Review Article

SOOT POLLUTION AND PATHOLOGICAL IMPLICATIONS

Abstract

Soot is a black particulate matter made up of carbon and results from the incomplete combustion of fossil fuels. Soot is considered as unwanted by-product derived from incomplete combustion of carbon containing materials. 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. Mechanism for respiratory disorder was described as direct contact-mediated dysfunction of the lung cells and involvement of immune response resulting in tissue remodeling and fibrosis. 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.

Introduction

Soot is a black particulate matter made up of carbon and results from the incomplete combustion of fossil fuels. Soot is considered as unwanted by product derived from incomplete combustion of carbon containing materials. It is powdery mass of black particles which consists of impure carbon, formed after the incomplete combustion of hydrocarbons [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]. Among hydrocarbons, the poly aromatic hydrocarbons (PAHs) are the main carcinogenic compounds in the soot. At elemental level, the most characterized diesel soot contains carbon as a major component, hydrogen, oxygen, sulfur, and trace amounts of metals. The major component of soot which is the Black Carbon causes premature human mortality and disability

Nations and economies are deeply concerned about public health. First, public health is a metric for determining whether or not an economy is productive. As a result, it is widely assumed that good health is essential for a country's growth and development. A healthy population is a

driving force behind economic growth. This suggests that an economy has a problem when individuals, especially the reproductive elements are poor in health. It is on this basis that public health is taken seriously [3].

For good health to be achieved in any economy, the environment must play a significant role as the relationship between both cannot be denied [4,5]. The environment is home to a wide range of resources that man uses to sustain his existence, in this sense clear air, potable water, nutritious food are environmental products that can help people live healthy lives. Similarly, advances in mineral exploitation have devalued the environment over time. One of the major environmental issues encountered here in Port-Harcourt, Rivers State is the issue of black soot also known as Black carbon or carbon Black [6].

EFFECTS OF SOOT IN THE ENVIRONMENT

Air and Water

Soot causes several environmental problems such as haze and acidification of lakes and rivers. Haze is formed when sunlight interacts with small particles in the atmosphere. Soot is the primary cause of haze, which severely decreases visibility and can lead to plane crashes or road accidents. [6]

Terrestial Environment

Soot particles carried by wind or water can end up depleting the nutrients in the soil and damaging sensitive farm crops, and changing the nutrient balance in the river basins along coastlines, and in forests. Acidification through soot pollution can also stain stone (rocks??) and erode it, slowly discolouring and damaging important national monuments and iconic buildings. [6]

TOXICITY AND SIDE EFFECTS OF BLACK SOOTS

MAJOR DISEASES AND RARE PATHOLOGICAL MANIFESTATIONS DUE TO SOOT AND CARBON BLACK

Over three centuries, the linkage of soot and carbon black with different diseases has been observed. Soot and carbon blacks causes many diseases but only three are understood to some

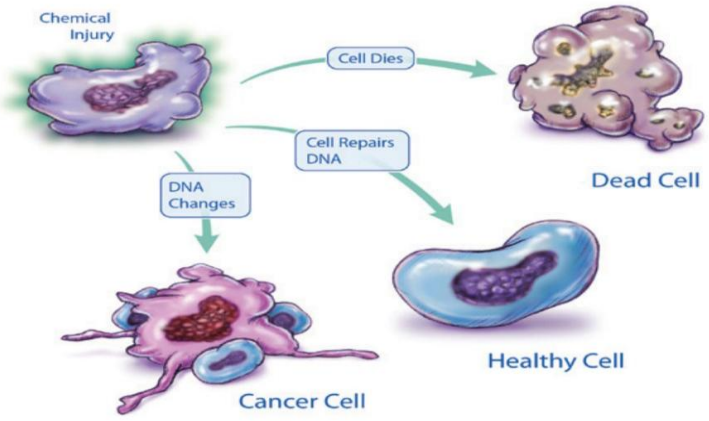
details. The more complex disease associated with the soot and CB is the occurrence of cancer. Soot- and CB-induced cancers are localized and systemic in nature [7]. The second major health issue with soot and CB is respiratory disorders, which sometimes can be very severe. The third one is the cardiovascular dysfunctions. Apart from these diseases, some unique pathological observations have also been seen in response to soot or CB exposures. In a study, prenatal exposure of printex-90 caused sexual and neuroinflammatory changes in mice [8]. Surprisingly, lung exposure of diesel engine exhaust significantly influenced proinflammatory markers of the rat brain. In another study, Printex-90 lowered the sperm production [9]. Similarly, carbon nanoparticles were found to adversely affect the male reproductive system of mice. Recently, it was known that Carbon black exerts developmental toxicity by the immune activation in the male offspring of mice. Soot from a transformer fire was also seen to induce salivary gland duct metaplasia in guinea pigs. These studies show the involvement of systemic response of the body in the development of different pathologies that further needs extensive exploration.

