Soot in Rivers State and Haematological Insults

Abstract

Soot is considered as unwanted by-products derived from incomplete combustion of carbon-containing materials. Soot is also known as black carbon (BC) or carbon blacks (CBs) which can cause many health issues in humans and animals. Soot arises from the illegal activities of refinery of crude and burning of petroleum products. Soot emissions are reportedly largest in developing countries. It is associated with various diseases in the human population; these diseases include respiratory diseases, cardiovascular diseases, cancers, and probably reproductive system defects. Rivers State is a highly populated State with multinational companies that are involved in one form of exploration and mining or another. The increasing level of soot in the air and environment of Rivers State is mainly due to two factors, the activities of security agencies that burn off illegally bunkered petroleum products and the activities of unlawful oil refinery operators who run their operations from the creeks and surroundings of some local communities and Port Harcourt, the State headquarters. The aim of this review was to assess the effects soot has on the haematological parameters of individuals living in Rivers State. Soot has been shown by several studies to cause some deviation in the haematological parameters such as a marked decrease in the PCV, Hb and RBC. However, it also brings about an elevation in the total white blood cell count, neutrophils and lymphocytes. This review provides information on the haematological insults associated with soot exposure in Rivers State.

Comment [u1]: The mechanisms of soot toxicity in relation to suppression of bone marrow and some other mechanisms should be included in the abstract for more clarity. There should be clear conclusion in the abstract.

Keywords: Soot, Hematological insults, Rivers State, Red Blood Cell, White Blood Cell.

Introduction

The environmental soot [black carbon (BC)] or carbon black (CB) cause many health issues in humans and animals (Buchner *et al*., 2012; Agarwal *et al.,* 2013). The terms soot and CB have been used interchangeably but, both are physically and chemically distinct entities (Medalia *et al., 1983;* Long *et al.*, 2013). Soot is considered as unwanted by-products derived from incomplete combustion of carbon-containing materials (Medalia *et al., 1983;* Long *et al.,* 2013;). In contrast, the CBs are manufactured under the controlled conditions in the rubber, printing and painting industries for commercial use (Medalia *et al., 1983;* Long *et al.,* 2013;). Soot is a powdery mass of fine black particles (Chuang *et al.,* 2011). It consists of

impure carbon, formed after the incomplete combustion of hydrocarbons (Canagaratna *et al.*, 20109). The main source of environmental soot is the combustion of fossil-based fuels and biomass burning at the Earth's surface (Glaser *et al.*, 2005). The other examples of soot may include coal, charred wood, petroleum coke, cenospheres, and tars (Birky & Voorhees, 1989; Scheepers & Bos, 1992). To a smaller extent, quartz/halogen bulbs with settled dust, cooking, oil lamps, smoking of plant matter, fireplaces, candles, house fires, furnaces, and local field burning also contribute to the soot production (Kamboures *et al*., 2013). Soot particles range from about 10 nm to 1 mm in size (Niessner, 2014; China, 2013). The relative amount of elemental carbon inside soot is considered to be less than 60% of the total mass of particle (Watson & Valberg, 2001; Cain *et al*., 2010). Among hydrocarbons, the poly aromatic hydrocarbons (PAHs) are the main carcinogenic compound in the soot (Cain *et al*., 2010– Wang *et al*., 2001). At elemental level, the most characterized diesel soot contains carbon (as a main component), hydrogen, oxygen, sulfur, and trace amount of metals (Fernandes & Brooks, 2003 Yang *et al*., 2012). The major component of soot, the BC, causes premature human mortality and disability (Goto, 2014).

Soot is particulate matter with varying sizes. They exit in the range of 10nm to 1mm in size and have less than 60% of carbon in their total mass (Elechi-Amadi *et al*., 2019). Regrettably, soot emissions are reportedly largest in developing countries (Elechi-Amadi *et al*., 2019). It is associated with various diseases in the human population, which include respiratory diseases, cardiovascular diseases, cancers, and probably reproductive system defects (Elechi-Amadi *et al*., 2019).

