Soot in Rivers State and Haematological Insults

Abstract

Soot is known as unwanted by-products gotten from incomplete burning of materials that contain carbon. Soot is also known as black carbon (BC) or carbon blacks (CBs). It is capable of causing many health issues in both humans and animals. When illegal refining and burning activities are carried out, the end product is usually the production of soots. Cases of soot emissions rising in developing countries. It is one cause of diseases in the human population; these diseases include respiratory diseases, cardiovascular diseases, cancers, and sometimes reproductive system abnormality. Rivers State is a highly populated State with lots of companies carrying out one form of refining and exploration or another. Two factors are responsible for the rising increase of soot in Rivers State, the burning of illegal petroleum product by security agencies and the operation of unlawful illegal refineries carried out 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 decrease in the PCV, Hb and RBC. However, it also brings about an increase 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.

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

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Introduction

Soot which is also known as black carbon (BC) or carbon black (CB) is responsible for many health challenges in both humans and animals (Buchner *et al*., 2012; Agarwal *et al.,* 2013). The word soot and carbon black is usually used interchangeably but, they both have physical and chemical differences(Medalia *et al., 1983;* Long *et al.,* 2013). Soot is known as unwanted by-products gotten from incomplete burning of substances containing carbon (Medalia *et al., 1983;* Long *et al.,* 2013;). while the Carbon Blacks are products found in the rubber, printing and painting industries which are produced under controlled condition for commercial use (Medalia *et al., 1983;* Long *et al.,* 2013;). Soot is powdery in nature and black in colour (Chuang *et al.,* 2011). It is made up of carbon that are impure which is formed when there is

an incomplete combustion of hydrocarbons products (Canagaratna *et al.*, 20109). The primarily source of soot is the burning of fossil fuels and the burning of materials on earth(Glaser *et al.*, 2005). Examples of other materials that produces 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 materials, fireplaces, candles, house fires, furnaces, and local field burning also add to the production of soot in the environment (Kamboures *et al*., 2013). The size of the particleof soot is from 10nm to 1mm in size (Niessner, 2014; China, 2013). The amount of elemental carbon inside soot is known to be 60% of the total mass of soot particle (Watson & Valberg, 2001; Cain *et al*., 2010). The main carcinogenic carbon fount in soot among hydrocarbon is the poly aromatic hydrocarbons (PAHs) (Cain *et al*., 2010– Wang *et al*., 2001). Diesel soot at the very elemental level contain a known amount of soot (as a main component), hydrogen, oxygen, sulfur, and trace amount of metals (Fernandes & Brooks, 2003 Yang *et al*., 2012). Soot components are the reasons for premature mortality and disability (Goto, 2014).

Sootis made of of different sizes. There sizes ranges fom 10nm to 1mm and contain 60% of the soot particles in their total mass (Elechi-Amadi *et al*., 2019). Soot production is more in developing countries than in developed countries (Elechi-Amadi *et al*., 2019is is one of the cause of different kind of diseases in humans and animals, which include respiratory diseases, cardiovascular diseases, cancers, and sometime reproductive system abnormality (Elechi-Amadi *et al*., 2019).

Rivers State is a highly populated State with a lot of companies carrying out different activities that bring about the production of soot such as mining and exploration. Two factors are responsible for the rising amount of soot in the air and environment around Rivers State; the burning off of illegal refineries by security agenciesand the activities of illegal refining of petroleum which is run from the creeks and surroundings of some of the communities around Port Harcourt, the State headquarters.

There are various mechanism through which remains of soot combustion can affect human health. One of which is what is known to bring about oxidative stress which consequently bring about an increase in Reactive Oxygien Species (ROS) or free radicals in the human body. The exposure to soot brings about the release of inflammatory cytokines, leucocytes and macrophages. When soot stimulate cytokine production it brings about the activation of

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mitogen-activated protein kinase (MAPK) in cells which responds to inflammation (Shi *et* al., 2019). This will futher bring about the release of proinflammatory cytokines which which consequently lead to 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.

Characteristics of Soot

Different types of soot exists based on their sources such as from a turbo diesel engine operated using various mechanism, biodiesel fuel, spark discharge generator and soot produced with a standard propane burner in a diffusion flame at varying carbon to oxygen (C/O) ratios.