THE PATHOLOGICAL MECHANISMS OF CANCER DUE TO SOOT AND CARBON BLACKS

As introduced in the previous section, soot is the first known carcinogen responsible for the development of different types of cancer in humans and experimental models. These cancers may have local or distal appearance from the site of exposure. It was noted that despite the efforts of 200 years to control the safety in ~~the~~ soot-related work, chimney sweeps still show increased mortality from cancer. In line of this, a case report from Gerber described that the development of penis carcinoma in chimney sweeps was caused due to soot exposure. In Swedish chimney sweeps, the cancer excess was also reported due to soot and asbestos exposure [10]. Soot is absorbed and transported to blood by airway epithelium and majority of the cancers in the distal body parts may be accompanied due to this mechanism of soot transportation. A population-based study showed that, occupational exposures to polycyclic aromatic hydrocarbons, a component of soot is responsible for respiratory and urinary tract cancers. Another case–control study in rubber manufacturing industry showed CB as a major contributor to the early cancer of skin. Subsequently, it became clear that exposure to polycyclic aromatic hydrocarbons (PAHs) in diesel soot are responsible in the development of prostate cancer. Furthermore, CB nanoparticle exposure-mediated human health risk was confirmed by gene

expression profiling [11]. Contradictory to the other studies, the International Agency for Research on Cancer (IARC) in Montreal, QC, Canada reported that the subjects with occupational exposure to titanium dioxide, industrial talc, CB, and cosmetic talc did not experience any detectable excess risk of lung cancer [12]. However, this study was limited to the lung pathology alone, and no other organs were investigated. In addition to the above described evidences, experimental models also provided data to further support the cancer causing properties of soot and CB. Study conducted on dogs demonstrated that the absorption of soot through alveolar epithelium is means of entry into the circulation of un-metabolized PAHs. It has been shown in rat model that soot particle interactions with lung tissue is responsible for morphological changes in the lungs. The diesel exhaust (DP) and CB when regularly inhaled by rats showed toxic and pulmonary carcinogenic properties. In vitro study on the carcinogenic potency of CB confirmed the genotoxic basis of soot toxicity [13]. Also, a mutation at the hprt locus was observed in the metabolically competent AHH-1 cell line (human lymphoblast cell line) in response to soot. It was further confirmed in an in vitro study of cultured cells that the soot causes the mutation in the DNA and induces genotoxic effect [14].

Figure 1 : Toxicant-induced carcinogenesis



Illustrating the mechanism of toxicant-induced carcinogenesis [15]

THE MECHANISMS OF PATHOPHYSIOLOGY IN RESPIRATORY DISEASES DUE TO SOOT ~~PRODUCTION~~ (EXPOSURE??)

The respiratory epithelium of the lungs is the first tissue to get constant exposure with different kinds of soot and CBs present in the environment. Soot toxicity causes the interruption of respiratory process by alteration in lung functions. These toxicological mechanisms may be of two kinds. The first mechanism is the direct contact-mediated dysfunctions of lung cells that include ROS generation, cell hyperplasia, cell death, or apoptosis of lung airway epithelium and other adjacent cells [16]. The second mechanism includes the involvement of systemic immune response resulting in the development of tissue remodeling and fibrosis that causes problem in breathing and lung dysfunctions. In this section, we would discuss these two types of toxicities caused by soot or CB in the context of human clinical and animal studies. The two respiratory diseases that are mainly reported in humans due to soot exposure are chronic obstructive pulmonary disease (COPD) and asthma. The pathophysiology of asthma involves the inflammation of airways, tissue remodeling and fibrosis, obstruction of airflow intermittently, and hyper responsiveness of bronchi [17]. The pathophysiology of COPD includes airway inflammation, mucociliary dysfunctions, and structural changes. There are numerous evidences that support the linkage of soot or CB with asthma and COPD. A study reported that an early exposure to the air pollution leads to the development of childhood asthma. Ultrafine particles (UFPs) (soot) and carbon monoxide concentrations are associated with asthma enhancement in the urban children [18]. DEP initiate the alveolar epithelial cell movement by alterations of polarity mechanisms. An epidemiological study reported that healthy subjects were affected by agriculture crop burning with their altered peak expiratory flow rate and pulmonary functions [19]. The patients prone to COPD or asthma already exhibit preexisting oxidative stress and hence are more susceptible toward soot-mediated oxidative damage. Interestingly, it is known that CB causes adverse effects via ROS and may have worse manifestations in these susceptible persons. The evidences from animal models also supported soot- and CB-mediated mechanisms of toxicity. A rat model of study described that the flame-generated ultrafine soot increased the ROS and upregulated Nrf2 antioxidants in the lungs [20]. Oxidative stress produced by soot or CB is subsequently linked with systemic immune response (inflammation) in the lungs, which results in the development of asthma and other diseases [21].

