Chapter 42

Nanoparticles in Environmental Epidemiology: Effects on Human Health

Haleema Sadia[1*], Nawal Chaudary[2], Fatima Siddiqa[2], Imama Nasir[1], Umar Mehmood Randhawa[3], Muhammad Imran[3], Khurram Adrian Shah[4] and Mohsin Rasool[5]

[1]Department of Epidemiology and Public Health, University of Agriculture, Faisalabad
[2]Institute of Soil and Environmental science, University of Agriculture Faisalabad
[3]Department of Parasitology, University of Agriculture, Faisalabad
[4]The University of Veterinary and Animal Sciences Swat, Pakistan
[5]Department of Entomology, University of Agriculture Faisalabad, Pakistan
*Corresponding author: dr.haleemasadia890@gmail.com

ABSTRACT

Nanoparticles (NPs) are becoming more and more significant every day because of their profound effect on human health. The ability of NPs levels to differentiate between natural and artificial causes of air pollution makes them an essential indicator. Due to their ultrafine size, which enables them to stay suspended in the environment for prolonged periods of time, they can cause a range of health issues and move farther. Indoor and outdoor conditions can both cause respiratory and cardiovascular diseases caused by NPs. Exposure to nicotine products at work, home, and through passive smoking has been associated with side effects such as dyspnea, thin septum and elevated levels of interleukin protein and tumor necrosis factor (TNF-α), which can lead to tumor growth in the exposed population. This comprehensive volume compiles information on the origin, exposure and impacts of NPs on numerous organ systems. For researchers exploring air nanoparticles in the fields of epidemiology, this chapter provides background information and scientific data.

KEYWORDS
Nanoparticles, Tumor necrosis factor,

Received: 29-Jun-2024
Revised: 29-Jul-2024
Accepted: 09-Aug-2024

A Publication of Unique Scientific Publishers

Cite this Article as: Sadia H, Chaudary N, Siddiqa F, Nasir I, Randhawa UM, Imran M, Shah KA and Rasool Mohsin, 2024. Nanoparticles in environmental epidemiology: effects on human health. In: Rubio VGG, Khan A, Altaf S, Saeed Z and Qamar W (eds), Complementary and Alternative Medicine: Nanotechnology-I. Unique Scientific Publishers, Faisalabad, Pakistan, pp: 372-381. https://doi.org/10.47278/book.CAM/2024.409

INTRODUCTION

NPs, which have diameters ranging from 1 to 100 nm, are one of the different sizes of particles that can be found in the environment. Particles in the environment that range in diameter from 0.1 to 10 µm stay there for approximately one week. The only mechanisms that can possibly remove small particles from the mixture are coagulation and diffusion; settling can remove larger particulate debris.Furthermore, the main reasons why NPs are difficult to remove from the atmosphere and represent a risk to human health are their fine size and extended atmospheric retention duration (Sonwani et al., 2021). In addition to being created in the bodies of insects, plants and people, nanoparticles are also released via combustion processes, forest fires, automobile exhaust, and industrial emissions (Jeevanandam et al., 2018). The high rates of urbanization, industrialization, vehicle emissions, extreme events (such as dust storms, volcanic eruptions, forest fires, etc.) and episodic events (like fireworks and crop residue burning) in Asia contribute to the region's high concentration of nanoparticles in the atmosphere (Saxena et al., 2020 and Sonwani et al., 2001).

Titanium dioxide nanoparticles, or TiO2 NPs, are the most commonly produced nanomaterial and are used in personal hygiene products, food additives, pigments and photocatalysis. These nanoparticles represent the cutting edge of a rapidly expanding discipline of nanotechnology. Titanium dioxide (TiO2) is the most often utilized and longest-produced of all these nanomaterials. These TiO2 nanoparticles are extensively utilized in the commercial sector, particularly in the cosmetics industry. High consumption in this method has made the effects of human population toxicity worse. Numerous investigations have demonstrated that the lungs, heart, liver, spleen, kidneys, alimentary canal and heart all gathered TiO2 NPs following oral exposure or inhalation.

Furthermore, they disrupt the equilibrium of glucose and lipids in mice and rats. TiO2 nanoparticles are mostly harmful because they induce oxidative stress, which can lead to immunological reactions, genotoxicity, inflammation, and cell damage. Degradation type and intensity are significantly influenced by the physical and chemical properties of TiO2

nanoparticles, which regulate their reactivity and bioavailability. According to research, TiO2 NPs have the ability to break DNA strands and harm chromosomes. The consequences of genotoxicity are influenced by a number of factors, including exposure route, size and change of the particle surface. The majority of these symptoms can be the consequence of a very high TiO2 NP dosage. Although more TiO2 NPs are being produced and employed, epidemiological data are still scarce (Shabbir et al., 2021). The potential health harm that the increasing amount of nanoparticles (NPs) in our environment may pose to human health must be investigated as soon as possible. Thus, more research is required to evaluate various biological endpoints and use various human cell models as targets.

Source of Nanoparticles

Natural and manmade processes are the two potential sources of nanoparticles in the atmosphere.

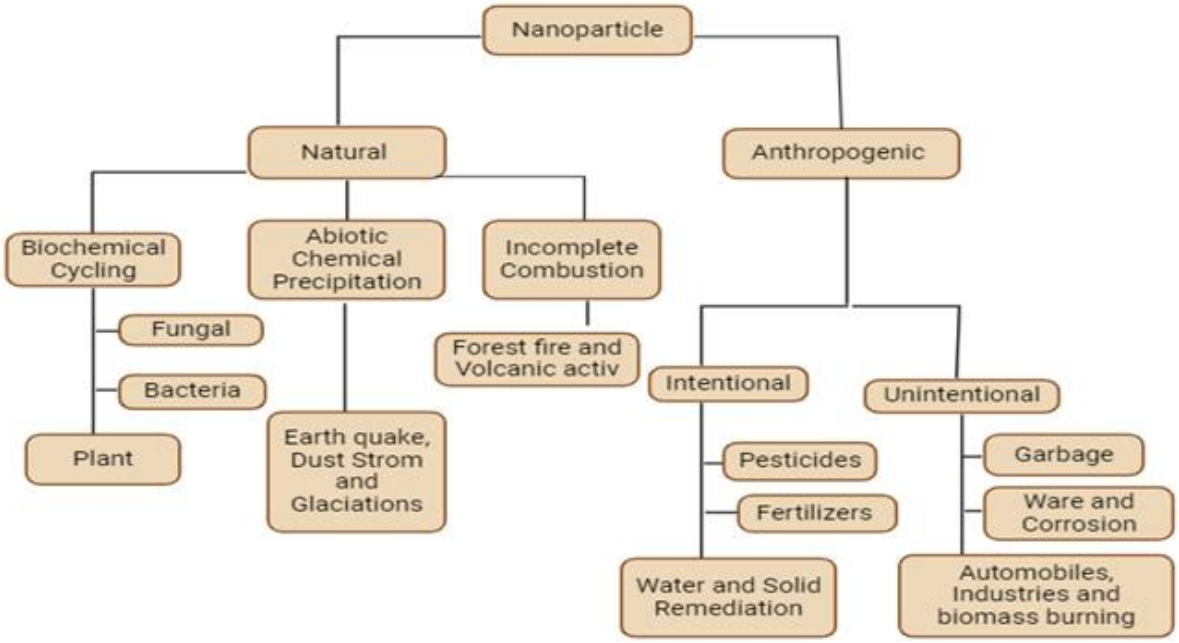

Fig. 1: Sources of nanoparticle emissions into the environment, both man-made and natural (adapted from Buzea and Pacheco, 2017).

