

Nanotoxicology: Nano Toxicity in Humans

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Abstract

Nanoparticles (NPs) have attracted a lot of attention in the fields of electronics, biology, and astronautics because of their unique physicochemical and electrical characteristics. NPs are materials with at least one dimension of fewer than 100 nanometres that are commercially manufactured (Bahadar et al., 2016; Vishwakarma et al., 2010). In the medical field, drugs, proteins, DNA, and monoclonal antibodies are all being delivered via NPs (Hussain et al., 2021).

Key words: nanotoxicology; physicochemical; biomedical use

Introduction

Although the name "nanotoxicology" may cause concern among the general public, it is a relatively young discipline of toxicology that tackles the information gap about toxicity caused by NPs. This new area of toxicology, according to Priyanka et al., presents techniques to access the toxicity generated by nanoparticles. For toxicity evaluation in people and the environment, this section comprises a fundamental understanding of the physicochemical impacts of NPs and their routes of exposure/uptake processes (Ganguly et al., 2018). The overall purpose of nanotoxicology is to define criteria for making safe nanoparticles. This necessitates a thorough, systemic approach to examining NP toxicity and its impact on cells, tissues, organs, and the entire organism (Donaldson et al., n.d.). The physical and chemical features of NPs, such as size, shape, specific surface area, surface charge, catalytic activity, and the presence or absence of a shell and active groups on the surface, all influence their toxicity (Sukhanova et al., 2018). Because of their small dimensions and unique features, NPs are frequently exploited as nanomedicine and drug nanocarriers. Their toxicity towards normal, healthy human cells, tissues, and organs might be due to their size, shape, surface functional groups, and dose-dependent characteristics. Numerous researchers have discovered that chemically synthesized NPs are more harmful to human cells than biosynthesized NPs with biocompatible surface functional groups due to the presence of synthetic chemicals as surface functional and capping agents. On the other hand, certain biosynthesized NPs can be harmful when they react with cells and disintegrate into simpler forms or accumulate (Egbuna et al., 2021). Nanotoxicology has just recently arisen, years after NPs first boom when many nanomaterials had already been used in a variety of industrial processes and products (Elsaesser & Howard, 2012).

NPs' physicochemical qualities have an impact on how they interact with cells and, as a result, on their total potential toxicity. Understanding these qualities can lead to the creation of NPs that are safer. Recent research has begun to pinpoint the characteristics that make certain NPs more dangerous than others. In theory, particle size might have a role in cytotoxicity. Smaller NPs have a bigger specific surface area (SSA) than larger NPs with the same mass, and hence a more accessible surface area to interact with biological components such as nucleic acids, proteins, fatty acids, and carbohydrates. Because of its tiny size, it is more likely to infiltrate cells and cause harm. Toxicity was discovered to be a function of both size and SSA in certain NPs. When evaluating the number of reactive oxygen species (ROS) generation per surface area within a given size range, the size of anatase TiO₂ was demonstrated to correlate with ROS production (Huang et al., 2017). In summary, the following are the most typical pathways of NPs cytotoxicity (Sukhanova et al., 2018):

1. NPs can induce oxidation by forming reactive oxygen species (ROS) and other free radicals;
2. NPs can perforate cell membranes, causing harm.
3. NPs disrupt intracellular transport and cell division by damaging cytoskeleton components.
4. NPs disrupt transcription and damage DNA, speeding up the mutagenesis process;
5. NPs harm mitochondria and disrupt their metabolism, resulting in an energy imbalance in the cell;
6. NPs prevent the development of lysosomes, impairing autophagy and macromolecule breakdown as well as causing apoptosis;

7. NPs alter the structure of membrane proteins and disrupt the movement of substances into and out of cells, including intercellular transport;

8. NPs trigger the production of inflammatory mediators by disrupting normal cell, tissue, and organ metabolism;

NPs' structure and physicochemical characteristics must be extensively defined and understood to accurately assess NPs'safety. To develop

particular nanoparticle structure-activity/toxicity functional connections, reported harmful effects can be better linked to certain NPs features. Because NPs structural features influence toxicity, evaluating the safety of NPs with substantial changes in physicochemical qualities, as observed in natural and accidental nanoparticles, is considerably more difficult (Yang et al., 2021).

Nanoparticle class and type	Exposure route, dose, duration, number of humans subjects	Adverse effect	Toxicity mechanism	Toxicity assessment	Reference(s)
Incidental Ag	Inhalation NA Chronic 76 human subjects	Genotoxicity	DNA damage in mononuclear leukocytes due to oxidative stress-induced by Ag nanoparticles	Blood analysis for DNA damage using alkaline comet assay and analysis of total antioxidant status, total oxidant status, total thiol, and ceruloplasmin in human blood plasma samples	(Aktepe et al., 2015)
Engineered PEGylated liposomes (Doxil, ~100 nm)	IV 40–306 mg Acute (infusion for 1 h) 29 human subjects	Immunotoxicity	Hypersensitivity reaction and anaphylatoxin release due to complement activation of PEGylated liposomes	Analysis of human blood samples for complement terminal complex (SC5b-9) to correlate complement activation with hypersensitivity reaction	(Chanan-Khan et al., 2003)
Natural Fe ₃ O ₄ (<20 nm)	NA NA Chronic (many years) 22 human subjects	Neurotoxicity	Abnormal, age-associated biomineralization of Fe ₃ O ₄ in the brain	Quantitative magnetometry; correlation between Fe ₃ O ₄ nanoparticle concentration in the human brain and Alzheimer's disease	(Pankhurst et al., 2008)
Incidental Chemically complex mixtures (10–80 nm)	Inhalation 30,000 NPs/cm ³ (>10 times background levels) Acute (6 h/day for 3 days) 17 human subjects	Pulmonary toxicity	Upper airway inflammation and systemic oxidative stress with generation of proinflammatory cytokines	Analysis of 14 cytokines in nasal lavage samples and analysis of 8-OH-dG and creatinine in human urine samples	(Khatri et al., 2013)
Incidental Diesel exhaust nanoparticles (<100 nm)	Inhalation 1.2 × 10 ⁶ NPs/cm ³ Acute (up to 14 days) 16 human subjects	Vascular dysfunction	Increased systolic blood pressure and attenuated vasodilation due to nanoparticle-induced vascular oxidative stress	Measurement of forearm blood flow and blood pressure and biomarker analysis of human blood samples	(Mills et al., 2011)

Table 1: Examples of nanoparticle toxicity in human subjects

Conclusion

Because of their physicochemical and behavioral distinctiveness, nanoparticles have found extensive biomedical use, yet worries about their harmful consequences in the biological system are now attracting the attention of the worldwide health community. This involves the research and comprehension of the impacts based on the cellular and molecular processes by which they produce these effects. As a result, drawing broad generalizations about nanoparticle toxicity is difficult since nanotoxicity is based on complicated interactions between many physicochemical features and the associated biological context. The public is concerned about widespread human exposure to nanoparticles. As a result, improved techniques for assessing the safety of nanoparticles are required. The current state of knowledge on the impacts of nanomaterials on humans is quite limited.

Conflicts Of Interest

The authors declare no conflicts of interest.

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