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A SHORT REVIEW ON THE TOXICITY OF THE NANOMATERIALS

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Abstract

In the last decade, nanoscience has greatly evolved as it has moved from bench-top research to applied technology. In a wide range of consumer products, such as computer components, athletic equipment, sun creams and biomedical applications, nanomaterials are currently used. It is imperative that both staff and end-users be shielded from inhalation of potentially harmful NPs because nanomaterials are increasing a portion of daily consumer goods, manufacturing processes, and medical products. It also implies that NPs may need to be sequestered into goods so that during the life of the product or during recycling, the NPs are not released into the atmosphere.Furthermore, in order to clarify potential toxic effects, non-inhalation routes of NP absorption, including dermal and medical injectables must be researched. To date, fewer studies have examined whether the body can potentially remove nanomaterials to avoid the build-up of particles in tissues or organs.

Keywords: *Athletic equipment, Biomedical,Nanomaterials, Nanoscience, Organ, Tissues..*

I. INTRODUCTION

Numerous scientific and technical fields have recognized major variations in the physico-chemical properties of nanomaterials compared to the bulk process[1]. Nanomedicine is a modern area of science focused on the dramatically improved properties of nanoparticles (NPs) (e.g. semiconducting, metallic, magnetic and polymeric nano-systems), allowing early detection and new therapies for disaster-related diseases such as multiple sclerosis, atherosclerosis and cancer[2][3]. For example, superparamagnetic iron oxide NPs (SPIONs), which are in clinical development as imaging agents,6 and preclinical studies for theranosis applications, are one of the most promising NP systems (i.e. simultaneous diagnosis and treatment). Furthermore, for magnetic marking, cell isolation, hyperthermia and controlled drug release, SPIONs have been used.

For biomedical applications, many commercial nano-agents are already available and several nano-medicine devices are close to receiving final approval for clinical use. NPs are commercially used in items such as electronic components, scratch-free paint, sports equipment, cosmetics, food color additives, and surface coatings in addition to biomedical applications. Our exposure to nanomaterials is therefore important and growing, but there is little awareness of the specific toxicological properties of NPs and their long-term effects on human health. Inhalation, absorption, skin penetration or injections are capable of penetrating the human body, and NPs have the ability to associate over long periods of time with intracellular structures and macromolecules.

The number of publications focused on nanomaterials has increased significantly over the years; however, most publications concentrate on the synthesis and production of novel nanomaterials and less than one percent concentrate on the biological effect of NPs. While the toxicity of many bulk materials is well understood, due to nano-scopic dimensions, it is not known at what concentration or scale they will begin to exhibit new toxicological properties[4]. A significant disparity exists between the available data on the processing of nanomaterials and the assessment of toxicity. The absence of data on toxicity will preclude the secure design of NPs.

Effect of physicochemical properties of NPs on toxicity

In determining the toxic potential of nanomaterials, the characteristic parameters of NPs, including dissolution, chemical composition, size, shape, agglomeration state, crystal structure, specific surface area, surface energy, surface charge, surface morphology and surface coating, influence the biological interaction of NPs, and it is therefore important to evaluate these properties.

Effect of the Size

Particle size and surface area are important features of the substance.

From a toxicological point of view, interactions usually take place on the surface of the NP between nanomaterials and biological organisms. As the size of the particles decreases, the surface area increases exponentially and a larger proportion of the atoms or molecules of the particles will be reflected on the surface rather than inside the material's bulk. Thus, with decreasing scale, the nanomaterial surface becomes more reactive to itself or surrounding biological components, and the possible catalytic surface increases for chemical reactions.

Impact of the shape of particles

Two additional main variables which decide the toxicity of NPs are particle shapes and aspect ratios. Nanomaterials, including fibers, spheres, loops, rings, and planes, may have very distinct shapes. Much of the shape-dependent toxicity information is based on in vitro studies. In vivo, nanomaterial shape-dependent toxicity is typically imparted by its particle shape influence. Particle shapes and aspect ratios are two additional main factors that determine the toxicity of NPs. Nanomaterials, including fibers, spheres, loops, rings, and

planes, may have very distinct shapes.Much of the shape-dependent toxicity information is based on in vitro studies. In vivo, nanomaterials' shape-dependent toxicity is generally imparted by its adverse effect on endocytosis or macrophage clearance, as shape can affect the process of membrane warping during endocytosis or phagocytosis.

