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***Review Article***

# Petroleum Industry-Derived Obesogens in Nigeria's Niger Delta Region: Mechanistic Insights, Clinical Impacts, and Public Health Implications

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## Abstract

The Niger Delta region of Nigeria represents one of the most ecologically degraded industrialized zones globally, characterized by over six decades of intensive crude oil extraction, gas flaring, and systemic oil spills. While the conventional paradigm of environmental toxicity in this region focuses heavily on onco-genesis, mutagenicity, and respiratory pathologies, an emerging frontier in environmental medicine im-plicates local pollutants as potent "obesogens." These environmental obesogens are xenobiotic compounds that disrupt lipid metabolism, adipogenesis, and metabolic homeostasis, thereby promoting obesity and metabolic syndrome independent of caloric intake or physical exertion. This review article comprehensively elucidates the molecular, cellular, and systemic patho-mechanisms by which petroleum and natural gas industry contaminants—specifically Polycyclic Aromatic Hydrocarbons, Volatile Organic Compounds (VOCs) like benzene, toluene, ethylbenzene, and xylene (BTEX), and heavy metals—induce obesogenic states within the resident population of the Niger Delta. We dissect the specific disruptions of nuclear re-ceptor signaling, including Peroxisome Proliferator-Activated Receptor gamma and Retinoid X Receptor, the induction of chronic low-grade systemic inflammation, epigenetic modifications, and the profound alteration of the gut microbiome. Furthermore, this paper analyzes the specific exposure pathways unique to the Niger Delta, such as the consumption of hydrocarbon-contaminated seafood, artisanal refining ac-tivities, and the inhalation of gas flaring particulate matter. By mapping these molecular cascades, this review provides a strong mechanistic link between oil-industrial ecotoxicity and the rising burden of non-communicable metabolic diseases in the Niger Delta region of Nigeria, providing a critical framework for environmental health policy and targeted therapeutic interventions.

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# 1. Introduction

The Niger Delta region of Nigeria, a globally significant basin for petroleum and natural gas exploration, is among the most ecologically stressed environments in the world [1–6]. While the socio-economic impli-cations of Nigeria's oil and gas industry are profound, the environmental degradation associated with its activities—including widespread oil spills, effluent discharges, and continuous gas flaring—poses severe, long-term public health threats [4–6]. Historically, the health discourse in the Niger Delta has focused on acute toxicities, respiratory diseases, and oncogenic risks [4, 5]. However, a growing body of toxicological and epidemiological evidence indicates that the complex mixture of environmental pollutants permeating the region's air, water, and soil plays a critical, insidious role in obesity and obesity-related metabolic dis-orders [7, 8].

Epidemiological data from Nigeria's Niger Delta region reveal a high prevalence of adult obesity ranging from 14.3% to 52.0%, alongside escalating rates of type 2 diabetes mellitus [9, 10]. Regional studies indicate generalized obesity rates reaching 49.6% and 52% among oil company workers and women, respectively, alongside an elevated burden of other obesity-related metabolic disorders linked to environmental petroleum operations [9, 10]. The residents in these oil-polluted communities face a type 2 diabetes mellitus prevalence of 17.4% compared to 6.6% in non-polluted areas, underscoring an urgent public health concern [11]. Furthermore, rising pre-diabetes and diabetes prevalence has been documented to exceed 50% in specific adult cohorts in the area [9, 10].

Specifically, these pollutants exhibit obesogenic properties; they interfere with normal hormonal signaling and metabolic pathways, predisposing exposed populations to abnormal weight gain, adiposity, and obe-sity-related metabolic dysfunction [7–13]. Hence, this review critically examines the obesogenic pathways triggered by environmental pollutants, such as the Polycyclic Aromatic Hydrocarbons (PAHs), Volatile Organic Compounds (VOCs) like benzene, toluene, ethylbenzene, and xylene (BTEX), toxic heavy metals, and gas flaring particulate matter from the petroleum and natural gas industry activities in the Niger Delta and how each of these pollutants trigger obesity and obesity-related non-communicable disorders (NCDs). Understanding pathways is vital for addressing the NCDs burden in vulnerable Niger Delta communities and informing evidence-based public health interventions.

# 2. Methods

## 2.1. Search Strategy and Information Sources

To ensure a comprehensive gathering of literature, systematic searches were conducted across four primary electronic databases: PubMed, ScienceDirect, Google Scholar, and African Journals OnLine (AJOL). The search string targeted three core thematic pillars: exposure/source (petroleum, flaring, hydrocarbons), ge-ographic setting (Nigeria, Niger delta), and clinical outcomes (obesity, obesogens, metabolic disorders, cardiovascular disorders). A combination of Medical Subject Headings (MeSH) terms and free-text key-words will be deployed using Boolean operators (AND, OR). The search string will be formatted as follows: ("Niger Delta" OR "Nigeria" OR "Port Harcourt" OR "Warri" OR "Bayelsa") AND ("petroleum" OR "gas flaring" OR "oil spill" OR "crude oil" OR "polycyclic aromatic hydrocarbons" OR "PAHs" OR "heavy metals" OR "VOCs" OR "particulate matter") AND ("obesogen" OR "obesity" OR "adipogenesis" OR "weight gain" OR "endocrine disruption" OR "metabolic syndrome"). To capture local context and gray literature that may not be indexed in major international databases, manual searches were performed on the repository sites of Nigerian universities, the Federal Ministry of Environment, and regional environmental protection agencies. Reference lists of all retrieved articles were hand-searched to identify additional rel-evant studies. The search was limited to articles published in English, spanning from January 2000 to 2026, capturing over two decades of intense oil and gas exploitation and concurrent rising metabolic health trends in the region. Figure 1 summarizes the selection processes of all the reviewed studies.

## 2.2. Study Selection and Eligibility Criteria

Studies were screened using a two-stage process. First, two independent reviewers evaluated the titles and abstracts of all unique citations identified during the initial search. Second, full-text articles of potentially eligible studies were retrieved and evaluated against pre-defined inclusion and exclusion criteria. Any disagreements between reviewers at either stage were resolved through consensus or via consultation with a most senior reviewer.

### Inclusion Criteria

**Study Type:** Peer-reviewed original research (epidemiological, toxicological, in vivo, in vitro, and ecological studies) and authoritative institutional reports.

**Exposure Source:** Environmental pollutants directly linked to petroleum and gas operations, including gas flaring emissions, crude oil spills, produced water, and petrochemical waste. Specific chemical components of interest include PAHs, volatile organic compounds (VOCs), heavy metals (lead, cadmium, nickel), and particulate matter ($PM_{2.5}$ and $PM_{10}$).

**Outcome:** Direct or indirect obesogenic mechanisms, such as adipocyte differentiation, disrupted lipid metabolism, alterations in appetite-regulating hormones (leptin, ghrelin), epigenetic modifications, and endocrine-disrupting pathways.

**Setting:** Studies must either be contextualized in Nigeria's Niger Delta region or use pollutant concentra-tions and exposure profiles directly representative of the Niger Delta environment.

### Exclusion Criteria

Studies focusing on general obesity risk factors (e.g., diet, genetics, sedentary lifestyle) without evaluating environmental chemical exposures. Editorials, commentary pieces, conference abstracts without full text, and studies published in non-English languages.

### 2.3. Data Extraction and Management

All identified citations were imported into reference management software (EndNote or Covidence) to automatically identify and remove duplicates. Data from the finalized list of included studies was system-atically extracted using a standardized, pre-piloted Microsoft Excel spreadsheet. For each study, the fol-lowing information was extracted:

**Bibliographic data:** Author, year of publication, and study design.

**Exposure profiling:** Specific pollutant evaluated (e.g., specific PAH congeners, heavy metals), environ-mental medium sampled (air, soil, water, biota, or human tissue), and exposure concentrations.

**Mechanistic/biological endpoints:** Pathways altered (e.g., PPAR-gamma activation, glucocorticoid re-ceptor signaling, oxidative stress, inflammation, mitochondrial dysfunction).

**Key findings:** Observed statistical associations, dose-response relationships, or biological effects on adi-pogenesis and metabolic disruption.

### 2.4. Data Synthesis and Analysis

Given the anticipated heterogeneity in study designs (ranging from molecular in vitro assays to human epidemiological cohorts), a qualitative narrative synthesis approach was utilized. Data were not pooled quantitatively for a meta-analysis. Instead, the synthesis was structured textually and conceptually around the biological mechanisms through which these regional pollutants act as obesogens. The synthesis mapped the extracted data across three distinct mechanistic tiers:

**Molecular and cellular alterations:** Documenting how oil-derived toxins trigger adipocyte hyperplasia or hypertrophy via receptor-mediated pathways (e.g., Peroxisome Proliferator-Activated Receptor gamma).

**Systemic disruption:** Analyzing impacts on endocrine signaling, neuroendocrine appetite control, and chronic pro-inflammatory states.

**Epidemiological realities:** Correlating environmental monitoring data from the Niger Delta with regional trends in metabolic disorders.

