Journal of Public Health and Environmental Research, 2(2):44–51, July-December: 2026.
DOI: https://doi.org/10.58612/jpher225

Journal of Public Health and Environmental Research
ISSN (Online): 3108-3196

Journal homepage: https://www.fpub.org/journal-details.php?journal-id=41

Review Article

Diabetogenic Petroleum Industry-Induced Pollutants in Nigeria's Niger Delta: Laboratory Diagnostic Challenges for Type 2 Diabetes Management

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Article Info

Keywords: *Nigeria, Petroleum, Environmental Pollutants, Environmental Exposure, Type 2 Diabetes Mellitus, Clinical Laboratory Services.*

Received: *09.08.2026;*
Accepted: *20.09.2026;*
Published: *26.09.2026*

Abstract

Background: The Niger Delta region of Nigeria experiences chronic environmental pollution driven by oil and gas extraction. Emerging evidence links these industrial pollutants to endocrine disruption, specifically targeting metabolic pathways that lead to Type 2 Diabetes Mellitus (T2DM). **Objective:** This narrative review synthesizes current knowledge on diabetogenic environmental pollutants from the petroleum sector in the Nigerian Niger Delta and critically evaluates the pre-analytical, analytical, and post-analytical challenges these contaminants impose on T2DM laboratory medicine workflows. **Methods:** A comprehensive literature search was conducted across major electronic databases for peer-reviewed articles published between 2000 and 2026. Keywords focused on heavy metals, polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), oil spills, gas flaring, diabetes mellitus, and clinical laboratory services within the Nigerian context. **Results:** Flaring and oil spills release massive quantities of diabetogenic agents, including heavy PAHs, VOCs, and heavy metals. These pollutants induce metabolic dysfunction through systemic pathways: chronic systemic inflammation, oxidative stress, beta-cell apoptosis, and insulin receptor antagonism. For laboratory medicine workflow, these pollutants create complex diagnostic challenges. They introduce pre-analytical (altering erythropoiesis, hence glycated hemoglobin A1c status), analytical (chemical interferences in assays), and post-analytical (confounding biomarkers of organ failure) challenges to T2DM diagnostics. **Conclusion:** Environmental diabetogenesis represents an escalating public health crisis in Nigeria's Niger Delta. To mitigate this threat, clinical laboratories must evolve. There is an urgent need to standardize diagnostic protocols to account for pollutant interference, establish locally adapted biomarker reference intervals, and integrate environmental toxicological monitoring into routine metabolic disease management.

How To Cite: C. Amadi, I. Nmehielle-Oluwadare, R.C. Amadi, S.C. Amadi, E.C. Amadi and C. John. Diabetogenic Petroleum Industry-Induced Pollutants in Nigeria's Niger Delta: Laboratory Diagnostic Challenges for Type 2 Diabetes Management. *Journal of Public Health and Environmental Research*, 2(2):44–51, 2026. https://doi.org/10.58612/jpher225

1. Introduction

The Niger Delta region of Nigeria, stretching over approximately 70,000 square kilometers of wetlands, mangroves, and creeks, contains one of the highest concentrations of oil and gas extraction infrastructure globally [1, 2]. Since commercial oil exploitation began in the late 1950s, the region has been subjected to relentless environmental degradation [1, 2]. Decades of pipeline sabotage, structural equipment failures, illegal artisanal refining, and systemic gas flaring have introduced vast quantities of xenobiotic compounds into the local ecosystem [2–6].

While the ecological and carcinogenic consequences of this pollution are well documented, its role as a driver of metabolic diseases—specifically Type 2 Diabetes Mellitus (T2DM)—is an emerging crisis in environmental health and clinical toxicology [5–10]. Historically viewed as a disease of lifestyle, urbanization, and genetic predisposition, diabetes mellitus is increasingly understood to have strong environmental determinants [7, 8]. Modern environmental medicine categorizes specific exogenous chemicals that interfere with metabolic homeostasis as "metabolic disruptors" or "diabetogens" [7–15]. In Nigeria's Niger Delta, the population is chronically exposed to a complex mixture of these diabetogenic agents through contaminated drinking water, bioaccumulated dietary sources (such as local seafood), and ambient air pollution driven by unceasing gas flaring [3–6]. This environmental burden directly complicates the clinical landscape of metabolic disease management in the region [3–5]. Laboratory medicine serves as the bedrock of diabetes screening, diagnosis, and therapeutic monitoring [5–10]. However, the unique chemical footprint of petroleum pollution introduces unprecedented challenges to clinical laboratories operating within the Niger Delta.

