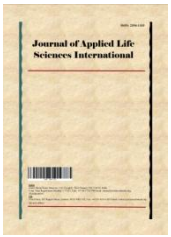

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Soot Pollution and Pathological Implications

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Carbon-based soot, a black particulate material, is produced when fossil fuels burn partially. Soot is seen as an unwelcome byproduct that results from the insufficient combustion of carbon-containing compounds. The environment is affected by the deposition of soot in water, air and soil. These environmental components serves as a means of exposure to man although, the effect of water and soil exposure are poorly studied. This reviewed has also shown that three pathological conditions may occur as a result of soot exposure; they include- respiratory disorder, cardiovascular disease and cancer. Poly aromatic hydrocarbon (PAH) was reported as carcinogenic substance in soot that causes cancer. Direct contact-mediated lung cell failure and immune response involvement that results in cellular proliferation and fibrosis have been proposed as the mechanisms underlying respiratory illness. Inflammation of myocardiac tissues was considered the pathogenesis of cardiovascular disease in soot exposure individuals. Treatment options were based on the mechanism of soot pathology. Based on existing literatures, this review has demonstrated that soot polluted environment can lead to cancer, cardiovascular disease and respiratory disorder.

Keywords: Cancer; environment; pollution toxicity; carbon; soot.

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1. INTRODUCTION

Carbon-based soot, a black particulate material, is produced when fossil fuels burn partially. Soot is seen as an unwelcome byproduct that results from the insufficient combustion of carbon-containing compounds. Incomplete combustion of hydrocarbons results in the formation of this powdery mass of black particles, which are impure carbon [1]. Since the quarter of 2016, soot has become an environmenal issue for residents in Niger delta particularly Port-Harcourt. It has been reported to cause about 1.6 million lung problems annually in this region [2]. The primary carcinogenic components in soot are poly aromatic hydrocarbons (PAHs), which are hydrocarbons. The most recognizable diesel soot is composed primarily of carbon, hydrogen, oxygen, sulfur, and trace amounts of metals at the elemental level. Black carbon, the main component of soot, contributes to early human death and disability.

Public health is a major concern for nations and economies. First, public health can be used as a gauge of how productive an economy is. As a result, it is generally accepted that maintaining good health is necessary for a nation to flourish and grow. Economic growth is fueled by a populace in good health. This shows that when people, especially the reproductive parts, are in bad health, the economy has a problem. Public health is taken seriously because of this. [3].

The environment must play a key role for good health to be realized in any economy because there is no denying the connection between the two [4,5]. Clear air, potable water, and nutrient-rich food are examples of environmental products that can support people in leading healthy lifestyles. The environment is the source of a vast variety of resources that man needs to sustain his existence. Similar developments in mineral mining have over time diminished the value of the environment. In Port-Harcourt, Rivers State, black soot, sometimes known as black carbon or carbon black, is one of the main environmental problems [6].

2. EFFECTS OF SOOT IN THE ENVIRONMENT

2.1 Air and Water

A few environmental issues caused by soot include smog and the acidity of lakes and rivers. When sunlight interacts with airborne particles, haze is created. Haze, which significantly reduces vision and increases the risk of a plane crash or a car accident, is primarily caused by soot [6].

2.2 Terrestial Environment

By being dispersed by the wind or water, soot particles have the potential to alter the nutritional balance in river basins, along coasts, and in forests, as well as deplete soil nutrients and harm delicate agricultural crops. Important national monuments and iconic structures can gradually lose their color and suffer damage due to acidification caused by soot pollution, which can also discolor and dissolve stone [6].