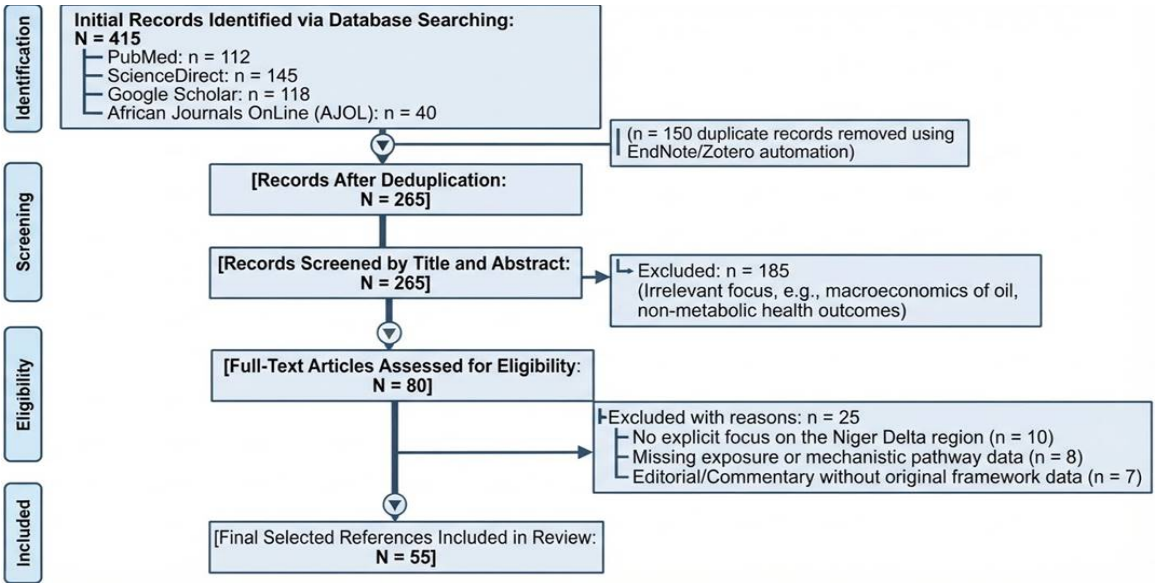


**Figure 1:** Flow diagram summarizing the selection processes of all the reviewed studies

## 3. Paradigm Shift: From Cytotoxicity to Obesotoxicity

In the past, acute toxicity and traditional chronic diseases have been the main subjects of epidemiological and toxicological studies in the Niger Delta [5, 6]. Dermatological lesions, acute respiratory distress syn-drome, hematological malignancies (including leukemia associated with exposure to benzene), reproductive failures, and genotoxic damage leading to increased cancer clusters are among the documented health effects [5, 6]. The increasing incidence of metabolic illnesses, such as type 2 diabetes mellitus, atherogenic dyslipidemia, and visceral obesity among the region's populations, is a significant epidemiological shift that has largely gone neglected, although these deadly maladies are still crucial [5–13]. The thermodynamic paradigm of energy imbalance—the excess of caloric intake over energy expenditure—has historically been used to understand obesity [9, 13]. The increasing rates of metabolic syndrome in rural and semi-urban Niger Delta communities, where access to ultra-processed Western diets is restricted and traditional livelihoods (such as artisanal fishing and subsistence farming) necessitate high levels of physical activity, are not adequately explained by this traditional model [1–4]. The Environmental Obesogen Hypothesis must be accepted due to this disparity [12, 13]. This theory, which was first put forth by Bruce Blumberg in 2006, suggests that some chemical substances, referred to as "obesogens," might modify energy balance pathways and artificially program the growth of fat cells, increasing fat accumulation [12, 13]. The numerous chemical byproducts of petroleum extraction and processing in the Niger Delta act as subtle endocrine and metabolic disruptors that actively promote adipogenesis and metabolic dysfunction in addition to being systemic poisons [7, 8].

## 4. Characterization of Petroleum-Derived Obesogens

The complex chemical mixture polluting the Niger Delta can be categorized into three major classes of potent metabolic disruptors: Polycyclic Aromatic Hydrocarbons (PAHs), Volatile Organic Compounds (VOCs), heavy metals, and alkylphenols [1–4]. PAHs are fused benzene ring structures generated during the incomplete combustion of organic materials, making them a primary constituent of gas flaring smoke and crude oil residues [4, 14].

In the Niger Delta, low molecular weight PAHs (such as phenanthrene, anthracene, and fluorene) and high molecular weight PAHs (such as benzo[a]pyrene, chrysene, and fluoranthene) contaminate the air, soil, and aquatic trophic chains. Benzo[a]pyrene (BaP), a well-established carcinogen, is also a highly potent obe-sogen [4, 14]. PAHs are highly lipophilic, allowing them to easily cross biological membranes, bioaccumulate in adipose tissues, and persist within the human body for extended durations [14]. The volatile fraction of petroleum products consists largely of the BTEX group: Benzene, Toluene, Ethylbenzene, and Xylene (BTEX) isomers. These compounds evaporate rapidly from open oil spill sites, borrowing pits, and the rudimentary distillation setups used in illegal artisanal refineries [3, 14]. Because these artisanal sites operate within densely forested and inhabited areas, atmospheric concentrations of BTEX in parts of Rivers, Bayelsa, and Delta states frequently exceed international safety limits by several thousand-fold [2, 14]. While acutely neurotoxic and myelotoxic, chronic low-dose inhalation of BTEX compounds disrupts systemic endocrine regulation, targeting hepatic and adipose tissue metabolic axes [14].

Crude oil extraction and the discharge of untreated industrial drilling muds release significant quantities of heavy metals into the Niger Delta's water tables and agricultural soils. Chief among these are Lead, Cad-mium, Chromium (particularly the hexavalent form), and Arsenic [4, 14]. These metals do not degrade; instead, they undergo biomagnification. For instance, periwinkles *(Tympanotonus fuscatus)*, tilapia, and mudfish—staples of the local diet—accumulate high tissue concentrations of Cadmium and lead [2, 14]. Beyond their established nephrotoxic and neurotoxic profiles, these heavy metals act as metallo-estrogens and enzymatic inhibitors that disrupt the delicate endocrine feedback loops governing appetite, thermo-genesis, and lipolysis [4, 14]. These structural compounds are present within the heavier polar fractions of crude oil and are frequently left behind as persistent organic pollutants in regional soils and river sediments [15]. When released into the ecosystem through oil spills, gas flaring, or refining, these fractions bioac-cumulate in food chains and alter normal human metabolic programming by mimicking or interfering with endogenous endocrine signaling pathways.

Alkylphenols mimic natural 17$\beta$-estradiol [15]. This artificial estrogenic stimulation alters the homeostatic regulation of the hypothalamus, compounding leptin resistance, skewing appetite control, and triggering early-stage adipogenesis [15].

## 5. General Obesogenic Mechanisms of Petroleum Industry Pollutants

### 5.1. Molecular/Cellular Induction of Adipogenesis Pathways

The transformation of an undifferentiated mesenchymal stem cell (MSC) into a mature, lipid-laden adi-pocyte is a highly orchestrated transcriptional process [3, 16]. Petrochemical pollutants hijack this differ-entiation program at multiple molecular checkpoints [3, 13]. Peroxisome Proliferator-Activated Receptor gamma (PPAR$\gamma$) is recognized as a master regulator of adipogenesis [16]. No cell can transition into a mature adipocyte without the activation of this ligand-activated transcription factor. Under physiological conditions, PPAR$\gamma$forms a functional heterodimer with the Retinoid X Receptor (RXR), binding to specific Peroxisome Proliferator Response Elements (PPREs) in the promoter regions of adipogenic genes [16]. Toxicological studies demonstrate that components of crude oil and PAH fractions act as exogenous ligands or allosteric modulators of the PPAR$\gamma$:RXR complex. For example, specific alkylated PAHs bind directly to the ligand-binding pocket of either PPAR$\gamma$ or RXR [16]. This inappropriate activation forces pluripotential Mesenchymal Stem Cells (MSCs) to commit preferentially to the adipocyte lineage at the expense of osteogenic (bone-forming) or myogenic (muscle-forming) pathways. Consequently, the stem cell niche is skewed toward an overproduction of pre-adipocytes, laying the cellular architecture for the eventual hyperplastic obesity [16]. Adipose tissue is not a uniform energy storage depot; it comprises White Adipose Tissue (WAT), which stores energy, and Brown/Beige Adipose Tissue (BAT), which dissipates energy as heat through non-shivering thermogenesis [17, 18].

This thermogenic dissipation is driven by Uncoupling Protein 1 (UCP1) located on the inner mitochondrial membrane. Heavy metals (Cadmium, lead) and volatile organic compounds derived from Niger Delta oil spills disrupt mitochondrial integrity. These toxins inhibit key complexes of the mitochondrial Electron Transport Chain (ETC)—particularly Complex I and Complex IV [18]. This inhibition has a dual metabolic consequence [17, 18]. First, it drastically reduces adenosine triphosphate (ATP) production, signaling a cellular energy crisis that triggers compensatory cellular overeating and decreases systemic basal metabolic rate (BMR). Secondly, it downregulates the expression of UCP1 and PPAR$\gamma$ coactivator 1-alpha (PGC-1$\alpha$). This prevents the "browning" of white adipose tissue, thereby locking the body into a state in which it cannot efficiently burn excess calories through thermogenesis [17, 18].