This narrative review explores the molecular mechanisms linking oil-derived pollutants to T2DM, defines the specific chemical entities driving this epidemic, and critically evaluates the core pre-analytical, analytical, and post-analytical challenges faced by laboratory medicine workflow and professionals in polluted environments.

2. Methodology

This narrative review synthesizes published literature on the diabetogenic effects of Niger Delta oil and gas pollutants, focusing on challenges to laboratory medicine practice. Relevant published articles and reports were systematically searched from relevant literature in databases using PubMed, MedlinePlus, Google Scholar, African Index Medicus, African Journals Online, and Bielefeld Academic Search Engine as the major search engines. The search techniques employed were keywords, Boolean Operators, Phrase Searching, and Field Searching. The search terms fed into the search engines were, but not restricted to, combinations of Medical Subject Headings (MeSH) terms and free-text keywords focused on three primary areas, namely:

1) Pollutants profile: "Niger Delta", "petroleum pollution", "oil spills", "gas flaring", "heavy metals", "polycyclic aromatic hydrocarbons", "heavy metals", "volatile organic compounds".
2) Pathophysiological mechanisms of pollutant-induced diabetes mellitus: "type 2 diabetes mellitus", "insulin resistance", "environmental pollutants", "chemical-induced diabetes mellitus", "endocrine disruptors", "oxidative stress".
3) Diagnostic/clinical challenges within regional clinical laboratories: "clinical laboratory services", "diagnostic services", "diagnostic errors", "health service accessibility".

Included were peer-reviewed original research, toxicological reports, and epidemiological studies detailing local environmental contamination and endocrine-disrupting chemicals. Articles were screened for relevance to the Niger Delta eco-zone published between January 2000 and May 2026. Non-English articles and studies lacking specific data on oil and gas pollutants were excluded. Data were synthesized qualitatively to frame environmental toxicity from a clinical pathology perspective. Abstracts and titles were first screened for relevance, followed by retrieval of full articles when available. Additional articles were identified through reference lists of the selected articles to enhance the comprehensiveness of the search. All authors searched for relevant articles, performed independent verification, and selected articles for appropriateness.

3. Landscape of Environmental Pollution in The Niger Delta

The environmental degradation of the Niger Delta is unique in its geographic scope and chronicity. The primary vectors of pollution are categorized into three major domains: petroleum spills, gas flaring, and artisanal refining activities [1–4]. Thousands of oil spills occur annually in the Niger Delta, releasing millions of barrels of crude oil into mangrove swamps, agricultural soils, and freshwater systems. Crude oil is a highly complex matrix consisting of thousands of distinct chemical entities, primarily classified into aliphatic and aromatic hydrocarbons [1–3]. Once released into the environment, these compounds undergo "weathering"—a process involving evaporation, dissolution, and photo-oxidation that often concentrates the more persistent, bioaccumulative, and toxic fractions, such as high-molecular-weight Polycyclic Aromatic Hydrocarbons (PAHs) [1–4]. Nigeria remains one of the world's top contributors to gas flaring, a practice in which natural gas associated with petroleum extraction is burned openly into the atmosphere [1–3]. This incomplete combustion releases a continuous plume of toxic gases and particulates into the troposphere. The flare stacks, often located near residential communities, generate a hazardous mixture of carbon monoxide, sulfur dioxide, nitrogen oxides, volatile organic compounds (VOCs), and fine particulate matter (PM2.5 and PM10) coated with adsorbed trace metals and organic toxins [1–4].

In the past two decades, illegal local refining of stolen crude oil has emerged as a dominant environmental threat, particularly in Rivers, Bayelsa, and Delta states [1–3]. These crude, uncontrolled thermal distillation setups discharge massive volumes of unrefined residues directly into surrounding soils and rivers [1–3]. More visibly, they have blanketed major urban centers like Port Harcourt with a persistent layer of airborne black soot (particulate carbon matter) [1, 2]. This soot acts as a structural vehicle, carrying concentrated mixtures of heavy metals and PAHs deep into the respiratory systems of exposed populations, from where they translocate into the general systemic circulation [1–4].