Natural Source

About 10.5% of the aerosols in the atmosphere are produced by human activities; the remaining 90% are created by nature (Pipal et al., 2014; Jeevanandam et al., 2018; Sonwani and Saxena, 2021). In nature, nanoparticles are everywhere and originate from a multitude of sources, including sea spray, forest fires, volcanic eruptions, landslides, dust storms and biogenic emissions. Because of their tendency to react with air particles and clouds, organic chemicals make up a significant fraction of atmospheric nanoparticles.

Nitrogen oxides, volatile organic compounds, and primary organic aerosols are abundant in ambient air due to both natural and anthropogenic sources. These substances eventually combine to form secondary organic aerosols, or SOA (Tiwari and Saxena, 2021). Numerous studies (Sonwani et al., 2016) estimate that 90% of SOA generated globally is produced by biogenic volatile organic compounds (bVOCs).

Location-specific factors, such as the kinds of local sources and their relative contributions, greatly influence the chemical composition of nanoparticles (NPs). The roles of NPs in varying ambient atmospheres have not been extensively researched. According to a Pittsburgh study, organic carbon (OC) and ammonium and sulfate salts, which make up 45–55% and 35–40% of NPs, respectively, are the main components of NPs. Furthermore, incomplete burning and geological origins make up the other main atmospheric causes of nanoparticle production.The main combustion-generated sources are forest fires and volcanic eruptions, while the main geological sources are earthquakes, glaciers, dust storms and volcanic eruptions (Strambeanu et al., 2015).

Human-based Source

In comparison to natural sources, anthropogenic sources of nanoparticles are found primarily in urban areas. Intentional and inadvertent anthropogenic sources are distinguished. Burning biomass, incinerating non-biodegradable garbage, and incomplete combustion from autos and factories are examples of unintentional sources. One deliberate source of NPs is the application of pesticides and fertilizers, which generates large amounts of them. Non-primary and secondary sources are another division of NPs based on origin. Transportation-related activities, industrial emissions,

resource mining, and energy generation are examples of primary sources. Both stationary and mobile primary sources are possible. Stationary sources of emissions include mining operations, thermal power plants, and the chemical and metallurgical sectors. The largest source of NPs is thermal power plants, especially in metropolises and semi-urban/rural areas where there are more of them than anywhere else (Sonwani et al., 2021). The bulk of the sources of mobility, according to (Strambeanu et al., 2015), include engines, extra-atmospheric rocket launches, automobiles, ships, submarines, airplanes, and engines.

The sharp increase in the number of cars on the road is one of the main causes of air pollution. In 2002, there were more than one billion cars on the road worldwide, and the number has only increased since then. Carbon monoxide (CO), hydrocarbons (HCs), nitrogen oxides (NOx), sulfur oxides (SOx), particulate matter (PM), volatile organic compounds (VOCs) and their secondary consequences are the main components of exhaust, according to Banerjee and Christian (2018). The second-largest sources of anthropogenic NPs emissions into the atmosphere are the mining and construction sectors. Indirect processes including decantation, sedimentation, and flotation can also produce nanoparticles (NPs); direct production of NPs can occur via surface mining and excavation through mine shafts (Strambeanu et al., 2015). Meteorology and demolition methods both have an impact on the amount of NPs in the atmosphere. In addition to dust, one can find lead, glass, wood and other dangerous particles at demolition sites, as well as respirable asbestos fibers. These particles can travel quite far into the atmosphere and sometimes they form dust clouds that can reach several kilometers, affecting nearby areas (Kumar et al., 2013).

Movement of NPs Across Borders

An additional significant factor in enhancing the pollution load and atmospheric concentration of particulates in a given area is the transboundary migration of air pollutants. Since air mass migration from desert and ocean areas includes a range of minerals and salts in addition to tiny particles, it has a considerable impact on the quality of the air in remote areas (Sonwani and Saxena, 2021). For instance, seasonal disturbances are brought to the Caribbean basin by the transportation of mineral dust from Africa (Buzea and Pacheco, 2017). The forest fires in 1997 and the volcanic eruptions in Iceland in 2011 caused comparable environmental destruction in Asia, especially in Singapore and Malaysia.Numerous writers from all over the world, especially in South Asia, have reported on the transportation of fine particle emissions from anthropogenic sources (Saxena et al-2020). Roughly 70–80 million tons of crop residue are burned each year, contributing significantly to air pollution and aggravating respiratory disorders. In October and November, daily newspapers in Northern India carried stories on the frequent occurrences of dense clouds of smog caused by farmers burning agricultural waste, which reduced visibility and raised the Air Quality Index (AQI) to an extreme level. In pre-monsoon Asia (March to early June), temperatures and wind speeds are higher than in other seasons, which makes dust storms more frequent (Badarinath et al., 2009).

Table 1: Indicates Effects of nano-particles on various organ of body

S. No	Biological System	Target Organ	Effect of Nanoparticles	Reference
1	Neural System	Epithelial lining and Brain	DNA damage, apoptosis And hormonal imbalance	Valdiglesias et al., 2013; Pujalté et al., 2011
2	Endocrine System	Epithelial lining, Thyroid and Hormone Receptors	Oxidative Stress, Apoptosis, Overproduction of T3 hormone and blocking of signal cascades	Jaing et al., 2019 Leso et al., 2018
3	Respiratory System	Lungs	Inflammation, Oxidative Stress and Genotoxicity	Clif et al., 2014 Sharma et al., 2012
4	Cardiovascular System	Heart	Increase blood pressure and Decrease Heart Rate	Yu et al., 2016
5	Excretory System	Kidney Epithelial lining	Nephrotoxicity, DNA damage, Shrinkage of Kidney Cell and Vasoconstriction	Sramkova et al., 2019
6	Digestive System	Stomac Intestine	Increase mucus production, Inflammation and Accumulation in lamina propria	Georgantzopoulou et al., 2015

