WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Flaring Ignition: Unraveling the Health and Environmental Crisis in Nigeria's Niger Delta; The Toxic Effects of Particulate Matter (PM) on Human Health

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A B S T R A C T

The Niger Delta region of Nigeria has endured decades of devastating environmental degradation and health crises due to relentless gas flaring activities. This comprehensive review examines the toxic effects of particulate matter (PM) emissions from gas flaring on human health. Gas flaring releases harmful pollutants, including PM2.5 and PM10, which exacerbate respiratory issues, cardiovascular disease, birth defects, and cancer. Exposure to PM also affects neurological development and cognitive function. This study synthesizes existing literature to underscore the dire consequences of gas flaring on local communities. The analysis reveals alarming rates of respiratory illnesses, infant mortality, and reduced life expectancy among residents. Furthermore, socioeconomic disparities exacerbate vulnerability to these health risks. The review emphasizes the urgent need for policy reforms, stricter regulations, and alternative energy solutions to mitigate gas flaring detrimental impacts. Effective mitigation strategies require collaborative efforts from government agencies, oil corporations, and local stakeholders. This research aims to inform evidence-based interventions, safeguarding the health and well-being of Niger Delta communities and protecting the region's fragile ecosystem.

Keywords: Gas Flaring, Particulate Matter, Niger Delta, Environmental Pollution, Health Impacts, Policy Reform.

INTRODUCTION

The Niger Delta region of Nigeria, richly endowed with vast oil and gas reserves, has been plagued by decades of unmitigated environmental degradation and devastating health crises. At the heart of this ecological disaster lies the persistent practice of gas flaring, a byproduct of petroleum extraction that has ravaged the region's fragile ecosystem and jeopardized the well-being of its inhabitants. (Okoro and Oyebanji, 2019) (Akpoyomare and Odjugo, 2014). Gas flaring, the deliberate burning of natural gas associated with oil production, releases an array of toxic pollutants into the atmosphere, including particulate matter (PM), volatile organic compounds, and greenhouse gases. (Okoro and Oyebanji, 2019). Particulate matter, a complex mixture of fine particles (PM2.5) and coarse particles (PM10), poses particularly alarming health risks. Prolonged exposure to PM has been linked to increased morbidity and mortality rates, exacerbating respiratory and cardiovascular diseases, neurological damage, and even cancer. (Pope, C. A., et al.(2018) (Chen, G., et al. (2020) The World Health Organization (WHO) estimates that 99% of the global population breathes air that exceeds recommended PM2.5 levels, with low- and middle-income countries bearing the brunt of this pollution (WHO, 2021).

Nigeria's Niger Delta region is among the most critically affected areas, with gas flaring activities concentrated in densely populated communities. The region's unique cultural, socioeconomic, and environmental vulnerabilities compound the adverse effects of gas flaring, disproportionately affecting vulnerable populations, including children, pregnant women, and the elderly. Despite international regulations and domestic policies aimed at reducing gas flaring, the practice persists, fueled by inadequate enforcement, lack of transparency, and economic interests.

This review aims to unravel the complex relationships between gas flaring, particulate matter emissions, and human health in the Niger Delta. By synthesizing existing literature and empirical evidence, this study seeks to:

1. Examine the magnitude and characteristics of PM emissions from gas flaring in the Niger Delta.
2. Investigate the toxic effects of PM on human health, with a focus on respiratory, cardiovascular, and neurological impacts.
3. Analyze the socioeconomic and environmental factors exacerbating vulnerability to gas flaring-related health risks.
4. Identify policy and regulatory gaps hindering effective mitigation of gas flaring and associated health crises.
5. Inform evidence-based interventions and advocacy efforts to safeguard the health, well-being, and environmental rights of Niger Delta communities.

By shedding light on the devastating consequences of gas flaring and PM pollution, this review aims to catalyze urgent action towards a sustainable, equitable, and healthy future for the Niger Delta region.

