OPEN ACCESS

Scholars International Journal of Anatomy and Physiology

Abbreviated Key Title: Sch Int J Anat Physiol
ISSN 2616-8618 (Print) |ISSN 2617-345X (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com

Review Article

The Impact of Air Borne Toxins from Gas Flaring on Cardiopulmonary and Other Systemic Functions

Wami-Amadi, Chisom Faith (MBBS, UPH, MSc, RSU)[1*]

[1]Department of Human Physiology, Faculty of Basic Medical Sciences, Rivers State University, Nigeria

DOI: https://doi.org/10.36348/sijap.2025.v08i01.003 | **Received:** 19.01.2025 | **Accepted:** 24.02.2025 | **Published:** 26.02.2025

***Corresponding author:** Wami-Amadi, Chisom Faith
Department of Human Physiology, Faculty of Basic Medical Sciences, Rivers State University, Nigeria

Abstract

Gas flaring and illegal refining in Nigeria's Niger Delta region contribute significantly to environmental pollution, public health crises, and economic losses. These activities release particulate matter ($PM_{2.5}$, PM_{10}), volatile organic compounds (VOCs), nitrogen oxides (NO), sulfur dioxide (SO_2), carbon monoxide (CO), heavy metals, and dioxins, which have been linked to cardiovascular diseases, respiratory disorders, neurological impairments, and adverse reproductive outcomes. Studies conducted in Rivers, Bayelsa, and Delta States reveal that pollutant levels exceed WHO-recommended safety limits by over 400%, correlating with increased risks of hypertension, myocardial infarction, chronic obstructive pulmonary disease (COPD), neurotoxicity, and adverse pregnancy outcomes (Ehumadu, Uyigue, & Ndekwu, 2021). The economic burden of gas flaring is estimated at $7.4 billion annually due to healthcare costs, lost productivity, and environmental damage (World Bank, 2022). This paper integrates air quality data, pathophysiological mechanisms, and epidemiological evidence to assess health risks.

Keywords: Gas Flaring, Cardiovascular Disease, Air Pollution, Endothelial Dysfunction, Oxidative Stress, Respiratory Health, Economic Burden.

1. INTRODUCTION

Gas flaring is the controlled burning of natural gas released during oil extraction. It is commonly used by oil companies when infrastructure to capture and utilize the gas is unavailable or economically unviable. Despite its harmful environmental and health consequences, gas flaring remains a widespread practice in Nigeria, particularly in the Niger Delta region, where oil exploration is concentrated. Nigeria ranks second globally in gas flaring emissions, contributing 10% of the world's total gas flaring-related carbon emissions (World Bank, 2022).

The history of gas flaring in Nigeria dates back to the 1950s when commercial oil production began. Despite legislative efforts such as the Associated Gas Re-Injection Act of 1979 and the Flare Gas (Prevention of Waste and Pollution) Regulations of 2018, enforcement has been weak and inconsistent. The Nigerian government has set multiple deadlines to end routine gas flaring, yet over 7 billion cubic meters of gas are still flared annually (Ehumadu *et al*., 2021).

The environmental and health consequences of gas flaring are severe and multifaceted. The pollutants released into the atmosphere contribute to climate change, acid rain, and loss of biodiversity. Additionally, communities living near gas flaring sites are disproportionately affected, experiencing higher rates of respiratory diseases, cardiovascular conditions, and neurological impairments.

This study aims to provide a comprehensive analysis of the health impacts, air quality assessments, epidemiological data, and mitigation strategies related to gas flaring in Nigeria's oil-producing regions. Special attention will be given to the cardiovascular effects, as prolonged exposure to air pollutants has been linked to hypertension, endothelial dysfunction, and an increased risk of myocardial infarction and stroke.

2. The Burden of Gas Flaring

Gas flaring presents a significant burden on human health, the environment, and the economy. The effects are most pronounced in oil-producing regions, where flare stacks burn continuously, releasing a complex mixture of toxic gases and particulate matter.

Figure 1: Gas Flaring in Nigeria (The Guardian, 2023)

Gas flaring is a major contributor to environmental degradation in the Niger Delta. The combustion of natural gas releases large quantities of Carbon(iv)Oxide (CO_2) and Methane (CH_4), both of which are potent greenhouse gases that exacerbate climate change. Nigeria's gas flaring activities account for a significant portion of global emissions, making it a key player in the global climate crisis. Beyond climate change, gas flaring also contributes to local environmental problems. For instance, the release of Sulphur Dioxide (SO_2) and Nitrous Oxides (NO) leads to the formation of acid rain, which damages crops, degrades soil quality, and harms aquatic ecosystems. Studies have shown that acid rain from gas flaring has reduced agricultural productivity in the Niger Delta, threatening food security for local communities.

In addition to air pollution, gas flaring contaminates soil and water systems. The deposition of heavy metals such as lead, cadmium, and nickel, as well as polycyclic aromatic hydrocarbons (PAHs), has been documented in soil and water samples near flaring sites. These pollutants accumulate in the food chain, posing risks to both human health and biodiversity. For example, research by Osuji and Onojake (2006) found elevated levels of heavy metals in soil and water near flaring sites, highlighting the widespread contamination caused by this practice.

2.1. Health Impacts of Gas Flaring

The health effects of gas flaring exposure have been well-documented in epidemiological studies. The primary pollutants of concern include $PM_{2.5}$, NO_2, CO, SO_2, and VOCs, which have been linked to a range of health conditions.

- **Cardiopulmonary Diseases:**

Studies have shown that long-term exposure to $PM_{2.5}$ and NO_2 increases blood pressure and promotes atherosclerosis, leading to a higher incidence of hypertension, heart attacks, and strokes. Populations living within 5 kilometers of gas flaring sites experience 35% higher rates of hypertension compared to those in non-flaring areas (Ehumadu *et al*., 2021).

The inhalation of SO_2 and fine particulate matter causes airway inflammation, bronchial constriction, and reduced lung function. Research indicates that asthma prevalence is 2.5 times higher in flaring-exposed communities, with increased cases of chronic obstructive pulmonary disease (COPD) and bronchitis (Orugun *et al*., 2023). Exposure to pollutants from gas flaring is a major risk factor for respiratory diseases. Another study by Ana *et al.*, (2009) found that residents of Rivers State, Nigeria, who were exposed to gas flaring had significantly higher rates of respiratory symptoms such as coughing, wheezing, and shortness of breath.

Similarly, long-term exposure to flaring emissions has been associated with cardiovascular diseases, including hypertension and ischemic heart disease. Research by Ukpebor *et al*., (2011) revealed that air quality in flaring communities was significantly worse than in non-flaring areas, with higher concentrations of PM2.5, SO_2, and NOx.

- **Neurological Effects:**

Heavy metal exposure, particularly lead (Pb) and mercury (Hg), has been linked to cognitive decline, neurodevelopmental disorders, and an increased risk of neurodegenerative diseases such as Parkinson's and Alzheimer's (Iwegbue, Emoyan, & Nwajei, 2022). The neurological impacts of gas flaring are particularly concerning, especially for children. Research by Nduka and Orisakwe (2010) found elevated levels of heavy metals in the blood of children living near flaring sites, raising concerns about the long-term effects on their cognitive and behavioral development.

- **Maternal and Infant Health:**

Pregnant women exposed to high levels of SO_2 and $PM_{2.5}$ have higher rates of miscarriage, preterm birth, and stillbirth. Infants born in flaring-exposed communities have a higher risk of low birth weight and developmental disorders (World Bank, 2022). A study by Ezejiofor *et al*., (2014) found that maternal exposure to flaring pollutants was associated with reduced fetal growth and developmental delays. Additionally, exposure to heavy metals and VOCs from gas flaring has been linked to reduced fertility and hormonal imbalances, further highlighting the reproductive health risks posed by this practice.

- **Cancer:**

The release of carcinogenic pollutants such as benzene, formaldehyde, and PAHs from gas flaring increases the risk of cancer in exposed populations. Studies have documented elevated levels of these carcinogens in the blood and urine of individuals living near flaring sites. For example, Orisakwe *et al*., (2012) found that residents of Bayelsa State had higher levels of PAH metabolites in their urine, indicating increased cancer risk. Similarly, Kponee *et al*., (2015) reported elevated levels of urinary benzene in individuals living near flaring sites, further underscoring the carcinogenic risks associated with gas flaring.

2.2. Environmental and Economic Costs

In addition to human health effects, gas flaring contributes to significant environmental and economic losses.

- **Soil and Water Contamination:** The release of acidic gases and heavy metals into the atmosphere leads to acid rain, which degrades soil quality and reduces agricultural productivity. Farmers in Bayelsa and Delta States report 40% lower crop yields due to soil acidification and chemical deposition from flare emissions (Ehumadu *et al*., 2021).
- **Economic Losses:** Nigeria loses over $3 billion annually in wasted flared gas that could otherwise be captured for energy production. The healthcare costs associated with treating pollution-related diseases amount to $7.4 billion per year (World Bank, 2022).

3. Geospatial Analysis of Gas Flaring in Nigeria

The distribution of gas flaring in Nigeria highlights its concentration in the Niger Delta region, particularly in Rivers, Bayelsa, and Delta States. These regions are home to major oil and gas extraction activities, making them the most affected by flaring pollution.

3.1. Case Study: Rivers State – The Black Soot Crisis

Since 2016, Port Harcourt and its surroundings have been engulfed in black soot, a phenomenon linked to gas flaring and illegal refining activities. A study by Ehumadu *et al*., (2021) found that:

- $PM_{2.5}$ levels exceeded WHO safety thresholds by over 400%.
- Residents reported increased cases of asthma, bronchitis, and lung fibrosis.
- Heavy metal contamination was detected in soil and water sources.

3.2. Case Study: Bayelsa State – High Flaring Emissions and Cardiovascular Risks

Bayelsa has one of the highest gas-flaring rates in Nigeria, with 78% of communities reporting pollution-related illnesses. Studies confirm that:

- Hypertension prevalence is 35% higher in flaring-exposed populations (Orugun *et al.*, 2023).
- SO_2 and $PM_{2.5}$ levels exceed WHO limits by 500%.