### 5.2. Endocrine and Metabolic Disruption Pathways

Beyond cellular adipogenesis, chronic exposure to petroleum pollutants induces systemic alterations in endocrine networks that govern hunger, satiety, glucose disposal, and lipid transport [15, 16].1 The stressful environmental conditions of the Niger Delta—characterized by constant ambient noise from gas flaring, the economic devastation of traditional livelihoods, and the inhalation of volatile toxic chemicals—induce chronic biological stress [5, 19, 20]. This stress activates the Hypothalamic-Pituitary-Adrenal (HPA) axis, resulting in continuous release of Adrenocorticotropic Hormone (ACTH) and sustained elevations of sys-temic cortisol [5, 19, 20]. At the molecular level, PAHs and heavy metals like cadmium amplify this effect by altering the expression of the enzyme 11$\beta$-Hydroxysteroid Dehydrogenase Type 1 (11$\beta$-HSD1) [20]. This enzyme is highly expressed in visceral adipose tissue, where it converts inactive cortisone into active cortisol locally [20]. Petrochemical exposure selectively upregulates 11$\beta$-HSD1 activity within visceral fat depots [21]. This localized hypercortisolemia acts independently of circulating cortisol levels, driving visceral adipocyte hypertrophy, increasing glucocorticoid receptor (GR) density, and promoting the classic phenotype of truncal/visceral obesity, which carries the highest risk of cardiovascular disease [20, 21].

Healthy adipose tissue acts as an endocrine organ, secreting adipokines that communicate metabolic status to the brain and peripheral tissues [22]. One of the most critical adipokines is leptin (the satiety hormone). Chronic exposure to industrial solvents (BTEX) and heavy metals disrupts leptin secretory balance [23]. Hypertrophied adipocytes exposed to benzene show a paradoxical hypersecretion of

leptin. However, this excess leptin fails to cross the blood-brain barrier effectively and induces leptin receptor desensitization in the arcuate nucleus of the hypothalamus [23]. This establishes a state of leptin resistance, where the brain perceives a state of starvation despite massive peripheral fat stores, causing continuous appetite stimulation and accelerated systemic insulin resistance, leading to metabolic obesity [23]. Thyroid hormones (T3 and T4) are the primary determinants of the body's basal metabolic rate [24]. Halogenated compounds, alkylated phenols, and trace elements present in refinery effluents act as Thyroid Disrupting Chemicals (TDCs) [25]. These pollutants compete with thyroxine for binding sites on transport proteins like Transthyretin. Fur-thermore, heavy metals like selenium analogs or lead inhibit the hepatic Deiodinase enzymes (Type 1 and Type 2), which are responsible for converting the prohormone T4 into the metabolically active T3 [24, 25]. This induced state of peripheral subclinical hypothyroidism slows down systemic lipid clearing, decreases energy expenditure, and promotes weight gain even under calorie-restricted conditions [25]. Alkylphenols and organic esters act as xenoestrogens [15]. They bind directly to estrogen receptors ($\alpha$) and ($\beta$), hijacking normal estrogen signaling [25]. Because natural estrogen plays a crucial role in preventing fat accumulation and regulating lipid homeostasis, the erratic signaling caused by these pollutants tricks the body into storing excess triglycerides. Moreover, heavy metals and PAHs can down-regulate or inhibit aromatase activity, causing a localized collapse in sex steroid ratios [26]. This imbalance triggers adipose tissue expansion and leptin resistance, shifting the body's metabolic setpoint toward calorie storage and, finally, metabolic obesity [25].

### 5.3. Inflammatory and Oxidative Stress Pathways

Obesity is intrinsically linked with chronic, low-grade systemic inflammation in the Niger Delta region of Nigeria [9, 10, 27]. The pollution profile of the Niger Delta accelerates this inflammatory state through specific cellular interactions in the adipose tissue microenvironment [9, 10, 27–29]. Adipose tissue contains resident macrophages that maintain tissue homeostasis [30, 31]. In a lean state, these are primarily M2-polarized macrophages, which secrete anti-inflammatory cytokines like IL-10, which promotes tissue repair, prevents fibrosis, and maintains insulin sensitivity [30]. However, the accumulation of lipophilic PAHs (BaP) and heavy metals within adipocytes triggers the production of chemokines, primarily Mon-ocyte Chemoattractant Protein-1 (MCP-1/CCL2) [30, 31]. This chemokine signal recruits circulating monocytes into the visceral fat pads, where they polarize into the classical, pro-inflammatory M1 phenotype. These M1 macrophages organize themselves in a distinct spatial pattern around dying, over-distended adipocytes, forming histologic configurations known as "Crown-Like Structures" (CLS) [31]. These macrophage clusters release large quantities of pro-inflammatory cytokines into the systemic circulation, including: Tumor Necrosis Factor-alpha (TNF-$\alpha$), Interleukin-6 (IL-6), and Interleukin-1$\beta$ (IL-1$\beta$) [30, 31].

TNF-$\alpha$ directly cleaves and degrades Insulin Receptor Substrate-1 (IRS-1) via c-Jun N-terminal kinase (JNK) activation, halting insulin signaling cascades [30]. IL-6 travels to the liver, where it stimulates the hyperproduction of C-Reactive Protein and accelerates hepatic gluconeogenesis, elevating fasting blood glucose levels. IL-1$\beta$ damages pancreatic $\beta$-cell function, reducing insulin secretory capacity over time. The resulting metabolic failure here is insulin resistance. Insulin resistance enhances the chronic secretion of TNF-$\alpha$, IL-6, and IL-1$\beta$ by M1 macrophages and impairs insulin signal transduction in healthy adipocytes, leading to insulin resistance [30, 31]. Because the tissue can no longer store lipids properly due to compromised insulin signaling, the body diverts and stores excess lipids ectopically (in the liver and muscle) and forces hypertrophy in existing fat cells, driving weight gain and further accelerating the obesity epidemic [30, 31].

PAHs (like Benzo[a]pyrene) and VOCs act as powerful Aryl Hydrocarbon Receptor (AhR) agonists. PAHs and VOCs are classic exogenous ligands for the AhR, a cytosolic transcription factor involved in xenobiotic metabolism [32, 33]. Upon binding a PAH molecule, AhR translocates into the nucleus, heterodimerizes with the AhR Nuclear Translocator (ARNT), and binds to Xenobiotic Response Elements to induce Cy-tochrome P450 enzymes (such as CYP1A1 and CYP1B1). However, prolonged hyper-activation of the AhR pathway by Niger Delta gas flaring emissions triggers significant metabolic cross-talk issues. AhR competes directly with PPAR$\gamma$, and ARNT competes with RXR for limited cellular pools of co-activators [32]. While this cross-talk can sometimes inhibit normal adipogenesis in acute high-dose exposures, chronic low-dose activation of AhR shifts cellular dynamics toward a highly dysfunctional state [33]. It promotes the expression of inflammatory cytokines and causes a profound defect in lipid storage capability. This forces lipids out of subcutaneous storage and into visceral depots and ectopic organs like the liver and skeletal muscle, driving non-alcoholic fatty liver disease and classic metabolic obesity [32, 33].

The biotransformation of PAHs and BTEX by CYP450 enzymes generates substantial quantities of Reac-tive Oxygen Species (ROS), including superoxide anions and hydrogen peroxide [34–36]. The localized depletion of endogenous antioxidants—such as Glutathione, Superoxide Dismutase, and Catalase—due to heavy metal binding (cadmium and lead possess high affinity for sulfhydryl groups on these enzymes) induces severe oxidative stress [35]. This oxidative imbalance directly targets the Endoplasmic Reticulum, the organelle responsible for protein folding and lipid synthesis. The accumulation of oxidized and mis-folded proteins within the ER lumen initiates the Unfolded Protein Response (UPR), mediated through three transmembrane sensors: PERK, IRE1$\alpha$, and ATF6 [34]. Chronic UPR activation upregulates the transcription factor CHOP (C/EBP homologous protein), which triggers adipocyte apoptosis, intensifies macrophage infiltration, and activates the NF-$\kappa$B (Nuclear Factor kappa-light-chain-enhancer of activated B cells) pathway [35, 36]. This sequence sustains a chronic inflammatory cycle that locks the patient into a state of metabolic dysfunction and progressive obesity [34–36].

### 5.4. Epigenetic and Transgenerational Pathways

One of the most concerning aspects of environmental obesogen exposure in the Niger Delta is its potential to permanently reprogram human metabolism across generations through epigenetic modifications, inde-pendent of alterations to the primary DNA sequence [3, 37]. DNA methylation involves the covalent addi-tion of a methyl group to the 5-carbon position of cytosines within CpG islands, a process catalyzed by DNA methyltransferases (DNMTs) [35, 36].

Exposure to heavy metals such as arsenic disrupts this process by depleting the cellular pool of S-adenosylmethionine (SAM), the primary methyl donor. In populations exposed to oil spills, researchers observe altered methylation profiles in the promoters of genes regulating growth and metabolism [38, 39].