II. ASSOCIATED MECHANISM

NP toxicity in the body can be caused by many different mechanisms, but most intracellular and in vivo toxicity from NPs results from the production of excess reactive oxygen species (ROS). During the dissolution of iron-based NPs, which catalyzes ROS generation and the formation of O-O-H and OH radicals from H_2O_2 through the Fenton reaction, one mechanism of NP-induced oxidative stress occurs. In addition, certain inert nanomaterials do not produce spontaneous development of ROS, but are able to induce production of ROS under biological conditions, based on the capacity of the NPs. In order to target mitochondria physiologically, ROS is both essential and potentially destructive. In the modulation of many cellular events, moderate levels of ROS play specific roles, including signal transduction, proliferative response, gene expression and protein redox regulation. High levels of ROS are indicative of oxidative stress and can kill cells via lipid peroxidation, protein modification, DNA disruption, signaling function interference, and gene transcription regulation, eventually resulting in cancer, renal disease, neurodegeneration, cardiovascular or pulmonary disease. In the cell membrane, ROS will steal electrons from lipids, resulting in a decrease in physiological activity and cell death. For example, oxidative stress associated with TiO2 NPs results in early inflammatory responses, such as an increase in polymorphic nuclear cells, disrupted phagocytosis of macrophages, and/or fibro-proliferative changes in rodents. In human endothelial cells, TiO2 NPs may also cause pro-inflammatory effects. It has been shown that carbon NPs cause oxidative stress in the brain cells of fish and pulmonary inflammation in rats. Oxidative stress and apoptosis have been linked with the exposure of human keratinocytes to insoluble carbon NPs.Owing to the high content of unsaturated fatty acids that are vulnerable to per-oxidation, toxicity from ROS can be more severe in the central nervous system (CNS). ROS also plays a function in the production of vasculo-pathies, including those that describe post-angioplasty atherosclerosis, hypertension, and restenosis[5]. Accumulation in the lungs of the NPs.