MAGNITUDE 0F PARTICULATE MATTER EMISSIONS

Gas flaring in the Niger Delta region of Nigeria releases significant amounts of particulate matter (PM) into the atmosphere. Studies have estimated that gas flaring in the region emits approximately 12.6 million tons of CO2-equivalent greenhouse gases annually, with PM comprising a substantial fraction of these emissions (Ekebafe, Ogbeide, and Okiemen 2011) (Ubani and Onyejekwe, 2013) A study conducted by Ogbodo, Ogbu and Amadi in 2015 measured PM concentrations in selected communities in the Niger Delta and reported average PM10 and PM2.5 levels of 814.5 μg/m^3 and 345.6 μg/m^3, respectively. These levels exceed the World Health Organization's (WHO) recommended guidelines of 20 μg/m^3 for PM10 and 10 μg/m^3 for PM2.5 (WHO, 2006). Another study by Adeyemi, Ukpebor, and Omonona, B. (2017) estimated that gas flaring in the Niger Delta emits approximately 34,000 tons of PM10 and 12,000 tons of PM2.5 annually.

CHARACTERISTICS OF PARTICULATE MATTER EMISSIONS

The characteristics of PM emissions from gas flaring in the Niger Delta vary depending on factors such as flare type, gas composition, and combustion efficiency (Adeyemi, Ukpebor, and Omonona, 2017). However, studies have identified the following characteristics:

1. **Particle size distribution**

   PM emissions from gas flaring in the Niger Delta are primarily composed of fine particles (PM2.5), with a median diameter of 0.1-0.5 μm

   (Ogbodo, Ogbu, and Amadi, 2015).

2. **Chemical composition**:

PM emissions contain high levels of organic compounds, including polycyclic aromatic hydrocarbons (PAHs), benzene, toluene, ethylbenzene, and xylene (BTEX) (Ite, Ibok and Umoh, 2013).

3. **Metals**

PM emissions also contain heavy metals such as lead, cadmium, chromium, and nickel, which are known to have adverse health effects (Adeyemi, Ukpebor, and Omonona, 2017).

4. **Volatile organic compounds (VOCs)**

Gas flaring in the Niger Delta emits VOCs, including methane, ethane, and propane, which contribute to ozone formation and air pollution (Ekebafe, Ogbeide, and Okiemen 2011)

FACTORS INFLUENCING PPARTICULATE MATTER EMISSIONS

Several factors influence PM emissions from gas flaring in the Niger Delta, including:

1. **Flare Type**

The type of flare used affects PM emissions, with older flares emitting higher levels of PM (Ogbodo, Ogbu, and Amadi, 2015) (Solomon, Giwa, Collins, Nwaokocha, Sidikat, Kuye, Kayode and Adama, 2019).

2. **Gas Composition**

The composition of the gas being flared influences PM emissions, with gases containing higher levels of hydrocarbons emitting more PMs

(Ite Ibok, and Umoh, 2013).

3. **Combustion Efficiency**

Inefficient combustion leads to higher PM emissions (USEPA,2022).

4. **Distance from Flares**: Communities located closer to gas flares experience higher PM levels (Olusegun, Fawole, Xiaoming Cai, Olawale, Abiye, and MacKenzie, 2019).

TOXIC EFFECTS OF PM25 ON HUMAN HEALTH

1. Respiratory Problems

PM2.5 can penetrate deep into the lungs, causing inflammation and damage to lung tissue, leading to conditions like bronchitis, asthma, and chronic obstructive pulmonary disease (COPD). (Xing,, Xu, Shi, and Lian, 2016) (Wang and Liu, 2023). It has also been found that children living near gas flaring sites had increased risk of respiratory problems.(Johnston, Enebish, Eckel, Navarro,and Shamasunder, 2021) (Alimi and Gibson 2022).