3.3. Case Study: Delta State

Delta State is a major oil and gas hub with severe pollution from Warri, Ughelli, and Sapele. Findings from Iwegbue *et al*., (2022) revealed that:

- Children in Delta State exposed to flaring had significantly lower lung function scores.
- Neurotoxic effects of lead (Pb) and mercury (Hg) increased cognitive impairments.

4. Chemical Composition of Flared Gas

Gas flaring is the combustion of natural gas associated with crude oil extraction, and the resulting emissions contain a complex mixture of harmful pollutants. The chemical composition of flared gas depends on factors such as oilfield type, combustion efficiency, and flare conditions. In Nigeria, studies indicate that flared gas consists primarily of hydrocarbons, carbon monoxide, sulfur compounds, nitrogen oxides, particulate matter, and heavy metals (Orugun, Adebayo, & Onwuneme, 2023).

4.1 Composition of Flared Gas:

Flared gas consists of a mixture of gaseous and particulate pollutants, including:

4.1.1 Hydrocarbons (Methane, Ethane, Propane, and Volatile Organic Compounds - VOCs)

Hydrocarbons such as methane (CH_4), ethane (C_2H_6), and propane (C_3H_8) are major constituents of flared gas. Methane is a potent greenhouse gas, with a global warming potential (GWP) 25 times greater than carbon dioxide (CO_2) over a 100-year period (World Bank, 2023). VOCs include toxic compounds such as benzene, toluene, ethylbenzene, and xylene (BTEX), which have been linked to neurological damage, liver toxicity, and cancer in exposed populations (Iwegbue, Emoyan, & Nwajei, 2022).

4.1.2 Carbon Dioxide (CO_2) and Carbon Monoxide (CO)

Flared gas contains significant amounts of CO_2, a major contributor to global warming. Additionally, incomplete combustion leads to the formation of carbon monoxide (CO), a colorless and odorless gas that reduces the blood's oxygen-carrying capacity, leading to hypoxia, cardiovascular stress, and neurological impairment (Ehumadu, Uyigue, & Ndekwu, 2021).

4.1.3 Sulfur Compounds (Sulfur Dioxide - SO_2, Hydrogen Sulfide - H_2S)

Flared gas contains high concentrations of sulfur dioxide (SO_2) and hydrogen sulfide (H_2S). SO_2 is a primary contributor to acid rain, which damages crops, soil, and water bodies (Nature, 2023). Prolonged exposure to H_2S, even at low concentrations, can cause respiratory distress, irritation of the eyes and throat, and, in high doses, central nervous system failure (World Bank, 2023).

4.1.4 Nitrogen Oxides (NO_x - NO and NO_2)

Nitric oxide (NO) and nitrogen dioxide (NO_2) are produced during the high-temperature combustion of natural gas. NO_2 is a major contributor to ground-level ozone (O_3), smog, and respiratory diseases (Orugun *et al*., 2023). Long-term exposure to NO_2 increases the risk of asthma, bronchitis, and lung infections.

4.1.5 Particulate Matter ($PM_{2.5}$ and PM_{10})

Particulate matter (PM) consists of fine solid and liquid particles, including soot and heavy metals, that remain suspended in the air. $PM_{2.5}$ particles are particularly harmful because they penetrate deep into the lungs and bloodstream, leading to chronic obstructive pulmonary disease (COPD), cardiovascular diseases, and lung cancer (World Bank, 2023).

4.1.6 Heavy Metals (Lead, Mercury, Arsenic, Cadmium)

Trace metals such as lead (Pb), mercury (Hg), arsenic (As), and cadmium (Cd) are often present in gas flaring emissions. These heavy metals accumulate in soil, water, and biological systems, causing neurological damage, kidney failure, and developmental disorders (Iwegbue *et al*., 2022).

4.2 Comparison of Gas Flaring Emissions in Nigeria vs. Other Oil-Producing Countries

This table illustrates that Nigeria is one of the top gas-flaring nations, with flaring emissions significantly exceeding those of Canada and Norway, where strict regulations and advanced gas utilization technologies have reduced emissions.

4.3 Air Quality Assessments in Niger Delta

Air quality monitoring studies have consistently shown that gas flaring releases high levels of $PM_{2.5}$, NO_2, SO_2, CO, and VOCs, exceeding recommended health thresholds. A recent World Bank (2023) study that analyzed satellite data found a strong correlation between gas flaring emissions and respiratory diseases, particularly in children under five years old. The study indicated that exposure to pollutants from flaring significantly increases the risk of pneumonia, bronchitis, and fever among children in oil-producing areas (World Bank, 2023).

5. Pathophysiology of Gas Flaring-Related Cardiovascular Diseases

Gas flaring releases a complex mixture of pollutants, including particulate matter ($PM_{2.5}$, PM_{10}), carbon monoxide (CO), nitrogen oxides (NO_x), sulfur dioxide (SO_2), volatile organic compounds (VOCs), and heavy metals. These pollutants trigger a cascade of pathophysiological mechanisms, leading to cardiovascular diseases (CVDs) such as tension, atherosclerosis, ischemic heart disease, myocardial infarction, and stroke (Ehumadu, Uyigue, & Ndekwu, 2021).

The three major pathophysiological mechanisms linking gas flaring exposure to cardiovascular diseases are:

- Endothelial Dysfunction and Atherosclerosis
- Hypertension and Myocardial Infarction
- Oxidative Stress and Systemic Inflammation

5.1 Endothelial Dysfunction and Atherosclerosis

The endothelium is a thin layer of cells that lines blood vessels and regulates vascular tone, clotting, and immune function. Chronic exposure to air pollutants damages the endothelium, leading to endothelial dysfunction, arterial stiffness, and atherosclerosis. A study conducted in Bayelsa State, Nigeria, found that individuals living within 5 kilometers of gas flaring sites had 35% higher rates of atherosclerosis and endothelial dysfunction compared to those living in non-flaring areas (Ehumadu *et al*., 2021).

Mechanisms of Endothelial Dysfunction in Gas Flaring Exposure

1. **Reduced Nitric Oxide (NO) Availability:**
   - Nitric oxide (NO) is a vasodilator that maintains blood vessel elasticity.
   - Pollutants such as NO_2, CO, and $PM_{2.5}$ reduce NO bioavailability, causing vasoconstriction and increased blood pressure (Orugun, Adebayo, & Onwuneme, 2023).

2. **Inflammation and Vascular Injury:**
   - Gas flaring emissions increase levels of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP).
   - These inflammatory markers promote endothelial cell apoptosis, vascular remodeling, and arterial plaque formation, increasing the risk of atherosclerosis and heart disease (World Bank, 2023).

3. **Increased Blood Coagulation and Thrombosis:**
   - Gas flaring pollutants increase platelet activation and blood clot formation, raising the risk of deep vein thrombosis (DVT), stroke, and myocardial infarction (Iwegbue, Emoyan, & Nwajei, 2022).

5.2 Hypertension and Myocardial Infarction

Chronic exposure to $PM_{2.5}$, CO, and NO_2 from gas flaring has been strongly linked to increased blood pressure, left ventricular hypertrophy, and a higher incidence of myocardial infarction (heart attacks). A study in Port Harcourt, Rivers State, found that individuals exposed to high levels of NO_2 and $PM_{2.5}$ had a 2.3-fold increase in hypertension rates compared to unexposed individuals (Iwegbue *et al*., 2022). The World Bank (2023) study reported that populations living near gas flaring sites had a significantly higher prevalence of myocardial infarction, with increased hospital admissions for cardiovascular-related emergencies.

Mechanisms of Hypertension in Gas Flaring Exposure

1. **Autonomic Nervous System Dysregulation:**
   - $PM_{2.5}$ and NO_2 activate the sympathetic nervous system (SNS), leading to vasoconstriction and elevated blood pressure (Orugun *et al*., 2023).
   - Increased catecholamine release (epinephrine and norepinephrine) further contributes to hypertension and arrhythmias.

2. **Impaired Oxygen Delivery (Hypoxia):**
   - Carbon monoxide (CO) binds to hemoglobin with 200 times the affinity of oxygen, reducing oxygen transport and delivery to cardiac tissues.
   - Chronic hypoxia results in myocardial ischemia, increasing the risk of heart failure and sudden cardiac arrest (World Bank, 2023).

3. **Increased Blood Vessel Stiffness (Arterial Remodeling):**
   - Continuous exposure to pollutants thickens arterial walls and increases vascular resistance, further exacerbating hypertension and heart disease.

5.3 Oxidative Stress and Systemic Inflammation

One of the primary mechanisms by which gas flaring exposure leads to cardiovascular diseases is through oxidative stress. A study conducted in Delta State found that individuals exposed to high levels of $PM_{2.5}$ had elevated oxidative stress markers, including malondialdehyde (MDA) and reduced glutathione (GSH), which correlate with increased cardiovascular risk (Iwegbue *et al.*, 2022). WHO (2022) air quality studies confirm that long-term exposure to airborne particulate matter significantly raises inflammation levels and oxidative damage in cardiovascular tissues.

Mechanisms of Oxidative Stress in Gas Flaring Exposure

1. **Production of Reactive Oxygen Species (ROS):**
   - Pollutants such as NO_2, SO_2, and $PM_{2.5}$ generate reactive oxygen species (ROS), including superoxide (O_2^-) and hydroxyl radicals (OH^-).
   - ROS cause lipid peroxidation, protein oxidation, and DNA damage, leading to cellular apoptosis and cardiovascular dysfunction (Orugun *et al*., 2023).

2. **Inflammatory Cytokine Activation:**
   - $PM_{2.5}$ exposure triggers the release of pro-inflammatory cytokines, leading to chronic inflammation and vascular injury.
   - Increased CRP, TNF-α, and IL-6 levels have been directly linked to atherosclerosis, insulin resistance, and metabolic syndrome (World Bank, 2023).

3. **Endothelial Cell Dysfunction:**
   - ROS reduce nitric oxide (NO) bioavailability, leading to vasoconstriction, increased vascular resistance, and hypertension.

