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Urban Air Pollution and Oxidative Stress Among Commercial Bus Drivers in Benin Metropolis

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Abstract- Background
Commercial bus drivers in urban Nigeria experience prolonged exposure to vehicular emissions and traffic-related air pollution, which can induce oxidative stress and elevate the risk of cardiovascular and respiratory diseases.
Objective
To assess air pollution exposure levels and oxidative stress biomarkers among commercial bus drivers in Benin Metropolis, using office workers as a comparison group.
Methods
A comparative cross-sectional study was conducted among 200 male participant comprising 100 commercial bus drivers and 100 office workers in Benin City, Edo State, between March and May 2024. Ambient air quality (PM_{2.5} and NO_2$) was measured across four major motor parks. Blood samples were analyzed for Malondialdehyde (MDA), Superoxide Dismutase (SOD), Glutathione (GSH), and C-reactive protein (CRP). Additionally, respiratory symptoms and blood pressure were evaluated.
Results
The mean $PM_{2.5}concentration at the motor parks was 78.4 ± 12.6 µg/m³, which is 3.1 times higher than the World Health Organization limit. Compared to the control group, bus drivers exhibited significantly higher levels of MDA (4.87 ± 1.11 vs. 3.02 ± 0.89 µmol/L) and CRP, alongside significantly lower levels of SOD and GSH (p < 0.001). Furthermore, 42% of drivers reported chronic cough, and 28% presented with pre-hypertension. Duration of driving (years) and daily hours spent on the road emerged as significant predictors of oxidative stress.
Conclusion
Commercial bus drivers in Benin Metropolis suffer from heightened oxidative stress driven by heavy urban air pollution. Targeted occupational health interventions and stricter air quality regulations are urgently required.

Keywords - Air Pollution; PM_{2.5}; Oxidative Stress; Commercial Drivers; Benin City; Occupational Health

I. INTRODUCTION

Urban air pollution is a critical global environmental risk factor, responsible for an estimated 4.2 million deaths annually (WHO, 2021). In low- and middle-income countries like Nigeria, rapid urbanization, an aging vehicular fleet, poor fuel quality, and severe traffic congestion have collectively exacerbated air quality deterioration.

Benin Metropolis, the capital of Edo State, accommodates over 500,000 registered vehicles alongside intense commercial transport operations. Commercial bus drivers routinely spend 8 to 14 hours daily immersed in heavy traffic, directly inhaling complex mixtures of exhaust fumes, particulate matter (PM{2.5} and $PM{10}), nitrogen dioxide, and polycyclic aromatic hydrocarbons. Vehicular emissions serve as a major source of reactive oxygen species (ROS). Chronic inhalation of these pollutants triggers oxidative stress—a pathological imbalance between ROS production and endogenous antioxidant defenses, such as superoxide dismutase (SOD) and glutathione (GSH) (Kelly, 2003). Persistent oxidative stress is a well-established precursor to hypertension, atherosclerosis, asthma, and lung cancer.

Despite these severe health implications, empirical data regarding occupational exposure and its biological sequelae among commercial drivers in South-South Nigeria remain sparse. Therefore, this study aims to assess ambient air pollution levels and biomarkers of oxidative stress among commercial bus drivers in Benin Metropolis.

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II. LITERATURE REVIEW: MECHANISMS OF AIR POLLUTION-INDUCED OXIDATIVE STRESS

Mechanisms of Oxidative Stress from Air Pollution

Over recent decades, urban air pollution driven by vehicular traffic and combustion processes has become a major public health concern. Particulate matter (PM) and gaseous pollutants are recognized as primary urban air hazards, with multiple mechanisms proposed to explain their adverse cardiopulmonary effects (Alavanidis et al., 2005). Although individual pollutants possess distinct toxicological profiles, agents such as ozone, nitrogen oxides, and suspended particulates share a common property: they are potent oxidants. They inflict damage either directly on lipids and proteins or indirectly by activating intracellular oxidant pathways (Boldo, Linares, & Lumbreras, 2011).