Impact of Nanoparticles on the Respiratory System and Inhalation Exposure

When humans inhale nanoparticles, they can cause a variety of respiratory disorders. According to Esztati et al. (2004), indoor air pollution from burning home fuel is the eighth-largest risk factor for the worldwide burden of disease. The primary cause of indoor nanoparticle pollution in homes is smoke from inefficient stoves and biomass fuels, which are most frequently used in rural regions. Ignorance and customs are the main barriers preventing rural populations from switching to contemporary chulas and eco-friendly fuels. Unprocessed biomass solid fuel, which is 50 times more polluting than gas stoves, is the most popular fuel used for residential cooking in rural areas (Ravindra et al., 2019). Another frequent cause of NPs exposure is cigarette smoking. It has been observed that a single cigarette releases nearly 8.8 × 109 nanoparticles. There are few research on distinguishing the various types of particles found in cigarette smoke, perhaps due to the large concentrations of particles and their quick dilution in air. An investigation conducted in the Netherlands provided experimental evidence of the direct correlation between a greater number of NPs and a longer puff length and

higher tar concentration in a single cigarette.

NP exposure at work poses health risks that are more prevalent in poor and underdeveloped countries. Traffic officers have been found to have higher incidences of respiratory and cardiovascular conditions as a result of their prolonged exposure to vehicle emissions. In a joint study conducted in 2017 by the United Kingdom and India, various medical tests of 532 traffic cops exposed to air nanoparticles were compared with 150 office workers working interior environments. According to the findings, there were 50% higher incidences of thick sputum, joint discomfort, and dyspnea in the traffic cops (Bajaj et al., 2017).

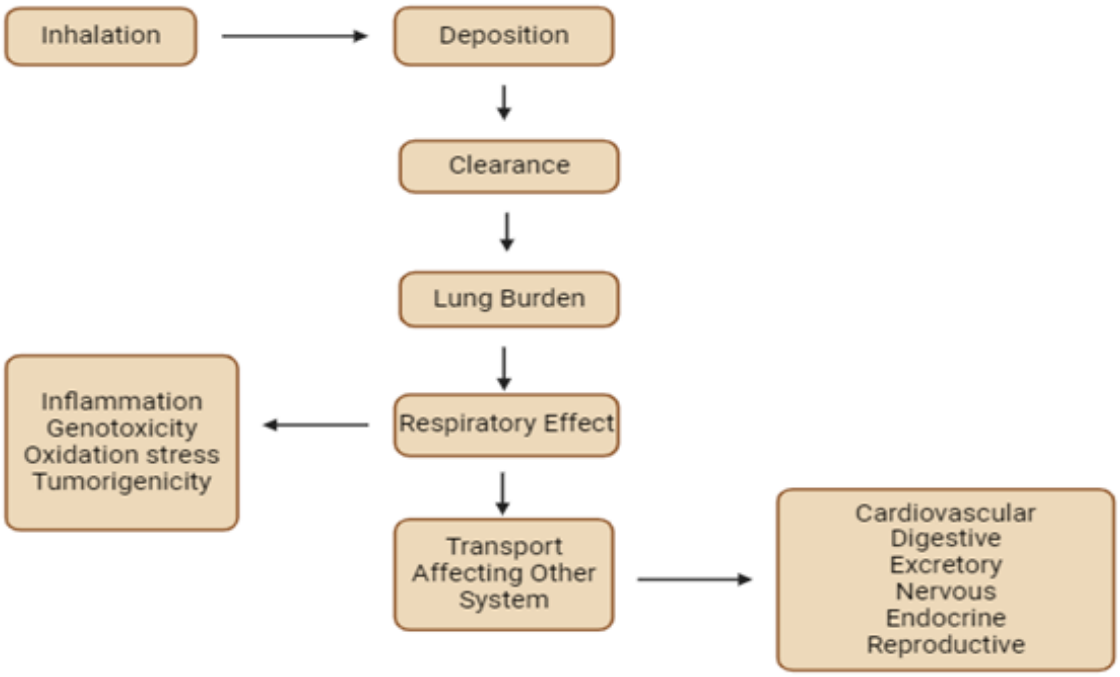

Fig. 2: Overview of pathway of nanoparticles from inhalation to disease site.

Inhalation

The human lung has an internal surface area of approximately 75–140 m2 and approximately 3,00,106 alveoli. The lungs are the main site of entry for nanoparticles into the body because of their direct contact with the external environment. According to Pacurari et al. (2016), they are also thought to be important and the primary focus of study on the effects of nanoparticles. Air enters the body through the mouth, nose, and throat, travels through the tracheobronchial tree, and then arrives in the alveoli. The Weibel bifurcating tubes model can be used to depict the conducting zone and respiratory zone, which were previously covered in depth and establish the contour of the respiratory airway. Particle transit is influenced by this structure. Furthermore, some of them can enter the cardiovascular system and other internal organs by overcoming alveolar epithelium and capillary endothelial cells (Qiao et al., 2015). Furthermore, deep into the cytoplasm and karyoplasm of the pulmonary epithelium and mesothelial cells of the lungs, electron imaging demonstrates that nanoparticle penetration can occur in both the outer and inner cellular compartments (Bakand et al., 2012).

Deposition

The aerodynamic dimension of the particle determines where NP will deposit in the respiratory tract (Ferreira et al., 2013). Larger diameter fibers are mainly deposited in the respiratory airways' "saddle points" where they branch off. As a result, they are unable to enter the respiratory system very far. For smaller particles, Brownian motions dominate the deposition process. Aerosol nanoparticles (NPs) measuring 20 nm or less have been found to elude most clearance systems and end up in the alveolar region of the lungs after 24 hours of inhalation, according to data gathered by energy filtering transmission electron microscopy (EFTEM).

Lung Burden

It is demonstrated that there are notable differences in the lung behavior of soluble and insoluble particulate matter (NPs). Once dissolved in aqueous solution, soluble nanoparticles enter the circulatory systems. By the way of macrophage phagocytosis or mucociliary escalator, on the other hand, the insoluble NPs (black carbon) are eliminated (Buzea et al., 2007). According to reports, insoluble particulate matter is responsible for increased inflammation, lung tumors, and tissue damage (Ferreira et al., 2013). Lung damage results from the insoluble particulates accumulating more quickly than the ability of macrophages to clean them. As a result, the lungs' defense mechanisms are unable to function. Additionally, it was found that the bronchoalveolar lavage fluid's shortened half-life of IL-1β and TGF-β1 induces acute lung inflammation.

Increased exposure durations, however, lead to the production of collagen, which irritates the lungs and can induce pulmonary fibrosis (Lin et al., 2014).