4. Molecular Architecture of Diabetogenic Pollutants

The primary chemical agents driving the environmental etiology of T2DM in the Niger Delta can be subclassified into three major classes: PAHs, VOCs, and toxic metals [11–15]. PAHs contain two or more fused benzene rings [11–13]. Principal representatives found in high

concentrations in Niger Delta soils and seafood include benzo [a] pyrene (BaP), phenanthrene, anthracene, and fluoranthene [11–13]. Because they are lipophilic, PAHs accumulate efficiently in adipose tissues, bypassing normal cellular elimination pathways and exerting long-term toxic effects on insulin-sensitive organs [12, 13]. The monoaromatic hydrocarbons Benzene, Toluene, Ethylbenzene, and Xylene (BTEX) are highly volatile liquids ubiquitous around oil flow stations, gas flare sites, and petrol dispensing stations. Inhalation represents the primary route of exposure [12]. Once inside the body, BTEX compounds undergo hepatic biotransformation via the Cytochrome P450 enzyme system (predominantly CYP2E1), yielding highly reactive intermediates capable of inducing systemic oxidative stress and metabolic derangements [12]. Petroleum operations, combined with the disturbance of geological strata, lead to the release of trace metals into the environment. Heavy metals such as arsenic, cadmium, lead, and mercury are widely dispersed in the soils, drinking water, and agricultural products of the Niger Delta [13–15]. Arsenic is frequently found in groundwater in oil-producing regions; chronic exposure to arsenic is heavily linked to the destruction of pancreatic beta-cells and the impairment of insulin secretion [13–15]. Cadmium accumulates in the human body, particularly in the kidneys and liver, and has been shown to interfere with insulin biosynthesis and promote insulin resistance by inducing oxidative stress [13–15]. Lead, when elevated in blood, is associated with systemic inflammation and altered glucose metabolism, contributing to a higher risk of metabolic syndrome, a precursor to T2DM [13–15].

5. Mechanisms of Pollution-Induced Diabetogenesis

The molecular pathogenesis of pollutant-induced T2DM involves a multifaceted network of cellular pathways [16–27]. These run parallel to, and frequently accelerate, classical lifestyle-driven metabolic pathways [16–27]. The unifying mechanism underlying the diabetogenicity of PAHs, VOCs, and heavy metals is the generation of excessive Reactive Oxygen Species (ROS) alongside the depletion of endogenous antioxidant defenses [16–20]. Pancreatic β-cells are highly vulnerable to oxidative injury due to their unusually low expression of key antioxidant enzymes, including superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx) [16]. When heavy metals like cadmium or lead accumulate in pancreatic tissue, they bind directly to the sulfhydryl (-SH) groups of mitochondrial membrane proteins [16–20]. This impairs the mitochondrial electron transport chain (ETC), specifically inhibiting Complexes I and III. The resulting electron leakage drives intracellular ROS generation, which triggers the opening of the mitochondrial permeability transition pore (mPTP). This releases cytochrome c into the cytosol, activating caspase-3 and caspase-7 enzymatic cascades, and ultimately causing apoptosis of pancreatic β-cells. The loss of functional β-cell mass directly leads to impaired insulin synthesis and defective glucose-stimulated insulin secretion (GSIS) [16–20].