Carcinogenicity of Soot

Soot enters the human body either through ambient or indoor inhalation, gotten through the ingestion of food that is contaminated or through the skin. Studies carried out on the carcinogenicity has shown thatsoot causes cancers in humans (International Agency for Research on Cancer (IARC), 1985). A study carried out by a British surgeon Percivall Pott in 1775 were he discovered a relationship between scrotal cancer and exposed chimney sweeps. This was further confirmed by several other epidemiological studies inwhich it was discovered that there was an increased risk of scrotal and other skin cancer among chimney sweeps (Evanoff *et al*., 1993). Similarly, there is also strong evidence from research that shows great association among occupational exposure (chimney sweeps) death from lung cancer, in some of European countries. Another study also confirmed a great risk of leukaemia, cancer of the esophagus and liver cancer with exposure to soot (Centre for Disease Control and Prevention, 2017). A corresponding follow-up study also confirmed an increased risk of esophageal, prostrate, haemopoietic, urinary bladder and lymphatic cancer among Swedish chimney sweeps (International Agency for Research on Cancer, 1987).

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). After the build up of soot in the air, it gets in contact with surfaces through random collision with those surfaces. As these particles collide and their size increases, they accumulates enough mass that is deposited later due to gravity

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(Elem, 2015). The time for soot deposited is shortened by other environmental factors, but the soot is consequently deposited onto surfaces anyways. 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 causes several health challenges. It has more impact on the most vulnerable in the society, children, elderly and those with pre-existing heart and lung conditions (Ana *et al., 2011).* Several studies have shown the relationship between air pollution, ill-health and premature death, and the very dangerous effects of pollutants are established. In Rivers state, a lot of attention is given to pollution caused by oil industries but little or no attention is given to those caused by transportation sources.

Different researchers have done a lot of studies on the impact of environmental pollution on public health. In a study carried out by Munguti (1988) on environmental degradation and disease in Kenya he discovered that the cause of premature deaths in Kenya is caused by environmental pollution. Another study by Kelishadi, (2012) on environmental pollution, health effects, and operational implications for pollutants removal. Kelishadi, (2012) also observed that environmental pollution causes great health challenges to human. Marchwinska-Wyrwal *et al.,* (2011) also confirmed that in their studies. All these researchers agreed that air pollution exixt and its the cause of about two million premature death globally. As the years went by, a new dimension to this discovery occurred due to (air pollution) increasing activities of artisanal refining and other activities which produces soot over time. The artisanal refinaries rely on oil theft for their operation (Goodnews & Wordu, 2019). To minimize or possibly bring this to an end, the Joint Military Taskforce (JTF) was established in order to moniotor the activities of these illegal refineries. At the time of this study, illegal refineries are soread all over the coastal communities and cannot be easily stopped. In a verge to monitor the activities of these refineries, the JTF sets them on fire. Sadly, the operators of these illegal refineries set up new ones immediately their old refineries are burnt down by the JTF. The circle continuous and the release of soot also increases.

A study by Niranjan and Thakur (2017) shows that soot exposure brings about a genetic mutation which affects humans by damaging the Deoxyribonucleic acid (DNA). These researchers confirmed that exposure to soot causes skin cancer, leukaemia, esophageal and liver cancer. The research also shows that the incidence of cancer in Rivers State is also very high and it is as a result of increase in the presence of soot in the study area. Another study done by Akutu (2018) on “Health issues to know about soot, preventive measures” revealed

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that pneumonia can be caused by long-term exposure to soot. This can consequently expose patients to other respiratory conditions like asthma. The proceedings of the National Academy of Science study in 2019 confirmed the findings proposed by other scholars' on the effect of soot in public health. The study exposed 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" asserts that the pollution level of soot in Rivers state is even higher than the World Health Organisation recommendation. High cases of Morbidities and mortalities in Rivers State can be attributed to the high level of air pollution in the area. The research by Nwachukwu *et al.,* (2012) reveal that cerebrospinal meningitis (CSM), chronic bronchitis, pertussis, pulmonary tuberculosis, pneumonia, and hyper respiratory tract infection were the most prevalent in the years of the of their study and is attributed to environmental pollution. It was concluded by Nwachukwu *et al.,* (2012) that the highest number of death cases in that year can be attributed to pneumonia. Weli and Adekunle (2014) in their 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, and miscarriage.