Rivers State is a highly populated State with multinational companies that are involved in one form of exploration and mining or another. The increasing level of soot in the air and environment of Rivers State is mainly due to two factors, the activities of security agencies that burn off illegally bunkered petroleum products and the activities of unlawful oil refinery operators who run their operations from the creeks and surroundings of some of the communities around and Port Harcourt, the State headquarters.

The soot exposure derived from the remnants of combustion products can affect health through various mechanisms. One of the mechanisms suspected to play a role is the occurrence of oxidative stress resulting in increased reactive oxygen species (ROS) or free radicals in the body. Soot exposure stimulates the release of inflammatory cytokines in addition to macrophages and leukocytes. The stimulation of soot to produce cytokines is

associated with the activation of mitogen-activated protein kinase (MAPK) in cells which responds to inflammation (Shi *et* al., 2019). The occurrence of activation of MAPK will trigger the release of proinflammatory cytokines which will eventually result in extensive tissue damage in blood vessels. (Sandra *et al.*, 2015). It is therefore important to evaluate the haematological damages caused by exposure to sooth in Rivers State.

Comment [u2]: This needs reversal. How can soot be stimulated?

Characteristics of Soot

There are many types of soot based on the sources of the soot such as from a turbo diesel engine operated under various loads, biodiesel fuel, spark discharge generator and soot generated with a standard propane burner in a diffusion flame at various carbon to oxygen (C/O) ratios.

Carcinogenicity of Soot

The routes of entry of soot into the human body include ambient and indoor air inhalation, ingestion of contaminated food, and dermal contact. Based on sufficient evidence from human carcinogenicity studies, soot causes cancers in humans (International Agency for Research on Cancer (IARC), 1985). In 1775, British surgeon Percivall Pott established a correlation between scrotal cancer and exposed chimney sweeps. In subsequent years, substantial amounts of epidemiological studies have since confirmed the elevated risk of scrotal and other skin cancers among chimney sweeps (Evanoff *et al*., 1993). Similarly, research establishes strong association between occupational exposure (chimney sweeps) mortality from lung cancer, in a number of European countries. In a separate study, leukemia and cancer of the esophagus, as well as risks of liver cancer correlated with exposure to soot (Centre for Disease Control and Prevention, 2017). Correspondingly, follow-up studies among Swedish chimney sweeps revealed that risks for esophageal, hematopoietic, prostate, urinary bladder, and total lymphatic cancer were elevated (International Agency for Research on Cancer, 1987).

Comment [u3]: Rather obsolete. There may be recent studies in favour or against

2.2 Soot and its effect on human health

Soot pollution is a result of long years of careless, reckless, irresponsible and unsustainable burning and use of fossil fuel (Akutu, 2018). When soot builds up in the air, it eventually deposits onto surfaces due to random collisions with surfaces. When particles collide and grow in size, they gain enough mass to deposit due to gravity. (Elem, 2015). Other

environmental factors may possibly shorten the time necessary for soot to deposit, but it should be understood that eventually the soot will deposit onto surfaces. Soot is hazardous to health as the toxic pollutants of inhale-able sizes of between 10 -2.5 micrometer can penetrate into human lungs and blood stream to cause respiratory, cardiovascular diseases and cancer (Feng *et al., 2015)*. Air pollution is associated with a number of adverse health impacts. Air pollution particularly affects the most vulnerable in society: children and older people, and those with pre-existing heart and lung conditions (Ana *et al., 2011).* There are studies and reports that have confirmed the links between air pollution and ill health and premature death, and the complex cumulative impacts of different pollutants on health are increasingly well understood. In Rivers state, much attention is given to general industrial pollution and pollution in oil industries with little reference on damage of pollution caused by mobile transportation sources of air pollution.