Lipophilic pollutants, particularly benzo [a] pyrene (Bap) and related PAHs, act as potent ligands for the Aryl Hydrocarbon Receptor (AhR), a cytosolic transcription factor involved in xenobiotic metabolism [21]. Upon ligand binding, the AhR translocates to the nucleus, heterodimerizes with the AhR Nuclear Translocator (ARNT), and binds to Xenobiotic Response Elements (XRE) on DNA [21]. Crucially, ARNT is identical to Hypoxia-Inducible Factor 1-beta (HIF-1β), a factor vital for maintaining β-cell glucose sensing and insulin gene transcription. Chronic overactivation of the AhR pathway by environmental PAHs sequesters ARNT, depriving the cell of the machinery needed for normal HIF-1β-mediated transcription [18–26]. This transcriptional interference deregulates glucose transporter-2 (GLUT2) and glucokinase expression, desensitizing β-cells to ambient blood glucose concentrations [21]. Additionally, AhR activation drives the expression of inflammatory cytokines like tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), promoting chronic, low-grade systemic inflammation that worsens metabolic health [22]. In peripheral tissues like skeletal muscle, liver, and white adipose tissue, chemical pollutants disrupt the canonical insulin signaling cascade [23–26]. Under normal physiological conditions, insulin binding to its receptor triggers tyrosine phosphorylation of Insulin Receptor Substrate-1 (IRS-1) [23]. This recruits Phosphoinositide 3-kinase (PI3K) and activates Protein Kinase B (Akt), driving the translocation of glucose transporter-4 (GLUT4) vesicles to the plasma membrane to facilitate glucose uptake [23]. Xenobiotics like arsenic, cadmium, lead, VOCs (e.g., benzene metabolites), and PAHs disrupt this pathway by activating stress-responsive serine/threonine kinases [16–20, 24–26]. These include c-Jun N-terminal kinase (JNK), inhibitor of nuclear factor-κ B kinase subunit β (IKKβ), and p38 Mitogen-Activated Protein Kinase (MAPK). These kinases induce inhibitory serine phosphorylation (e.g., Ser307) of IRS-1, blocking downstream PI3K/Akt activation. Consequently, GLUT4 vesicles remain trapped in the intracellular compartment, causing profound insulin resistance. In the liver, this loss of insulin signaling prevents the inactivation of glycogen synthase kinase-3 (GSK-3) and downregulates FoxO1 phosphorylation. This accelerates unchecked gluconeogenesis and glycogenolysis, worsening fasting hyperglycemia [16–20, 24–26]. Table 1 below Summarizes each diabetogenic pollutant class with its representative xenobiotic, primary target, mechanisms, and clinical endpoint.

Table 1: Summary of each diabetogenic pollutant class with their representative xenobiotic, primary target, mechanisms, and clinical endpoint

Pollutant Class	Representative Xenobiotic	Primary Cellular Target	Molecular Mechanisms	Clinical Endpoints
PAHs	Benzo [a] pyrene	Pancreatic β-cells, Adipose tissue [18, 25, 26]	AhR activation; ARNT sequestration; CYP1A1/ 1B1 overexpression; TNF-α production [18, 25, 26]	β-cell desensitization, systemic insulin resistance [18, 25, 26]
VOCs	Benzene, Toluene	Hepatic parenchyma, Skeletal muscle [19, 20, 25]	CYP2E1 biotransformation; ROS generation; JNK kinase activation [19, 20, 25]	Inhibitory serine phosphorylation of IRS-1; blunted GLUT4 translocation [19, 20, 25]
Heavy Metals/ Metalloids	Lead, Cadmium, Arsenic	Mitochondria, Pancreatic Islets, Hepatic enzymes [17, 18, 24]	-SH group binding; Complexes I/III inhibition; mPTP opening; Caspase-3/7 activation [17, 18, 24]	β-cell apoptosis; defective glucose-stimulated insulin secretion [17, 18, 24]

6. Epidemiological Realities in the Niger Delta

Epidemiological profiles in Nigeria's Niger Delta show a clear correlation between environmental oil and gas pollution and metabolic diseases [27–30]. Multiple cross-sectional and cohort studies across oil-bearing communities in Rivers, Delta, Bayelsa, and Akwa Ibom states reveal a significantly higher prevalence of impaired fasting glucose, metabolic syndrome, and overt T2DM among residents living near gas flare stacks and oil spill sites than in unexposed control populations in non-oil-producing regions [29]. Biomonitoring studies frequently show elevated concentrations of blood lead, urinary cadmium, and hydroxylated PAH metabolites (such as 1-hydroxypyrene) in local populations. These elevated body burdens correlate with higher homeostatic model assessment of insulin resistance values, elevated glycosylated hemoglobin (HbA1c), and an atherogenic lipid triad (high triglycerides, low high-density lipoprotein cholesterol, and elevated small, dense low-density lipoprotein cholesterol particles) [5]. Notably, these metabolic abnormalities are common even among individuals with normal body mass index [30]. This indicates that toxin-induced diabetes in this region bypasses classical obesity-dependent pathways, presenting instead as a form of lean or atypical diabetes driven by environmental cytotoxicity [30].