3. TOXICITY AND SIDE EFFECTS OF BLACK SOOTS

3.1 Soot and Carbon Black-Related Major Diseases and Rare Pathological Manifestations

The association between soot and carbon black and many diseases has been noted for more than three centuries. Although many diseases are caused by soot and carbon black, only three of them are somewhat well understood. The occurrence of cancer is the most complicated condition linked to soot and CB. Localized and systemic malignancies are caused by soot and CB. [7]. Respiratory diseases, which can occasionally be very serious, are the second significant health problem associated with soot and CB. Cardiovascular dysfunctions are the third. In addition to these illnesses, some unusual pathological findings have also been linked to soot or CB exposures. In a study, prenatal exposure to pritex-90 led to alterations in mice's neuroinflammatory and sexual development [8]. Surprisingly, rat brain proinflammatory marker levels were dramatically affected by lung exposure to diesel engine exhaust. In a different investigation, Printex-90 reduced sperm production [9]. Likewise, it was discovered that carbon nanoparticles had a negative impact on mice's male reproductive system. It has recently come to light that carbon black causes immunological activation in the male mouse pups, which causes developmental damage. In guinea pigs, soot from a transformer fire was also observed to cause salivary gland duct metaplasia. These research demonstrate the role of the body's systemic reaction in the emergence

of many illnesses, which still requires in-depth investigation.

3.2 The Pathological Mechanics of Cancer Caused by Soot and Carbon Blacks

As previously stated, soot is the first known carcinogen to be linked to the emergence of many cancer forms in both people and laboratory animals. These tumors may arise nearby or far away from the exposure location. It was noticed that chimney sweeps still exhibit higher cancer mortality rates after 200 years of efforts to restrict the safety in soot-related activities. In keeping with this, a case study from Gerber stated that soot exposure led to the development of penile cancer in chimney sweeps. The increased risk of cancer among Swedish chimney sweeps was also attributed to exposure to asbestos and soot. [10]. The majority of malignancies in the distal body parts may be accompanied by soot transportation because soot is absorbed and carried to blood via airway epithelium. According to a population-based study, occupational exposure to polycyclic aromatic hydrocarbons, a soot component, causes malignancies of the urinary tract and lungs. Another case-control research in the rubber manufacturing sector identified CB as a significant risk factor for early-stage skin cancer. It was later discovered that exposure to polycyclic aromatic hydrocarbons (PAHs) found in diesel soot is what causes prostate cancer. Furthermore, gene expression profiling supported the link between CB nanoparticle exposure and human health risk. [11]. Unlike the other studies, the International Agency for Research on Cancer (IARC) in Montreal, Quebec, Canada found no measurable increase in the risk of lung cancer in the patients who had occupational exposure to titanium dioxide, industrial talc, CB, and cosmetic talc [12]. However, no other organs were looked into; this study was just focused on lung pathology. Experimental models also produced data to support the notion that soot and CB have cancer-causing qualities in addition to the evidence already mentioned. A study done on dogs showed that soot absorption through the alveolar epithelium is how unmetabolized PAHs enter the bloodstream. The morphological abnormalities in the lungs are caused by soot particle interactions with lung tissue, as demonstrated in a rat model. When frequently breathed by rats, diesel exhaust (DP) and CB demonstrated hazardous and pulmonary carcinogenic effects. The genotoxic underpinnings of soot toxicity were validated by an *in vitro* research on the carcinogenic potential of CB [13]. The metabolically competent AHH-1 cell line (a human lymphoblast cell line) also showed a mutation at the hprt gene in response to soot. Further evidence that soot induces genotoxic effects and causes DNA mutations came from an *in vitro* investigation of grown cells [14].