Nano-Toxicity

The pseudostratified epithelium that makes up the lung-blood stream barrier is present in human lungs. The mucous layer covers the thin columnar epithelium, bronchial epithelium (3-5 mm) and bronchiolar epithelium (0.5-1 mm), which make up the airways (Prapathawatvet et al., 2020). Since there are more than forty different cell types that make up lung tissue, a number of cell models were developed and assessed to look into the overall effects of nanoparticles on the lungs. The main focus of NP exposure is the epithelium of the respiratory system. A549, which is derived from human lung adenocarcinomas, is the most often used cell line for toxicity testing; Calu-3, 16HBE14o- and BEAS-2B cell lines are utilized as models for the bronchial barrier system.

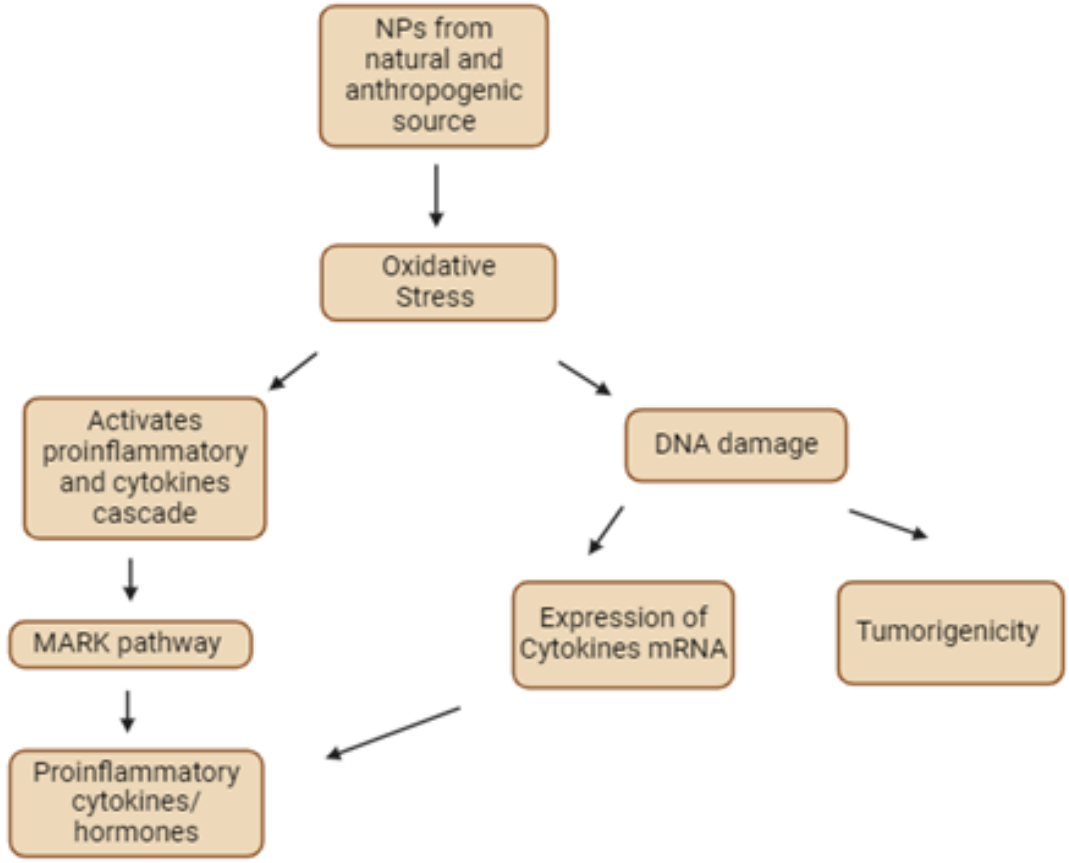

Fig. 3: Effects of toxicity caused by nanoparticles on human body (adapted from Li et al., 2010; Chakraborty et al., 2018).

According to Donaldson et al. (2005), the damage that nanoparticles (NPs) inflict to the respiratory system is inversely proportionate to their size, since smaller particles are more easily deposited in the distal portions of the throat. Herein, four primary categories of nanoparticle toxicological impacts are examined.

- Stress Caused by Oxidation
- Inflammation
- Genotoxicity
- The ability to grow

Oxidative Stress

The main effect of nanotoxicity is oxidative stress, which is brought on by an imbalance between antioxidants and free radicals in the body. According to Sharma et al. (2012), this leads to an imbalance in the respiratory system when excess free radicals with an unbalanced number of electrons accidentally react with other molecules. Reactive oxygen species (ROS) production. The production of reactive oxygen species (ROS) is a major oxidative mechanism that damages human lungs. According to Donaldson et al. (2010), NPs' involvement in the electron transport chain of the cell's mitochondria causes an excess of reactive oxygen species (ROS). According to Martin and Sarkar (2017), metallic NPs can trigger Fenton-type reactions that lead to free-radical-mediated toxicity, whereas carbon NPs are known to affect mitochondrial functions. According to Li et al. (2008), Fenton's reaction is the process that produces exceptionally reactive hydroxyl radicals (OH·)

when hydrogen peroxide (H2O2) has an enhanced potential due to specific metals' unique oxygen transfer characteristics.

The surface groups' catalytic activity determines how much ROS is created by a specific NP. About 10% of the molecules in a particle with a size of 30 nm are expressed, compared to only about 20% and 50% of the molecules in particles with a size of 10 and 3 nm, respectively (Hao and Chen, 2012). As nanoparticles are deposited, molecular oxygen-dependent superoxide anion radicals (O2−2), H2O2 and hydroxyl radicals (OH·) are produced. It has been demonstrated that BEAS-2B bronchial epithelial cells respond cytotoxically to several chemical species. Additionally, it was noted that the respiratory system releases neutrophils and macrophages, which are inflammatory phagocytes, in reaction to particulates.

Inflammation

There are two types of immune systems in humans: innate and adaptive. The innate immune system is the body's initial line of defense against any foreign particle that gets inside. If the foreign particle (antigen) cannot be neutralized by the innate immune system, the much more powerful and complex adaptive immune system is activated. The body's dendritic cells carry out this activation. Because they are foreign particles, nanoparticles also trigger the activation of dendritic cells, which release ROS, chemokines and cytokines and stimulate naïve T-cells and different inflammasomes. Factors that Determine Inflammation. NPs trigger an inflammatory response in the respiratory system, just like they do in other body systems (Padmanabhan and Kyriakides, 2015). The zeta potential (ξP) of a particle is a well-known indicator of its capacity to induce inflammation. The electric potential generated by the interaction of charged groups (found on a particle's surface) with the suspension medium is known as ξP. A particle with more positively charged groups on its surface will be more soluble in the acidic medium of the human body. This will enhance the particle's interaction with macrophages, leading to an inflammatory response (Schins, 2013).