7. Challenges to Laboratory Medicine Diagnostic Workflows

The pervasive presence of environmental contaminants in the Niger Delta compromises the entire workflow of clinical laboratory medicine, presenting major challenges across the pre-analytical, analytical, and post-analytical laboratory phases [31–45]. Figure 1 summarizes how diabetogenic environmental petroleum pollutants within Nigeria's Niger Delta region impact T2DM-related laboratory diagnostic workflow.

7.1. Pre-Analytical Challenges

The pre-analytical phase remains the most error-prone component of laboratory medicine, a vulnerability worsened by the conditions in the Niger Delta. Transporting clinical specimens from remote riverine communities (e.g., communities in Southern Ijaw or Bonny Island) to centralized tertiary laboratory facilities in urban centers requires traversing difficult aquatic terrains [34]. Due to the lack of cold-chain infrastructure and widespread regional grid failures, specimens are frequently exposed to high ambient temperatures for extended periods. This accelerates *in vitro* glycolysis in unseparated whole blood tubes, causing falsely low plasma glucose readings unless sodium fluoride/potassium oxalate tubes are used properly. Furthermore, high ambient concentrations of volatile hydrocarbons can diffuse through standard plastic blood collection tubes, altering sample matrix integrity before analysis [35]. Patients living in these zones carry high baseline body burdens of environmental toxicants. Chronic exposure to heavy metals like lead and cadmium induces subclinical hemolysis and alters erythropoiesis in bone marrow. This shortens red blood cell lifespan, causing a falsely lower HbA1c measurement relative to a patient's true long-term glycemic state. Consequently, using HbA1c as a standalone diagnostic tool can lead to significant underdiagnosis or underestimation of diabetes severity in these communities [36].

7.2. Analytical Challenges

The analytical integrity of modern clinical chemistry instruments relies on stable optical paths, precise antibody-antigen recognition, and predictable enzymatic kinetics. The presence of oil and gas pollutants directly undermines these processes. Automated clinical chemistry analyzers measure plasma glucose using enzymatic reactions, primarily the glucose oxidase-peroxidase (GOD-POD) method or the hexokinase method [37]. Both of these methods rely on spectrophotometric absorbance changes at specific wavelengths (e.g., 505 nm for GOD-POD) [37]. Circulating volatile hydrocarbons and lipid-soluble components of crude oil can alter plasma turbidity [38].

This creates unpredictable light scattering and false absorbance readings, which can mimic or mask the chromogens generated during glucose quantification, leading to inaccurate results [38]. The hexokinase method for glucose testing requires divalent magnesium (Mg^{2+}) as a cofactor for enzymatic phosphorylation [37]. Heavy metals like lead (Pb^{2+}) and cadmium (Cd^{2+}) can displace Mg^{2+} from the active site of the hexokinase enzyme due to their higher coordinate binding affinities [39, 40]. This causes artificial enzyme inhibition within the assay system, leading to falsely depressed glucose measurements. Similarly, arsenic can bind to the sulfhydryl groups of reagent enzymes used in lipid and metabolic panels, impairing assay kinetics and reducing diagnostic accuracy [39, 40]. Quantifying endogenous metabolic hormones, such as insulin, C-peptide, and glucagon, relies on immunoassay platforms like Enzyme-Linked Immunosorbent Assay or Chemiluminescent Immunoassay. Circulating PAHs and heavy metals can bind directly to clinical analytes or to the hypervariable regions of diagnostic monoclonal antibodies used in these assays. This steric hindrance prevents proper antigen-antibody binding, leading to variable analytical errors. Depending on whether a competitive or sandwich immunoassay design is used, this interference can cause either falsely elevated or falsely suppressed hormone measurements, misrepresenting a patient's true pancreatic reserve.

7.3. Post-Analytical Challenges

In the post-analytical phase, pathologists face the challenge of interpreting data using inadequate reference frameworks. Most clinical laboratories in Nigeria use reference intervals copied from Western textbooks or manufacturer package inserts [1]. These reference values are derived from populations living in unpolluted environments with entirely different baseline chemical exposures. In the Niger Delta, a "normal" population sample likely carries measurable concentrations of blood lead, cadmium, and PAHs [2]. If reference intervals are established locally without filtering out individuals with high toxicant burdens, the baseline reference ranges for fasting plasma glucose, insulin, liver enzymes, and inflammatory biomarkers will be shifted upward [1, 2]. This shifts the baseline and normalizes subclinical metabolic disease, delaying clinical intervention until advanced, irreversible tissue damage has occurred. Diagnosing diabetic nephropathy and hepatosteatosis relies on monitoring serum creatinine, urea, and the transaminases. However, heavy metals and petroleum solvents are directly nephrotoxic and hepatotoxic [42, 43]. When a clinician observes elevated serum creatinine or microalbuminuria in a diabetic patient from the Niger Delta, it is difficult to determine whether these markers indicate classical diabetic microvascular complications or direct toxicant-induced tubulointerstitial nephritis [42, 43]. This diagnostic ambiguity complicates clinical staging, risk stratification, and therapeutic management [42, 43].