6. Pathophysiology of Respiratory Diseases Due to Gas Flaring

Gas flaring in Nigeria releases toxic pollutants, including particulate matter ($PM_{2.5}$, PM_{10}), nitrogen oxides (NO_x), sulfur dioxide (SO_2), carbon monoxide (CO), volatile organic compounds (VOCs), and heavy metals. These pollutants penetrate deep into the respiratory tract, causing airway inflammation, oxidative stress, and impaired lung function. Chronic exposure increases the risk of asthma, bronchitis, chronic obstructive pulmonary disease (COPD), lung

fibrosis, and lung cancer (Ehumadu, Uyigue, & Ndekwu, 2021).

The mechanisms by which gas flaring emissions contribute to respiratory diseases, including:

- Airway inflammation and oxidative damage
- Lung function impairment and obstructive pulmonary diseases

1. Fibrosis and long-term lung damage

2. Airway Inflammation and Oxidative Stress

The primary mechanism by which gas flaring pollutants cause respiratory diseases is airway inflammation triggered by oxidative stress. Inhaled pollutants such as $PM_{2.5}$, SO_2, and NO_2 enter the lungs and cause irritation, mucus hypersecretion, and inflammation.

a) Role of Particulate Matter ($PM_{2.5}$ and PM_{10}) in Airway Inflammation: $PM_{2.5}$ particles are small enough to penetrate deep into the alveoli (air sacs) of the lungs, causing alveolar inflammation and oxidative stress (Orugun, Adebayo, & Onwuneme, 2023). PM induces macrophage activation, leading to the release of pro-inflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP), which worsen lung inflammation. Chronic exposure to $PM_{2.5}$ leads to increased airway resistance, lung stiffness, and respiratory infections.

b) Effects of Sulfur Dioxide (SO_2) and Nitrogen Oxides (NO_x) on Lung Function: SO_2 and NO_2 dissolve in the respiratory tract, forming sulfuric and nitric acid, which cause airway irritation, bronchoconstriction, and mucus hypersecretion (World Bank, 2023). NO_2 triggers bronchial inflammation, leading to airway hyperreactivity, which is a hallmark of asthma and COPD. A study in Rivers State, Nigeria, found that children living near gas flaring sites had increased airway hyperresponsiveness, wheezing, and chronic bronchitis symptoms (Iwegbue, Emoyan, & Nwajei, 2022).

3. Lung Function Impairment and Obstructive Pulmonary Diseases

A population study by Pope III *et al*., (1993) demonstrated that for every 100 µg/m³ increase in PM_{10}, FEV_1 decreased by 2%, a pattern that is now being observed in gas flaring regions. Recent studies confirm that gas flaring exposure leads to measurable reductions in FVC and FEV_1, consistent with obstructive lung disease (Chattopadhyay *et al.*, 2007).

a) Asthma and Chronic Bronchitis: $PM_{2.5}$ and SO_2 exposure leads to recurrent airway inflammation, increasing the risk of asthma attacks, chronic cough, and dyspnea (shortness of breath) (Orugun *et al.*, 2023). Epidemiological data indicate that asthma prevalence in gas flaring communities is 2.5 times higher than in non-exposed populations (World Bank, 2023).

b) Chronic Obstructive Pulmonary Disease (COPD): Long-term exposure to $PM_{2.5}$, NO_2, and SO_2 damages the lung parenchyma, leading to irreversible airflow obstruction and COPD. Studies have shown that individuals in gas flaring regions exhibit reduced forced expiratory volume in one second (FEV_1), a key marker of COPD (Ehumadu *et al.*, 2021). COPD progression is accelerated by chronic inflammation, mucus hypersecretion, and fibrosis, leading to permanent lung damage and respiratory failure.

3. Pulmonary Fibrosis and Long-Term Lung Damage

a) Mechanisms of Fibrosis Due to Gas Flaring Pollutants

Pulmonary fibrosis is characterized by excessive scarring of lung tissue, leading to stiff lungs and reduced oxygen exchange. This process is triggered by:

- Oxidative stress from $PM_{2.5}$ and NO_2 exposure, which causes lung fibroblast activation. Upregulation of transforming growth factor-beta (TGF-β), a key cytokine in fibrosis development (Iwegbue *et al.*, 2022). Deposition of extracellular matrix proteins, leading to thickened alveolar walls and reduced lung compliance.

b) Gas Flaring Exposure and Lung Cancer Risk

- Polycyclic aromatic hydrocarbons (PAHs) and benzene from flared gas are classified as Group 1 carcinogens by the International Agency for Research on Cancer (IARC). Long-term exposure increases lung cancer risk by 3.5 times in communities near flaring sites (World Bank, 2023). DNA damage and genetic mutations caused by PM and VOC exposure contribute to lung adenocarcinoma development.

7: Pathophysiology of Neurological Effects of Gas Flaring

Gas flaring releases neurotoxic pollutants, including heavy metals (lead, mercury, arsenic), volatile organic compounds (VOCs), carbon monoxide (CO), nitrogen oxides (NO_x), and fine particulate matter ($PM_{2.5}$, PM_{10}). These pollutants cross the blood-brain barrier (BBB), trigger oxidative stress, cause neuroinflammation, and disrupt neurotransmitter function, leading to cognitive impairment,

neurodegenerative diseases, and developmental disorders (Ehumadu, Uyigue, & Ndekwu, 2021).

The pathophysiological mechanisms linking gas flaring exposure to neurological diseases include:

- Neurotoxicity and Oxidative Stress
- Blood-Brain Barrier (BBB) Disruption and Neuroinflammation
- Neurotransmitter Imbalance and Cognitive Decline
- Developmental and Behavioral Disorders in Children

1. Neurotoxicity and Oxidative Stress

a) Heavy Metals and Neuronal Damage

Gas flaring releases heavy metals such as lead (Pb), mercury (Hg), arsenic (As), and cadmium (Cd), which accumulate in brain tissues and disrupt neuronal function.

- **Lead (Pb):** Lead crosses the BBB, accumulating in brain tissue and causing mitochondrial dysfunction, oxidative damage, and neuronal apoptosis (Orugun, Adebayo, & Onwuneme, 2023). Lead exposure in children has been linked to lower IQ scores, memory deficits, and attention disorders.
- **Mercury (Hg):** Methylmercury (MeHg) interferes with neurotransmitter release, causing excitotoxicity and neuronal degeneration. Chronic exposure leads to tremors, cognitive dysfunction, and neuromuscular impairment (Iwegbue, Emoyan, & Nwajei, 2022).
- **Arsenic (As):** Induces oxidative stress and inflammatory cascades, leading to neurodegeneration and increased risk of Parkinson's disease.

2. Particulate Matter ($PM_{2.5}$, PM_{10}) and Oxidative Stress

$PM_{2.5}$ particles are small enough to enter the bloodstream and penetrate brain tissue, triggering inflammatory responses and mitochondrial dysfunction. Exposure to $PM_{2.5}$ increases reactive oxygen species (ROS) production, leading to lipid peroxidation and neuronal apoptosis (World Bank, 2023). Studies have linked $PM_{2.5}$ exposure to cognitive decline, increased risk of Alzheimer's disease, and stroke.

3. Blood-Brain Barrier (BBB) Disruption and Neuroinflammation

The blood-brain barrier (BBB) is a protective structure that prevents harmful substances from entering the central nervous system (CNS). Gas flaring pollutants damage the BBB, leading to neuroinflammation, glial cell activation, and increased permeability to toxins.

Mechanisms of BBB Disruption

a) Inflammatory Mediators: Gas flaring pollutants stimulate the release of pro-inflammatory cytokines (TNF-α, IL-6, IL-1β), increasing BBB permeability. Glial cell activation (astrocytes and microglia) causes chronic neuroinflammation, damaging neurons (Ehumadu *et al*., 2021).

b) Oxidative Stress and Endothelial Damage: NO_2 and $PM_{2.5}$ trigger oxidative damage in endothelial cells, disrupting tight junction proteins in the BBB. Increased BBB permeability allows neurotoxins, heavy metals, and inflammatory molecules to enter the brain.

4. Neurotransmitter Imbalance and Cognitive Decline

Gas flaring pollutants alter dopamine, serotonin, and glutamate levels, leading to mood disorders, memory impairment, and neurocognitive deficits.

a) Effects on Dopaminergic and Serotonergic Systems: Lead (Pb) exposure reduces dopamine levels, increasing the risk of Parkinson's disease and depression. Carbon monoxide (CO) binds to hemoglobin, reducing oxygen transport and leading to neuronal hypoxia (World Bank, 2023). Serotonin depletion due to oxidative stress contributes to anxiety, depression, and sleep disturbances.

b) Cognitive Impairment and Alzheimer's Risk: Studies show that individuals exposed to high levels of $PM_{2.5}$ exhibit faster cognitive decline. Amyloid beta (Aβ) plaque accumulation is increased in individuals exposed to gas flaring pollutants, a key marker of Alzheimer's disease (Orugun *et al.*, 2023).

Developmental and Behavioral Disorders in Children

Children are more vulnerable to neurotoxic effects of gas flaring emissions, as their developing brains are highly sensitive to toxins. Prenatal exposure to gas flaring pollutants increases the risk of autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD). Maternal exposure to NO_2 and $PM_{2.5}$ has been linked to lower birth weight, impaired motor function, and cognitive deficits in newborns (Iwegbue *et al*., 2022).

A study in Port Harcourt, Nigeria, found that children living near gas flaring sites had lower cognitive performance and higher rates of hyperactivity (Ehumadu *et al*., 2021). Long-term exposure to VOCs and CO reduces IQ levels and memory function in children.

