

## NF-KB - A Key Transcription Factor in Progression of Oral Cancer

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**Received:** July 05, 2021; **Published:** July 29, 2021

### Abstract

Oral cancer is a leading cancer in south East Asia and India due to extensive use of tobacco and alcohol. Tobacco and alcohol induced release of inflammatory mediators activate NF-KB, a key ubiquitous transcription factor involved in tumor progression by activation of inflammatory mediators. This article brief about the role of NF-KB transcription factor in progression of oral cancer.

**Keywords:** Oral Cancer; NF-KB Transcription Factor; Tobacco; Alcohol

### Introduction

Oral cancer killing mankind around the world. In South East Asia India has the largest group around 70% of oral cancer load globally. About 2.25 million oral cancer patients are in India. Around 1.2 million new oral cancer patients are every year adding and around 0.8 million annually die. WHO told Indian oral cancer patients increase five times in next decade. Global oral cancer burden 2012 reports 369,200 new oral cancer cases and 145,328 of oral cancer patients die worldwide annually. According to GLOBOCAN 2018 new lip and oral cancer cases of about 354,864 cases reported worldwide and 177,384 oral cancer patients die every year worldwide [1]. Etiological factors for oral cancer are tobacco use in the form of chewing or smoking, alcohol, viruses such as HPV and EBV.

### NF-KB: Transcription factor in progression of oral cancer

External environmental factors such as tobacco, alcohol, viruses activate IL-1, TNF- $\alpha$  and COX-2 inflammatory mediators and LPS (Lipopolysaccharide) activate NF-KB a key transcription factor. NF-KB a key transcription factor control more than 500 genes present in cytosol of an each cell. NF-KB transcription factor activation in immune cells involved in activation of immune cells involved in development of immunity and inflammation [2,8-12]. Generalized activation of NF-KB involved in cells involved in chronic inflammation and tumor progression. Inflammatory mediators activated by activation of NF-KB, a key transcription factor involved in cell proliferation by (Cyclin D, Cyclin E), cell survival by (BCL-2, BCL-XL), angiogenesis by (IL-8, COX-2, VEGF, HIF-1 $\alpha$ ), genomic instability by (ROS, RNS, AID, iNOS), immune modulation by (IL-4, IL-5, IL-10, IL-13, TGF- $\beta$ ), invasion and metastasis by (Mmp's 2,9, UPA). NF-KB along with STAT-3 transcription factor activated by inflammatory mediators such as L-6, EGF, FGF and PDGF involved in cell proliferation and cell survival [2-7].

NF-KB a key transcription factor antagonize P53 by ROS, RNS, iNOS, AID (Activation induced cytidine deaminase) enzyme expressed by NF-KB transcription factor [12-23]. NF-KB a key transcription factor acts as a key therapeutic target, which is involved in all stages of oral cancer progression eventually improve the overall prognosis in oral cancer patients.

### Conclusion

Understanding of NF-KB a key transcription factor and its activation, mechanisms of actions and its dual role in cancer helps in future therapeutic application and prognostic marker.

### Bibliography

1. Freddie B., *et al.* "Global cancer statistics 2018: Globocon estimates of incidence and mortality worldwide for 36 cancers in 185 countries". *CA: A Cancer Journal for Clinicians* 68.6 (2018): 394-424.
2. Shrihari TG. "Dual role of inflammatory mediators in cancer". *E Cancer Medical Science* 23.11 (2017): 1-9.
3. Coussens LM and Werb Z. "Inflammation and cancer". *Nature* 420 (2002): 860-867.
4. Grivennikov S I., *et al.* "Immunity, Inflammation and cancer". *Cell* 2140 (2010): 883-1013.
5. Glanben L., *et al.* "Chronic inflammation and cytokines in the tumor microenvironment". *Journal of Immunological Research* 6 (2014): 1-20.
6. Nathan C. "Points of control in inflammation". *Nature* 420 (2002): 846-852.
7. Fernandes J V., *et al.* "The role of the mediators of inflammation in cancer development". *Pathology and Oncology Research* 21 (2015): 527-534.
8. Philip M., *et al.* "Inflammation as a tumor promoter in cancer induction". *Seminars in Cancer Biology* 14 (2004): 433-439.
9. Blackwill F and Mantovani A. "Inflammation and cancer: Back to Virchow?" *Lancet* 357 (2001): 539-545.
10. Candido J. "Cancer-related inflammation". *Journal of Clinical Immunology* 33 (2013): 579-584.
11. Ioannis L A., *et al.* "How do cytokines trigger genomic instability?" *Journal of Biomedicine and Biotechnology* 6 (2012): 1-12.
12. Brett B., *et al.* "Tumor induced perturbations of cytokines and immune cell networks". *Biochimicaet BiophysicaActa (BBA)- Reviews on Cancer* 2 (2014): 182-201.
13. Korniluk A., *et al.* "From inflammation to cancer". *Irish Journal of Medical Science* 10 (2016): 45-52.
14. Mantovani A and Sica A. "Macrophages, Innate immunity and cancer: Balance, tolerance and diversity". *Current Opinion in Immunology* 22 (2010): 231-237.
15. Shrihari TG. "Inflammation related cancer-Highlights". *Journal of Carcinogenesis and Mutagenesis* 7 (2016): 1-2.
16. Lippitz BE. "Cytokine patterns in patients with cancer :A systematic review". *The Lancet Oncology* 14 (2013): 218-228.
17. Shrihari TG and Ramesh DNSV. "Chronic inflammation induced immunosuppression in Tumor microenvironment of oral cancer". *Global Journal of Medical Research: Journal of Dentistry and Otolaryngology* 16 (2016): 1-8.

18. Facciabene A. "Tumor hypoxia promotes tolerance and angiogenesis via CCL28 and Treg cells". *Nature* 475 (2011): 226-230.
19. Masako N and Daniel WS. "Multifaceted roles of PGE2 in inflammation and cancer". *Seminars in Immunopathology* 35 (2013): 123-137.
20. Oian BZ and Pollard JW. "Macrophage diversity enhances tumor progression and metastasis". *Cell* 141 (2010): 39-51.
21. Noy R and Pollard JW. "Tumor associated macrophages : From mechanisms to therapy". *Immunity* 41 (2014): 49-61.
22. Schetter AJ. "Inflammation and cancer: Interweaving micro RNA, free radical ,Cytokine and P53 pathways". *Carcinogenesis* 31 (2010): 37-49.
23. Shrihari TG., *et al.* "Potential Co-relation between Chronic Periodotitis and Cancer-An emerging concept". *Gulf Journal of Oncology* 20 (2016): 20-24.

**Volume 20 Issue 8 August 2021**

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