Haematological insults associated with soot exposure in Rivers State

Environmental pollutants which include soot are caused by the illegal activities of refinery of crude and the burning of petroleum products. This has been shown by several studies to bring about a significant decrease in the Packed Cell Volume (PCV), Haemoglobin (Hb) and Red Blood Cell (RBC) (Elechi-amadi *et al.,* 2019). However, there was a significant increase in the Total White Blood Cell Count (WBC), Neutrophil and Lymphocytes in individuals exposed to soot. As shown by the study carried out by Elechi –amadi *et al., (*2019), this can be attributed to haemolysis caused by the inflammatory response of immune system and bone marrow to soot exposure. A number of carbon with high molecular weight is highly toxic to humans. For example, at different concentrations, benzene is toxic to humans, and is capable of causing haematotoxicity and bone marrow depression, when it is inhaled for long period. Another compound known as naphthalene can cause a destruction in the membrane of red blood cells leading to haemolysis if they are inhaled in very large amounts. (Elechi-amadi *et al.,* 2019).

A study carried out on the effect of flared gases on humans showed that there is relationship between high exposure to flared gases and toxicity (Kindzierski, 1999). There was also

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similar decrease in the PCV, Hb, and RBC among petrol station attendants who are exposed to hydrocarbon through inhalation (Okoro *et al*., 2006).

Some studies showed contradictory results on WBC count. Some studies reported an increase while others reported a decrease in the WBC count of individuals exposed to soot ((Ovuru & Ekweozor, 2004; Owu *et al*., 2005; Elechi-Amadi *et al*., 2019). Stress induced changes in the haematopoietic pathway can attribute for an increase in WBC count as a result of soot exposure. A study carried out by Adienbo and Nwafor, (2010) showed an increase in the abnormality of red blood cell morphology and WBC count with a corresponding drop in PCV, Hb and RBC as a result of concentrated environmentally associated pollutants brought about by long term exposure to oil and gas activities in the environment. Stained smears of red blood cells from nestling herring gulls that ingested Prudhoe crude oil were observed to have reduced red cell count, Hein body formation, anisocytosis, poikilocytosis and reticulocytosis (Leighton *et.al.*, 1985); it has also been observed that exposure to flared gases caused by incomplete combustion is associated to its effect on humans (Kindzierski, 2000). Benzene is known to be toxic to human at any concentration when inhaled for long period. It causes haematotoxicity and bone marrow depression. Nwhen naphthalene is inhaled or ingested in large amount can cause a damage of red blood cell membrane consequently leading to a breakdown. This may have also contributed to the decrease in the haematological parameters and change in morphology. Stress-induced changes in the haematopoitic pathway may be the reason for an increase in WBC count.

Possible Therapeutic intervention to Combat Soot Associated Disorders

Some therapeutic strategies have been put in place in recent years to fight the disorders caused by the exposure to soot. The mechanism of soot toxicity can be attributed to immune cells, mediators of inflammation, and molecules of oxidative stress responsive pathways as proposed by early studies carried out (Allan *et* al., 2010; Patella *et al.,* 2015). These mechanisms can be exploited for the development of therapeutics (Cho, 2011). Examples of these therapies include:

The antioxidant therapy which can be used to treat soot and carbon black toxicity (Hoffman *et al*., 2015; Provotorov *et al*., 2015). Some literatures have shown the antioxidant therapy for pulmonary toxicity (Allen *et al*., 2009). An antioxidant known as Zerumbone, is shown to attenuate Th2 responses induced toxicity brought about by ovalbumin this study was carried

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out in mice and the antioxicant decresed airway inflammation in the mice used for study (Shieh *et al.,* 2005). Similarly, another flavinoid antioxidant known as naringin, has also been shown to attenuate airway inflammation in a mouse model of asthma (Guihua *et al*., 2015).

Immune cells such as mast cells, eosinophils, T cells, and neutrophils are the main targets in soot and its toxicity. These can be used to develop new therapies (Kato *et al.,* 1992). One of such is a monoclonal antibody known as mepolizumab which is used against the eosinophils activation. This is currently in clinical trials against severe eosinophilic asthma (DREAM) (Pavord *et al*., 2012). Such strategies can be used against eosinophils and other mediators of immune response to soot and carbon black toxicity. The mast cell can as well be exploited to develop new therapies against soot toxicity (Hugle, 2014). Notably, CGS 9343B, which is a strong inhibitor of calmodulin family, has been shown to inhibit histamine release by mast cells in rat models (Veerappan *et al*., 2013). The research shows that Inhibitors of dectin-1 signaling (R406) downregulated mast cells activation, and can also be used as a new therapy to target soot-induced mast cell's toxicity (Veraldi *et al*.,2016).