For example, hypermethylation of the promoters of the Leptin gene or POMC (pro-opiomelanocortin) in the fetus occurs when pregnant women ingest contaminated water or inhale gas flaring fumes [38]. This hypermethylation silences these essential satiety-signaling genes, permanently resetting the offspring's hypothalamic appetite-control set point toward hyperphagia (overeating) from birth. Conversely, hypo-methylation of the PPAR$\gamma$ promoter has been documented, lowering the transcriptional threshold required to trigger adipogenesis

throughout life [38, 39]. Histone tails undergo post-translational modifica-tions—including acetylation, methylation, ubiquitination, and phosphorylation—that dictate the structural accessibility of chromatin (euchromatin versus heterochromatin) [40, 41]. Petrochemical toxins disrupt the enzymatic balance between Histone Acetyltransferases (HATs) and Histone Deacetylases (HDACs) [37]. Specifically, exposure to nickel and chromium compounds found in refinery waste stream effluents inhibits HDAC activity [41]. This results in hyperacetylation of core histones (H3 and H4) at the promoters of lipogenic genes such as SREBP-1c and FAS. The resulting open chromatin configuration allows basal transcription factors to bind continuously, leading to baseline elevations in lipogenesis and a tendency towards obesity regardless of nutritional status [40, 41].

MicroRNAs are short, non-coding RNA molecules (21-25 nucleotides) that bind to the 3'-untranslated regions (3'-UTRs) of target messenger RNAs (mRNAs), leading to translational repression or transcript degradation [42, 43]. Inhalation of VOCs and fine particulate matter (PM2.5) from gas flares alters the ex-pression profiles of circulating and tissue-specific miRNAs [39]. For example, exposure downregulates miR-143 and miR-103 [40]. Under normal physiological conditions, miR-143 promotes proper gating of adipocyte differentiation, while miR-103 regulates insulin sensitivity by targeting caveolin-1 [43]. Their systematic downregulation by petrochemical exposure imposes downstream constraints on pre-adipocyte proliferation, leading to uncontrolled adipocyte expansion and severe peripheral insulin desensitization, ultimately leading to metabolic obesity [42, 43].

The most striking evidence of the environmental obesogen framework is its potential for transgenerational impact [38–44]. When a pregnant woman in a Niger Delta oil-producing community is exposed to PAHs and endocrine disruptors, three generations are simultaneously exposed: the mother (F0), the fetus (F1), and the primordial germ cells within that fetus (F2) [38–41]. However, toxicological models demonstrate that the metabolic phenotypes of obesity, insulin resistance, and hepatic steatosis can persist into the F3 and F4 generations, which were never exposed to the index chemical [38–41]. This transgenerational inheritance is mediated by stable, un-erased epigenetic marks passed through the germline (sperm and oocytes) [38–41]. The environmental pollution occurring today in the Niger Delta is likely programming metabolic vulner-ability and obesity into generations of Nigerians yet unborn [44].

### 5.5. Gut Microbiome Alterations (Dysbiosis) and Metabolic Toxemia Pathways

The human gastrointestinal tract hosts trillions of microorganisms that play a fundamental role in harvesting energy from the diet, maintaining mucosal barrier integrity, and regulating systemic immunity [45–48]. The ingestion of hydrocarbon-polluted water and food in the Niger Delta exerts a severe antimicrobial and disruptive effect on this fragile ecosystem, with a potential tendency toward metabolic obesity [42–45]. Crude oil fractions and heavy metals exhibit selective antimicrobial toxicity[45, 46]. Ingesting these compounds kills sensitive, beneficial commensal bacteria while allowing resistant, often pathogenic phyla to proliferate [45, 46]. This induces profound *gut dysbiosis* [45, 46]. Specifically, environmental toxicant exposure consistently shifts the ratio of the two dominant bacterial phyla: it increases *Firmicutes* and decreases *Bacteroidetes* [46]. These altered bacterial communities (raised *Firmicutes-to-Bacteroidetes* ratio) are highly efficient at breaking down and fermenting otherwise indigestible dietary polysaccharides into monosaccharides and Short-Chain Fatty Acids (SCFAs). *Firmicutes* possess an advanced enzymatic potential for harvesting energy from otherwise indigestible dietary fibers and increase the extraction of monosaccharides and SCFAs from the colonic lumen, presenting the liver with an increased calorie load from the same volume of food [45, 46]. Therefore, the host absorbs these excess monosaccharides directly in the small intestine, resulting in higher daily caloric yields from the same food intake with a tendency to-wards metabolic obesity [45, 46].

The intestinal epithelial lining is held together by an intricate network of apical junctional complexes known as tight junctions, consisting of proteins like Claudins, Occludin, and Zonula Occludens-1 [45, 47]. Chronic ingestion of heavy metals (Arsenic/Cadmium) and volatile hydrocarbons directly breaks down these tight junction proteins through oxidative stress and localized cell death [45, 47]. This structural breakdown results in an abnormally permeable intestinal barrier, commonly termed "leaky gut syndrome." This loss of integrity allows large macromolecular components to pass unhindered from the intestinal lumen directly into the lamina propria and the portal venous system [45, 47]. The most critical metabolic consequence of this increased permeability is the systemic translocation of Lipopolysaccharide (LPS) [45, 48]. LPS is a major structural component of the outer membrane of Gram-negative bacteria, acting as a potent endotoxin. Under dysbiosis, Gram-negative populations expand, and their shed LPS crosses the damaged intestinal wall into the portal vein, entering the liver and systemic circulation—a clinical condition known as *Metabolic Endotoxemia* [45, 48]. Circulating LPS is recognized by Lipopolysaccharide-Binding Protein and delivered to the CD14/Toll-Like Receptor 4 (TLR4) complex present on the surfaces of hepatocytes, skeletal myocytes, and adipocytes. Activation of TLR4 triggers a downstream intracellular signaling cascade [45,48]. Once inside the nucleus, NF-$\kappa$B drives the transcription of pro-inflammatory cytokines, causing continuous, low-grade systemic inflammation that is directly linked to insulin resistance and impaired fat burning [45]. This specific, endotoxin-driven pathway also impairs insulin receptor autophosphorylation, induces systemic leptin resistance, alters fat storage kinetics, and promotes progressive weight gain and metabolic syndrome [45, 48]. Figure 2 below illustrates how some key environmental petroleum pollutants contribute to the onset and progression of obesity and obesity-related Disorders

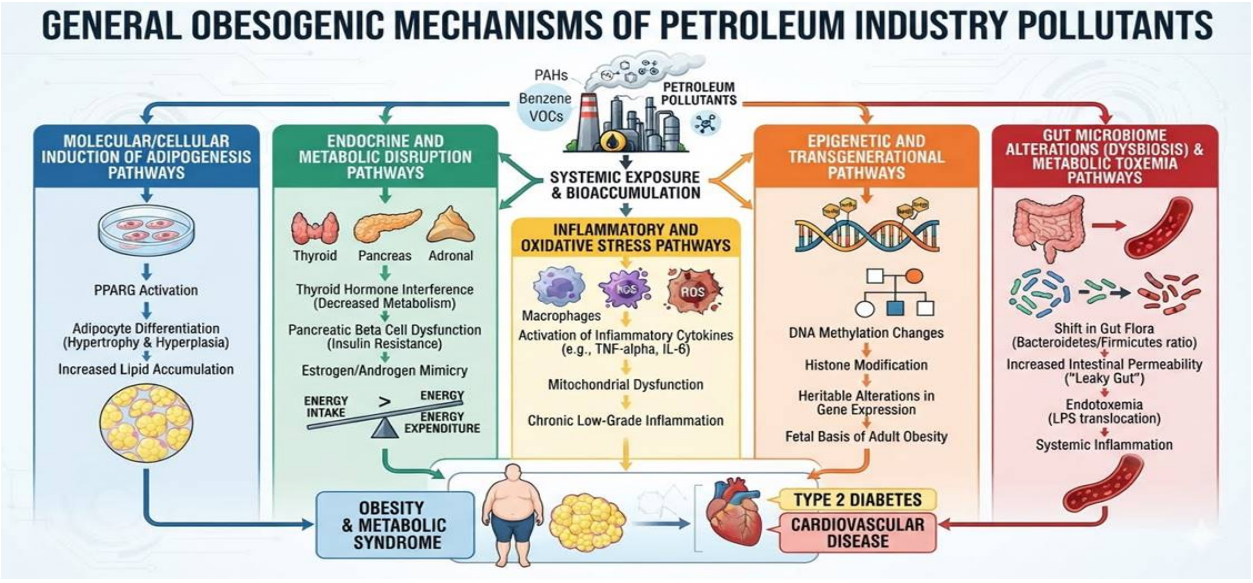


**Figure 2:** Pathway illustrating how some key environmental petroleum pollutants contribute to the onset and progression of obesity and obesity-related disorders

## 6. Specifically Validated Obesogenic Mechanisms Reported Within Nigeria's Niger Delta

### 6.1. Insulin Resistance and Hyperinsulinemia

A prominent mechanism linking crude oil pollution to weight gain and metabolic syndrome is the promo-tion of systemic insulin resistance [7]. Mechanistic and epidemiological data from the region demonstrate that populations with prolonged exposure to oil and gas flaring pollutants exhibit elevated homeostatic model assessment indices [7]. Flaring emissions, which contain high levels of polycyclic aromatic hydro-carbons (PAHs), alter glucose transport kinetics and stimulate localized cellular inflammation [7]. The resulting hyperinsulinemia drives an obligate lipogenic state. It shifts systemic fuel utilization from fat oxidation to triglyceride storage, which clinically manifests as visceral adiposity and elevated body mass index [7–11].