2. Cardiovascular Disease

Exposure to PM2.5 has been linked to an increased risk of cardiovascular disease, including heart attacks, strokes, increasing blood pressure and other cardiovascular events. (Aryal, Harmon and Dugas, 2021). These events occur through the following processes:

a. Damaging of Blood Vessels

PM2.5 exposure can cause endothelial dysfunction, leading to reduced blood vessel flexibility and increased blood pressure (Brook, Rajagopalan, Pope, Brook, Bhatnagar, Diez-Roux and Kaufman, 2010) (Liang, Zhang, Ning, Du, Liu, Batibawa , Duan and Sun, 2020). A study published in the Journal of the American College of Cardiology found that PM2.5 exposure increased the risk of cardiovascular disease by damaging blood vessels. (Huang, Wang,Lu, Kipen,, Wang, and Zhang, 2012

b. Increasing Blood Pressure

PM2.5 exposure has been shown to increase systemic blood pressure in both healthy individuals and those with pre-existing hypertension (Urch, Silverman, Corey, De Souza, Mudiyanselage, and Brook, (2017) (Maduka and Tobin-West, 2017) . A study published in the European Heart Journal that investigated the relationship between long-term fine particulate matter (PM2.5) exposure and cardiovascular disease in patients with hypertension found that PM2.5 exposure increased blood pressure and cardiovascular risk in patients with hypertension (Chen, Yin, Meng, Wang, Liu, Zhang and Zhou, 2018).

c. Cardiovascular Events

Exposure to PM2.5 has been linked to an increased risk of heart attacks, strokes, and other cardiovascular events (Cesaroni, Forastiere, Stafoggia, Andersen, Badaloni, Beelen and Weinmayr, 2014).). A study published in the New England Journal of Medicine which investigated the relationship between long-term fine particulate matter (PM2.5) exposure and mortality, using data from the Harvard Six Cities study, found that PM2.5 exposure increased the risk of cardiovascular mortality (Lepeule, Laden, Dockery and Schwartz, 2012). In a diverse cohort of 3.7 million adults, by Stacey, Kamala, Stephen , Joel, Noelle and Stephen (2023), the cohort study found that long-term PM2.5 exposure was associated with an increased risk of incident acute myocardial infarction, ischemic heart disease mortality, and cardiovascular disease mortality, and these associations were more pronounced in low socioeconomic status communities. This same study also found evidence of associations at moderate concentrations of PM2.5 below the current regulatory standard of 12μg/m^3.

3. Lung Cancer

PM2.5 contains various polycyclic aromatic hydrocarbons (PAHs), including benzo(a)pyrene, benzo(b)fluoranthene, and indeno(1,2,3-cd)pyrene (Fang, Zhang, Zeng, Liu, Yang, Wang, Wang and Wang, 2020). These PAHs are known carcinogens and can cause genetic mutations, leading to cancer (Bostrom, Gerde, Hanberg, Jernström, Johansson, Kyrklund and Törnqvist, 2002). Based on these facts, exposure to PM2.5 has been linked to an increased risk of lung cancer. Exposure to PM2.5 has been classified as "carcinogenic to humans" by the International Agency for Research on Cancer (IARC, 2013). A study published in the Journal of the National Cancer Institute found that PM2.5 exposure increased the risk of lung cancer by 12% for every 10 μg/m^3 increase in PM2.5 concentration (Pope, III, Burnett, Thun, Calle, Krewski, Ito and Thurston, 2002).

Another study published in the American Journal of Respiratory and Critical Care Medicine found that long-term PM2.5 exposure increased the risk of lung cancer by 35% (Laden, Schwartz, Speizer and Dockery, 2006)..

Mechanisms

PAHs in PM2.5 can bind to DNA, causing genetic mutations and epigenetic changes that lead to cancer (Knaapen, Borm, Albrecht, and Schins, 2004).

PM2.5 exposure can also lead to inflammation, oxidative stress, and immune system suppression, which can contribute to cancer development (Donaldson, Stone, Seaton, and MacNee, 2001).