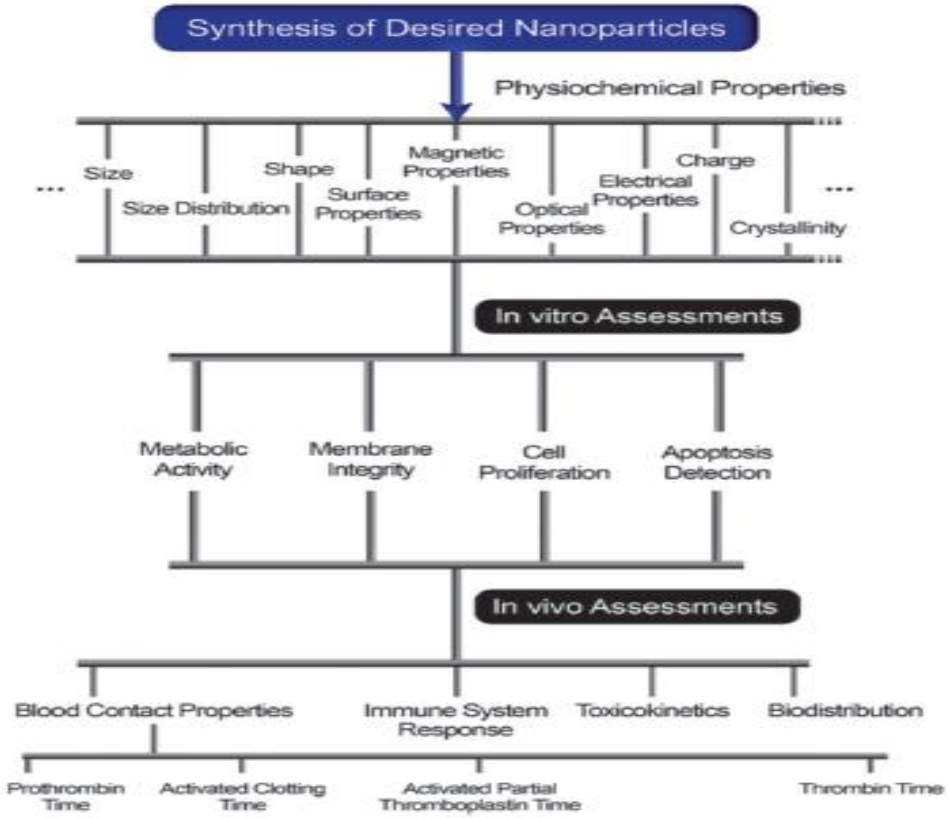

Figure 1: Shows the in vitro and in vivo synthesis for the study of Nano-toxicity

In addition to the prevalence of various phagocytic cells, the reticuloendothelial system (RES) imbalances ROS homeostasis and antioxidant defenses, rendering oxidative stress primary targets for the liver and spleen. As defined by Nel et al, nanoparticle-induced oxidative stress affects cell signaling in three stages[6]. Via transcription factor nrf2, a low level of oxidative stress increases transcription of defense genes. Inflammation signalling through NFkB is triggered by a higher level of oxidative stress and very high levels are correlated with activation of apoptotic pathways and necrosis. The carcinogenic effects of NPs are associated with modifying these signaling pathways in cells. The ROS toxicity of NPs against the cell nucleus and DNA content was investigated by Peterson and Nelson. Double strand breaks, which are considered the most lethal form of oxidative damage to DNA, may result from the accumulation of single strand breaks and oxidative induced base lesions. Mitochondrial DNA (mtDNA) may also be damaged by excess quantities of ROS[7]. Several clinical syndromes such as neurogenic muscle weakness, ataxia and retinitis pigmentosa, mitochondrial encephalo-myopathy lactic acidosis, stroke like episodes, retinitis pigmentosa, cardiac conduction defect and elevated protein of cerebrospinal fluid are reported to be associated with damage to mtDNA.

Some new steps in NP architecture have been taken in order to mitigate ROS impact. Cerium oxide nanoparticles, which contain oxygen defects that scavenge free radicals, have recently been formed. It was found that NPs of cerium oxide similarly prevented oxidative stress as

well as N-acetyl cystine in mice with liver toxicity caused by tetrachloride. Some physicochemical properties of NP can also cause toxicity, apart from ROS effects. Minchin et al., for example, recently showed that certain NPs allow fibrinogen to unfold, thereby encouraging its association with the Mac-1 integrin receptor. Activation of this receptor upregulates the signaling pathway of NFkB, leading to inflammatory release. Activation of this receptor upregulates the signaling cascade of NFkB, causing inflammatory cytokines to be released.

III. CONCLUSION

The toxicity of nanomaterials, much like the parent bulk materials, is influenced by their structure. However, in assessing the toxicity of nanomaterials, additional physicochemical properties play a crucial role, such as scale, surface chemistry, shape, gradient of protein absorption, and surface smoothness or roughness. Thus, by the modification of many physicochemical properties, the toxicity of chemically similar products may be substantially altered. Scientific efforts to reduce the substantial information gap between the production and in vivo toxicity of NPs require considerable effort. To research the physiological effects of acute and chronic exposure to NPs in the population.For the future design of safe nanotechnology, a fundamental understanding of the biological interactions of NPs with cells, proteins, and tissues is essential. NP-products must be shown to have a high degree of biocompatibility with minimal harmful effects on blood components, genetic content, and cell viability prior to their broader use in everyday products and their clinical use.In-haled nanoparticles from airborne sources, including carbon-based materials, are of concern due to minimal studies conducted in the last few years. It is imperative that employees are properly shielded from inhaling NPs during the manufacture of nano-based materials, as the long-term impacts of exposure are still unknown.This also implies that to avoid subsequent release during use or disposal, NPs should be properly integrated or sequestered into items. In addition, to clarify potential toxic effects, dermal touch and other non-inhalation routes of exposure to nanoparticles must be examined..

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