease. The identification of TP53 mutations in oral cancer patients has several implications. First, it can aid in early detection, as TP53 mutations are often present in premalignant lesions. Second, it can help in identifying residual tumor cells after surgery, which may guide further treatment. Finally, it can predict surgical outcomes, as TP53 mutations are associated with a higher risk of recurrence and metastasis. This review explores the role of TP53 gene mutations in oral cancer and discusses the implications for early detection, residual cancer identification, and surgical outcomes.

TP53

Mechanism of TP53 mutation

TP53 is a tumor suppressor gene that encodes a protein that plays a central role in preventing cancer. The protein is normally inactive, but is activated in response to DNA damage. Once activated, the protein can initiate a cascade of events that leads to cell cycle arrest, DNA repair, or apoptosis. Mutations in the TP53 gene can lead to a loss of function of the protein, which can increase the risk of cancer.

Consequences of 53 deficiency

Deficiency of the TP53 protein can lead to a variety of consequences, including an increased risk of cancer, developmental abnormalities, and shortened lifespan. In mice, TP53 deficiency leads to a high incidence of cancer, particularly in the liver and lung. In humans, TP53 mutations are associated with a variety of cancers, including breast, colon, and lung cancer.

TP53 Mutation in Oral and Oropharyngeal Cancers

Frequency of TP53 mutation

TP53 mutations are found in approximately 50% of oral and oropharyngeal cancers.

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