Epidemiological Evidence

A meta-analysis of 22 studies found a significant association between PM2.5 exposure and lung cancer risk (Hamra, Guha, Cohen, Laden, Raaschou-Nielsen, Samet, and Loomis 2014). The study examined the association between mean long-term ambient PM2.5 concentrations and lung cancer mortality among 188,699 lifelong never-smokers drawn from the nearly 1.2 million Cancer Prevention Study-II participants enrolled by the American Cancer Society in 1982 and followed prospectively through 2008. Another cohort study of over 300,000 individuals found that PM2.5 exposure increased the risk of lung cancer, even at low concentrations (Turner, Krewski, Pope, III, Chen, Gapstur, and Thun, 2011). These findings strengthen the evidence that ambient concentrations of PM2.5 measured in recent decades are associated with measurable increases in lung cancer mortality.

4. Neurological Problems

Exposure to PM2.5 has been associated with an increased risk of neurological problems. PM2.5 can cause neuro-inflammation, leading to conditions like Parkinson's disease, Alzheimer's disease, and other neurodegenerative disorders (Wang, Xiong, and Tang, 2017).

a. Neuro-Inflammation

PM2.5 exposure can lead to neuro-inflammation, characterized by the activation of microglia and the release of pro-inflammatory cytokines (Block, Elder, Auten, Bilbo, Chen, Chen, and Costa, 2007). Nanoparticle exposure induces inflammation and oxidative stress in the brain. Environmental Health Perspectives, 115(7), 943–948. doi: 10.1289/ehp.9834. Neuro-inflammation can cause oxidative stress, mitochondrial dysfunction, and damage to neuronal cells. Recent studies show that in response to certain environmental toxins and endogenous proteins, microglia can enter an over-activated state and release reactive oxygen species (ROS) that cause neurotoxicity. Pattern recognition receptors expressed on the microglial surface seem to be one of the primary, common pathways by which diverse toxin signals are transduced into ROS production. (Morgan, Todd, Davis, David, Iwata, Nobuhisa, Tanner, Jennifer, Snyder, David, and Finch, Caleb, 2011)

b. Parkinson's Disease

Exposure to PM2.5 has been linked to an increased risk of Parkinson's disease (Liu, Xueyan, Chen, Rong, Wang, Wei, 2016). The mechanism involves the activation of microglia, leading to the release of pro-inflammatory cytokines and the damage of dopaminergic neurons (Caudle, Michael, Jason, Richardson, Ming , Gary, 2012). Epidemiology studies have shown that exposure to PM could bring about neurotoxicity and play a significant role in the etiology of CNS disease, which has been gradually corroborated by in vivo and in vitro studies

c. Alzheimer's Disease

PM2.5 exposure has also been linked to an increased risk of Alzheimer's disease (Chen, Kwong, Copes, Tu, Villeneuve, and van Donkelaar, 2018).

The mechanism involves the activation of microglia, leading to the release of pro-inflammatory cytokines and the accumulation of amyloid-β plaques (Levesque, Duchesne, Garant, Rhainds, Dales, and Blanchard, 2011).

d. Other Neurodegenerative Disorders

PM2.5 exposure has also been linked to other neurodegenerative disorders, including amyotrophic lateral sclerosis (ALS) and Huntington's disease (Kumar, Bjørge, Bråtveit, and Nielsen, 2018) (Li, Chen, Caldwell, Kowal, and Wilson, 2019)

Mechanism of Action

PM2.5 can cross the blood-brain barrier and directly interact with neuronal cells, leading to inflammation and oxidative stress (Tian, Liu, and Sun, 2020).

PM2.5 can also activate microglia, leading to the release of pro-inflammatory cytokines and the damage of neuronal cells (Xu, Wang, Zhao and Sun, 2020).

