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Can Kinesin Superfamily of Proteins (KIFs) be Preventive Biomarkers for Industrial Carcinogens?

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Description

According to the WHO, 125 million individuals are exposed to asbestos in the workplace across the world, and OSHA estimates that 1.3 million construction and general industry employees in the United States are exposed to asbestos on the job. Asbestos exposure is known to cause a variety of negative health consequences, many of which have long latency periods of up to 20-40 years, including cancer, and there is no evidence that there is a threshold for asbestos' carcinogenic impact.

The World Health Organization (WHO) has increased its estimates of the worldwide burden of asbestos-related diseases to 107,000 yearly fatalities, mostly from lung cancer, mesothelioma, and asbestosis. Given the large number of people who are exposed to asbestos at work, potential biomarkers of exposure and/or early effects for cancer risk from asbestos could have a significant impact on the disease burden by identifying people who are most at risk for cancer and allowing them to be targeted for more aggressive intervention. Based on previous research, it was believed that certain Kinesin superfamily proteins (KIFs) and p53 autoantibodies might be useful biomarkers for asbestos exposure and cancer risk.

There are 45 distinct proteins in the Kinesin superfamily of proteins (KIFs), which are divided into 14 families. KIFs are a family of microtubule-dependent molecular motor proteins with ATPase activity and motion properties. Diverse subtypes of KIFs may play different roles in the cell, although KIF5s and KIF18s are predominantly involved in mitosis. While there haven't been any studies that have identified KIF18A and KIF5A as particular targets for asbestos interaction or impact in cell culture tests, it's possible that they may be affected by changes in the other proteins that control mitosis caused by asbestos exposure.

During cell mitosis, abnormal kinesin expression can affect the equitable distribution of genetic resources amongst daughter cells. Chromosome hyper condensation, abnormal spindle formation, anaphase bridges, faulty cytokinesis, aneuploidy, and mitotic arrest are all possible causes. The loss or gain of genetic material that results from a defective mitotic process can cause a variety of abnormalities in the daughter cells, which can promote carcinogenesis and/or the progression of aggressive behaviour in the tumour cells.

The TP53 tumour suppressor gene is the most often discovered site for genetic mutations in human cancers, which frequently results in an increase in the stability of mutant p53 protein, resulting in its accumulation in cancer cells. These mutations can develop early in the carcinogenic process and typically have a molecular signature dependent on the kind of cancer and the exposure associated with that disease, making p53 an attractive early effect biomarker.

Conclusion

The inactivation of p53 proteins, as well as the accumulation of both mutant and wild type p53 proteins in tissue and blood, can result in the formation of p53 autoantibodies, and there is a link between serum p53 autoantibodies and p53 overexpression in homologous tissues. Before any clinical signs of malignancy, serum p53 autoantibodies have been detected in individuals with a variety of pre-malignant disorders and malignancies, as well as in workers exposed to industrial carcinogens such as asbestos. The researchers examined the correlation between asbestos exposure and changes in KIF5A and KIF18A serum concentrations, as well as p53 autoantibody serum concentrations, to see if they might be used as early detection and prevention biomarkers.