The amount of white blood cells (WBCs) in the blood is another sign of inflammation in the body. An elevated WBC count above normal indicates inflammation and consequently, a reduction in immunity. Inhaled nanoparticles trigger the release of pro-inflammatory hormones when they settle in the lungs and interact with the main immune system, the alveolar macrophages. Thus, the dormant macrophages are aroused to facilitate the transport of several pro-inflammatory cytokines (IL-1 family, IL-6, IL-8, and i-CAM pro-inflammatory protein synthesis) to the affected location (Clift et al., 2011; Foldbjerg et al., 2011).

Genotoxicity

Inflammation or oxidative stress can produce genotoxicity indirectly, or NPs can directly interact with DNA to cause it. Genotoxicity can be separated into major and secondary categories based on various studies and the ensuing effects. Primary genotoxicity is the state in which minuscule particles penetrate the nucleus and modify DNA. There is another significant indirect process that is linked to the DNA repair cascade. Secondary genotoxicity is caused by NP-induced oxidative stress and inflammation (Zhu et al., 2013; Magdolenova et al., 2014).

DNA oxidation causes carcinogenic changes, such as adenine and guanine hydroxylation, which results in the creation of DNA adducts (Berube et al., 2007). When specific carcinogens are present in DNA, covalent changes take place that lead to DNA adducts. Particulate carcinogens can penetrate cellular membranes and the nucleus directly. Examples of such substances include asbestos and crystalline silica. According to Magdolenova et al. (2014), they then interact with and disturb the many components of the mitotic spindle's process and functionality, leading to dysfunctionalities. All biological functions, including mitosis, DNA replication and DNA transcription into mRNA, can be interfered with by these foreign particles. NPs unintentionally promote mistakes in nuclear processes indirectly by binding to the nuclear proteins responsible for DNA repair mechanisms and interfering with their function. Topoisomerases are the enzymes responsible for carrying out this repair cascade. Human topoisomerase-II alpha has been shown to bind to Carbon-60 fullerenes in the ATP-binding domain, which can interfere with the enzyme's ability to function as an enzyme (Magdolenova et al., 2014).

Tumourigenecity

A network of proteins in a cell called the MAPK/ERK pathway, also known as the Ras-Raf-MEK-ERK pathway, is responsible for transmitting signals from surface receptors to DNA found in the nucleus. When a signaling molecule attaches to the epidermal growth factor receptor (EGFR) receptor on the cell surface, the signal is initiated. When this receptor is in its normal state, external ligands like epidermal growth factors bind to it, phosphorylate it, activate it and trigger a series of docking proteins that eventually produce mRNA, which is then translated to build other proteins. Here, these receptors are interacting with ROS and occasionally direct NPs, and because of the altered DNA code, aberrant proteins may or may not arise. The formation of many cancers begins at a fundamental stage when one of the pathway's proteins mutates and becomes stuck in the "on" or "off" position. The body produces higher amounts of 8-hydroxy-2′-deoxyguanosine (8-OHdG) as a result of the entrance of NPs. Tumors can arise from 8-OHdG-induced G-to-T transversion mutations in important genes implicated in the development of cancer (Guo et al., 2017). As a result, the incidence of carcinogenicity, which reduces immunity, is directly correlated with an elevated level of 8-OHdG.

Effect of NPs on Digestive System

The digestive system, sometimes referred to as the gastrointestinal tract, is primarily composed of the oesophagus, stomach, and intestines. Food and drink are the main routes via which NP reaches the intestines. Interleukin-8 is elevated

by silver nanoparticles, and this is directly associated with inflammation and increased mucus formation (Georgantzopoulou et al., 2015). Certain nanoparticles (NPs) can disturb the layers of mucous and epithelial cells by penetrating blood vessels and avoiding the junctions between intestinal epithelial cells. That depends on how big they are.

Depending on their size, certain nanoparticles (NPs) have the ability to break through the intestinal epithelial cell junctions and cause damage to the mucus and epithelial cell layers in blood vessels. Furthermore, they eventually congregate in the intestinal lamina propria, where they impede the function of goblet cells. The rate of NP formation rises if an individual already has underlying medical disorders that cause inflammation in the intestinal regions, such as Crohn's disease or ulcerative colitis (Jones et al., 2015). As a result, toxicity levels rise, increasing the incidence of carcinomas and colon cancer.

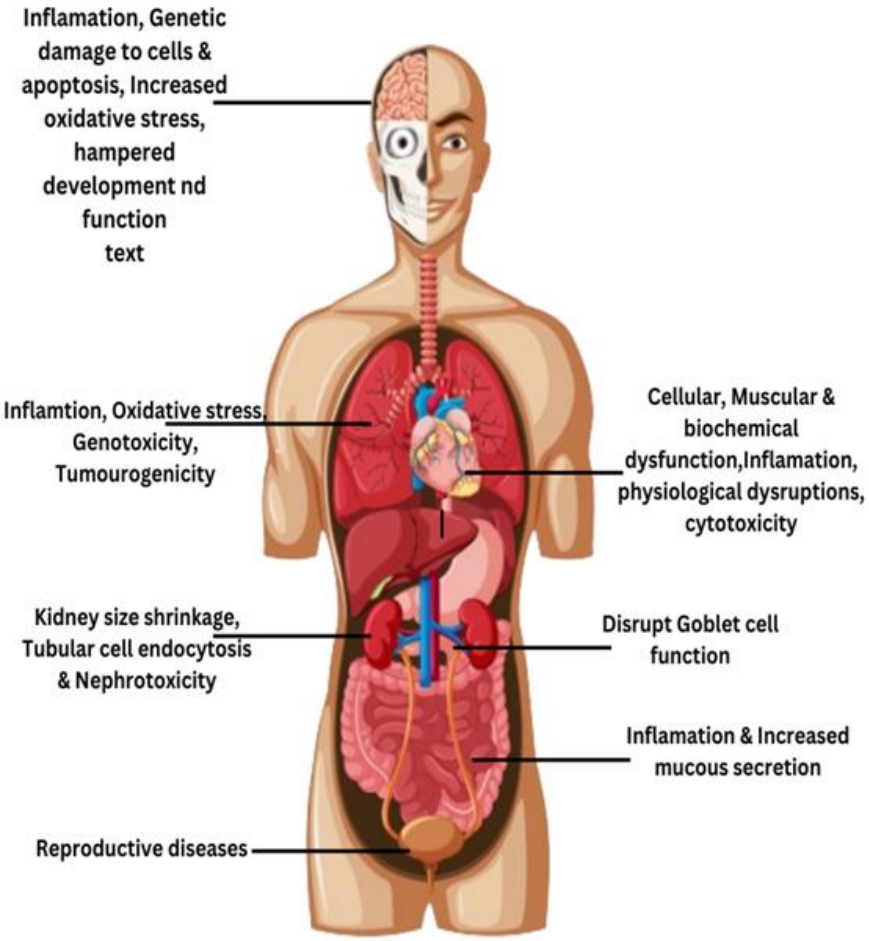

Fig. 4: Impact of Nanoparticles on various systems of the human body.