5. Cognitive Impairment

PM2.5 exposure has been linked to reduced cognitive function, memory, and Intelligence Quotient (IQ) in children and adults. (Fu, Guo, Zhang, Liu, and Chen, 2019). (Chen, Wang, Wellenius, Serre, and Kan, 2018).

a. Children

PM2.5 exposure has been shown to reduce cognitive development in children, including lower IQ scores and reduced memory (Perera, Wang, Li, Chen, Zhng, Qu and Tang, ,2018)

A study published in the Journal of the American Medical Association found that PM2.5 exposure was associated with reduced cognitive development in children aged 4-7 years (Chen, Wang, Wellenius, Serre, and Kan, 2018). Another study published in the journal: Environmental Health Perspectives found that PM2.5 exposure was linked to reduced working memory and attention in children aged 8-11 years (Wang, Li, Chen, Liu, Ren, Zhang and Tang, 2020)

Studies on "Exposure to Fine Particulate Matter and Cognitive Development in Nigerian Children" found a significant association between PM2.5 exposure and reduced cognitive development in Nigerian children aged 4-7 years. (Adegoke, Oyedeji, Olopade, Agboola, and Ogunyemi, 2020) (Oluwole, Adewuyi, Otulana, Olaniyan, Arinola, 2018). Another study on “Fine Particulate Matter Exposure and Working Memory in Nigerian School Children" showed that PM2.5 exposure was linked to reduced working memory in Nigerian school children aged 8-12 years. (Nwosu, Nwagbara, Ubani, and Eme, 2019).

b. Adults

PM2.5 exposure has been linked to reduced cognitive function in adults, including lower IQ scores and reduced memory (Ailshire, Clarke, and Strauss, 2018). A study published in the journal Environmental Research found that PM2.5 exposure was associated with reduced cognitive function in adults aged 50-75 years (Kioumourtzoglou, Power, Hart, Manson, and Weisskopf, 2019)Similar studies that were carried out in different parts of Nigeria equally showed a relationship between exposure to particulate materials and decline in cognitive function (Igwe, Nwagbara, and Ubani, 2020 (Ezemonye et al, 2019 (Akpomudaye, Nwagbara, and Ubani, 2018). (Eze, Nwagbara, and Ubani, 2019).

Mechanisms

PM2.5 exposure can lead to neuro-inflammation, oxidative stress, and damage to brain cells, which can impair cognitive function (Block, Elder, Auten, Bilbo, Chen, Chen and Morgan, 2012)..

PM2.5 exposure can also disrupt the blood-brain barrier, allowing toxins to enter the brain and damage cognitive function (Levesque, Duchesne, Garant, Rhainds, Dales, and Blanchard, 2011).

6. Increased Mortality

Long-term exposure to PM2.5 can reduce life expectancy and increase the risk of premature death. (Adejumo, Nwagbara, and Ubani, 2020) (Eze, Nwagbara, and Ubani, 2019).

Mechanisms of Action

a. **Inflammation:** PM2.5 exposure can lead to chronic inflammation in the body, which can damage organs and tissues, increasing the risk of premature death (Pope, Burnett, Thurston, Thun, Calle, Krewski and Godleski, 2004)..

b. **Oxidative stress:** PM2.5 exposure can cause oxidative stress, which can damage cellular components, including DNA, proteins, and lipids, leading to cell death and increasing the risk of premature death (Donaldson et al, 2001) (Ifenkwe et al 2022).

c. **Cardiovascular disease:** PM2.5 exposure can increase the risk of cardiovascular disease, including heart attacks, strokes, and other cardiovascular events, which can eventually lead to premature death (Brook, Franklin, Cascio, Hong, Howard, Lipsett, and Smith, 2010).

d. **Respiratory Disease:** PM2.5 exposure can exacerbate respiratory diseases, such as chronic obstructive pulmonary disease (COPD) and lung cancer, which can lead to premature death (Cohen, Brauer, Burnett, Anderson, Stroud, Devon and Frostad, 2017).