THE MECHANISM OF CARDIOVASCULAR DYSFUNCTIONS DUE TO SOOT AND CARBON BLACK EXPOSURES

The cardiovascular diseases due to soot and CB exposure are of major concerns because of their distal appearance from the site of exposure and involvement of more systemic responses. A sufficient amount of clinical and epidemiological data linked soot and CB to cardiovascular dysfunctions. A case-crossover study showed that personal soot exposure is linked with acute myocardial infarction. Soot was also seen responsible in the incidence of myocardial infection. In London, air pollution (BC) caused the activation of implantable cardioverter defibrillators (a device used to treat cardiovascular dysfunctions). In Darwin, Australia, the risk of cardiovascular hospitalization was high in people exposed with bushfire particulates [22]. Furthermore, air pollution was considered as a major risk factor to the ST-segment (a measure in electrocardiogram) depression in patients suffering from coronary artery disease (CAD). Notably, traffic emission sources of primary organic carbon particles enhanced platelet activation, systemic inflammation, and potentially reduced antioxidant enzyme activity in old people, suffered from CAD [23]. It was observed that CB particles were associated with accelerated cardiovascular changes, which may compromise “healthy aging” and may trigger cardiovascular diseases [24]. Many studies on experimental models demonstrated the mechanistic basis of soot toxicity leading to cardiovascular dysfunctions. A study revealed that CB affects cardiac autonomous nervous system functions in mice. This indicated that the CB can cause the cardiovascular dysfunctions independent of apparent myocardial and pulmonary injury. CB nanoparticles exposure also caused endothelial changes via modulating nitric oxide synthase expression when it is orally given to the rats. The long-term exposure of soot (fine particulate air pollution) was found associated with the adverse cardiovascular outcomes [25]. The fact that biodiesel particles are more toxic to health and can cause more cumbersome cardiovascular health issues was shown in a mice model of study. In this study, heart rate (HR) and mean corpuscular volume were increased compared with control. Interestingly, leukocytes, reticulocytes, platelets, metamyelocytes, neutrophils, and macrophages were also increased compared with control.

POSSIBLE THERAPEUTIC INTERVENTIONS TO COMBACT SOOT AND CARBON BLACK (CB) ASSOCIATED DISORDERS.

In recent years, some therapeutic strategies have been suggested to combat the adverse effects of soot or carbon black [26]. As understood from the existing literature and from above discussions, the mechanism of soot toxicity involves immune cells, mediators of inflammation, and various molecules of oxidative stress responsive pathways. Therefore, these all may contribute as important targets for the development of novel therapeutics. These can possibly be used to treat soot- or CB-induced toxicities. The antioxidant therapy can be an important way of treating soot and CB toxicity. The existing literature already reported some examples of antioxidant therapy for the pulmonary toxicities [27]. Zerumbone, an antioxidant, attenuated Th2 responses induced by ovalbumin and decreased airway inflammation in a mice model of study. Similarly, naringin, a flavinoid antioxidant, also attenuated airway inflammation in a mouse model of asthm. *Allium cepa* extract and quercetin also showed protective effect in a mice model of asthma [28]. Crocus sativus, a natural antioxidant, and its main constituents, safranal and crocin, have shown the protective effects against the oxidative stress in the mice model of asthma [29]. Resveratrol, a well-known antioxidant, has also shown its protective effects in a mice model of asthma. The clear example of antioxidant therapy to the soot or carbon black caused injury came from the effect of artesunate, which significantly decreased the levels of oxidative biomarkers, 3-nitrotyrosine, and 8-isoprostane, in a mice model of lung injury. Similarly, melatonin, a natural antioxidant, was found to reduce airway inflammation in an asthma model [30].

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.

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.

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