1. Pathophysiology of Endocrine and Reproductive Health Risks Due to Gas Flaring

Gas flaring releases endocrine-disrupting chemicals (EDCs), volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), heavy metals (lead, mercury, arsenic), nitrogen oxides (NO_x), sulfur dioxide (SO_2), and fine particulate matter($PM_{2.5}$, PM_{10}). These pollutants interfere with hormonal balance, reproductive function, fetal development, and metabolic regulation (Ehumadu, Uyigue, & Ndekwu,2021). The

pathophysiological mechanisms linking gas flaring exposure to endocrine and reproductive disorders include:

- Endocrine Disruption and Hormonal Imbalances
- Reproductive Health Risks in Women
- Male Infertility and Sperm Damage

1. Endocrine Disruption and Hormonal Imbalances

Endocrine-disrupting chemicals (EDCs) interfere with hormone synthesis, secretion, transport, and receptor binding, leading to metabolic and reproductive dysfunction.

Mechanisms of Endocrine Disruption by Gas Flaring Pollutants

a. Mimicking or Blocking Natural Hormones: VOCs (e.g., benzene, toluene, xylene) and PAHs bind to estrogen and androgen receptors, disrupting hormonal signaling. Heavy metals (lead, mercury) interfere with thyroid hormone metabolism, causing hypothyroidism (Orugun, Adebayo, & Onwuneme, 2023).

b. Alteration of Hormone Synthesis and Secretion: NO_2 and $PM_{2.5}$ cause oxidative stress in the hypothalamus and pituitary gland, reducing gonadotropin-releasing hormone (GnRH) secretion. Chronic exposure to SO_2 is linked to insulin resistance and increased diabetes risk.

Metabolic Disorders Associated with Endocrine Disruption

Gas flaring pollutants alter thyroid-stimulating hormone (TSH) levels, leading to hypothyroidism, goiter, and developmental delays in children (World Bank, 2023). Chronic exposure to $PM_{2.5}$ increases systemic inflammation and disrupts pancreatic beta-cell function, contributing to type 2 diabetes (Iwegbue, Emoyan, & Nwajei, 2022).

2. Reproductive Health Risks in Women

Female reproductive health is highly sensitive to environmental toxins, and exposure to gas flaring emissions is associated with menstrual irregularities, infertility, and pregnancy complications.

c) Menstrual and Ovarian Dysfunction: PAHs and benzene from gas flaring interfere with estrogen and progesterone balance, causing irregular menstrual cycles. Lead (Pb) and mercury (Hg) reduce ovarian reserve and impair egg maturation (Ehumadu *et al*., 2021). Chronic exposure to VOCs has been linked to increased risk of polycystic ovary syndrome (PCOS). Gas flaring pollutants induce oxidative stress in the ovaries, leading to decreased fertility rates. Studies in Nigeria show that women living near gas flaring sites have a 2.8-fold increased risk of infertility (Orugun *et al*., 2023).

3. Male Infertility and Sperm Damage

Gas flaring emissions significantly affect male reproductive health by reducing testosterone levels, impairing sperm production, and increasing oxidative stress in the testes. Lead (Pb) and mercury (Hg) accumulate in the testes, causing reduced sperm count and motility. Arsenic exposure is linked to DNA fragmentation in sperm, increasing miscarriage risk (Iwegbue *et al.,* 2022).

Benzene and PAHs reduce testosterone levels, affecting libido and erectile function. Oxidative stress from $PM_{2.5}$ exposure damages sperm DNA and increases abnormal sperm morphology. A study in Rivers State found that men living near gas flaring sites had 40% lower sperm motility and 30% lower sperm concentration compared to controls (World Bank, 2023). Higher levels of benzene and NO_2 exposure were associated with decreased testosterone and increased sperm DNA fragmentation.

4. Pregnancy Complications and Adverse Birth Outcomes

Maternal exposure to gas flaring pollutants increases the risk of adverse birth outcomes, including miscarriage, preterm birth, low birth weight, and congenital abnormalities.

Mechanisms of Pregnancy Complications

NO_2 and CO exposure reduce placental blood flow, causing fetal hypoxia and growth restriction (Orugun *et al*., 2023). $PM_{2.5}$ induces placental inflammation, increasing the risk of preterm labor and stillbirth (World Bank, 2023).

Key Findings:

- Women in gas flaring regions have a significantly higher risk of pregnancy complications.
- Miscarriage rates are 2.37 times higher in exposed populations.
- Exposure to benzene, PAHs, and NO_2 is strongly associated with congenital abnormalities.

2. **Pathophysiology of Renal Damage From Gas Flaring**

The kidneys are highly vascularized and thus susceptible to damage from systemic toxins such as heavy metals and VOCs in gas flaring emissions. Heavy metals (e.g., cadmium and lead) accumulate in renal tissues, impairing nephron function and causing oxidative stress. VOCs like benzene and toluene affect renal metabolism through ROS generation and direct toxicity.

Lead and cadmium cause proximal tubule dysfunction, impairing the reabsorption of essential electrolytes and causing nephrotoxicity. PAHs induce

glomerular injury by promoting inflammatory cytokines such as TNF-α and IL-6.

Reactive oxygen species damage renal cells, causing acute tubular necrosis (Orisakwe *et al*., 2012).

Health Impacts

Chronic exposure to heavy metals can lead to tubular necrosis and reduced glomerular filtration rate (GFR) (Nriagu *et al*., 2016). Chronic Kidney Disease (CKD): Long-term exposure to heavy metals disrupts renal blood flow, increasing the risk of CKD. Impaired kidney function affects sodium and potassium balance, causing cardiac arrhythmias and neuromuscular dysfunction.

3. Effect of Gas Flaring on The Hematological System

Pathophysiological Mechanisms

Gas flaring emissions significantly affect hematopoiesis and erythrocyte stability due to exposure to carbon monoxide (CO), lead, and benzene. CO interferes with oxygen transport by forming carboxyhemoglobin, reducing oxygen delivery to tissues. Benzene metabolites impair bone marrow function, leading to hematotoxicity.

- Benzene: Causes bone marrow suppression, reducing the production of red blood cells, white blood cells, and platelets.
- Heavy Metals: Lead inhibits heme synthesis, causing anemia.
- Oxidative Damage: PAHs induce lipid peroxidation in erythrocyte membranes, reducing cell viability.

Health Impacts

1. Anemia: Lead and benzene exposure cause microcytic anemia and aplastic anemia (Kponee *et al.*, 2015).
2. Leukopenia: Bone marrow suppression leads to low white blood cell counts, increasing susceptibility to infections.
3. Thrombocytopenia: Platelet production is reduced, increasing the risk of bleeding disorders.

4. Effects of Gas Flaring on the Immunological System

Exposure to gas flaring pollutants, particularly PAHs, $PM_{2.5}$, and VOCs, modulates immune function by triggering chronic inflammation and immune suppression. Cytokine dysregulation results from $PM_{2.5}$ exposure, while heavy metals impair lymphocyte activity.

- $PM_{2.5}$: Activates macrophages, producing pro-inflammatory cytokines (IL-1β, TNF-α, IL-6).
- Heavy Metals: Disrupt T-cell and B-cell activity, reducing adaptive immunity.
- Oxidative Stress: Induces apoptosis of immune cells, impairing host defense.

Health Impacts

1. Chronic Inflammation: Persistent cytokine activation leads to autoimmune diseases (Ezejiofor *et al*., 2014).
2. Immunosuppression: Heavy metal exposure reduces resistance to infections.
3. Allergic Sensitization: VOCs increase susceptibility to allergic asthma and other hypersensitivity reactions.

5. Musculoskeletal System

Gas flaring pollutants, particularly heavy metals (lead, mercury, cadmium), $PM_{2.5}$, and VOCs, disrupt musculoskeletal health by causing oxidative stress, inflammation, and calcium dysregulation. The musculoskeletal system is indirectly affected by systemic inflammation and reduced nutrient absorption resulting from gastrointestinal and renal dysfunction.

- Heavy Metals: Lead and cadmium disrupt calcium metabolism, impairing bone mineralization and causing osteopenia and osteoporosis (Nriagu *et al*., 2016).
- Inflammation: Chronic exposure to $PM_{2.5}$ triggers systemic inflammation, leading to joint degeneration and arthritis.
- Neurotoxicity: VOC-induced nerve damage contributes to muscle weakness and cramps.

Health Impacts

1. Bone Disorders: Chronic exposure to heavy metals increases the risk of osteoporosis and fractures.
2. Muscle Wasting: Oxidative stress leads to muscle atrophy and reduced muscle function.
3. Arthritis: Systemic inflammation exacerbates osteoarthritis and joint pain.

13. Gastrointestinal (GI) System

Pollutants from gas flaring, including heavy metals and PAHs, interfere with gastrointestinal integrity by damaging the mucosal barrier, altering gut microbiota, and inducing oxidative stress in GI tissues.

- Heavy Metals: Lead and arsenic impair nutrient absorption and induce toxicity in the liver and gut lining.
- PAHs and VOCs: Disrupt gut microbiota composition, promoting gut inflammation and increasing intestinal permeability ("leaky gut").
- Oxidative Stress: ROS damage intestinal epithelial cells, impairing barrier function and nutrient uptake.

Health Impacts

1. Gastroenteritis: Increased exposure to heavy metals and VOCs results in diarrhea, abdominal pain, and vomiting.

2. Hepatotoxicity: Pollutants affect liver function, leading to elevated liver enzymes and hepatocyte injury (Osuji & Onojake, 2006).
3. Nutritional Deficiencies: Impaired absorption of essential nutrients leads to deficiencies in iron, calcium, and vitamins.

14. Cutaneous System

The skin is directly exposed to airborne pollutants from gas flaring, such as heavy metals, $PM_{2.5}$, and VOCs. These pollutants generate oxidative stress and inflammatory responses in skin cells, leading to cellular damage, premature aging, and carcinogenesis.

- $PM_{2.5}$: Causes lipid peroxidation in skin cells, disrupting the skin barrier and increasing susceptibility to infections and irritation.
- Heavy Metals: Accumulate in skin tissues, causing dermatitis and pigmentation disorders.
- PAHs: Promote the formation of DNA adducts, increasing the risk of skin cancer.

Health Impacts

1. Dermatitis: Chronic exposure leads to irritant and allergic dermatitis.
2. Pigmentation Disorders: Heavy metal exposure disrupts melanin production, causing hyperpigmentation or hypopigmentation.
3. Skin Cancer: PAHs and VOCs are carcinogenic, increasing the risk of basal cell carcinoma and melanoma (Orisakwe *et al*., 2012).