7. Eye, Nose, and Throat Irritation

PM2.5 can cause irritation and inflammation in the eyes, nose, and throat due to its small size and ability to penetrate deep into these sensitive areas.

a. Eye Irritation

1. PM2.5 can cause eye irritation, including redness, itching, and tearing (Khillare et al, 2018) (Adejumo, Awosika, and Omidiji, 2020).
2. The fine particles can penetrate the eye's mucous membranes, leading to inflammation and discomfort (Liu, SLi, Zou, Wang, and Li, 2020)
3. Studies have shown that PM2.5 exposure is associated with increased risk of dry eye syndrome and conjunctivitis (Zhang, Wang, Chen and Wang, 2020) (Hwang and Lee, 2017).

b. Nose Irritation

1. PM2.5 can cause nasal irritation, including runny nose, congestion, and sneezing (Igwe, Nwagbara and Ugwu, 2019) (Hwang, and Lee, 2017).
2. The particles can damage the nasal mucosa, leading to inflammation and impaired nasal function (Zhang, Wang, Chen and Zhang, 2020).
3. Studies have shown that PM2.5 exposure is associated with increased risk of rhinitis and sinusitis (Li, Wang, and Liu, 2019). (Ji, Wang and Li, 2020).

c. Throat Irritation

1. PM2.5 can cause throat irritation, including soreness, coughing, and difficulty in swallowing (Eze, Nwagbara, and Ugwu, 2018)
2. The particles can penetrate the throat's mucous membranes, leading to inflammation and discomfort (Liu, Li, Zou, Wang, and Li, 2020)..
3. Studies have shown that PM2.5 exposure is associated with increased risk of pharyngitis and laryngitis (Zhang, Wang, Chen and Wang, 2019)

Mechanisms

1. PM2.5 can cause oxidative stress and inflammation in the eyes, nose, and throat, leading to irritation and discomfort (Tao, Gonzalez-Flecha, and Kobzik, 2003). This study investigated the effects of fine particulate matter (PM2.5) exposure on human epithelial lung cells, finding:Changes in gene expression related to oxidative stress and inflammation Increased expression of pro-inflammatory cytokines and chemokinesActivation of oxidative stress pathways
2. The particles can activate immune cells, leading to the release of pro-inflammatory cytokines and chemokines (Chen, 2017).

8. Immune System Suppression

PM2.5 can weaken the immune system, making people more susceptible to infections. Qian, Z. M., & Lin, H. L. 2016), through the following processes:

1. **Reduced Phagocytosis:** PM2.5 impairs macrophage function, reducing phagocytosis with resultant increase in bacterial load (Khillare, Meena, and Singh, 2018).
2. **Impaired T-Cell Function:** PM2.5 reduces T-cell proliferation and cytokine production, compromising adaptive immunity (Liu, Zhang, Liu, Wang, 2020).

Mechanisms of Action

1. **Oxidative Stress:** PM2.5 generates reactive oxygen species (ROS), which damage immune cells and disrupt immune function (Liu, Wang, Zhang, and Cao, 2019).
2. **Inflammation:** PM2.5 induces chronic inflammation, leading to immune cell exhaustion and impaired immune response (Chen, Liu, Zhang, and Wang, 2020).
3. **Cytokine Imbalance:** PM2.5 alters cytokine production, disrupting the balance between pro-inflammatory and anti-inflammatory cytokines (Wang, Liu, Zhang and Cao, 2019).
4. **Immune Cell Dysfunction:** PM2.5 impairs immune cell function, including macrophages, neutrophils, and T-cells (Zhang, Liu, Wang, and Cao, 2020).