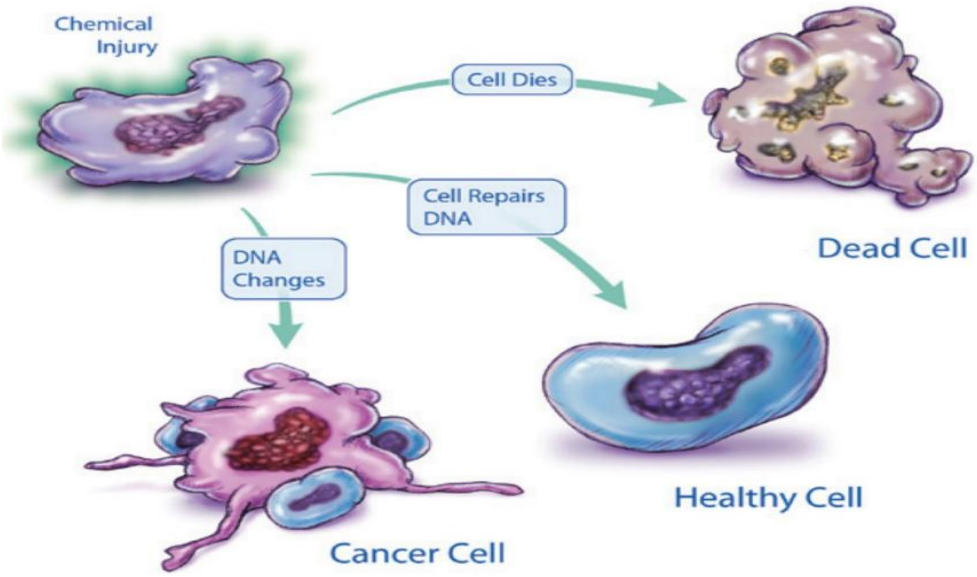

Fig. 1. Toxicant-induced carcinogenesis
Illustrating the mechanism of toxicant-induced carcinogenesis [15]

3.3 The Pathophysiological Mechanisms of Respiratory Diseases Caused by Soot Production

The first tissue to continuously be exposed to the many types of soot and CBs found in the environment is the respiratory epithelium of the lungs. By altering lung functioning, soot poisoning prevents the respiratory process from occurring. There could be two different types of toxicological processes. The first mechanism is the direct contact-mediated dysfunctions of lung cells, which include the production of ROS, cell hyperplasia, cell death, or apoptosis of lung airway epithelium and other nearby cells [16]. The next pathway involves the systemic immune response, which results in tissue remodeling and fibrosis, which impairs lung function and causes breathing problems. In the context of human clinical trials and animal investigations, we will explore these two categories of toxicities brought on by soot or CB in this section. Asthma and chronic obstructive pulmonary disease (COPD) are the two respiratory conditions that are most frequently observed in people as a result of soot exposure. The pathogenesis of asthma includes airway inflammation, tissue remodeling and fibrosis, intermittent airflow obstruction, and bronchial hyper-reactivity [17]. Airway inflammation, mucociliary dysfunctions, and structural alterations are all part of the pathogenesis of COPD. Numerous studies have shown a connection between soot or CB and asthma and COPD. According to a study, asthma in children is brought on by early exposure to air pollution. Smog, ultrafine particles (UFPs), and carbon monoxide levels are linked to worsening asthma in metropolitan children [18]. DEP change polarity mechanisms to start the migration of alveolar epithelial cells. According to an epidemiological investigation, crop burning had an impact on healthy volunteers' peak expiratory flow rate and pulmonary functioning [19]. Patients who are predisposed to COPD or asthma already display oxidative stress, making them more vulnerable to soot-mediated oxidative damage. It's interesting to note that CB is known to negatively affect people via ROS and may exacerbate their symptoms in vulnerable individuals. Animal model results also backed soot- and CB-mediated mechanisms of toxicity. The ultrafine soot produced by the flame boosted ROS and elevated Nrf2 antioxidants in the lungs, according to research done on rats [20]. As a result of the oxidative stress caused by soot or CB and the accompanying systemic immunological response (inflammation) in the lungs, asthma and other illnesses might occur [21].

