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Association between COX-2 gene polymorphisms and head and neck cancers risk in a Chinese Han population

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Background: COX-2 is an inducible enzyme that catalyzes prostaglandins through inflammatory response, which can involve in autoimmune diseases and cancer pathogenesis. Two potentially functional genetic single nucleotide polymorphisms (SNPs) (COX-2-1195G>A and 8473T>C) were supposed to contribute to HNSCC susceptibility. The aim of this study was to determine the association of these polymorphisms with HNSCC susceptibility in a Chinese Han population.

Methods: In this study, two SNPs were genotyped by Taqman methods in a case-control study including 260 HNSCC patients and 1047 cancer-free controls in Chinese Han population.

Results: We found significant difference in the frequency of alcohol consumption between HNSCC patients and controls (P<0.001), but the genotype frequencies of the two polymorphisms were not significantly different between cases and controls. Further stratified analysis indicated that none of the genotypes was associated with increased risk of HNSCC.

Conclusion: The COX-2-1195G>A and 8473T>C polymorphisms were not involved in the development of HNSCC in the Chinese Han population. However, further perspective studies are warranted to test these findings and further investigate the potential interactions involving the COX-2 polymorphism and HNSCC.

Keywords: COX-2, polymorphism, head and neck squamous cell carcinoma, and susceptibility.

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Novel therapeutic strategy targeting autophagy in cancer

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Recent findings have revealed that a wide range of cancer therapeutic agents activate autophagy, a process that often functions as a pro-survival ("self-cleaning") mechanism responsible for limited efficacy of and resistance to such agents. However, because autophagy can act as a "double-edged sword" in cancer (e.g., in addition to its cytoprotective actions, excessive autophagy can also trigger cell death), whether autophagy should be inhibited or activated in cancer treatment has become the subject of considerable debate. Currently, it is widely accepted that the mechanisms by which autophagy is perturbed may determine the outcome of autophagy-targeting therapies. This lecture will discuss a variety of issues and challenges related to current autophagy-targeting therapeutic strategies. It will then introduce a novel approach targeting the cargo-loading process of selective autophagy. The net effect of this strategy is to disable cargo loading into autophagosomes, a critical step required for efficient removal of misfolded or unfolded proteins by selective autophagy through fusion with lysosomes. It will also present a novel cross-talk between autophagy and apoptosis, via which a cytoprotective autophagic resopnse can be switched to pro-death response to benefit various cancer therapies. These new findings will provide a theoretical basis for an entirely new autophagy-directed strategy which could be immediately translatable to the treatment of human cancers.

Biography

Yun Dai has studied cancer cell biology and targeted therapy for 12+ years, during which he has authored about 70 peer-reviewed papers. He has served on the editorial boards for the Journal of Cancer Research & Therapy and as a peer-reviewer for Cancer Biology & Therapy, Cancer Research, Journal of Cellular and Molecular Medicine, Molecular Cancer Therapeutics, Molecular Pharmacology, PLoS ONE, Cell Proliferation, Food and Chemical Toxicology, Current Cancer Drug Targets, and Cancer Letter. He is an active member of the American Association for Cancer Research and American Society of Hematology, and an executive committee member of US-Chinese Anti-Cancer Association.