

Nanoparticle-Induced Oxidative Stress: Mechanisms and Implications for Human Health and Environmental Safety

Newton Neogi^{*1}, Kristi Priya Choudhury¹, Ibrahim Hossain¹, Sabbir Hossain¹, MD. Golam Sazid¹¹ Department of Environmental Research, Nano Research Centre, Sylhet 3114, Bangladesh

ABSTRACT

Nanoparticles (NPs), which possess unique physicochemical qualities such as large surface area and reactivity, have brought about a revolution in a variety of sectors, including medicine and electronics. The growing ubiquity of these substances, on the other hand, has given rise to worries over the toxicological effects they have on human health and ecosystems. The condition known as oxidative stress, which is caused by an imbalance between the formation of reactive oxygen species (ROS) and antioxidant defenses, is one of the key processes that contribute to the toxicity of NPs. An excessive amount of ROS may cause damage to cellular components such as lipids, proteins, and DNA, which can result in detrimental consequences such as inflammation, apoptosis, and the development of cancer. NP-induced oxidative stress is investigated in this work, which focuses on the molecular mechanisms that are responsible for it. These processes include mitochondrial dysfunction, catalytic redox cycling, and the release of metal ions from particle disintegration. On top of that, we investigate how the features of NPs, such as their size, shape, surface charge, and composition, affect their capacity to produce ROS. Additionally, the consequences of oxidative stress for both acute and chronic health effects are examined, in addition to the function that it plays in the toxicity of the environment. The use of antioxidants and alterations to the surface of NPs are two examples of mitigation measures that are discussed in this article. The findings of this study highlight the significance of gaining knowledge of the processes behind oxidative stress to ensure the safe design and deployment of nanoparticles.

INTRODUCTION

Nanoparticles (NPs) are transformative in medicine, electronics, and environmental applications due to their small size, large surface area, and high reactivity. Yet, these same features raise concerns about adverse effects on human health and ecosystems. A critical toxicity pathway is oxidative stress, caused by imbalance between reactive oxygen species (ROS) and antioxidant defenses. Excessive ROS damages DNA, proteins, and lipids, leading to inflammation, apoptosis, and carcinogenesis. Physicochemical properties of NPs—such as size, shape, surface charge, and composition—strongly influence oxidative potential. Understanding these mechanisms is essential for risk assessment and strategies to mitigate harmful effects.

PHYSICOCHEMICAL PROPERTIES OF NPS

(A) Surface Effects

Surface area



Bulk material

Nanoparticles

- Dispersed nanoparticles → Large surface area & high particle number per mass
- Enhanced fraction of surface atoms
- Surface atoms → fewer neighboring atoms
- Result → Higher surface to volume

(B) Quantum Effects

- Size-dependent features (1–100 nm)
- Quantum confinement (radius \approx exciton Bohr radius)
- Bulk non-magnetic metals → magnetic at nanoscale (e.g., Pd, Pt, Au)

MOLECULAR MECHANISMS OF INDUCED OXIDATIVE STRESS

(A) Mitochondrial Dysfunction

- NPs interact with mitochondria, leading to electron leakage in the respiratory chain.
- This leakage generates excessive ROS, damaging cellular components.

(B) catalytic Redox Cycling

- Certain NPs (e.g., transition metals) catalyze redox reactions on their surface.
- This accelerates continuous ROS formation.

(C) Metal Ion Release

- Dissolution of metal-based NPs (e.g., Zn, Ag, Fe) releases toxic ions.
- These ions interact with biomolecules, enhancing ROS generation.

(D) Inflammatory signaling

- NPs activate immune cells (e.g., macrophages)
- Overproduction of inflammatory mediators further amplifies ROS levels.

