ENM-induced pulmonary inflammation: Challenges to Control ENM Exposure, Risks and Their Management

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The promises of engineered nanomaterials (ENM), technologies using ENM as well as consumer products containing them have gained recently increasing attention. They enable new types of products for industrial purposes and consumer use, and are likely to have solutions for energy production, improving of semiconductors, surface coatings, new drugs, and even water purification.

There are hundreds of thousands of different ENM, and the number of technologies utilizing them is vast. However, next to nothing is known of the potential health hazards of possible exposure to of effects of ENM hence rendering their risk assessment and management demanding. Thus, identifying of hazardous ENM among less hazardous or harmless ENM remains difficult, and new approaches need to be found. In this context, the importance of ENM-induced pulmonary inflammation was used to assess the potential health consequences of exposure to titanium dioxide. When mice were exposed via inhalation to 10 mg/m^3 of nanosized titanium dioxide, nanosized titanium dioxide coated by nanosized amorphous silica, or nanosized amorphous silica alone, only nanosized titanium dioxide coated by nanosized amorphous silica could elicit a clear pulmonary inflammation in animals as indicated by increased numbers of inflammatory cells in BAL, and increased levels of proinflammatory mediators. We hypothesize that surface reactivity of the active material, rather than other metrics of the material such as surface area might explain the results because there was no correlation between inflammation and the commonly used metrics.

In another study LPS-primed human primary macrophages were exposed to a variety of different types of multi-walled carbon nanotubes (MWCNT) or crocidolite asbestos, and the role of IL-1β in inflammation was explored. The macrophages were transfected with small interfering RNA for NLRP3 inflammasome and IL-1β was analyzed. Of the studied materials, only long fibrous MWCNT and asbestos induced the secretion of IL-1β, and it was inhibited by gene silencing of the NLRP3 inflammasome suggesting a similar inflammatory mechanism for MWCNT and asbestos. These examples emphasize the diversity of mechanisms behind the harmful effects of different ENM. There is an obvious need to explore means for rapid screening of potentially harmful effects of ENM. These novel methods may provide an avenue for identifying methods for assessing of risks and their management, and for providing understanding of acceptable levels of ENM exposure. Supported by EU FP7 CP-IP 211464 (NANODEVICE).