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Perspective

Diseases Associated with Occupational Chemical Exposure and Toxicology

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DESCRIPTION

Emissions from the mining, metallurgical, chemical, and other sectors continue to pose serious problems for indoor and outdoor air quality. Negative health consequences and environmental and occupational illnesses can be caused by workplace factors. Regarding the chemical component, it's significant to notice the lack of high-efficiency filtering devices that provide complete air purification. Industrial employees are at the most risk because they are exposed to the most dangerous byproducts, which are ultrafine particles that are released into the environment and endanger the general public. Additionally, the widespread usage of nanomaterials in engineering, several national economic sectors, and medicine may result in nanoparticle exposure. To create suitable predictive diagnostic approaches, identify premorbid states, design, and put into practise preventative and rehabilitative interventions, it is essential to comprehend the processes causing the detrimental impact of these unfavourable circumstances. The use of everyday things to determine the hazardous effects of chemicals is a global trend in today's toxicology and nanotoxicology study. Compare the effects of the three cancer-causing mineral fibres crocidolite, chrysotile, and erionite on the three kinds of macrophages: non-activated (M0), pro-inflammatory (M1), and alternatively activated (M2).

Three mineral fibres exert their effects through various toxic processes. Crocidolite's biodurability, generation of Reactive Oxygen Species (ROS), cytokines, and DNA damage are the main causes of its harmful effects. Chrysotile that degrades readily has harmful effects linked to the release of hazardous metals and the generation of ROS and cytokines. The toxicity of biopersistent fibrous erionite, which produces less ROS and hazardous metal release but has the ability to disrupt the intracellular homeostasis of significant cations, is explained by other processes. There are similarities in the behaviour of the three macrophage phenotypes' production of pro-inflammatory mediators, notwithstanding their variances. Although the M2 phenotype is recognised as a cell type that is used in the cases of asbestos fibres and erionite to reduce the inflammatory state, helps the process along by providing pro-inflammatory mediators. This study is part of a long-term Italian research project of national interest (PRIN) that was started in 2017 and aims to identify the biochemical processes that cause negative *in vivo* consequences linked to exposure to mineral fibres. Understanding the mechanisms of inhaled mineral fibre toxicity and carcinogenicity represents an essential first step towards a quantitative classification of mineral fibre toxicity/carcinogenicity for preventive medicine and the creation of successful treatment plans for both at-risk workers and the general public. Molecular biology has advanced to the point that it is now possible to investigate transcriptome alterations brought on by exposure to various substances. Next-Generation Sequencing (NGS)-based transcriptomics methods are extremely effective and sensitive and enable the simultaneous investigation of a vast quantity of gene expression. Transcriptomic methods can be extremely useful in revealing the molecular principles behind intricate biological processes. Conduct a comprehensive investigation based on transcriptome profiling that aims to determine how exposure to Toluene Diisocyanate (TDI) affects the expression of all genes worldwide. Dual-Specificity Phosphatase 6 (DUSP6) is one of the genes significantly altered by TDI exposure, according to their transcriptome investigation of human bronchial epithelial cells (BEAS-2B) following TDI therapy. The extracellular signal-regulated kinase 1/2 activity is attenuated by TDI exposure due to transient receptor potential ankyrin 1 receptor activation, which also raises the mRNA level of p53 as well as its protein and activity, which trans-activates DUSP6. The lethal effects of TDI exposure are attenuated by the apoptotic response, suggesting that TDI exposure aids in the survival of cancer cells.

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