15. Neoplasia and the Carcinogenic Effects of Gas Flaring

Neoplasia, the abnormal and uncontrolled growth of cells, is a serious health concern linked to prolonged exposure to environmental carcinogens. Gas flaring, a practice commonly associated with oil extraction, releases numerous toxic pollutants, including polycyclic aromatic hydrocarbons (PAHs), benzene, heavy metals (lead, mercury, arsenic, cadmium), dioxins, and fine particulate matter ($PM_{2.5}$, PM_{10}). These substances have been identified as potent carcinogens capable of inducing DNA damage, oxidative stress, chronic inflammation, and epigenetic modifications, all of which contribute to the initiation and progression of various forms of cancer (Ezejiofor *et al.,* 2014; IARC, 2022). Several organ systems, including the lungs, skin, liver, kidneys, hematopoietic system, and reproductive organs, are particularly vulnerable to the carcinogenic effects of gas flaring emissions.

Mechanisms of Gas Flaring-Induced Neoplasia

The development of cancer due to gas flaring exposure is driven by several interconnected biological mechanisms, including DNA damage, chronic inflammation, and epigenetic alterations.

DNA Damage and Mutagenesis

One of the primary ways gas flaring pollutants contribute to cancer formation is by damaging DNA and promoting genetic mutations. PAHs and benzene metabolites are known to form DNA adducts, which disrupt normal DNA replication and increase the likelihood of mutations that drive cancerous transformations (Kponee *et al*., 2015). Similarly, heavy metals such as arsenic, cadmium, and lead interfere with DNA repair mechanisms, leading to genomic instability and an increased likelihood of malignant transformation (Nduka & Orisakwe, 2010). Additionally, oxidative stress induced by reactive oxygen species (ROS) further damages nucleotides and enhances carcinogenesis (Calderón-Garcidueñas *et al*., 2016).

Chronic Inflammation and Cell Proliferation

Gas flaring emissions contain high levels of particulate matter ($PM_{2.5}$, PM_{10}), sulfur dioxide (SO_2), and nitrogen oxides (NO_x), all of which contribute to chronic inflammation. Inflammatory processes are characterized by the sustained release of pro-inflammatory cytokines such as IL-6, TNF-α, and IL-1β, creating an environment that favors uncontrolled cell growth and tumor formation (Buonocore *et al*., 2023). Chronic inflammation also inhibits normal apoptosis, allowing damaged cells to persist and proliferate, further increasing cancer risk (Osuji & Onojake, 2006).

Epigenetic Alterations and Tumorigenesis

Epigenetic modifications, such as abnormal DNA methylation, histone modifications, and altered microRNA expression, also play a significant role in gas flaring-induced carcinogenesis. These changes can lead to the silencing of tumor suppressor genes and activation of oncogenes, further promoting the development of malignancies (Ezejiofor *et al*., 2014).

Types of Neoplasia Associated with Gas Flaring Exposure

a) **Lung Cancer:** Lung cancer is one of the most common malignancies associated with gas flaring emissions. The inhalation of PAHs, benzene, and $PM_{2.5}$ leads to the accumulation of carcinogenic compounds in lung tissues, resulting in genetic mutations in critical genes such as KRAS and TP53, which drive lung cancer progression (Cushing *et al*., 2020). Epidemiological studies have shown a significantly higher incidence of lung cancer in communities near gas flaring sites due to long-term exposure to airborne carcinogens (Ukpebor *et al*., 2011).

b) **Hematological Malignancies (Leukemia, Lymphoma, Myeloma):** Gas flaring releases benzene, a well-established hematotoxin, which affects the bone marrow and disrupts the production of red and white blood cells. This increases the risk of acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), and multiple

myeloma (Orisakwe *et al*., 2012). Chronic benzene exposure has also been linked to bone marrow suppression, leading to aplastic anemia and immune dysfunction (Kponee *et al*., 2015).

c) **Liver Cancer (Hepatocellular Carcinoma - HCC):** PAHs, dioxins, and heavy metals from gas flaring accumulate in the liver, where they induce hepatotoxicity and fibrosis. Over time, these changes can lead to hepatocellular carcinoma (HCC), one of the most aggressive forms of liver cancer (Osuji & Onojake, 2006). Heavy metals like arsenic and cadmium further impair hepatocyte function and interfere with detoxification pathways, increasing the risk of malignant transformation.

d) **Skin Cancer (Basal Cell Carcinoma, Melanoma, Squamous Cell Carcinoma):** Direct exposure to PAHs and UV-activated pollutants significantly increases the risk of skin cancer. PAHs interact with skin cells, leading to the formation of basal cell carcinoma, squamous cell carcinoma, and melanoma (Ana *et al*., 2009). Arsenic and cadmium bioaccumulate in the skin, disrupting normal cell division and promoting neoplastic transformation (Ezejiofor *et al*., 2014).

e) **Kidney and Bladder Cancer:** Gas flaring pollutants such as cadmium, arsenic, benzene, and toluene are known nephrotoxic agents. These toxicants accumulate in the kidneys and bladder, leading to renal cell carcinoma and urothelial carcinoma (Nriagu *et al*., 2016).

f) **Reproductive Cancers (Breast, Ovarian, Prostate, Testicular Cancer):** Gas flaring releases endocrine-disrupting chemicals (EDCs) that interfere with hormonal balance. Disruptions in estrogen and testosterone levels have been linked to an increased risk of breast, ovarian, prostate, and testicular cancers (Cushing *et al.*, 2020).

SUMMARY

Gas flaring, the controlled burning of associated natural gas during oil extraction, remains a significant environmental and public health crisis in Nigeria's Niger Delta region. Despite regulatory frameworks, Nigeria continues to be one of the top gas-flaring nations, contributing to severe air pollution, climate change, and health hazards (World Bank, 2023). The emissions from gas flaring contain particulate matter ($PM_{2.5}$, PM_{10}), carbon monoxide (CO), nitrogen oxides (NO_x), sulfur dioxide (SO_2), volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), and heavy metals—all of which have profound effects on human health (Ehumadu, Uyigue, & Ndekwu, 2021).

Key Findings

Air pollution levels in gas flaring communities exceed WHO safety standards by 400%, with $PM_{2.5}$, NO_2, and SO_2 concentrations being dangerously high (Orugun, Adebayo, & Onwuneme, 2023).

- $PM_{2.5}$ and NO_2 exposure lead to endothelial dysfunction, oxidative stress, and systemic inflammation, increasing the risk of hypertension, atherosclerosis, and myocardial infarction (World Bank, 2023). Long-term CO exposure causes hypoxia, increasing the likelihood of heart failure and arrhythmias.
- Exposure to $PM_{2.5}$, SO_2, and NO_2 triggers airway inflammation, bronchial constriction, and lung tissue scarring. Asthma prevalence is 2.5 times higher in gas flaring communities, and COPD rates are significantly elevated (Iwegbue, Emoyan, & Nwajei, 2022).
- Heavy metals (lead, mercury, arsenic) from gas flaring accumulate in the brain, causing neuroinflammation, neurotransmitter imbalances, and cognitive deficits. Exposure to $PM_{2.5}$ is linked to a 1.5x higher risk of Alzheimer's and Parkinson's disease (Orugun *et al*., 2023).
- PAHs, VOCs, and heavy metals interfere with hormone synthesis and disrupt thyroid function, increasing the risk of metabolic disorders and diabetes. Women in gas flaring regions are 2.8 times more likely to experience infertility, while men have lower testosterone levels and reduced sperm quality (Ehumadu *et al*., 2021).
- Pregnancy complications, including miscarriage, preterm birth, and congenital abnormalities, are significantly higher in gas flaring communities.

REFERENCES

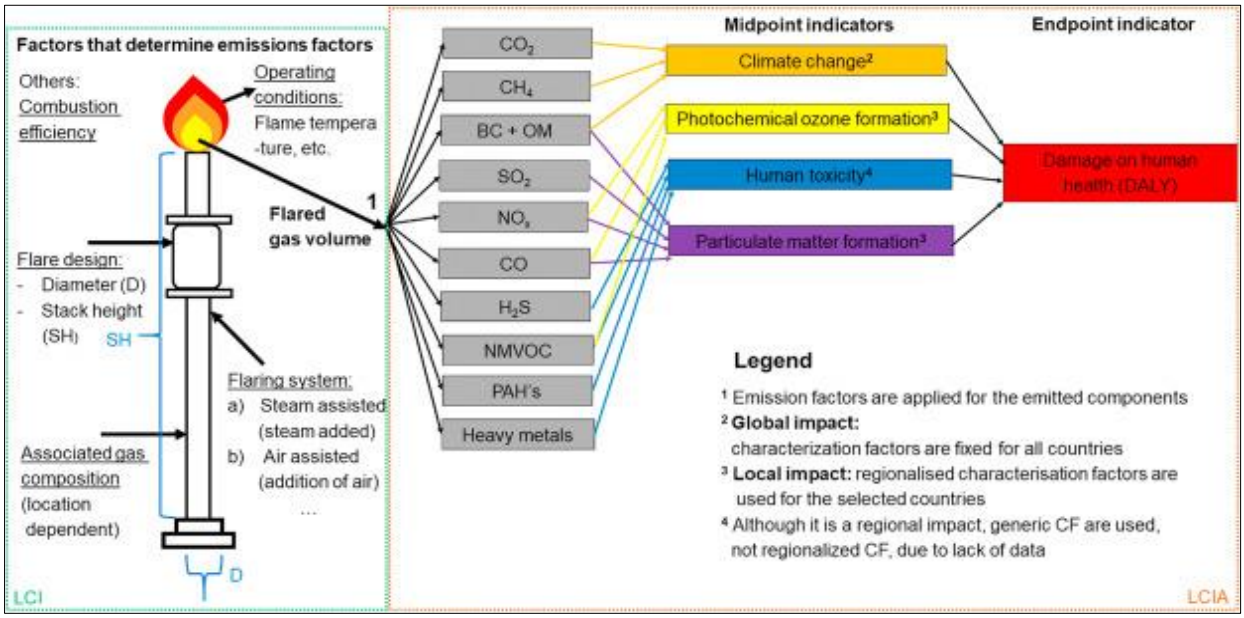
- Abu, E. M., Okafor, P. I., & Ugwumba, C. O. (2022). Geospatial analysis of gas flaring in Nigeria: Environmental and health implications. *Journal of Environmental Studies*, *45*(2), 112-128.
- Ana, G. R. E. E., Shendell, D. G., Odeshi, T. A., & Osinubi, O. O. (2009). Respiratory symptoms and lung function patterns in workers exposed to wood smoke and cooking oil fumes in Nigeria. *Journal of Occupational Medicine and Toxicology*, *4*(1), 1–8.
- Anejionu, O. C. D., Whyatt, J. D., Blackburn, G. A., & Price, C. S. (2015). Contributions of gas flaring to a global air pollution hotspot: Spatial and temporal variations, impacts, and alleviation. *Atmospheric Environment*, *118*, 184–193. https://doi.org/10.1016/j.atmosenv.2015.08.006
- Blundell, E., & Kokoza, C. (2022). Natural gas flaring, respiratory health, and distributional effects. *Bureau of Land Management Report*. Retrieved from https://eplanning.blm.gov
- Buonocore, J. J., Salas, R. N., Sohani, S., & Spengler, J. D. (2023). Oil and gas flaring linked to 7.4 billion in health damages annually. *Boston University School of Public Health*. Retrieved from