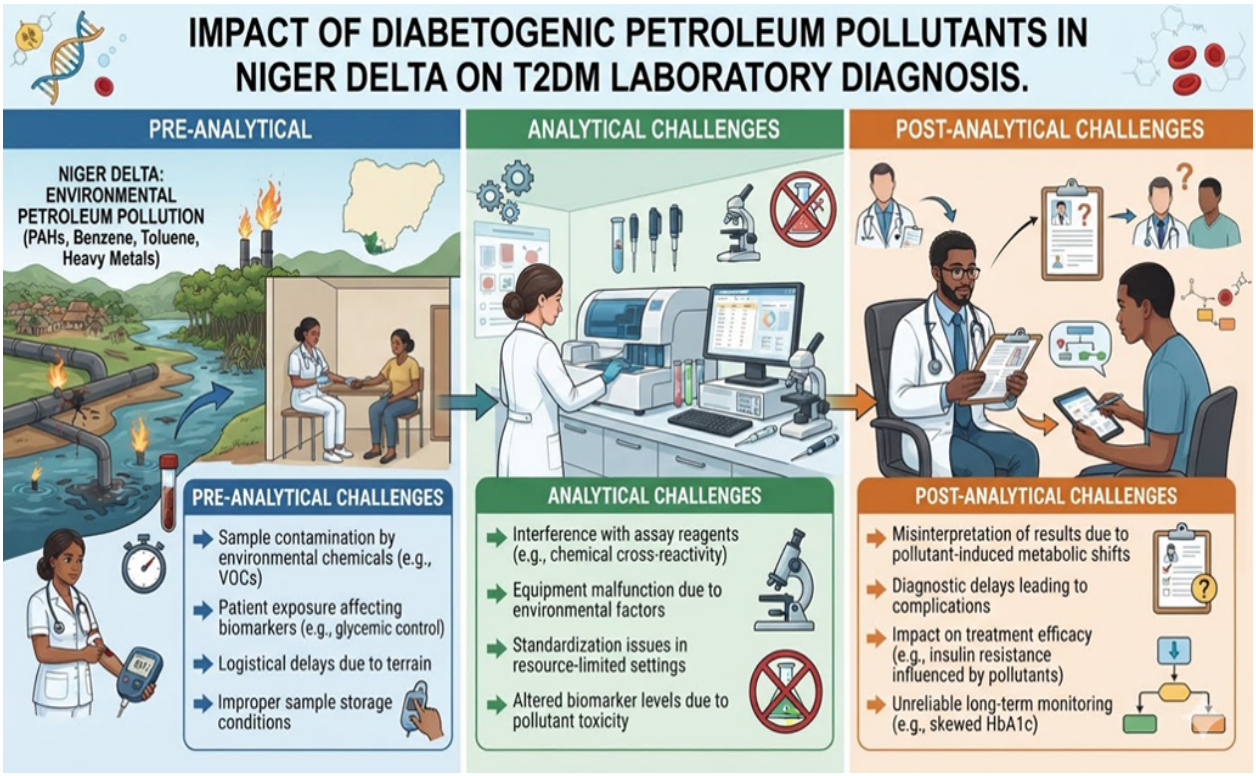

Figure 1: Image summarizing how diabetogenic environmental petroleum pollutants within Nigeria's Niger Delta region impact T2DM-related laboratory diagnostic workflow.