Impact of Nanoparticles on the Cardiovascular System

NPs can enter our bodies through the mouth or respiratory system and travel through the digestive tract before entering the circulatory system. These can significantly affect the way the system usually operates. According to Yu et al. (2016), some of the early effects of NPs on the cardiovascular system include changed vascular tone and dysfunction, a lowered heart rate, and elevated blood pressure. Nanoparticles (NPs) can affect our bodies in a variety of ways, depending on their concentration, physical characteristics, and duration of retention. These effects can include angiogenic or antiangiogenic, vasodilation or constriction, pro-oxidant or antioxidant, cytotoxic, apoptotic, or phagocytic (Gonzalez et al., 2016).

The two effects of silver nanoparticles (Silver NPs) on blood composition are antagonistic: angiogenesis (the formation of new blood vessels) and membrane permeability. These days, pacemakers, medications and related antibodies all contain silver nanoparticles. Garcia and associates (2016). TiO2 NPs can accumulate in the heart after prolonged exposure and result in inflammation, cellular necrosis, sparse cardiac muscle fibers and cardiac biochemical malfunction. In addition to cardiac ischemia damage and atrioventricular occlusion, a study on SiO2 NPs in aged rats showed elevated blood viscosity and Fbg levels (Yu et al., 2016).

Impact of Nano-particles on the Excretory Organ System

The toxicity of a particle rises with its size, according to Wang et al. (2009). Indicators of apoptosis, such as shrinkage and nuclear condensation, were observed in kidney cells treated to SiO2 nanoparticles at dosage levels of 20–100 µg/ml in this investigation on HEK293 cells, or cultured human embryonic kidney cells. Furthermore, oxidative stress activation and

ROS formation in HEK293 cells imply the possibility of nephrotoxicity for nanoparticles. Impaired nephrotoxic potential can cause cerebral epilepsy by disrupting nephron elasticity. To evaluate the cytotoxicity of NPs, Pujalté et al. (2011) additionally employed the glomerular mesangial (IP15) and epithelial proximal (HK-2) cell lines.

Impact of Nanoparticles on the Endocrine System

The kidney is highly vulnerable to xenobiotics and the bioaccumulation of toxins because each kidney nephron includes a network of blood capillaries that filter toxins from blood (Pujalté et al in 2011). After glomerular filtration, NPs typically gather in the proximal convoluted tubules (PCT), where tubular cells may endocytose the particles. TH1 cells treated to inorganic NPs were demonstrated to display DNA damage in addition to an acceleration of NP-induced nephrotoxicity (Sramkova et al., 2019). According to Pujalté et al. (2011), there was a similar result observed in another investigation when copper nanoparticles were introduced to the tubular cells of mice. This will lead to chromosomal anomalies as well as genetic diseases.

Impact of Nanoparticles on the Reproductive System

It has been established that the buildup of NPs is one of the primary causes of the present increase in cases of infertility. According to Zhang et al. (2020), sperm concentration and motility rates were found to be decreased and sperm abnormalities rates were elevated when silica NPs, which are frequently found in workplaces, were administered to rats in an experimental investigation that involved a high-fat diet. Significant sperm DNA integrity loss was seen as a result of the genotoxic effects of TiO2 NPs, which are demonstrated when they breach the blood–testis barrier and produce inflammation and cytotoxicity (Santonastaso et al., 2019).

According to one study, a subject administered MoO3 had a marked reduction in the weight of both the uterus and the right ovary, which eventually had a negative impact on reproduction (Asadi et al., 2019). Consequently, NPs play a major role in lowering birth rates and birth defects in their progeny. These conditions are frequently observed in females living in urban areas who are regularly exposed to air pollutants such particle matter (NPs).

Conclusion

Nanoparticle exposure can result in major respiratory and cardiovascular disorders, as is widely known from their health impacts. Epidemiological and toxicological studies have shown that nanoparticles are significantly more harmful than coarser particles because of their ultrafine size, chemical reactivity, and prolonged residence time in the environment. Furthermore, a correlation has been noted between the size of the particles and their capacity to go deep into the lungs. Nanoparticles can originate from man-made sources such as industrial emissions, forest fires, dust storms, volcanic eruptions and welding fumes, as well as natural sources like cigarette smoke and vehicle emissions. Nanoparticles (NPs) damage and delete segments of single- and double-stranded DNA and cause genetic material mutations that result in lung cancer and neoplasms. In addition to damaging the lungs, nanoparticles (NPs) also impair the cardiovascular and gastrointestinal systems, which can result in ulcers and cardiac arrest, respectively. Because of the buildup of NP, comorbid patients are constantly more vulnerable to many health issues.

REFERENCES

Bajaj, N., Sharma, T., Suneja, D., Jain, S., and Kumar, P. (2017). Determinants of respiratory and cardiovascular health effects in traffic policemen: a perception-based comparative analysis. *Journal of Transport and Health*, *4*, 30-39.

Asadi, F., Sadeghzadeh, M., Jalilvand, A., Nedaei, K., Asadi, Y., and Heidari, A. (2019). Effect of molybdenum trioxide nanoparticles on ovary function in female rats. *Journal of Advances in Medical and Biomedical Research*, *27*(121), 48-53.

Badarinath, K. V. S., Kharol, S. K., and Sharma, A. R. (2009). Long-range transport of aerosols from agriculture crop residue burning in Indo-Gangetic Plains—a study using LIDAR, ground measurements and satellite data. *Journal of Atmospheric and Solar-Terrestrial Physics*, *71*(1), 112-120.

Bakand, S., Hayes, A., and Dechsakulthorn, F. (2012). Nanoparticles: a review of particle toxicology following inhalation exposure. *Inhalation Toxicology*, *24*(2), 125-135.

Banerjee, T., and Christian, R. A. (2018). A review on nanoparticle dispersion from vehicular exhaust: Assessment of Indian urban environment. *Atmospheric Pollution Research*, *9*(2), 342-357.

BéruBé, K., Balharry, D., Sexton, K., Koshy, L., and Jones, T. (2007). Combustion-derived nanoparticles: mechanisms of pulmonary toxicity. *Clinical and Experimental Pharmacology and Physiology*, *34*(10).

Buzea, C., and Pacheco, I. (2017). Nanomaterial and nanoparticle: origin and activity. *Nanoscience and plant–soil systems*, 71-112

Chakraborty, A., Ghosh, S., Chakraborty, R., Chatterjee, S. M., and Hopper, W. (2018). Molecular mechanism of nanotoxicity-a critical review. *International Journal Current Biotechnology*, *6*, 1-12.