https://www.bu.edu/sph/news/articles/2024/oil-and-gas-flaring-linked-to-7-4-b-in-health-damages

- Calderón-Garcidueñas, L., Kavanaugh, M., Block, M., D'Angiulli, A., Delgado-Chávez, R., Torres-Jardón, R., & González-Maciel, A. (2016). Air pollution and your brain: What do you need to know right now. *Journal of Alzheimer's Disease, 54*(2), 597–611.
- Cushing, L. J., Vavra-Musser, K., Chau, K., Franklin, M., & Johnston, J. E. (2020). Flaring from unconventional oil and gas development and birth outcomes in the Eagle Ford Shale in South Texas. *Environmental Health Perspectives, 128*(7), 077003. https://doi.org/10.1289/EHP6394
- Ehumadu, C. O., Uyigue, L., & Ndekwu, B. O. (2021). Air pollution assessment of a gas flare site in the Niger Delta region. *Journal of Engineering Research and Reports, 20*(8), 116–126.
- Ezejiofor, T. I. N., Nwoke, E. A., & Orisakwe, O. E. (2014). Environmental health impacts of gas flaring in Nigeria: A review. *Journal of Environmental Science and Water Resources, 3*(1), 1–10.
- Iwegbue, C. M. A., Emoyan, O. O., & Nwajei, G. E. (2022). Neurotoxic and respiratory effects of gas flaring exposure in Delta State, Nigeria. *Journal of Environmental Toxicology, 19*(4), 327-345.
- Kponee, K. Z., Zhou, J., Xia, Y., Boulware, D. R., & Nash, D. (2015). Indoor air pollution from gas flaring and respiratory symptoms in a Nigerian community. *Environmental Research, 137*, 1–7.
- Motte, J., Alvarenga, R. A., Thybaut, J. W., & Dewulf, J. (2021). Quantification of the global and regional impacts of gas flaring on human health via spatial differentiation. *Environmental Pollution, 291*, 118213. https://doi.org/10.1016/j.envpol.2021.118213
- Nduka, J. K., & Orisakwe, O. E. (2010). Water-quality issues in the Niger Delta of Nigeria: A look at heavy metal levels and some physicochemical properties. *Environmental Science and Pollution Research, 17*(7), 1231–1240.
- Orisakwe, O. E., Akumuo, R. C., & Nduka, J. K. (2012). Heavy metal health risk assessment of rainwater harvested from gas flaring areas in Nigeria. *Environmental Monitoring and Assessment, 184*(5), 1–10.
- Orugun, M. I., Adebayo, A. O., & Onwuneme, C. C. (2023). Health impacts of gas flaring on communities in Bayelsa State. *Environmental Research, 245*, 115–127.
- Osuji, L. C., & Onojake, C. M. (2006). Field reconnaissance and estimation of petroleum hydrocarbon and heavy metal contents of soils affected by the Ebocha-8 oil spillage in Niger Delta, Nigeria. *Journal of Environmental Management, 79*(3), 273–279.
- Ukpebor, E. E., Obazee, E. I., & Ukpebor, J. E. (2011). Air quality assessment in gas flaring communities in the Niger Delta, Nigeria. *Journal of Environmental Chemistry and Ecotoxicology, 3*(10), 264–270.
- United Nations Environment Programme (UNEP). (2022). *Best practices for reducing gas flaring and transitioning to sustainable energy.* Retrieved from https://www.unep.org
- World Bank. (2022). *Global Gas Flaring Reduction Partnership (GGFR).* Retrieved from https://www.worldbank.org
- World Bank. (2023). *Impact of gas flaring on child health in Nigeria.* Retrieved from https://documents1.worldbank.org
- World Health Organization (WHO). (2022). *Air pollution and global health: WHO compendium on health and environment.* Retrieved from https://www.who.int/airpollution

APPENDIX

Figure 2: Location of Gas Flare Fields in Niger Delta. Source: Schick, L. Mapping Gas Flares in the Niger Delta. Code of Africa (Impact Africa). 2017



Legend: CO_2 – Carbon Dioxide, CH_4 – Methane, BC + OM – Black Carbon and Organic Matter, SO_2 – Sulfur Dioxide, NO_x – Nitrogen Oxides, CO – Carbon Monoxide, H_2S – Hydrogen Sulfide, NMVOC – Non-Methane Volatile Organic Compounds, PAHs – Polycyclic Aromatic Hydrocarbons, Heavy Metals

Figure 3: Gas Flare Composition

Source: (Mottee *et al.*, 2021)

Table 1: Annual Gas Flaring Emissions (in billion cubic meters) by Country

Country	Gas Flaring (billion m^3/year)	CO_2 Emissions (Mt CO_2)	Methane Emissions (Mt CH_4)
Russia	23.0	75.0	1.2
Nigeria	7.3	25.2	0.57
United States	7.0	22.5	0.45
Canada	1.8	5.9	0.12
Norway	0.3	1.2	0.03

(**Source**: World Bank, 2023)

Table 2: Measured Air Pollutant Concentrations at Different Distances from Gas Flaring Sites

Distance from Flare Site (m)	CO (µg/m^3)	NO_2 (µg/m^3)	SO_2 (µg/m^3)	$PM_{2.5}$ (µg/m^3)	WHO Limit
50	21.5	48.3	32.7	102.6	CO: 10, NO_2: 40, SO_2: 20, $PM_{2.5}$: 25
100	17.8	38.4	27.5	85.1	-
500	6.2	15.1	12.3	34.9	-
1000	3.1	8.7	6.1	21.5	-

(**Source**: Ehumadu *et al*., 2021)

These findings highlight that pollutant concentrations remain significantly elevated in communities located near gas flaring sites, explaining the higher prevalence of respiratory diseases in such regions.

Table 3: Pregnancy Outcomes in Gas Flaring Communities

Pregnancy Outcome	Gas Flaring Communities (%)	Non-Flaring Communities (%)	Relative Risk (RR)
Miscarriage	18.7%	7.9%	2.37
Preterm Birth	21.4%	9.2%	2.33
Low Birth Weight	16.9%	6.8%	2.49
Congenital Defects	7.2%	2.5%	2.88

(**Source**: Ehumadu *et al*., 2021)

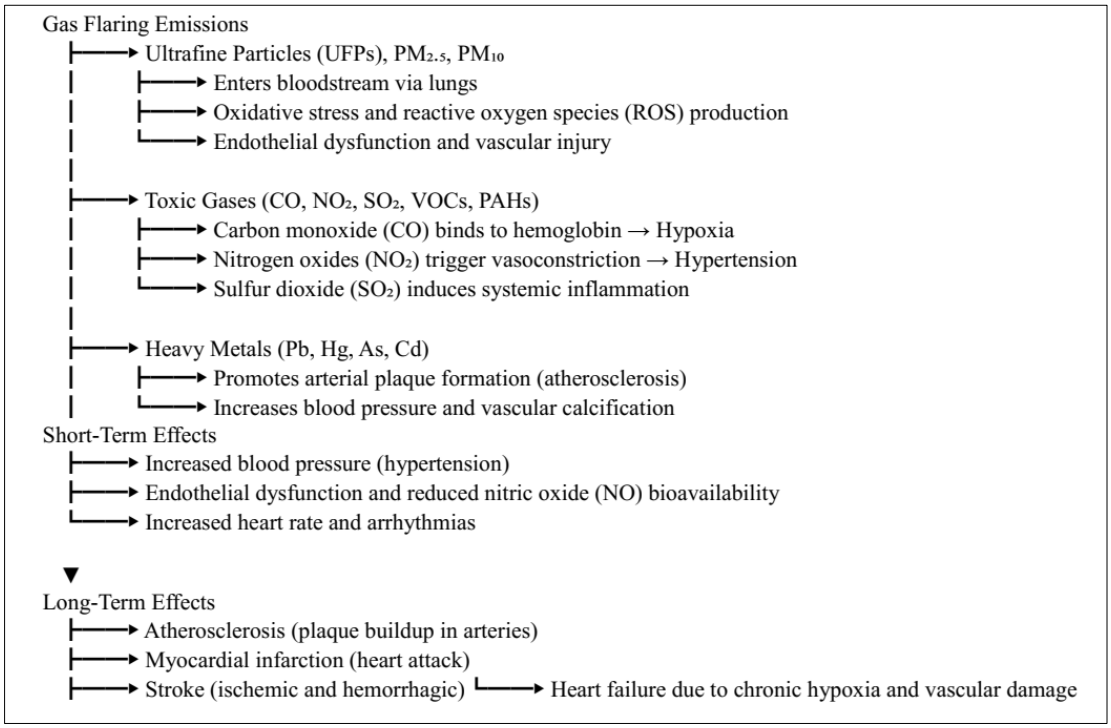


Figure 4: The Pathophysiological Mechanisms of Cardiovascular Disease Due to Gas Flaring
Source: Self