The influence of a polluted environment on public health has been established by different scholars. Munguti (1988) in a study of environmental degradation and disease in Kenya observed that the leading causes of morbidity in Kenya are environmentally based. Kelishadi, (2012) in his study of environmental pollution, health effects, and operational implications for pollutants removal equally observed that environmental pollutants have various adverse health effects on man. Marchwinska-Wyrwal *et al.,* (2011) also affirm that such relationship exists. For these scholars, air pollution exist and air pollution is a major cause of environmental health problem and that it caused about two million premature death globally. Over the years, a new dimension to this emerged due to (air pollution) which is a product of rising cases of artisanal refineries and other activities which over time produced soot. The operators of these artisanal refineries rely on oil theft (Goodnews & Wordu, 2019). To curb this, the government set up Joint Military Taskforce (JTF) to monitor the activities of these operators. As it stands, illegal refineries are virtually in all coastal communities and cannot be stopped easily. The taskforce operation technique is to set these refineries on fire. Sadly, as soon as the disruptions are made, the operators re-erect new ones. So the scenario is that of; erect, destroy and re-erect. As this cycle is maintained, so also is soot emission.

Niranjan and Thakur (2017) also revealed that soot particles from the 1991 oil fields of Kuwait induced genetic mutation which is capable of damaging Deoxyribonucleic acid (DNA). These scholars confirmed that soot causes leukemia, cancer of the liver, oesophagus and skin. The research equally asserts that there is a high incidence of cancer in the area of this study and that its prevalence is related to the presence of soot in the area of their study.

A separate study by Akutu (2018) titled "Health issues to know about soot, preventive measures" revealed that long-time exposure to soot can cause pneumonia. This condition makes patients prone to other respiratory diseases like asthma. Like other scholars, the proceedings of the National Academy of Science study in 2019 reaffirm other scholars' findings on the part of soot in public health. The findings from the study revealed that soot caused an estimated 131,000 premature deaths in Latino city of America in 2015. Nwachukwu *et al.,* (2012) in their study titled "the effects of air pollution on diseases of people of Rivers State, Nigeria" discovered that the pollution level in Rivers State is not only high but also higher than the World Health Organization recommendation. The rise in air pollution is reported to be the cause of high morbidities and mortalities in Rivers State. The research reveals that cerebrospinal meningitis (CSM), chronic bronchitis, measures, pertussis, pulmonary tuberculosis, pneumonia, and hyper respiratory tract infection were the most prevalent in the years of the study. Nwachukwu *et al.,* (2012) concluded that pneumonia accounts for the highest number of deaths in 2015. Weli and Adekunle (2014) in a study of environmental risk factors and hospital-based cancers in two Nigerian cities established a close relationship between air pollution including soot with morbidities like respiratory diseases, traumatic skin, outgrowth and respiratory health condition, child deformities, stillbirth, miscarriage.

Haematological insults associated with soot exposure in Rivers State

Environmental pollutants e.g. soot arises from the illegal activities of refinery of crude and burning of petroleum products this has been shown by several studies to cause a marked decrease in the PCV, Hb and RBC. (Elechi-amadi *et al.,* 2019). However, the total white blood cell count, neutrophils and lymphocytes were significantly elevated. As shown by the study carried out by Elechi –amadi *et al., (*2019), this may be as a result of inflammatory response of the immune system and bone marrow to soot with associated haemolysis. Many high molecular weight carbon compounds have one form of systemic toxicity. For instance, benzene is toxic in human at any concentration, and may cause haematotoxicity and bone marrow depression, when inhaled for prolonged period. Naphthalene, another compound if inhaled in large amount, is known to destroy the membrane of red blood cells leading to haemolysis. (Elechi-amadi *et al.,* 2019).

Comment [u4]: You mean bone marrow suppression?

Previous studies carried out reported the effect of flared gases on humans is related to the exposure of hazardous air pollutant emitted during incomplete combustion of the flared gases

(Kindzierski, 1999). Related studies also reported a similar decrease in the PCV, Hb, and RBC among petrol station attendants. (Okoro *et al*., 2006).