Clift, M. J., Gehr, P., and Rothen-Rutishauser, B. (2011). Nanotoxicology: a perspective and discussion of whether or not in vitro testing is a valid alternative. *Archives of Toxicology*, *85*, 723-731.

Clift, M. J., Gehr, P., and Rothen-Rutishauser, B. (2011). Nanotoxicology: a perspective and discussion of whether or not in vitro testing is a valid alternative. *Archives of Toxicology*, *85*, 723-731.

Donaldson, K., Poland, C. A., and Schins, R. P. (2010). Possible genotoxic mechanisms of nanoparticles: criteria for improved test strategies. *Nanotoxicology*, *4*(4), 414-420.

Ezzati, M., Lopez, A. D., Rodgers, A. A., and Murray, C. J. (2004). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. World Health Organization.

Ferreira, A. J., Cemlyn-Jones, J., and Cordeiro, C. R. (2013). Nanoparticles, nanotechnology and pulmonary nanotoxicology. *Revista portuguesa de pneumologia (English Edition)*, *19*(1), 28-37.

Ferreira, A. J., Cemlyn-Jones, J., and Cordeiro, C. R. (2013). Nanoparticles, nanotechnology and pulmonary nanotoxicology. *Revista portuguesa de pneumologia (English Edition)*, *19*(1), 28-37.

Foldbjerg, R., Dang, D. A., and Autrup, H. (2011). Cytotoxicity and genotoxicity of silver nanoparticles in the human lung cancer cell line, A549. *Archives of Toxicology*, *85*, 743-750.

Georgantzopoulou, A., Serchi, T., Cambier, S., Leclercq, C. C., Renaut, J., Shao, J., and Gutleb, A. C. (2015). Effects of silver nanoparticles and ions on a co-culture model for the gastrointestinal epithelium. *Particle and Fibre Toxicology*, *13*(1), 1-17.

Georgantzopoulou, A., Serchi, T., Cambier, S., Leclercq, C. C., Renaut, J., Shao, J., and Gutleb, A. C. (2015). Effects of silver nanoparticles and ions on a co-culture model for the gastrointestinal epithelium. *Particle and Fibre Toxicology*, *13*(1), 1-17.

Gonzalez, C., Rosas-Hernandez, H., Ramirez-Lee, M. A., Salazar-García, S., and Ali, S. F. (2016). Role of silver nanoparticles (AgNPs) on the cardiovascular system. *Archives of Toxicology*, *90*, 493-511.

Guo, C., Wang, J., Yang, M., Li, Y., Cui, S., Zhou, X., and Sun, Z. (2017). Amorphous silica nanoparticles induce malignant transformation and tumorigenesis of human lung epithelial cells via P53 signaling. *Nanotoxicology*, *11*(9-10), 1176-1194.

Hao, L., and Chen, L. (2012). Oxidative stress responses in different organs of carp (Cyprinus carpio) with exposure to ZnO nanoparticles. *Ecotoxicology and Environmental Safety*, *80*, 103-110.

Iavicoli, I., Fontana, L., Leso, V., and Bergamaschi, A. (2013). The effects of nanomaterials as endocrine disruptors. *International Journal of Molecular Sciences*, *14*(8), 16732-16801.

Jeevanandam, J., Barhoum, A., Chan, Y. S., Dufresne, A., and Danquah, M. K. (2018). Review on nanoparticles and nanostructured materials: history, sources, toxicity and regulations. *Beilstein Journal of Nanotechnology*, *9*(1), 1050-1074.

Jiang, Z., Shan, K., Song, J., Liu, J., Rajendran, S., Pugazhendhi, A., and Chen, B. (2019). Toxic effects of magnetic nanoparticles on normal cells and organs. *Life Sciences*, *220*, 156-161.

Jiang, Z., Shan, K., Song, J., Liu, J., Rajendran, S., Pugazhendhi, A., and Chen, B. (2019). Toxic effects of magnetic nanoparticles on normal cells and organs. *Life Sciences*, *220*, 156-161.

Jones, K., Morton, J., Smith, I., Jurkschat, K., Harding, A. H., and Evans, G. (2015). Human in vivo and in vitro studies on gastrointestinal absorption of titanium dioxide nanoparticles. *Toxicology Letters*, *233*(2), 95-101.

Leso, V., Fontana, L., Marinaccio, A., Leopold, K., Fanali, C., Lucchetti, D., ... and Iavicoli, I. (2018). Palladium nanoparticle effects on endocrine reproductive system of female rats. *Human and Experimental Toxicology*, *37*(10), 1069-1079.

Leso, V., Fontana, L., Marinaccio, A., Leopold, K., Fanali, C., Lucchetti, D., and Iavicoli, I. (2018). Palladium nanoparticle effects on endocrine reproductive system of female rats. *Human and Experimental Toxicology*, *37*(10), 1069-1079.

Li, N., Xia, T., and Nel, A. E. (2008). The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles. *Free Radical Biology and Medicine*, *44*(9), 1689-1699.

Lin, S., Wang, X., Ji, Z., Chang, C. H., Dong, Y., Meng, H., and Nel, A. E. (2014). Aspect ratio plays a role in the hazard potential of CeO2 nanoparticles in mouse lung and zebrafish gastrointestinal tract. *ACS nano*, *8*(5), 4450-4464.

Magdolenova, Z., Collins, A., Kumar, A., Dhawan, A., Stone, V., and Dusinska, M. (2014). Mechanisms of genotoxicity. A review of in vitro and in vivo studies with engineered nanoparticles. *Nanotoxicology*, *8*(3), 233-278.

Magdolenova, Z., Collins, A., Kumar, A., Dhawan, A., Stone, V., and Dusinska, M. (2014). Mechanisms of genotoxicity. A review of in vitro and in vivo studies with engineered nanoparticles. *Nanotoxicology*, *8*(3), 233-278.

Martin, A., and Sarkar, A. (2017). Overview on biological implications of metal oxide nanoparticle exposure to human alveolar A549 cell line. *Nanotoxicology*, *11*(6), 713-724.

Pacurari, M., Lowe, K., Tchounwou, P. B., and Kafoury, R. (2016). A review on the respiratory system toxicity of carbon nanoparticles. *International Journal of Environmental Research and Public Health*, *13*(3), 325.

Padmanabhan, J., and Kyriakides, T. R. (2015). Nanomaterials, inflammation, and tissue engineering. *Wiley Interdisciplinary Reviews: Nanomedicine and Nanobiotechnology*, *7*(3), 355-370.

Pipal, A. S., Taneja, A., and Jaiswar, G. (2014). Risk assessment and toxic effects of exposure to nanoparticles associated with natural and anthropogenic sources. In *Chemistry: The Key to our Sustainable Future* (pp. 93-103). Springer Netherlands.

Praphawatvet, T., Peters, J. I., and Williams III, R. O. (2020). Inhaled nanoparticles–An updated review. *International Journal of Pharmaceutics*, *587*, 119671.