### 6.2. Heavy-Metal Induced Oxidative Stress and Lipoperoxidation

Petroleum exploitation in the region releases complex mixtures of heavy metals, including lead, cadmium, and arsenic, into localized groundwater and food chains [17, 28, 29]. Studies focused on Niger Delta oil-host communities validate that chronic heavy metal exposure triggers severe systemic lipid peroxidation and depletes endogenous antioxidant defenses [28, 29]. Free radicals and toxic intermediates compromise the functional integrity of mitochondrial membranes in metabolically active tissues. This mitochondrial decay impairs routine beta-oxidation of fatty acids [17, 18]. Consequently, unmetabolized lipids accumulate within ectopic depots, expanding cellular adiposity and driving macro-metabolic derangements [28, 29].

### 6.3. Disruption of the Endocrine Axes

The chemical profile of Nigerian Bonny Light Crude Oil (BLCO) directly interferes with central endocrine pathways [49, 50]. Toxicological bioassays show that exposure to BLCO disrupts peripheral hormone clearance and elevates circulating thyroid hormones (thyrotropin, triiodothyronine, and thyroxine). While conventional clinical paradigms link hyperthyroidism to weight loss, chemical-induced disruption of the pituitary-thyroid-metabolic axis in these settings can provoke compensatory hyperphagia and dysregulate appetite-controlling networks [49, 50].

Furthermore, the lipophilic nature of PAHs and volatile organic compounds (VOCs) allows them to ac-cumulate within adipose tissue. This accumulation initiates a feed-forward cycle where expanding fat mass serves as a storage sink for incoming lipophilic pollutants, prolonging endogenous endocrine disruption.

## 7. Dynamics of Mixed Obesogenic Pollutants Exposures in Nigeria's Niger Delta

Populations residing in Nigeria's oil-rich Niger Delta are chronically exposed to complex, multi-class chemical mixtures originating from relentless petroleum exploration, gas flaring, and oil spills[14, 27–29]. While traditional toxicological literature isolates the adverse health outcomes of single pollutant categories, real-world exposure patterns involve a concurrent influx of PAHs, BTEX, heavy metals, and alkylphenols [1–5]. A critical scientific gap exists regarding how these co-occurring xenobiotics interact within human biological systems to synergistically drive the onset of obesity and associated cardiometabolic syndromes. Rather than operating as isolated insults, these mixed contaminants function as a complex network of en-vironmental obesogens. They converge on crucial endocrine and metabolic pathways to disrupt cellular energy homeostasis, accelerate adipogenesis, and promote metabolic dysfunctions [6–11]. The patho-physiological mechanisms of these industry-derived mixtures operate through additive and synergistic toxicological modes of action. Structurally diverse lipophilic pollutants, such as PAHs and alkylphenols, act as potent EDCs [1, 16]. They bind to and abnormally activate the PPAR-gamma

and Retinoid X Receptor (RXR) heterodimer [16]. This master transcriptional switch triggers adipocyte hyperplasia and hypertrophy, transforming undifferentiated mesenchymal stem cells into mature, dysfunctional fat cells. Concurrently, volatile organic fractions like BTEX compounds and heavy metals (e.g., lead, cadmium, and nickel) induce severe systemic oxidative stress and deplete total antioxidant capacity. This cellular strain damages pancreatic beta-cell morphology, which severely impairs glucose-stimulated insulin secretion and downregulates peripheral insulin receptor signaling [16–19].

Observational evidence from heavily impacted communities in the Niger Delta confirms the clinical reality of these toxicant mixtures. Local epidemiological evaluations establish a clear correlation between persistent exposure to gas flaring emissions and a marked escalation in cardiometabolic risk profiles. Residents of highly polluted zones demonstrate an elevated triglycerides-to-HDL-cholesterol ratio alongside age- and time-dependent progressions of hyperinsulinemia [27–29]. This clinical reality is further corroborated by elevated occupational obesity rates exceeding 49% among regional oil and gas workers, where chemical exposures overlap with sedentary work patterns [9–11]. Additionally, environmental health surveillance across crude oil-producing communities documents systemic toxicosis [3, 4, 17, 19]. This status is marked by elevated serum levels of toxic metals, altered serum electrolytes, and heightened lipid peroxidation, directly tying environmental exposure to metabolic strain. Experimental animal models using Nigerian crude oil variants confirm these compound metabolic interactions [17, 19]. In vivo studies demonstrate that crude oil fractions cause severe hepatotoxicity, hepatic steatosis, and systemic inflammation. The organometallic complex of the mixture triggers a vicious cycle: expanded, lipophilic adipose tissue serves as a biological sink that continuously retains bioaccumulative pollutants [17, 19]. This internal reservoir causes sustained low-grade systemic inflammation by upregulating pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha). Ultimately, the antagonistic or synergistic interplay within these pollutant mixtures paralyzes physiological energy balance. This molecular failure transforms the Niger Delta's landscape from a zone of ecological degradation into a potent environmental driver of metabolic diseases [14].

## 8. Obesogenic Pollutants Problem in the Niger Delta Compared to Other Oil-Producing Regions

### 8.1. The Ecuadorian Amazon

Similar to the Niger Delta, the Ecuadorian Amazon (particularly the Oriente region) suffers from chronic, legacy oil pollution and systemic regulatory failures [51]. Indigenous populations in both regions face direct dietary exposure to petrogenic hydrocarbons through contaminated drinking water and subsistence fishing [3, 51].

However, a major difference lies in the operations: while the Amazon's burden is primarily driven by his-torical dumping of toxic produced water into surface pits, the Niger Delta's exposure is continually exac-erbated by active, widespread gas flaring and highly toxic artisanal "bunkering" refining operations, which release volatile obesogenic organics directly into coastal communities [3, 51].

### 8.2. The Gulf Coast of the United States

The Gulf Coast represents a heavily industrialized oil-producing region, notably marked by catastrophic events like the Deepwater Horizon spill [52]. Gulf Coast populations are exposed to a parallel profile of PAHs and chemical dispersants, which have been shown to induce adipogenesis in mammalian models [52]. However, the risk paradigm is entirely different. The Gulf Coast benefits from robust regulatory oversight, highly advanced engineering controls, and rapid environmental remediation, which limit long-term human contact [52]. In contrast, the Niger Delta features dilapidated pipeline infrastructure near residential zones, meaning exposure is chronic and lack of remediation leads to a "vicious cycle" where lipophilic obesogens remain in the food web indefinitely [5].

### 8.3. The Middle East

In major oil-exporting nations of the Middle East, such as Saudi Arabia, petroleum-related pollution is heavily linked to metropolitan industrial processing and airborne particulate matter ($PM_{2.5}$) rather than direct ecosystem contamination [53]. Studies from crude-rich cities like Jeddah show that PAH-rich particulate pollution significantly increases the prevalence of metabolic syndrome and hyperglycemia [53]. While the Middle East presents an urban, inhalation-driven metabolic risk, the Niger Delta presents a multi-pathway crisis where inhalation of soot from gas flaring is compounded by high-dose dermal contact and ingestion of contaminated agricultural yields [3, 53].

**Table 1:** Summary table detailing the primary exposure pathways, toxicological mechanisms, and the state of human health evidence for the four major obesogenic pollutant categories associated with the petroleum industry