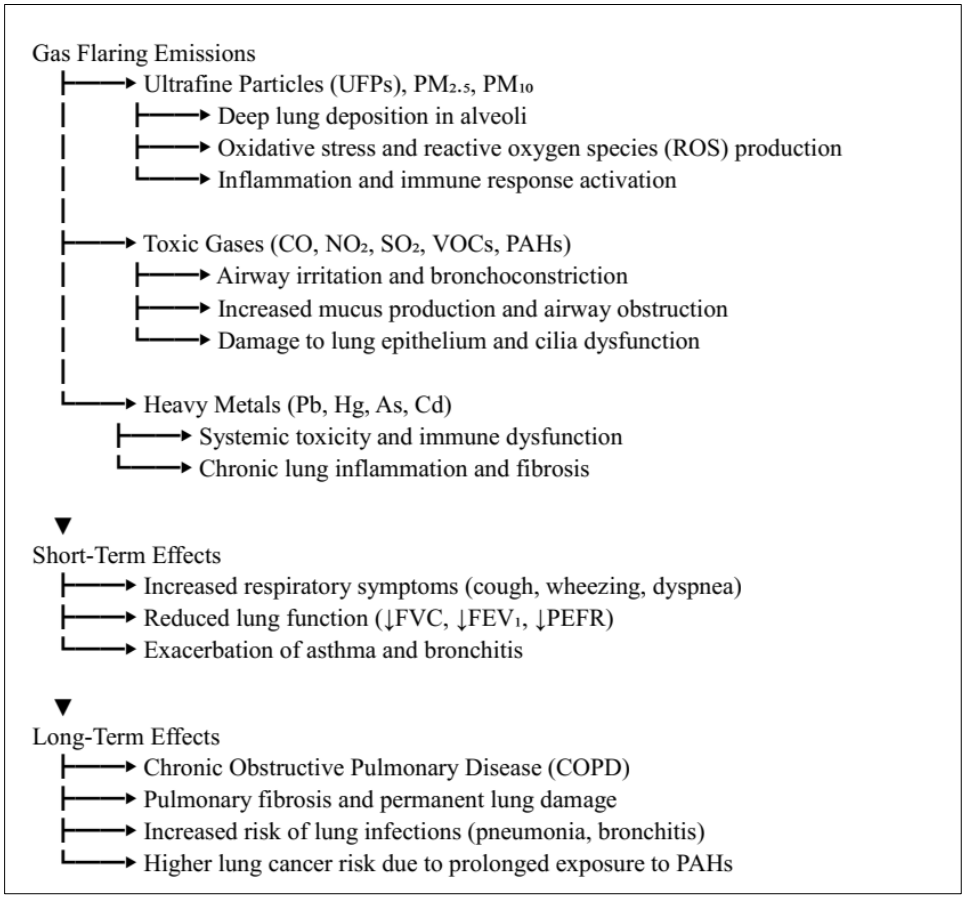


Figure 5: Pathophysiological Mechanism of Respiratory Disease Due to Gas Flaring
Source: Self

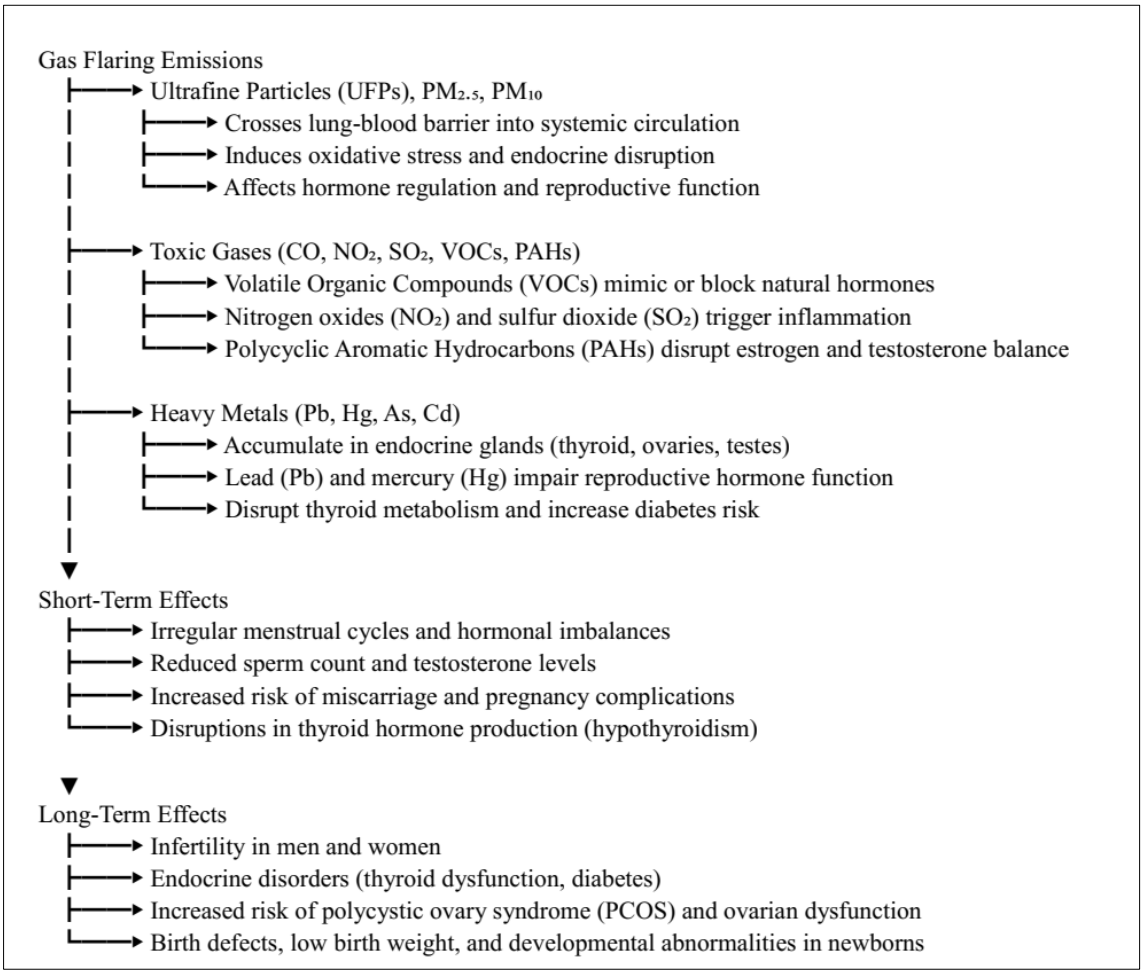


Figure 6: Pathophysiological Mechanism of Reproductive Disease Due to Gas Flaring
Source: Self

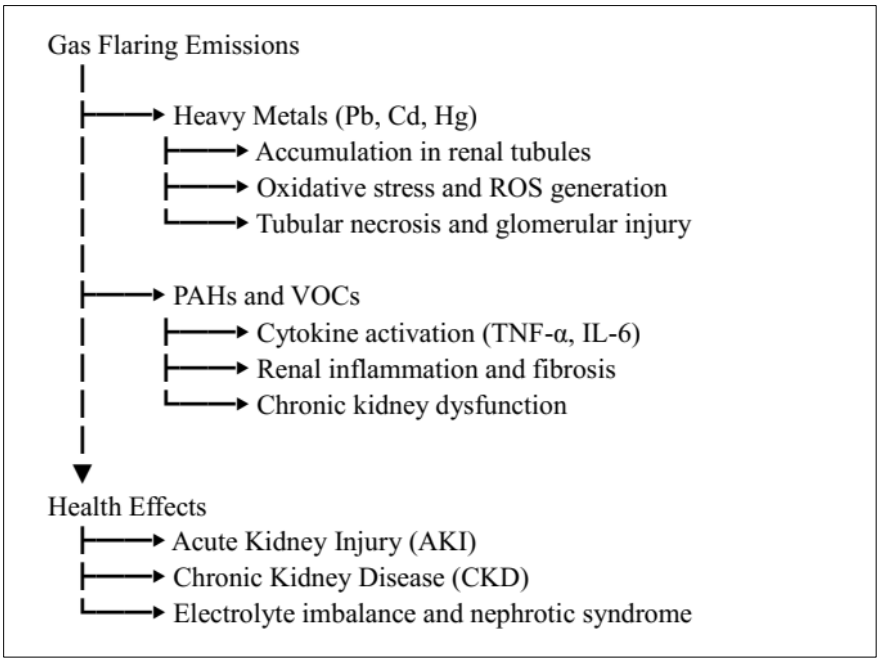


Figure 7: Pathophysiological Mechanism of Renal Disease Due to Gas Flaring
Source: Self