Reactive oxygen species (ROS) can be generated directly from particle surfaces carrying adsorbed polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs, and transition metals (such as iron, copper, chromium, and vanadium). These metals catalyze Fenton-type reactions (Fe^{2+} + H_2O_2 + H^+ \rightarrow Fe^{3+} + OH^\bullet + H_2O$), producing highly reactive hydroxyl radicals capable of inducing oxidative DNA damage (Alavanidis et al., 2011). Furthermore, particle-bound benzo(a)pyrene is bioavailable and induces DNA damage in systemic target organs, including the lungs and kidneys.

In ambient air, ozone and nitrogen dioxide frequently coexist with particulates, compounding the risk of oxidative DNA damage. Volatile organic compounds (VOCs) such as benzene also contribute to DNA oxidation (Bell, Peng, Dominici, & Samet, 2009). Additionally, secondary photochemical oxidants like ozone and peroxyacetyl nitrate, formed through sunlight-driven reactions involving hydrocarbons and NOx, further elevate oxidative stress (Boldo et al., 2011).

When ROS formation surges, it triggers mitochondrial damage, activates NADPH-oxidase isoform 4 (NOX4), and stimulates inflammatory cells (neutrophils, eosinophils, and monocytes), increasing macrophages capable of producing ROS and reactive nitrogen species (Bell et al., 2009).

- Phase 1 (Adaptive Response): Under low levels of oxidative stress, transcription factors like nuclear factor erythroid-2 related factor 2 (Nrf2) upregulate antioxidant and detoxification enzymes (e.g., catalase, superoxide dismutase, and glutathione S-transferase) to neutralize ROS and protect cells.
- Phase 2 (Pro-inflammatory Response): When antioxidant defenses are overwhelmed by excessive ROS production, a pro-inflammatory state ensues, accompanied by cytotoxicity. These pathways are mediated by redox-sensitive mitogen-activated protein kinase (MAPK) and NF-\kappaB cascades, which drive the expression of pro-inflammatory cytokines, chemokines, and adhesion molecules (Boldo et al., 2011).

Contributions of Atmospheric Gaseous Pollutants

Gaseous pollutants, largely originating from fossil fuel combustion and motor vehicle emissions, significantly alter atmospheric composition.

- Ozone (O_3): Formed in the troposphere via photochemical reactions between sunlight, nitrogen dioxide, and hydrocarbons, ozone acts as a powerful oxidizing agent. It initiates intracellular oxidative stress through ozonide and hydroperoxide formation, subsequently activating Nrf2, heat shock protein 70, $NF-\kappaB$, and oncogenes like c-Fos and c-Jun, while upregulating pro-inflammatory cytokines (TNF-\alpha, $IL-1\beta, and IL-8$).
- Nitrogen Dioxide (NO_2$): Primarily emitted via fossil fuel combustion from stationary and mobile sources, nitric oxide is rapidly converted into nitrogen dioxide by atmospheric oxidants like ozone (Kosmider, Loader, Murphy, & Mason, 2010). While less potent than ozone, NO_2 also activates oxidant pathways (Sprague & Khalil, 2009).
- Volatile Organic Compounds (VOCs) and Carbon Monoxide (CO): Inhaled VOCs and gases significantly impact cardiopulmonary health. Carbon monoxide is strongly linked to

cardiovascular disease development, whereas benzene induces hematological disorders and carcinogenesis (Sprague & Khalil, 2009). Benzene toxicity stems from its metabolic conversion into reactive metabolites like hydroquinone and benzoquinone, which undergo redox cycling to generate ROS. Occupational benzene exposure has been shown to induce oxidative stress, immunosuppression, and expression of the tumor suppressor gene p53$ in workers (Hayes & McMahon, 2008).

Body fluids utilize endogenous antioxidants—such as ascorbic acid, uric acid, and thiols—to scavenge O_3 and $NO_2 radicals, protecting airway lining fluids (Kosmider et al., 2010). However, once these defenses are overwhelmed, O_3 and NO_2 provoke lipid peroxidation and inflammatory gene expression (Bell et al., 2009).

Oxidative Stress from the Organic Fraction of Particulate Matter

Ambient particulate matter comprises complex mixtures derived from industrial emissions, incinerators, traffic, construction, and windblown dust. Evidence strongly indicates that fine and ultrafine particles (UFPs) are more hazardous to cardiovascular and respiratory health than larger particles due to their high surface area and composition (Amara, Bachoual, & Desmard, 2007).