Pujalté, I., Passagne, I., Brouillaud, B., Tréguer, M., Durand, E., Ohayon-Courtès, C., and L'Azou, B. (2011). Cytotoxicity and oxidative stress induced by different metallic nanoparticles on human kidney cells. *Particle and Fibre Toxicology*, *8*, 1-16.

Pujalté, I., Passagne, I., Brouillaud, B., Tréguer, M., Durand, E., Ohayon-Courtès, C., and L'Azou, B. (2011). Cytotoxicity and

oxidative stress induced by different metallic nanoparticles on human kidney cells. *Particle and Fibre Toxicology*, *8*, 1-16.

Qiao, H., Liu, W., Gu, H., Wang, D., and Wang, Y. (2015). The transport and deposition of nanoparticles in respiratory system by inhalation. *Journal Nanomater* 2015: 1–8.

Ravindra, K., Singh, T., Mor, S., Singh, V., Mandal, T. K., Bhatti, M. S., and Beig, G. (2019). Real-time monitoring of air pollutants in seven cities of North India during crop residue burning and their relationship with meteorology and transboundary movement of air. *Science of the Total Environment*, *690*, 717-729.

Santonastaso, M., Mottola, F., Colacurci, N., Iovine, C., Pacifico, S., Cammarota, M., and Rocco, L. (2019). In vitro genotoxic effects of titanium dioxide nanoparticles (n-TiO2) in human sperm cells. *Molecular Reproduction and Development*, *86*(10), 1369-1377.

Saxena, P., Srivastava, A., Verma, S., Shweta, Singh, L., and Sonwani, S. (2020). Analysis of atmospheric pollutants during fireworks festival 'Diwali'at a residential site Delhi in India. *Measurement, Analysis and Remediation of Environmental Pollutants*, 91-105.

Saxena, P., Srivastava, A., Verma, S., Shweta, Singh, L., and Sonwani, S. (2020). Analysis of atmospheric pollutants during fireworks festival 'Diwali'at a residential site Delhi in India. *Measurement, Analysis and Remediation of Environmental Pollutants*, 91-105.

Schins, R. (2013). Genotoxicity of nanoparticles. *Nanomaterials*, 60-64.

Shabbir, S., Kulyar, M. F. E. A., Bhutta, Z. A., Boruah, P., and Asif, M. (2021). Toxicological consequences of titanium dioxide nanoparticles (TiO2NPs) and their jeopardy to human population. *BioNanoScience*, *11*(2), 621-632.

Sharma, V., Singh, P., Pandey, A. K., and Dhawan, A. (2012). Induction of oxidative stress, DNA damage and apoptosis in mouse liver after sub-acute oral exposure to zinc oxide nanoparticles. *Mutation Research/Genetic Toxicology and Environmental Mutagenesis*, *745*(1-2), 84-91.

Sharma, V., Singh, P., Pandey, A. K., and Dhawan, A. (2012). Induction of oxidative stress, DNA damage and apoptosis in mouse liver after sub-acute oral exposure to zinc oxide nanoparticles. *Mutation Research/Genetic Toxicology and Environmental Mutagenesis*, *745*(1-2), 84-91.

Sonwani, S., and Saxena, P. (2021). Water-insoluble carbonaceous components in rainwater over an urban background location in Northern India during pre-monsoon and monsoon seasons. *Environmental Science and Pollution Research*, *28*(38), 53058-53073.

Sonwani, S., Madaan, S., Arora, J., Suryanarayan, S., Rangra, D., Mongia, N., and Saxena, P. (2021). Inhalation exposure to atmospheric nanoparticles and its associated impacts on human health: a review. *Frontiers in Sustainable Cities*, *3*, 690444.

Sonwani, S., Saxena, P., and Kulshrestha, U. (2016). Role of global warming and plant signaling in BVOC emissions. *Plant Responses to Air Pollution*, 45-57.

Sramkova, M., Kozics, K., Masanova, V., Uhnakova, I., Razga, F., Nemethova, V., and Gabelova, A. (2019). Kidney nanotoxicity studied in human renal proximal tubule epithelial cell line TH1. *Mutation Research/Genetic Toxicology and Environmental Mutagenesis*, *845*, 403017.

Sramkova, M., Kozics, K., Masanova, V., Uhnakova, I., Razga, F., Nemethova, V., and Gabelova, A. (2019). Kidney nanotoxicity studied in human renal proximal tubule epithelial cell line TH1. *Mutation Research/Genetic Toxicology and Environmental Mutagenesis*, *845*, 403017.

Strambeanu, N., Demetrovici, L., and Dragos, D. (2014). Anthropogenic sources of nanoparticles. In *Nanoparticles' Promises and Risks: Characterization, Manipulation, and Potential Hazards to Humanity and the Environment* (pp. 21-54). Cham: Springer International Publishing.

Strambeanu, N., Demetrovici, L., and Dragos, D. (2014). Anthropogenic sources of nanoparticles. In *Nanoparticles' Promises and Risks: Characterization, Manipulation, and Potential Hazards to Humanity and the Environment* (pp. 21-54). Cham: Springer International Publishing.

Tiwari, S., and Saxena, P. (2021). *Air pollution and its complications* (pp. 1-178). Springer-Nature, Singapore.

Valdiglesias, V., Costa, C., Kiliç, G., Costa, S., Pásaro, E., Laffon, B., and Teixeira, J. P. (2013). Neuronal cytotoxicity and genotoxicity induced by zinc oxide nanoparticles. *Environment International*, *55*, 92-100.

Wang, F., Gao, F., Lan, M., Yuan, H., Huang, Y., and Liu, J. (2009). Oxidative stress contributes to silica nanoparticle-induced cytotoxicity in human embryonic kidney cells. *Toxicology in vitro*, *23*(5), 808-815.

Yu, X., Hong, F., and Zhang, Y. Q. (2016). Bio-effect of nanoparticles in the cardiovascular system. *Journal of Biomedical Materials Research Part A*, *104*(11), 2881-2897.

Yu, X., Hong, F., and Zhang, Y. Q. (2016). Bio-effect of nanoparticles in the cardiovascular system. *Journal of Biomedical Materials Research Part A*, *104*(11), 2881-2897.

Zhang, L., Wei, J., Duan, J., Guo, C., Zhang, J., Ren, L., and Zhou, X. (2020). Silica nanoparticles exacerbates reproductive toxicity development in high-fat diet-treated Wistar rats. *Journal of Hazardous Materials*, *384*, 121361.

Zhu, X., Hondroulis, E., Liu, W., and Li, C. Z. (2013). Biosensing approaches for rapid genotoxicity and cytotoxicity assays upon nanomaterial exposure. *Small*, *9*(9-10), 1821-1830.