Conclusion

When an individual is exposed to flared gas or burning of petroleum products, this can lead to a significant decrease in some haematological parameters. Prolonged exposure to soot can bring about an alteration in some haematological parameters. This review suggests that living in a soot polluted environment is quite harmful and is capable of causing morbidity and mortality, those at more risks are patients with pre-existing chronic medical conditions.

References

Adienbo, O.M & Nwafor, I. (2010) Effect of prolong exposure to gas flaring on haematological parameters of Human in the Niger Delta region of Nigeria. *Journal of Applied Science and Environmental Management*, 14(1), 13-15.

Agarwal, R., Awasthi, A., Singh, N., Mittal, S.K. & Gupta, P.K (2013). Epidemiological study on healthy subjects affected by agriculture crop-residue burning episodes and its relation with their pulmonary function tests. *International Journal of Environmental Health Research*, 23(4):281–295.

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Allan, K., Kelly, F.J. & Devereux, G. (2010). Antioxidants and allergic disease: a case of too little or too much? Clinical Expression of Allergy 40(3):370–380.10.1111/j.1365-2222.2009.03413.x

Allen, S., Britton, J.R., Leonardi-Bee, J.A. (2009). Association between antioxidant vitamins and asthma outcome measures: systematic review and 3meta-analysis. *Thorax*, 64(7):610–619.

Ana, G.R (2011). Air pollution in the Niger Delta area: scope, challenges, and remedies. *Open access peer – review*.24-36.

Akutu, G. (2018) "The Guardian Newspaper 27/05/2018: futospace.futo.edu.ng › handle extracted 03/03/21 by 23:26 pm. Web results Health Issues To Know About Soot : Protective Measures

Birky, M.M. & Voorhees, K.J. (1989). The use of soot analysis as an investigative tool in aircraft fires. *Aviation of Space Environmental Medicine* 60(10) 2.

Buchner, N, Ale-Agha, N., Jakob, S., Sydlik, U., Kunze, K. & Unfried, K. (2013). Unhealthy diet and ultrafine carbon black particles induce senescence and disease associated phenotypic changes. *Experimental Gerontology*, 48(1):8–16.

Canagaratna, M.R., Onasch, T.B., Wood, E.C., Herndon, S.C., Jayne, J.T. & Cross, E.S. (2010). Evolution of vehicle exhaust particles in the atmosphere. *Journal of Air Waste Management Association*, 60(10):1192–1203.

China, S., Mazzoleni, C., Gorkowski, K., Aiken, A.C., Dubey, M.K. (2013). Morphology and mixing state of individual freshly emitted wildfire carbonaceous particles. *Natures Commununication* 4:2122.10.

Cho, J.Y. (2011). Recent advances in mechanisms and treatments of airway remodeling in asthma: a message from the bench side to the clinic. Korean Journal of Internal Medicine, 26(4):367–383.

Chuang, H.C., Jones, T., Chen, Y., Bell, J., Wenger, J. & BeruBe, K. (2011). Characterisation of airborne particles and associated organic components produced from incense burning. *Analytical and Bioanalytical Chemistry*, 401(10):3095–3102.

Elechi-Amadi, K. Batubo, N.P. Boma, H.O & Andrew, J. (2019). Toxicological effects of prolonged exposure of soot-polluted air on some haematological and biochemical parameters of residents of Port Harcourt. *International Journal of Research in Medical Sciences, 7(4):1063-1066.*

Elem, M. (2015). Primary health care and maternal mortality in selected areas in Rivers State. Ph.D Dissertation presented to University of Port Harcourt Graduate School.

Feng, S., Gao, D., Liao, F., Zhou, F. & Wang, X. (2015). The health effects of ambient PM2.5 and Potential mechanisms. *Ecotoxicology & environmental safety Journal,* (128)26-36.