The oxidative potential of fine particles is largely driven by organic carbon compounds, including quinones and PAHs, which arise from incomplete combustion. These include substituted and unsubstituted PAHs, nitro-PAHs, dinitro-PAHs, and peroxyacetyl nitrate (Amara et al., 2007).

- Metabolic Activation: PAHs undergo metabolic activation primarily via cytochrome P450 (CYP450) enzymes to form diol epoxides, radical cations, and redox-active quinones that cause DNA adduct formation.
- Synergistic Action: Redox-active transition metals, quinoids, and PAHs act synergistically to produce robust ROS generation (Uzma et al., 2010). Diesel exhaust particles (DEPs), consisting of a carbon core with adsorbed organics and redox-active metals, induce CYP1A1 expression, drive lipid peroxidation, and trigger oxidant-dependent $NF-\kappaB$ activation, initiating inflammatory damage.

Transition Metal-Mediated Oxidative Stress

Transition metals including iron, lead, mercury, cadmium, nickel, vanadium, chromium, manganese, and copper are frequently found on the surface of PM {2.5}$ and UFPs, where they catalyze ROS generation via Fenton reactions (Andersson et al., 2009). These metals originate from natural crustal sources as well as anthropogenic activities like combustion and industrial manufacturing. Notably, iron acts as a soot suppressant and a major element in metallic brake pads. Zinc is commonly derived from waste engine oil in traffic emissions, while copper is a major constituent of ceramic brake pads (10%–20% by mass). When soluble metals (e.g., Fe, Ni, V, Cu, Cr) are inhaled and deposited on airway epithelial cells, they provoke substantial cellular oxidative stress (Andersson et al., 2009).

Target Organ Damage: Cardiovascular and Pulmonary Systems

Cardiovascular System

Because of their minute size and expansive surface area, ultrafine particles penetrate deeply into the pulmonary interstitium and systemic circulation (Jaeschke, 2011). UFPs can reach the cardiac vasculature, triggering arrhythmias, reducing myocyte contractility, and impairing coronary blood flow.

- Fine particulate matter and ozone cause acute arterial vasoconstriction in healthy individuals, while long-term $PM_{2.5}$ exposure elevates blood pressure and fosters atherosclerosis (Wold et al., 2006; Schnelle-Kreis et al., 2009).
- Animal models demonstrate that particulate exposure accelerates atherosclerotic lesion progression, increases macrophage infiltration, and induces mitochondrial DNA damage via NADPH oxidase-dependent pathways (Møller & Loft, 2010). Furthermore, traffic-related pollutants have been associated with altered cardiac repolarization (prolonged QT interval) in vulnerable populations, such as diabetic, obese,

and elderly non-smokers (Kliucininkas, Martuzevicius, & Krugly, 2011).

Pulmonary System

A strong correlation exists between the concentration of redox-active compounds in PM and cellular damage in alveolar macrophages and bronchial epithelial cells (Kliucininkas et al., 2011). Organic compounds adsorbed onto particles promote inflammation through CYP1A1-mediated ROS generation and MAPK/$NF-\kappaB$ signaling pathways.

- Nitro-PAHs (such as 1-nitropyrene) induce DNA damage in human endothelial cells via ROS production (Andersson et al., 2011).
- Pro-inflammatory cytokines ($TNF-\alpha$ and $IL-8$) released in the lungs stimulate the migration of neutrophils and precursors into systemic circulation, causing acute tissue damage and prompting systemic organ-repair responses (Kliucininkas et al., 2011).

III. MATERIALS AND METHODS

Study Design and Setting

This study utilized a comparative cross-sectional design conducted in Benin Metropolis, Edo State, Nigeria. The exposed group was recruited from four major commercial motor parks (New Benin, Ekiosa, Central Park, and Uselu), while the control group was selected from four designated government office complexes within the metropolis to ensure minimal occupational exposure to traffic-related air pollution.

Study Population and Eligibility

The study population comprised 200 healthy adult males aged 20 to 55 years, divided equally into two cohorts:

Exposed Group: 100 commercial bus drivers with a minimum of two years of driving experience.

Control Group: 100 indoor office workers with minimal outdoor exposure.

