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Peran p53 sebagai jalur kritis pada mekanisme kontrol siklus sel sebagai pencegah terjadinya kanker mulut./ Herlia Nur Istindiah, Elza Ibrahim Auerkari

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Abstrak

In cell cycle control, p53 acts as an emergency brake, where its important checkpoint function is to maintain the genome integrity by preventing the formation and proliferation of mutant cells. P53 activity is increased by DNA damage occurs caused by agents (such as radiation, UV light or drugs) or oncogenes. Mdm2 protein can inhibit the p53 activation, but oncogenes can inhibit Mdm2 or activate p53. If DNA damage occurs, then p53 prevents the cells from replicating their DNA by arresting the cell cycle, so that the cells can repair the damage. Alternatively, p53 instructs the cells to undergo apoptosis by inducing bax gene expression, so that irregular cell growth, and cancer can be avoided. Cancer, including oral cancer, oftenthuolved cells with altered p53. Exogenous factors, such as tobacco and alcohol, presumably plays a role in triggering p53 mutations. Several techniques, such as immunohistochemistry and PCR can be used to investigation ther etiology and development of oral cancer. This paper discusses the role on p53 in preventing the occurrance and proliferation of mutated cells that lead to cancer, including oral cancer.