9. Genetic Damage

Fine particulate matter (PM2.5) contains genotoxic substances, including: Polycyclic aromatic hydrocarbons (PAHs), heavy metals (e.g., cadmium, chromium, arsenic), volatile organic compounds (VOCs) and nitroarenes. These substances can cause DNA damage through various mechanisms, including: Oxidative stress, DNA adduct formation and epigenetic modifications , including DNA methylation and histone modification, which can affect gene expression (Wang, Liu, Chen, Li, Huang, and Chen, 2019) (Bhatt and Tripathi. 2018). This DNA damage can lead to genetic mutations including point mutations, deletions, and chromosomal aberrations, which may result in:Cancer initiation and progression, Respiratory diseases (e.g., lung cancer, chronic obstructive pulmonary disease), Cardiovascular diseases and Neurological disorders (Møller and Loft, 2018).

10. Birth Abnormalities

Exposure to PM2.5 during pregnancy can increase the risk of low birth weight, premature birth, and birth defects.

a. Low birth weight:

PM2.5 exposure during pregnancy can lead to inflammation and oxidative stress, causing placental dysfunction and reduced fetal growth (Wang, Li, Huang, and Chen, 2019). The authors found that PM2.5 exposure was significantly associated with an increased risk of low birth weight, highlighting the importance of reducing exposure to air pollution during pregnancy to improve fetal health. PM2.5 exposure can also restrict fetal growth, leading to low birth weight (<2500g), which increases the risk of infant mortality, morbidity, and long-term health problems (Hu et al., 2020). Similar studies carried out in Port Harcourt and Lagos, Nigeria found significant association between PM2.5 exposure and low birth weight thus, affirming that PM2.5 exposure increased the risk of low birth weight. (Adegoke, Ojo, and Oyekunle, 2020) (Nwofor, and Ogunkoya, 2022).

b. Premature Birth:

PM2.5 exposure can cause inflammation and oxidative stress, leading to cervical ripening and premature labor (Chen, Li, Zhang and Zhao, 2018).). This is because PM2.5 particles can induce the production of pro-inflammatory cytokines, such as IL-6 and TNF-α, which can stimulate cervical ripening (Huang, Wei, Hu, and Li, 2018. Increase levels of reactive oxygen species (ROS) induced by PM2.5 can damage the cervical tissue and lead to premature labor (Li, Liu, Chen, and Wang, 2020). It can also disrupt the balance of the maternal-fetal immune system, leading to an increased risk of preterm labor (Wang, Liu, Li and Zhang, 2019).

c. Birth Defects

PM2.5 exposure can cause oxidative stress and DNA damage, leading to genetic mutations and birth defects including heart defects and cleft palate (Guxens, Ghassabian, Gong, and Wright, 2018).. A study on animals suggested that maternal air pollution exposure might cause inflammatory damage and oxidative stress to the cerebellum, contributing to reduced cognitive performance in mice offspring. (Jiajia et al, 2023). Prenatal period is critical for brain development. It is a complicated process determined by both genetic and external factors. Deleterious factors during the prenatal period might have severe and long-term adverse effects on brain structure and function, resulting in neuro-developmental disorders (Zhang, Yang, Al-Ahmady, Du, Duan, Liao, Sun, Wei, and Hua , 2022).

SOCIOECONOMIC AND ENVIRONMENTAL FACTORS EXACERBATING VULNERABILITY TO GAS FLARING-RELATED HEALTH RISKS IN THE NIGER DELTA

a. Socioeconomic Factors

1. **Poverty**: Communities in the Niger Delta predominantly have low income, limiting access to healthcare, clean water, and sanitation, thereby increasing vulnerability to gas flaring-related health risks (Adeyemi, Adewuyi and Ojewumi, 2017).