| Petroleum-Derived Pollutant Category | (Primary Exposure Routes | Key Molecular Targets of Obesogenesis | Metabolic Effects of Obesogenesis | Strength of Epidemiological Evidence |
|---|---|---|---|---|
| **PAHs** *(e.g., Benzo[a] pyrene, Naphthalene)* [4, 14] | • Inhalation of ambient air and fuel exhaust<br>• Ingestion of contaminated food/water<br>• Dermal contact | • **AhR** (Aryl Hydrocarbon Receptor) activation [4, 14]<br>• Downstream cross-talk disrupting**PAR$\gamma$** (Peroxisome Proliferator-Activated Receptor gamma) [4, 14]<br>• Inhibition of **AMPK** signaling | • Accelerates adipocyte differentiation and proliferation [4, 14]<br>• Upregulates de novo lipogenesis [4, 14]<br>• Triggers white adipose tissue inflammation and oxidative stress [4, 14, 30–33] | **Moderate to Strong** Extensive cohort data link high PAH exposure (e.g., from urban air or industrial settings) to increased body mass index, waist circumference, and altered lipid profiles in adults and children. |
| **BTEX** *(Benzene, Toluene, Ethylbenzene, Xylenes)* [2, 3, 14] | • Inhalation of volatile vapors (refineries, petrol stations)<br>• Ingestion of contaminated seafood/ groundwater<br>• Minor dermal absorption | • Activation of **PPAR$\gamma$** and **RXR** [14, 33]<br>• Disruption of Glucocorticoid Receptor pathways<br>• Induces **NF-$\kappa$B-mediated** systemic inflammation [32, 33] | • Alters hormonal regulation of appetite and satiety [14, 23, 32, 33]<br>• Promotes ectopic lipid deposition in the liver [14, 32, 33]<br>• Drives systemic insulin resistance and dyslipidemia [2, 3, 14, 23] | **Emerging to Moderate** While traditionally monitored for carcinogenicity and hematotoxicity, recent occupational and community health studies reveal a correlation between chronic BTEX exposure and metabolic syndrome components. |
| **Heavy Metals** *(e.g., Lead, Cadmium, Arsenic, Mercury)* [4, 14, 17] | • Ingestion of contaminated water and local biota/crops<br>• Inhalation of refining particulate matter<br>• Accidental soil ingestion | • **ER$\alpha$/ER$\beta$** estrogen receptor mimics [4, 14, 17]<br>• Epigenetic modifications via DNA **DNMTs**<br>• Disruption of thyroid hormone receptors [14, 18] | • Blocks glucose uptake in peripheral tissues (insulin resistance) [4, 14, 17, 18]<br>• Permanently alters early-life metabolic set-points during development [4, 23]<br>• Promotes fat storage over energy expenditure [45, 46] | **Strong** A robust body of international epidemiological studies (e.g., Korean NHANES, US NHANES) directly associates serum/urinary heavy metal levels with obesity, elevated low-density lipoprotein cholesterol, and metabolic dysfunction. |
| **Alkylphenols** *(e.g., Nonylphenol, Octylphenol from industrial surfactants)* [15] | • Ingestion via contaminated drinking water and aquatic food webs<br>• Dermal contact with petroleum-derived industrial detergents | • Potent **ER$\alpha$/ ER$\beta$** estrogen Receptor agonism (xenoestrogen) [15, 25]<br>• Antagonism of Androgen Receptors<br>• Interferes with hypothalamic appetite signaling circuits [15, 25] | • Induces standard white adipose tissue expansion [15, 25]<br>• Disrupts pancreatic beta-cell insulin secretion [15, 25]<br>• Pre-programs fetal stem cells toward the adipocyte lineage [15, 25] | **Moderate** Well-documented in xenoestrogen-specific research, showing clear, consistent associations between elevated alkylphenol biomarkers and increased fat accumulation/body weight across multiple age cohorts. |

PAH: polycyclic aromatic hydrocarbon; BTEX: Benzene, Toluene, Ethylbenzene, Xylenes; AhR: Aryl Hydrocarbon Receptor; PPAR$\gamma$: Pe-roxisome Proliferator-Activated Receptor gamma; AMPK: AMP-activated protein kinase; RXR: retinoid X receptor; DNMTs: DNA methyl-transferases; ER$\alpha$/Er$\beta$: Estrogen Receptor alpha and Estrogen Receptor beta; NF-$\kappa$B: Nuclear factor kappa-light-chain-enhancer of

activated B cells. Table 1 above details the primary exposure pathways, toxicological mechanisms, and the state of human health evidence for the four major obesogenic pollutant categories associated with the petroleum industry.

## 9. Clinical Impacts and Public Health Implications

The recognition of petroleum industry pollutants as metabolic disruptors requires a comprehensive overhaul of public health frameworks, environmental regulations, and clinical management strategies in Nigeria. The convergence of EDCs, nutritional transitions, and socioeconomic challenges in the Niger Delta creates a unique epidemiological profile. While undernutrition remains a concern in certain rural areas, the region—like much of urbanizing Nigeria—is witnessing a rapid rise in the prevalence of obesity and type 2 diabetes mellitus. The chronic exposure to obesogenic pollutants from the petroleum industry may act as a catalyst, accelerating the development of metabolic syndrome in susceptible individuals. Clinically, this manifests as abdominal obesity, dyslipidemia, elevated fasting blood glucose, and hypertension. Envi-ronmental pollutants disrupt metabolic homeostasis, so traditional weight-management strategies, such as caloric restriction and increased physical activity, may have limited efficacy in populations with a high body burden of EDCs.

Current health metrics in Nigeria underrepresent the metabolic burden within the Niger Delta region because health surveillance continues to focus primarily on infectious diseases and acute toxicities [1–4]. The Federal Ministry of Health, in partnership with state ministries, should establish longitudinal health registries in high-impact oil communities. These programs must move beyond basic anthropometric measurements to include bioelectrical impedance analysis for visceral fat mapping, fasting insulin levels, lipid subfraction profiles, and biomarkers of environmental exposure (such as urinary 1-hydroxypyrene for PAHs and blood heavy-metal levels). Long-term cohort studies modeled after global environmental health initiatives must be funded to definitively track the transgenerational progression of metabolic diseases across affected generations in this Niger Delta region. Clinicians managing obesity in the region must adapt their strategies to account for the environmental etiology of these conditions [54, 55]. Dietary recommendations should emphasize foods rich in natural ligands for protective pathways.

For instance, cruciferous vegetables (such as broccoli, kale, and Brussels sprouts) contain sulforaphane, a potent activator of the nuclear factor erythroid 2-related factor 2 pathway that helps detoxify PAHs and neutralize ROS [54, 55]. Dietary polyphenols like resveratrol and epigallocatechin gallate can help counteract pollutant-induced histone hyperacetylation and suppress excessive PPAR-$\gamma$ activation. Supplementation with high-quality, marine-derived omega-3 fatty acids (EPA/DHA) can also help reduce M1 macrophage polarization in adipose tissue while promoting the anti-inflammatory M2 phenotype, thereby enhancing tissue repair [54, 55].

Given the high prevalence of pollutant-induced dysbiosis, therapeutic protocols should include targeted probiotics (e.g., Lactobacillus and Bifidobacterium strains) alongside prebiotics (e.g., inulin and fruc-to-oligosaccharides) to repair the mucosal barrier, reduce circulating LPS levels, and mitigate metabolic endotoxemia [54, 55]. When lifestyle modifications yield limited results due to deep-seated epigenetic programming, early pharmacological options may be warranted [54, 55]. Medications such as Metformin, which activate the AMP-activated protein kinase pathway and counteract pollutant-induced insulin re-sistance, should be considered. In cases of confirmed heavy metal toxicity, carefully monitored clinical chelation therapies may help remove systemic burdens of lead and cadmium, restoring proper enzymatic and metabolic function [54, 55].

## 10. Conclusion

Shifts toward Western lifestyles and calorie imbalances are no longer the only factors contributing to the increased prevalence of obesity in Nigeria's Niger Delta. According to a substantial body of molecular, cellular, and systemic evidence compiled in this analysis, extensive, long-term oil and gas pollution in the area is a powerful cause of adipogenesis and metabolic disturbance. These environmental contaminants actively alter the human metabolic landscape through mechanisms such as the hyperactivation of the PPAR$\gamma$:RXR transcriptional machinery, induction of chronic tissue inflammation, modification of the gut microbiome, and stable transgenerational epigenetic reprogramming. Future studies must concentrate on determining the precise chemical combinations that provide the greatest metabolic danger to solve this covert public health emergency. In the Niger Delta, epidemiologists must explicitly incorporate environ-mental exposure measures into frameworks for preventing non-communicable diseases. In the end, coor-dinated measures involving environmental engineering, stringent regulatory enforcement, public health planning, and clinically focused therapy approaches will be necessary to reverse this metabolic tendency. Restoring metabolic health and ecological justice to the populations in Nigeria's Niger Delta region can only be achieved by treating the chemical causes of chronic illness.

**Abbreviations**

ACTH - Adrenocorticotropic Hormone
Ahr - Aryl Hydrocarbon Receptor
ATP - Adenosine Triphosphate
BAT - Brown/beige Adipose Tissue
Bap - Benzo[a]pyrene
BLOC - Bonny light crude oil
BTEX - Benzene, Toluene, Ethylbenzene, Xylene
CLS - Crown-like Structure
CRP - C-reactive Protein
EDCs - Endocrine-disrupting Chemicals
HATS - Histone Acetyltransferases
HDACs - Histone Deacetylases
HPA - Hypothalamic-pituitary Adrenal Axis
IL-1$\beta$ - Interleukin 1 Beta
IL-6 - Interleukin 6

JNK - c-Jun N-terminal Kinase
LPS - Lipopolysaccharide
MCP-1/CCL2 - Monocyte Chemoattractant Protein 1
NCDs - Non-communicable diseases
NF-$\kappa$B - Nuclear Factor Kappa-light-chain-enhancer of Activated B Cells
PAHs - Polycyclic Aromatic Hydrocarbons
PPAR-$\gamma$ - Peroxisome Proliferator-activated Receptor-gamma
ROS - Reactive Oxygen Species
RXR - Retinoid X Receptor
TDCs - Thyroid-disrupting Chemicals
TLR4 - CDS/Toll-like Receptor 4
TNF-1$\beta$ - Tumor-necrosis Factor 1 Beta
UPCP1 - Uncoupling Protein 1
UPR - Unfolded Protein Response
VOCs - Volatile Organic Compounds
WAT - White Adipose Tissue

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## References

[1] F. Gbadamosi and J. Aldstadt. The interplay of oil exploitation, environmental degradation, and health in the Niger Delta: A scoping review. *Trop Med Int Health*, 30(5):351–67, 2025. URL https://doi.org/10.1111/tmi.14108.