(E) Direct Interaction with Biomolecules

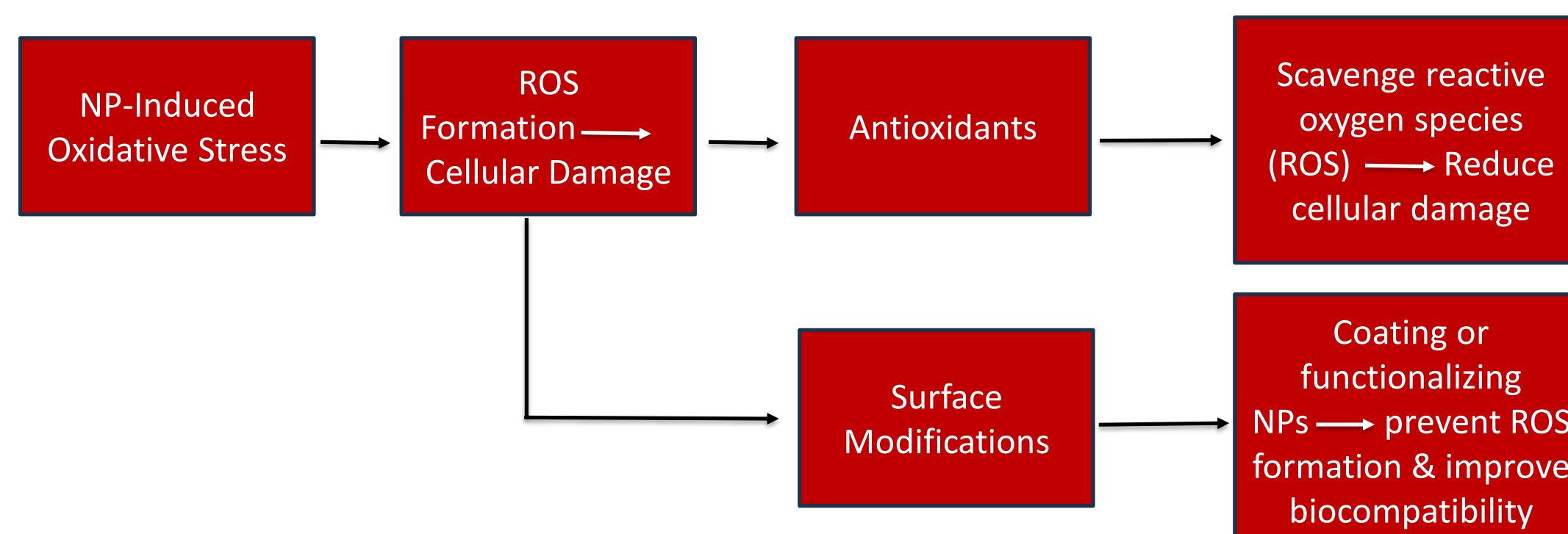
- NPs bind DNA, proteins and lipids.
- This promotes oxidative modification and functional impairment.

OXIDATIVE STRESS CAUSES HEALTH EFFECTS

System/organ	Health Effects
DNA / Cellular	DNA damage, chromosomal abnormalities, oncogene activation Carcinogenesis
cardiovascular	Atherosclerosis, heart disease, hypertension CVD
Neurological	Alzheimer's, Parkinson's, ALS, MS, depression, memory loss
Renal (Kidney)	Nephritis, renal failure, proteinuria, uremia
Reproduction	Delayed puberty onset, delayed sexual maturation

MITIGATION OF NP TOXICITY

- Antioxidants** : Scavenge reactive oxygen species → reduce cellular damage
- Surface modifications** : Coating or functionalizing NPs prevent ROS formation and improve biocompatibility



CONCLUSION

The growing integration of nanoparticles into diverse technological and biomedical applications underscores the urgency of evaluating their potential risks. Oxidative stress has been identified as a central mechanism in NP-induced toxicity, linking physicochemical properties to harmful effects on human health and the environment. Evidence indicates that oxidative stress contributes to cancer, cardiovascular, neurological, renal, and reproductive disorders, while also posing ecological hazards. Mitigation strategies, including the use of antioxidants and surface modifications, hold promise in reducing NP toxicity. Ultimately, advancing our understanding of the molecular pathways of oxidative stress is crucial for ensuring the safe and sustainable design, use, and disposal of nanoparticles in modern society.

REFERENCES

- E. Roduner, "Size matters: why nanomaterials are different", doi: 10.1039/b502142c.
- C. Buzea, I. I. Pacheco, and K. Robbie, "Nanomaterials and nanoparticles: Sources and toxicity," *Biointerphases* 2007 2:4, vol. 2, no. 4, pp. MR17–MR71, Dec. 2007, doi: 10.1116/1.2815690.
- S. J. Ikhmayies, "Characterization of Nanomaterials," *JOM*, vol. 66, no. 1, 2014, doi: 10.1007/s11837-013-0826-6.
- L. D. Geoffrion and G. Guisbiers, "Quantum confinement: Size on the grill!," *Journal of Physics and Chemistry of Solids*, vol. 140, p. 109320, May 2020, doi: 10.1016/J.JPCS.2019.109320.
- Y. Taniyama and K. K. Griendling, "Reactive Oxygen Species in the Vasculature Molecular and Cellular Mechanisms," 2003, doi: 10.1161/01.HYP.0000100443.09293.4F.
- M. Valko, M. Izakovic, M. Mazur, C. J. Rhodes, and J. Telser, "Role of oxygen radicals in DNA damage and cancer incidence," Kluwer Academic