2. **Limited Education**: Low educational levels hinder awareness of gas flaring's health impacts, reducing the ability to take preventive measures (Ikelegbe, 2005).
3. **Unemployment**: Unemployment drives residents to engage in hazardous occupations, increasing exposure to gas flaring pollutants (Ogbodo, Abah, and Ogbu, 2015).
4. **Population Density**: High population density in communities near gas flares increases exposure to pollutants (Ekebafe, Ogbeide, and Ologunorisa, 2011).
5. **Lack of infrastructure**: Inadequate healthcare facilities, roads, and communication networks hinder access to medical care and emergency services (Ite, Ibok, and Umoh, 2013).

b. Environmental Factors

1. **Proximity to Gas Flares**: Communities located near gas flares experience higher levels of pollution thereby increasing health risks (Adeyemi, Adewuyi, and Ojewumi, 2017),

   Climate Change: Rising temperatures and changing precipitation patterns exacerbate air pollution, increasing health risks (IPCC, 2013).

3. **Water Pollution**: Gas flaring contaminates water sources, reducing access to clean water and increasing health risks (Ogbodo, Abah, and Ogbu, 2015)
4. **Soil Degradation**: Gas flaring leads to soil contamination, affecting agricultural productivity and food security (Ekebafe, Ogbeide, and Ologunorisa, 2011).
5. **Biodiversity Loss**: Gas flaring destroys habitats, reducing ecosystem services and increasing vulnerability to environmental hazards (Ite, Ibok, and Umoh, 2013).

POLICY AND REGULATORY GAPS HINDERING EFFECTIVE MITIGATION OF GAS FLARING AND ASSOCIATED HEALTH CRISES IN NIGERIA

1. **Inadequate Regulations**: Existing laws, such as the Petroleum Act of 1969 and the Associated Gas Re-injection Act of 1979 have failed to prevent gas flaring due to lack of enforcement and repeated postponement of deadlines (Ite, Ibok, and Umoh, 2013).
2. **Insufficient Data**: Limited research on gas flaring's health impacts and inadequate data on flare emissions hinder effective policy-making.
3. **Ineffective Enforcement**: Weak enforcement of existing regulations and lack of penalties for non-compliance contribute to ongoing gas flaring.
4. **Lack of Transparency**: Inadequate disclosure of gas flaring data and operations obscures the issue's severity and hampers accountability hindering effective policy-making (Ekebafe, Ogbeide, and Ologunorisa, 2011).
5. **Inadequate Compensation**: Communities affected by gas flaring receive insufficient compensation for health and environmental damages.
6. **Inadequate Measurement and Reporting**: Lack of standardized measurement and reporting procedures hinders accurate tracking of gas flaring and emissions.

Interventions and Advocacy to Safeguard the Health, Well-Being and Environmental Rights of Niger Delta Communities.

To safeguard the health, well-being, and environmental rights of Niger Delta communities, several evidence-based interventions and advocacy efforts can be implemented.

1. Advocacy Efforts

- **Climate Change Mitigation and Adaptation**: Civil society organizations can advocate for government intervention in the plight of coastal communities affected by climate change, leveraging environmental occurrences to increase awareness and demand accountability on the implementation of mitigation and adaptation measures.
- **Environmental Laws and Sustainable Development**: Advocating for the enactment and strict enforcement of environmental laws can ensure sustainable development in the Niger Delta region.
- **Health Impacts of Climate Change**: Raising awareness about the health impacts of climate change, such as malnutrition, water and vector-borne diseases, can inform policymakers and the public.

2. Interventions

- **Research and Data Collection**: Conducting research studies to understand the health impacts of climate change and environmental degradation can inform policy decisions.

- **Multi-Sectoral Collaboration**: Fostering collaboration between relevant ministries, departments, and agencies across health, environment, agricultural, and economic sectors can ensure synergy and effectiveness.
- **Public Engagement and Education**: Educating the public on the link between environmental factors and health can drive action and behavior change.

Key Recommendations

- End flaring: Implement alternatives like gas reuse or capture.
- Increasing funding for research on climate change and health impacts.
- Enhancing public awareness and education on environmental health issues.
- Health surveillance: Establish health monitoring programs for nearby communities.
- Supporting community-led initiatives for sustainable development and environmental conservation.

By implementing these interventions and advocacy efforts, Niger Delta communities can better protect their health, well-being, and environmental rights.

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