Fernandes, M.B. & Brooks, P. (2003). Characterization of carbonaceous combustion residues: II. Nonpolar organic compounds. *Chemosphere* 53(5):447–458.10.1016/S0045-6535(03)00452-1

Glaser, B., Dreyer, A., Bock, M., Fiedler, S., Mehring, M. & Heitmann, T. (2005). Source apportionment of organic pollutants of a highway-traffic-influenced urban area in Bayreuth (Germany) using biomarker and stable carbon isotope signatures. *Environmental Science Technology,* 39(11):3911–3917.

Goodnews, M.M. & Wodu, S.A. (2019) Analysis of trend and emergent factors of artisanal refining in the Niger Delta region of Nigeria. *International journal of innovative human ecology and nature studies,* 7(1).

Guihua, X., Shuyin, L., Jinliang, G. & Wang, S. (2016). Naringin protects ovalbumin-induced airway inflammation in a mouse model of asthma. *Inflammation* 39(2):891–9.10.1007/s10753-016-0321-7

Haitham, H., Al-wakeel, Z.A., Abdul, K., Hussain, H., Al-kayiem & Mat-Jamlus, M.H (2012). Soot reduction strategy; a review. *Journal of Applied Sciences*, 12(23), 2338-2345.

Hugle, T. (2014) Beyond allergy: the role of mast cells in fibrosis. Swiss Med Wkly 144:w13999.10.4414/smw.2014.13999

UNDER PEER REVIEW

Kamboures, M.A., Hu, S., Yu, Y., Sandoval, J., Rieger, P., Huang, S.M. (2013). Black carbon emissions in gasoline vehicle exhaust: a measurement and instrument comparison. *Journal of Air Waste Management Association,* 63(8):886–901.

Kato, A., Kyono, H., Kuwabara, N. (1992). Electron-microscopic observations on rat lungs after long term inhalation of diesel emissions – non-neoplastic lesions. Nihon Kyobu Shikkan Gakkai Zasshi 30(2):238–47.

Kelishadi, R. (2012). Environmental pollution: Health effects and operation implication for pollutants removal. *Journal of environment and public health,* 201-304.

Kindzierski, W.B. (1999). Importance of human environmental exposure to hazardous air pollutants from gas flares. *Environmental Reviews*. 8(1):41-62.

Kettner, H. & Langmann, R.(1970). Air pollution by soot. *Offentel Gesundheitswes* 32(7):346–8.

Lewis, G.P. & Coughlin, L.(1973). Lung "soot" accumulation in man. *Atmospheric Environ* ment 7(12):1249–55.10.1016/0004-6981(73)90134-0

Long, C.M., Nascarella, M.A. & Valberg, P.A. (2013). Carbon black vs. black carbon and other airborne materials containing elemental carbon: physical and chemical distinctions. *Environmental Pollution* , 181:271–86.10.1016/j.envpol.2013.06.009

Marchwinska- Wyrwal, G., Oziubanek, G., Hajok I, Rusin, M., Oleksiuk, K. & Kubasiak, M. (2011) www.interchopen.com.

Medalia, A.I., Rivin, D. & Sanders, D.R.(1983). A comparison of carbon black with soot. *Science of the Total Environment* 31(1):1–22.10.1016/0048-9697(83)90053-0

Munguti, K.J. (2008). Environmental degradation and health in Kenya in International Development Research Conference. Environment and development in Kenya. National Academy of Science.

Niessner, R. (2014). The many faces of soot: characterization of soot nanoparticles produced by engines. *Chemical International Education England,* 53(46):12366–79.10.1002/anie.201402812

Niranjan, R. & Thakur, A.K. (2017). The toxicological mechanisms of environmental soot (black carbon) and carbon black: focus on oxidation stress and inflammatory pathways. Frontiers in immunology 8(763).

Nwachukwu, A.N., Chukwuocha, E.O. & Igbudu, O. (2012). A Survey on the Effects of Air Pollution on Diseases of the People of Rivers State, Nigeria. *African Journal of Environmental Science and Technology,* 6: 371–379.

Okoro, A.M., Ani, E.J., Ibu, J.O. & Akpogomeh, B.A. (2006). Effect of petroleum products inhalation on some haematological indices of fuel attendants in Calabar metropolis, Nigeria. *Nigerian Journal of Physiological Science*,21(1-2).

Ovuru, S.S &, Ekweozor, I.K. (2004). Haematological changes associated with crude oil ingestion in experimental rabbits. *African Journal of Biotechnology,* 3(6):346-8.

