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IS19. CHRONIC OBSTRUCTIVE AIRWAY DISEASES AND WORKPLACE EXPOSURES

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Oxygen- and nitrogen-derived reactive species are constantly generated in living organisms by endogenous and exogenous sources. Reactions of reactive species such as free radicals with DNA cause the formation of multiple mutagenic and cytotoxic lesions, leading to genetic instability, which is a hallmark of cancer. DNA repair mechanisms exist in living organisms to repair DNA lesions. Most effective cancer treatments work by causing DNA damage in malignant tumors. Just like in normal cells, however, DNA repair also exist in cancer cells. Thus, understanding of how DNA lesions are repaired is essential for the understanding of cancer development and treatment. Cancer cells develop greater DNA repair capacity than normal cells by overexpressing DNA repair proteins. Increased DNA repair capacity that removes DNA lesions before they become toxic is a major mechanism for development of resistance to therapy. Knowledge of DNA repair capacity and levels of DNA repair proteins may be a predictive biomarker for patient response to therapy, and guide development of novel treatments. DNA repair proteins constitute targets for inhibitors to overcome the therapy resistance. Inhibitors of DNA repair proteins for combination therapy or for monotherapy as single drugs may help selectively kill tumors, potentially leading to personalized therapy. Over the past decade, a variety of inhibitors have been developed for various DNA repair proteins and are being tested in clinical trials. The efficacy of some inhibitors in therapy has been successfully demonstrated in patients. More developments of inhibitors of DNA repair proteins are globally underway to help eradicate cancer.

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