[2] J. A. Jumbo, B. Ordinioha, P. Mmom, E. Okokon, M. B. Teere, and A. Tamunomiegbam. Environmental and health impacts of crude oil exploration in the Niger Delta. *Journal of Environment*, 3(2):41–62, 2023. URL https://econpapers.repec.org/article/bhxojtjoe/v_3a3_3ay_3a2023_3ai_3a2_3ap_3a41-62_3aid_3a1424.htm.

[3] O. E. Orisakwe. Crude oil and public health issues in the Niger Delta, Nigeria: Much ado about the inevitable. *Environmental research*, 194:110725, 2021. URL https://doi.org/10.1016/j.envres.2021.110725.

[4] F. U. Igwe, A. Dokubo, and C. P. Ukpaka. Overview of crude oil toxicity: a potential health and environmental hazard in the Niger Delta. Niger. *Nigerian Journal of Oil and Gas Technology*, 2(2):94–104, 2016. URL https://rsustnjogat.org/ojs/index.php/home/article/view/65.

[5] J. Nriagu, E. A. Udofia, I. Ekong, and G. Ebuk. Health risks associated with oil pollution in the Niger Delta, Nigeria. *International Journal of Environmental Research and Public Health*, 13(3):346, 2016. URL https://doi.org/10.3390/ijerph13030346.

[6] G. Richard, S. C. Izah, and M. Ibrahim. Air pollution in the Niger Delta region of Nigeria: Sources, health effects, and strategies for mitigation. *Journal of Environmental Studies*, 29(1):1–5, 2023. URL https://doi.org/10.21608/jesj.2023.182647.1037.

[7] A. N. Chuemere, B. L. Dum-awara, and O. Obia. Persistent exposure to toxic oil and gas flaring pollu-tants-mediated insulin resistance and hyperinsulinemia among populations in the Niger Delta. *Intern J Sci Res Updat*, 3(2):120–126, 2022. URL https://doi.org/10.53430/ijsru.2022.3.2.0031.

[8] A. G. Essiet. Prevalence and Determinants of Cardiovascular Diseases in Oil Producing Communities in Niger Delta, Nigeria: A Systematic Review. *Port Harcourt Journal of Public Health*, 1(1):105–114, 2025. URL http://phjph.com/index.php/pjph/article/view/35.

[9] A. U. Idung, F. Abasiubong, S. B. Udoh, and U. S. Ekanem. Overweight and obesity profiles in Niger Delta region, Nigeria. *African journal of primary health care & family medicine*, 6(1):E1–E5, January 2014. URL https://doi.org/10.4102/phcfm.v6i1.542.

[10] C. B. Onigbogi, S. Banerjee, and Z. J. McKinney. Prevalence and risk factors of obesity among workers in the oil and gas industry of the Niger Delta region. *Nigerian Medical Journal*, 63(4):288–294, 2022. URL https://doi.org/10.60787/NMJ-63-4-41.

[11] O. Maduka and J. N. Paul. Is there a relationship between residence in oil-bearing communities and the prevalence of diabetes mellitus? *Journal of Epidemiological Society of Nigeria*, 1:43–7, 2021. URL https://doi.org/10.5281/zenodo.6014879.

[12] J. J. Heindel and B. Blumberg. Environmental obesogens: mechanisms and controversies. *Annual Review of Pharmacology and Toxicology*, 59(1):89–106, 2019. URL https://doi.org/10.1146/annurev-pharmtox-010818-021304.

[13] F. Grün and B. Blumberg. Perturbed nuclear receptor signaling by environmental obesogens as emerging factors in the obesity crisis. *Reviews in Endocrine and Metabolic Disorders*, 8(2):161–71, 2007. URL https://doi.org/10.1007/s11154-007-9049-x.

[14] W. Igoniye, O. I. Horsfall, and F. D. Fyneface. From single pollutants to synergistic threats: A cumulative health risk assessment of multi-media exposure in Niger Delta's oil-producing communities. *International Journal of Modern Science and Research Technology*, 3(9):565–578, 2025. URL https://www.ijmsrt.com/.

[15] C. M. Markey, C. L. Michaelson, C. Sonnenschein, and A. M. Soto. *Alkylphenols and bisphenol A as envi-ronmental estrogens*, pages 129–153. Springer Berlin Heidelberg, Berlin, Heidelberg, 2001.

[16] C. Casals-Casas, J. N. Feige, and B. Desvergne. Interference of pollutants with PPARs: endocrine disruption meets metabolism. *international journal of obesity*, 32(Suppl 6):S53–s61, 2008. URL https://doi.org/10.1038/ijo.2008.207.

[17] B. M. Lolescu, A. V. Furdui-Linţa, C. A. Ilie, A. Sturza, F. Zară, D. M. Muntean, A. Blidişel, and O. M. Creţu. Adipose tissue as target of environmental toxicants: focus on mitochondrial dysfunction and oxidative inflammation in metabolic dysfunction-associated steatotic liver disease. *Molecular and Cellular Biochemistry*, 480:2863–2879, 2025. URL https://doi.org/10.1007/s11010-025-05221-2.

[18] A. Nwafor, F. C. Mmom, O. Obia, C. Obiandu, V. O. Hart, and B. C. Chinko. Relationship between blood pressure, blood glucose and body mass index and coexisting prehypertension and prediabetes among rural adults in Niger Delta Region, Nigeria. *Journal of Advances in Medicine and Medical Research*, 9(7):1–12, 2055. URL https://doi.org/10.9734/BJMMR/2015/14777.

[19] B. Ordinioha and S. Brisibe. The human health implications of crude oil spills in the Niger delta, Nigeria: An interpretation of published studies. *Nigerian Medical Journal*, 54(1):10–6, 2013. URL https://doi.org/10.4103/0300-1652.108887.

[20] J. R. Seckl and B. R. Walker. Minireview: 11$\beta$-hydroxysteroid dehydrogenase type 1- a tissue-specific am-plifier of glucocorticoid action. *Endocrinology*, 142(4):1371–1376, 2001. URL https://doi.org/10.1210/endo.142.4.8114.

[21] C. Formichi, S. Caprio, L. Nigi, and F. Dotta. The impact of environmental pollution on metabolic health and the risk of non-communicable chronic metabolic diseases in humans. *Nutrition, Metabolism and Cardiovascular Diseases*, 35(6):103975, 2025. URL https://doi.org/10.1016/j.numecd.2025.103975.

[22] M. Coelho, T. Oliveira, and R. Fernandes. Biochemistry of adipose tissue: an endocrine organ. *Archives of Medical Science*, 9(2):191–200, 2013. URL https://doi.org/10.5114/aoms.2013.33181.

[23] Y. Cui, Z. Mo, P. Ji, J. Zhong, Z. Li, D. Li, Q. Qin, L. amd Liao, Z. He, W. Guo, L. Chen, Q. Wang, G. Dong, W. Chen, Y. Xiao, and X. Xing. Benzene Exposure Leads to Lipodystrophy and Alters Endocrine Activity In Vivo and In Vitro. *Front Endocrinol (Lausanne)*, 13:937281, 2022. URL https://doi.org/10.3389/fendo.2022.937281.

[24] A. A. Jumbo, M. O. Monanu, and L. C. Chuku. Thyroid Hormone Dysregulation Potential of Gas Flare Ex-posure. *International Journal of Innovative Biochemistry & Microbiology Research*, 13(2):54–61, 2025. URL https://doi.org/10.5281/zenodo.15584730.

[25] M. Capezzone, G. Pellegriti, A. Ronchi, F. Gianì, A. Corsello, and R. M. Paragliola. The Thyroid Under Pressure: Heavy Metals, Endocrine Disruptors and Translational Insights into Carcinogenesis and Thyroid Dysfunctions. *International Journal of Molecular Sciences*, 27(12):5583, 2026. URL https://doi.org/10.3390/ijms27125583.

[26] U. Shahnazaryan, M. Wójcik, T. Bednarczuk, and A. Kuryłowicz. Role of Obesogens in the Pathogenesis of Obesity. *Medicina (Kaunas)*, 55(9):515, 2019. URL https://doi.org/10.3390/medicina55090515.

[27] A. G. Essiet, A. A. Gordon, I. B. Inyang, and S. B. Mkpan. The hidden impact of hydrocarbon pollution on car-dio-metabolic health in Akwa Ibom State. *Faculty of Natural and Applied Sciences Journal of Applied Chemical Science Research*, 2(1):58–68, 2024. URL https://fnasjournals.com/index.php/FNAS-JACSR/article/view/512.

[28] A. W. Oyeyemi, W. M. Owonikoko, T. D. Okoro, O. Adagbonyi, and K. O. Ajeigbe. Water contamination: A culprit of serum heavy metals concentration, oxidative stress and health risk among residents of a Nigerian crude oil-producing community. *Toxicology reports*, 12:375–388, 2024. URL https://doi.org/10.1016/j.toxrep.2024.03.006.