Owu, D.U., Udoete, U.B., Azah, N., & Eyong, E.U. (2005). Effect of bonny light crude oil on some haematological parameters of guinea pigs. *Biokemistri*. 17(2):165-70.

Pavord, I.D., Korn, S., Howarth, P., Bleecker, E.R., Buhl, R., Keene, O.N. (2012). Mepolizumab for severe eosinophilic asthma (DREAM): a multicentre, double-blind, placebo-controlled trial. *Lancet,* 380(9842):651–659.

Patella, V., Incorvaia, C., Minciullo, P.L., Oricchio, C., Saitta, S. & Florio, G. Oxidative stress markers in patients with hymenoptera venom allergy. Allergy Asthma Procedure 36(1):9–13.10.2500/aap.2015.36.3814

Provotorov, V.M., Budnevsky, A.V., Filatova, Y.I., Perfil'eva, M.V. (2015). Antioxidant therapy of bronchial asthma. Klinical Medicine (Mosk), 93(8):19–22.

Rosen, H., Hansen, A.D., Dod, R.L. & Novakov, T. (1980). Soot in urban atmospheres: determination by an optical absorption technique. *Science* (1980) 208(4445):741–4.10.1126/science.208.4445.741

Shi, X., Wang, S., Luan, H., Tuerhong, D., Lin, Y. & Liang, J. (2019). Clinopodium chinense Attenuates Palmitic Acid-Induced Vascular Endothelial Inflammation and Insulin Resistance through TLR4-Mediated NF-κ B and MAPK Pathways. *American Journal of Chinical Medicine.* 47(01):97–117.

UNDER PEER REVIEW

Sandra, F., Oktaviono, Y..H., Widodo, M.A., Dirgantara, Y., Chouw, A., Sargowo, D.(2015). Endothelial progenitor cells proliferated via MEK-dependent p42 MAPK signaling pathway. *Molecular and Cell Biochemistry*. 400(1–2):201–6.

Shieh, Y.H., Huang, H.M., Wang, C.C., Lee, C.C., Fan, C.K. & Lee, Y.L. (2015). Zerumbone enhances the Th1 response and ameliorates ovalbumin-induced Th2 responses and airway inflammation in mice. *International Journal of Immunopharmacology*, 24(2):383–91.

Scheepers, P.T. & Bos, R.P. (1992). Combustion of diesel fuel from a toxicological perspective. I. Origin of incomplete combustion products. *International Archive of Occupational and Environmental Health*, 64(3):149–161.10.

Veerappan, A., O'Connor, N.J., Brazin, J., Reid, A.C., Jung, A. & McGee, D. (2005). Mast cells: a pivotal role in pulmonary fibrosis. DNA Cell Biology 18.10.1089/dna.2013.2005

Veraldi, S., D'Agostino, A., Pontini, P., Guanziroli, E. & Gatt, P. (2016). Nasal polyps: a predisposing factor for cutaneous leishmaniasis of the lips? *International Journal of Dermatology*, 55(4):443–445.

Wang, J., Levendis, Y.A., Richter, H., Howard, J.B., Carlson, J. (Polycyclic aromatic hydrocarbon and particulate emissions from two-stage combustion of polystyrene: the effect of the primary furnace temperature. *Environ Science, 23(12*), 23-27.

Wang, Q., Schwarz, J.P., Cao, J., Gao, R., Fahey, D.W., Hu, T. (2014). Black carbon aerosol characterization in a remote area of Qinghai-Tibetan Plateau, Western China. *Science Total Environment*, 47(9–480):151–8.10.1016/j.scitotenv.2014.01.098

Watson, A.Y. & Valberg, P.A.(2001). Carbon black and soot: two different substances. *American Internationion Health Advances Journal* 62(2):218–28.

Weli, E.V. & Adekunle, I.S. (2015). Climate and Epidemiology of Malaria in Port Harcourt Region, Nigeria. *African Journal of Complementary medicine*, 4: 40–47.

Yang, H., Jin, H., Wang, X., Liu, Z., Yu, M. & Zhao, F. X-ray crystallographic characterization of new soluble endohedral fullerenes utilizing the popular C82 bucky

cage. Isolation and structural characterization of Sm@C3v(7)-C82, Sm@C(s)(6)-C82, and Sm@C2(5)-C82. *Journal of American Chemical Society*, 134(34):14127–14136.

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