WBC count reported from similar studies has been contradictory. Some reported a reduction of the WBC count; others have reported an increase as was observed in the study bu Elechi-Amadi *et al*., (2019) (Ovuru SS, Ekweozor, 2004; Owu *et al*., 2005). The increased in the WBC count may be associated to stress induced changes in the haemopoietic pathway by components of the soot. Another study carried out by Adienbo and Nwafor, (2010) showed that the concentrated environmentally associated pollutants arising from prolonged exposure to oil and gas activities in the environment can caused a marked increase in the abnormality of red blood cell morphology and WBC count with a corresponding decrease in PCV, Hb and RBC. Previous studies had reported that stained smears of red blood cells from nestling herring gulls that ingested Prudhoe crude oil were characterized by reduced red cell count, Hein body formation, anisocytosis, poikilocytosis and reticulocytosis (Leighton *et.al.*, 1985); and it has been suggested that the effect of flared gases on humans is related to the exposure of hazardous air pollutants emitted during incomplete combustion of the flared gases (Kindzierski, 2000). Benzene is a known systemic toxicant in human at any concentration, when inhaled for prolonged period, with haematotoxic and bone marrow depressant effects. Naphthalene, if inhaled or ingested in large amount, is known to destroy the membrane of red blood cells leading to its breakdown. This may have also contributed to the deterioration in the observed parameters as well as the change in morphology. The increase in WBC count may be associated to stress induced changes in the haemopoietic pathway.

Comment [u5]: Dont start statement with abbreviation

Comment [u6]: Heinz?

Possible Therapeutic intervention to Combat Soot Associated Disorders

In recent years, some therapeutic strategies have been suggested to combat the adverse effects of soot. As understood from the existing literature the mechanism of soot toxicity involves immune cells, mediators of inflammation, and various molecules of oxidative stress responsive pathways (Allan *et* al., 2010; Patella *et al.*, 2015). Therefore, these all may contribute as important targets for the development of novel therapeutics (Cho, 2011). Some of these therapies include:

The antioxidant therapy which can be an important way of treating soot and CB toxicity (Hoffman *et al*., 2015; Provotorov *et al*., 2015). The existing literature already reported some examples of antioxidant therapy for the pulmonary toxicities (Allen *et al*., 2009). Zerumbone,

Comment [u7]: Rewrite. Something is missing

an antioxidant, attenuated Th2 responses induced by ovalbumin and decreased airway inflammation in a mice model of study (Shieh *et al.,* 2005). Similarly, naringin, a flavinoid antioxidant, also attenuated airway inflammation in a mouse model of asthma (Guihua *et al*., 2015).

It is evidenced that immune cells such as mast cells, eosinophils, T cells, and neutrophils are the major culprit in soot and toxicity. Therefore, these may be targeted for the development of noble therapeutic approaches (Kato *et al.,* 1992). A monoclonal antibody mepolizumab against the eosinophils activation has been developed and is currently in clinical trials against severe eosinophilic asthma (DREAM) (Pavord *et al*., 2012). Similar strategies can be used against eosinophils and other mediators of immune response in soot- and CB-mediated toxicity. The mast cells may be the next important target for which a number of therapeutic interventions have been developed (Hugle, 2014). Notably, CGS 9343B, a strong inhibitor of calmodulin family, has a potential to inhibit histamine release by mast cells as shown in rats (Veerappan *et al*., 2013). Inhibitors of dectin-1 signaling (R406) downregulated mast cells activation, thus can also be used as a novel therapy to target soot-induced mast cell's toxicity (Veraldi *et al.*,2016).

Conclusion

Prolonged exposure to flaring of associated gas and by extension to oil production environment can cause marked deterioration in haematological parameters. Exposure to soot polluted air has the capacity to bring about marked alteration in some haematological parameters. This review suggests that leaving in a soot polluted environment is harmful and can cause morbidity and mortality especially in patients with underlined chronic medical conditions.

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