8. Strategic Solutions and Future Directions

Addressing the metabolic health crisis in the Niger Delta requires adapting clinical laboratory infrastructure to handle environmental toxicity, rather than relying solely on standard diagnostic models [44, 45]. Clinical laboratories in the region should transition to an integrated diagnostic framework that combines metabolic monitoring with environmental biomonitoring. Patients presenting with new-onset, atypical, or lean T2DM should be routinely screened using an Environmental Diabetogenesis Panel. This specialized testing panel should include Inductively Coupled Plasma Mass Spectrometry to quantify whole blood lead, urinary cadmium, and total arsenic; High-Performance Liquid Chromatography coupled with fluorescence detection to measure urinary 1-hydroxypyrene (a validated biomarker of PAH exposure), and Routine quantification of high-sensitivity C-reactive protein and free fatty acids (to trace the downstream inflammatory effects of xenobiotic exposure. The Pathology subspecialties across Nigerian tertiary institutions must lead initiatives to establish rigorous, locally adjusted reference intervals. This process requires selecting reference individuals from the local population who are confirmed to have toxicant levels below established thresholds. Filtering reference cohorts by environmental exposure levels ensures that reference intervals accurately reflect health, allowing for the early detection of metabolic deviations. To minimize analytical interference from hydrocarbons and trace metals, laboratories should invest in analytical platforms less prone to matrix disruptions. For example, using tandem mass spectrometry for steroid, hormone, and metabolic profiling bypasses the antibody-binding interferences common to automated immunoassays. Additionally, laboratories must implement strict matrix validation protocols. This includes conducting recovery studies by spiking patient samples with known quantities of glucose and insulin in the presence of varying concentrations of crude oil fractions, ensuring the accuracy and resilience of routine testing methods.

Given the logistical challenges of sample transport across riverine terrains, validating and deploying rugged, quality-controlled point-of-care testing devices is essential. Utilizing advanced biosensor-based point-of-care testing devices that employ direct electrochemical detection (amperometry) rather than optical spectrophotometry can bypass colorimetric interferences caused by environmental turbidity or soot particles. These devices should be supported by decentralized quality assurance networks linked to tertiary reference hubs.

9. Conclusion

The extensive oil and gas industry engagements in Nigeria's Niger Delta have created a challenging public health environment, where ecotoxicity and chronic metabolic diseases are deeply linked. Ubiquitous environmental pollutants—including PAHs, BTEX, and toxic heavy metals—act through complex molecular pathways to drive pancreatic β-cell death and systemic insulin resistance, accelerating a unique regional epidemic of environmentally induced T2DM. This metabolic crisis presents significant challenges across all phases of laboratory medicine. Resolving these diagnostic complexities requires systemic changes. The clinical laboratory can no longer operate in isolation from the surrounding ecosystem. There is an urgent need for Nigerian medical institutions, environmental regulatory bodies, and multinational energy corporations to invest in upgrading laboratory medicine infrastructure. By implementing advanced molecular biomonitoring, establishing localized toxicant-filtered reference intervals, and adopting analytical methods resilient to chemical interference, the laboratory medicine community can provide the accurate diagnostic data needed to combat this metabolic threat and safeguard the health of the Niger Delta.

Abbreviations

Ahr	Aryl Hydrocarbon Receptor
BaP	Benzo [a] pyrene
BTEX	Benzene, Toluene, Ethylbenzene, and Xylene
GLUT2	Glucose Transporter 1
GLUT4	Glucose Transporter 4
GOD-POD	Glucose Oxidase-Peroxidase
GPx	Glutathione Peroxidase
HbA1c	Glycated Hemoglobin A1c
IL-6	Interleukin Six
JNK	c-Jun N-terminal Kinase
Mptp	Mitochondrial Permeability Transition Pore
PAHs	Polycyclic Aromatic Hydrocarbon
POCT	Point-of-care Testing
ROC	Reactive Oxygen Species
SOD	Superoxide Dismutase
T2DM	Type 2 Diabetes Mellitus
TNF-α	Tumor Necrosis Factor-alpha
VOCs	Volatile Organic Acids

Article Information

Funding: No external funding was received for this study.

Author Contributions: C.A. - Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision; I.N. - Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision; R.C.A. - Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision; S.C.A. - Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision; E.C.A. - Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision; C.J. - Conceptualization, Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision.

Ethical Approval: Not applicable.

Informed Consent: Not applicable.

Conflict of Interest: The authors declare no competing interests.

Data Availability Statement: Not applicable.

Disclaimer (Artificial Intelligence): The author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.), and text-to-image generators have been used during writing or editing of manuscripts.

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