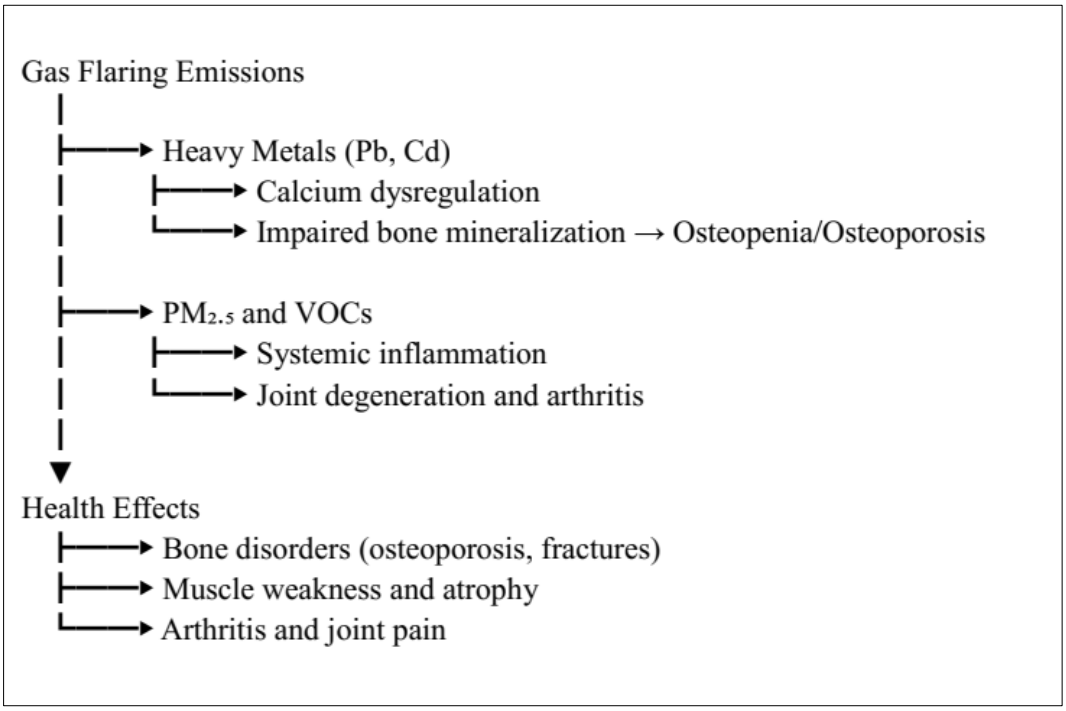


Figure 8: Musculoskeletal Effects of Gas Flaring

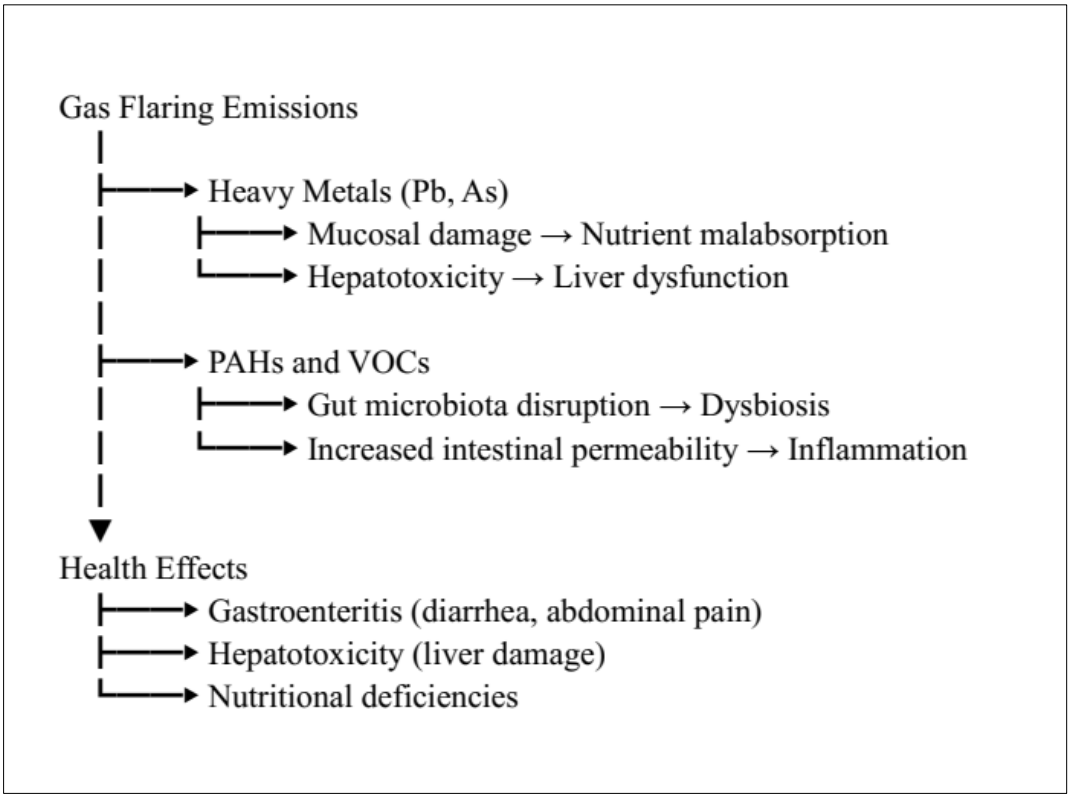


Figure 9: Gastrointestinal Effects of Gas Flaring

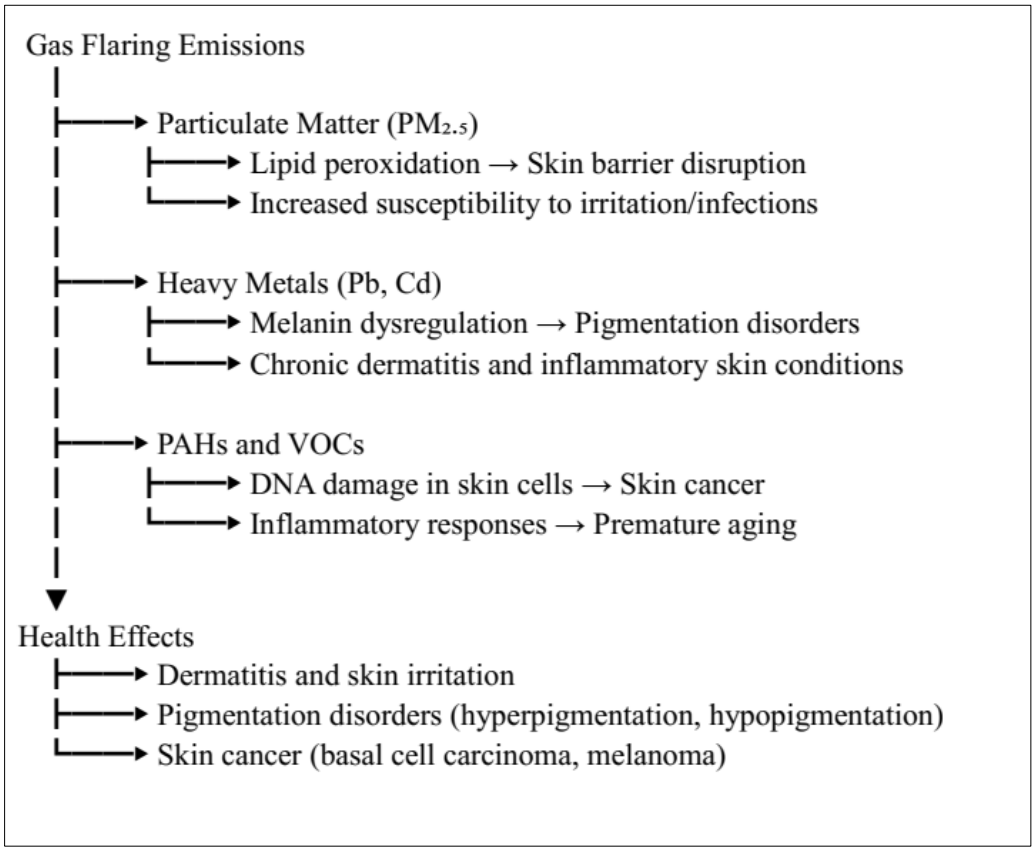


Figure 10: Cutaneous Effects of Gas Flaring

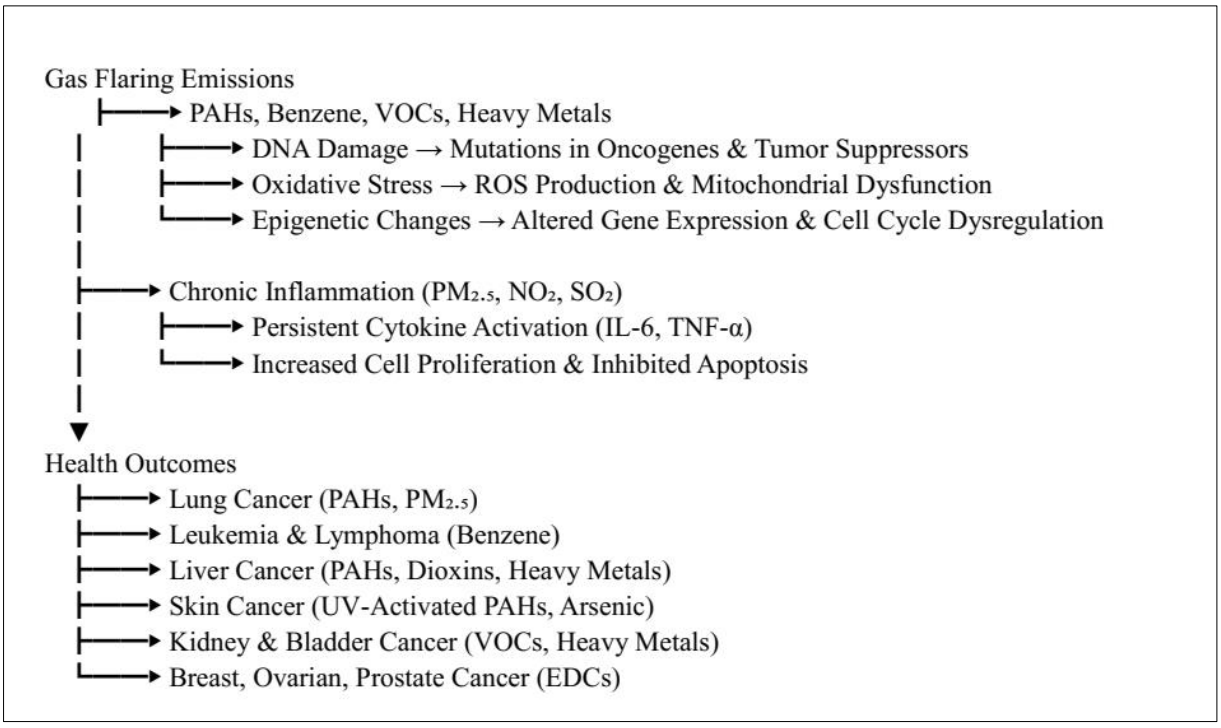


Figure 11: Neoplastic Effects of Gas Flaring