3.4 The Mechanism of Cardiac Dysfunctions Caused by Exposure to Soot and Carbon Black

Due to their distal manifestation from the site of exposure and participation of more systemic responses, cardiovascular illnesses brought on by soot and CB exposure are a serious cause for concern. Suitable clinical and epidemiological evidence connected CB and soot to cardiovascular dysfunctions. Personal soot exposure is associated with acute myocardial infarction, according to a case-crossover research. Myocardial infection incidence was also attributed to soot. Implantable cardioverter defibrillators in London were activated by air pollution (BC) (a device used to treat cardiovascular dysfunctions). People who were exposed to wildfire particles in Darwin, Australia, had a higher risk of hospitalization for cardiovascular disease [22]. In addition, air pollution was thought to be a significant risk factor for the ST-segment depression in patients with coronary artery disease (an ECG measurement) (CAD). Notably, primary organic carbon emissions from traffic increased platelet activation, systemic inflammation, and maybe decreased antioxidant enzyme activity in elderly persons with CAD [23]. CB particles were found to be linked to rapid cardiovascular alterations, which may undermine "healthy aging" and lead to cardiovascular illnesses [24]. Numerous research using experimental models showed how soot poisoning causes circulatory dysfunctions. According to a study, CB has an impact on mice's cardiac autonomous nervous system activities. This showed that despite apparent myocardial and pulmonary injury, the CB can still cause cardiovascular dysfunctions. When given orally to rats, CB nanoparticle treatment also affected endothelium alterations through altering the expression of nitric oxide synthase. It was discovered that long-term exposure to soot (fine particulate air pollution) was linked to negative cardiovascular outcomes. [25]. A study using mice demonstrated that biodiesel particles are more harmful to health and can worsen cardiovascular problems. Heart rate (HR) and mean corpuscular volume were higher in this study when compared to the control. Intriguingly, there was also an increase in neutrophils, macrophages, platelets, metamyelocytes, leukocytes, and reticulocytes when compared to control.

3.5 Therapeutic Actions that Could be Taken to Control Soot and Carbon Black (CB) Related Disorders

Some treatment methods have been proposed in recent years to counteract the negative effects of soot or carbon black [26]. According to the literature that is currently available and the explanation above, the mechanism of soot toxicity involves immune cells, inflammatory mediators, and different molecules of oxidative stress sensitive pathways. Therefore, each of these could serve as a key target for the creation of novel therapies. These may be used to address toxicities brought on by soot or CBs. Soot and CB poisoning may benefit greatly from antioxidant therapy. There are few examples of antioxidant therapy for pulmonary toxicity in the literature currently available [27]. In a mouse model of the study, the antioxidant zerumbone inhibited Th2 responses brought on by ovalbumin and reduced airway inflammation. In a rat model of asthma, the flavinoid antioxidant naringin also reduced airway inflammation. In a mice model of asthma, allium cepa extract and quercetin also demonstrated protective effects [28]. Natural anti-oxidant Crocus sativus and its primary components, safranal and crocin, have demonstrated protective benefits against oxidative stress in a mouse model of asthma [29]. A mouse model of asthma has demonstrated the protective effects of the well-known antioxidant resveratrol. The effect of artesunate, which significantly decreased the levels of oxidative biomarkers, 3-nitrotyrosine, and 8-isoprostane in a mice model of lung injury, provided a clear illustration of antioxidant therapy to the soot or carbon black produced injury. Likewise, it was discovered that the natural antioxidant melatonin decreased airway inflammation in an asthma model [30].

4. CONCLUSION

This review has shown that human exposure to sooth and carbon black contaminated environment can lead to physiological alterations which could lead to carcinogenesis, respiratory diseases and cardiovascular diseases among other diseases yet to be fully explored. Treatment options available are based on the identified mechanisms that results to the various diseases identified.

5. LIMITATION OF REVIEW

Most studies have considered the pathology of soot to be attributed to inhalation but from the earlier section, it was stated that air was just one out of the components of the environment that serves as a means of exposure to man. Water and soil sources and their effects on man are scarcely studied, thus poorly reviewed.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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