[29] T. B. Odinga, F. U. Igwe, E. Asogwa, and C. U. Gabriel-Brisibe. Impact of gas flaring on the serum antioxidant biomarkers of Eleme residents in Rivers State, Nigeria. *JSM Biochemistry & Molecular Biology*, 6(1):13–20, 2020. URL https://doi.org/10.5281/zenodo.15584730.

[30] A. Bright, F. Li, M. Movahed, H. Shi, and B. Xue. Chronic exposure to low-molecular-weight polycyclic aromatic hydrocarbons promotes lipid accumulation and metabolic inflammation. *Biomolecules*, 13(2):196, 2023. URL https://doi.org/10.3390/biom13020196.

[31] X. Wang, Z. Sun, Q. S. Liu, Q. Zhou, and G. Jiang. Environmental Obesogens and Their Perturbations in Lipid Metabolism. *Environ Health (Wash)*, 2(5):253–268, 2024. URL https://doi.org/10.1021/envhealth.3c00202.

[32] C. F. A. Vogel, L. S. Van Winkle, C. Esser, and T. Haarmann-Stemmann. The aryl hydrocarbon receptor as a target of environmental stressors - Implications for pollution-mediated stress and inflammatory re-sponses. *Redox biology*, 34:101530, 2020. URL https://doi.org/10.1016/j.redox.2020.101530.

[33] J. J. Heindel, S. Howard, K. Agay-Shay, J. P. Arrebola, K. Audouze, P. J. Babin, et al. Obesity II: Establishing causal links between chemical exposures and obesity. *Biochem Pharmacol*, 199:115015, 2022. URL https://doi.org/10.1016/j.bcp.2022.115015.

[34] S. Xu, J. Xi, T. Wu, and Z. Wang. The Role of Adipocyte Endoplasmic Reticulum Stress in Obese Adipose Tissue Dysfunction: A Review. *International Journal of General Medicine*, 16:4405–4418, 2023. URL https://doi.org/10.2147/IJGM.S428482.

[35] K. Ma, Y. Zhang, J. Zhao, L. Zhou, and M. Li. Endoplasmic reticulum stress: bridging inflammation and obesity-associated adipose tissue. *Front Immunol*, 15, 2024. URL https://doi.org/10.3389/fimmu.2024.1381227.

[36] T. Suzuki, J. Gao, Y. Ishigaki, K. Kondo, S. Sawada, T. Izumi, et al. ER Stress Protein CHOP Mediates Insulin Resistance by Modulating Adipose Tissue Macrophage Polarity. *Cell Reports*, 18(8):2045–2057, 2017. URL https://doi.org/10.1016/j.celrep.2017.01.076.

[37] E. U. Alum, H. E. Aloh, D. C. Obasi, P. N. Okoroh, U. C. Aniokete, and A. P. Emeruwa. Maternal Nutrition, Toxi-cants, and Epigenetic Programming of Obesity Across Generations. *Diabetes, Metabolic Syndrome and Obesity*, 18:4873–4911, 2025. URL https://doi.org/10.2147/DMSO.S579409.

[38] J. Stel and J. Legler. The Role of Epigenetics in the Latent Effects of Early Life Exposure to Obesogenic Endocrine Disrupting Chemicals. *Endocrinology*, 156(10):3466–3472, 2015. URL https://doi.org/10.1210/en.2015-1434.

[39] Z. Zhu, F. Cao, and X. Li. Epigenetic Programming and Fetal Metabolic Programming. *Front Endocrinol (Lausanne)*, 10:764, 2019. URL https://doi.org/10.3389/fendo.2019.00764.

[40] T. O. Oni, D. Chiweike, E. C. Destiny, and I. O. Victor. Impact of Heavy Metals and Chemicals on Epigenetic Changes. *International Journal of Interdisciplinary Studies and Innovation*, 1(2):12–15, 2022. URL https://doi.org/10.54117/iimj.v1i2.4.

[41] N. B. Chinedu, E. N. Orhuebor, O. U. Hannah, U. B. Essien, C. E. Ndubuka, I. S. Ayeni, N. P. Otu, E. M. Emmanuel, and Y. Rasheed. Petrochemical pollutants and their toxicological fate in sediment-water systems: a combined analytical and ecoto-xicological study of Oguta Lake and Njaba River, Imo State, Nigeria. *International Journal of Research and Scientific Innovation*, 13(3), 2026. URL https://doi.org/10.51244/IJRSI.2026.1303000242.

[42] C. F. Wami-Amadi. The impact of air-borne toxins from gas flaring on cardiopulmonary and other systemic functions. *Scholars International Journal of Anatomy and Physiology*, 8(1):12–28, 2025. URL https://doi.org/10.36348/sijap.2025.v08i01.003.

[43] O. U. Nze-Dike, K. M. Ezealisiji, A. N. Ezejiofor, A. M. Awanye, and C. S. Dike. Genotoxic and epigenetic con-cerns of black carbon/soot in recent times: a systematic review. *Discover Applied Sciences*, 7(10):1140, 2025. URL https://doi.org/10.1007/s42452-025-06942-1.

[44] E. U. Alum, H. E. Aloh, D. C. Obasi, P. N. Okoroh, U. C. Aniokete, and A. P. Emeruwa. Maternal nutrition, toxi-cants, and epigenetic programming of obesity across generations. *Diabetes, Metabolic Syndrome and Obesity*, pages 4873–911, 2025. URL https://doi.org/10.2147/DMSO.S579409.

[45] O. J. Ugbede. Gut health crisis? Investigating the impact of oil spillage on microbiome-related diseases in the Niger Delta. *Int'l Journal of Education Research and Scientific Development*, 7(2):244–257, 2025. URL https://doi.org/10.59795/ijersd.v7i2.225.

[46] W. Ma, X. Xiong, Z. Tian, L. Li, and Y. Huang. Environmental pollutants and the gut microbiota: mechanistic links from exposure to systemic disease. *Frontiers in microbiology*, 17:1737229, 2026. URL https://doi.org/10.3389/fmicb.2026.1737229.

[47] S. Ghosh, S. P. Nukavarapu, and V. R. Jala. Effects of heavy metals on gut barrier integrity and gut microbiota. *Microbiota Host*, 2(1):e230015, 2024. URL https://doi.org/10.1530/MAH-23-0015.

[48] E. R. Eworo, E. R. Egbe, Z. A. Okhormhe, B. K. Offor, B. I. Uduak, and A. Dauda. Anthropometric indices, lipid profile, and lipopolysaccharide-binding protein levels in metabolic endotoxemia: A case-control study in Calabar Metropolis, Nigeria. *Journal of Acute Disease*, 9(2):67–72, 2020. URL https://doi.org/10.4103/2221-6189.281320.

[49] I. A. Adedara, A. P. Ebokaiwe, P. P. Mathur, and E. O. Farombi. Nigerian bonny light crude oil induces endocrine disruption in male rats. *Drug and Chemical Toxicology*, 37(2):198–203, 2014. URL https://doi.org/10.3109/01480545.2013.834359.

[50] O. Otitoju and I. N. Onwurah. Preliminary investigation into the possible endocrine disrupting activity of Bonny light crude oil contaminated diet on Wistar rats. *Biokemistry*, 19(1):23–28, 2007. URL https://doi.org/10.4314/biokem.v19i1.56419.

[51] M. San Sebastián and A. K. Hurtig. Oil development and health in the Amazon basin of Ecuador: the pop-ular epidemiology process. *Soc Sci Med*, 60(4):799–807, 2005. URL https://doi.org/10.1016/j.socscimed.2004.06.016.

[52] S. E. Allan, B. W. Smith, and K. A. Anderson. Impact of the deepwater horizon oil spill on bioavailable pol-ycyclic aromatic hydrocarbons in gulf of mexico coastal waters. *Environ Sci Technol*, 46(4):2033–9, 2012. doi:10.1021/es202942q.

[53] M. Shamy, M. Alghamdi, M. I. Khoder, A. M. Mohorjy, A. A. Alkhatim, A. K. Alkhalaf, et al. Association between Exposure to Ambient Air Particulates and Metabolic Syndrome Components in a Saudi Arabian Population. *International Journal of Environmental Research and Public Health*, 15(1):27, 2017. URL https://doi.org/10.3390/ijerph15010027.

[54] R. Botchlett, S. L. Woo, M. Liu, Y. Pei, X. Guo, H. Li, and c. Wu. Nutritional approaches for managing obesi-ty-associated metabolic diseases. *Journal of Endocrinology*, 233(3):R145–R171, 2017. URL https://doi.org/10.1530/JOE-16-0580.

[55] B. Hennig, M. C. Petriello, M. V. Gamble, Y. J. Surh, L. A. Kresty, N. Frank, N. Rangkadilok, M. Ruchirawat, and W. A. Suk. The role of nutrition in influencing mechanisms involved in environmentally mediated diseases. *Rev Environ Health*, 33(1):87–97, 2018. URL https://doi.org/10.1515/reveh-2017-0038.