Exclusion Criteria: Individuals were excluded if they were current smokers, had a pre-existing clinical diagnosis of diabetes mellitus or cancer, or were actively taking antioxidant vitamin supplements.

Data Collection Procedures

Data collection encompassed four main components:

- Questionnaires: An interviewer-administered questionnaire was used to gather socio-demographic data, occupational history, and respiratory symptoms, adapting the standardized American Thoracic Society (ATS) respiratory questionnaire.
- Air Quality Monitoring: Ambient concentrations of $PM_{2.5}$ and NO_2 were measured at the motor parks over an 8-hour daily period across a two-week duration using calibrated portable air quality monitors.
- Clinical Evaluations: Standardized measurements were recorded for blood pressure, body mass index (BMI), and Peak Expiratory Flow Rate (PEFR).
- Laboratory Analyses: A 5 mL venous blood sample was collected from each participant. Serum levels of Malondialdehyde (MDA) were determined via the thiobarbituric acid reactive substances (TBARS) method. Superoxide Dismutase (SOD) and Glutathione (GSH) were quantified using spectrophotometry, C-reactive protein (CRP) was measured via ELISA, and blood lead levels were analyzed using atomic absorption spectrophotometry (AAS).

Statistical Analysis and Ethical Considerations

Quantitative data were analyzed using SPSS version 26. Group differences were evaluated using independent t-tests and Chi-square tests, while relationships between variables were assessed through Pearson correlation coefficients and multiple logistic regression models. Statistical significance was set at $p < 0.05$.

Ethical approval for the study was granted by the Health Research Ethics Committee of the University of Benin Teaching Hospital (UBTH), and written informed consent was obtained from all participants prior to enrollment.

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IV. ANALYSIS

1 Air Quality

Table 1: Ambient Air Pollutant Levels at Motor Parks

Pollutant	Mean ± SD	WHO 24hr Guideline	Times Above Limit
PM2.5	78.4 ± 12.6	25	3.1x
NO2	45.2 ± 8.9	25	1.8x

3.2 Participant Characteristics

Mean age of drivers: 38.2 ± 6.7 years. Mean driving experience: 9.4 ± 4.2 years. Mean daily hours: 11.2 ± 2.1 hrs.

Biomarkers of Oxidative Stress

Table 2: Comparison of Oxidative Stress and Inflammatory

Biomarker	Drivers	Controls	p-value
MDA	4.87 ± 1.11	3.02 ± 0.89	<0.001
SOD	142.3 ± 28.4	198.7 ± 31.2	<0.001
GSH	18.6 ± 4.2	26.9 ± 5.1	<0.001
CRP	5.4 ± 2.1	2.8 ± 1.3	<0.001
Blood Lead	12.4 ± 3.8	5.9 ± 2.2	<0.001

Health Outcomes

Chronic cough: 42% vs 14%. Wheezing: 31% vs 9%. Pre-hypertension: 28% vs 12%. All p<0.01

V. RESULTS.

Correlation Analysis

Pearson correlation analysis revealed a strong positive relationship between duration of driving and serum Malondialdehyde levels ($r = 0.61, p < 0.001$), alongside a significant negative correlation with Superoxide Dismutase activity ($r = -0.54, p < 0.001$). Furthermore, multiple logistic regression identified daily hours spent on the road as an independent predictor of high oxidative stress ($AOR = 3.42, 95\% CI: 1.87-6.25$).

VI. DISCUSSION

This study demonstrates that commercial bus drivers in Benin Metropolis experience exposure to $PM_{2.5}$ concentrations exceeding World Health Organization guidelines by threefold, resulting in pronounced oxidative stress.

The elevated serum MDA levels alongside reduced SOD and GSH concentrations reflect heightened lipid peroxidation coupled with the depletion of systemic antioxidant defenses due to inhaled pollutants. Additionally, elevated CRP and blood lead levels point toward systemic inflammation and toxic metal exposure, likely stemming from historical leaded fuel residues, ambient traffic emissions, and vehicular brake wear (Kelly, 2003).

The 42% prevalence of chronic cough observed among the drivers aligns with findings from urban centers like Lagos and Accra, emphasizing the heavy respiratory burden borne by transport workers (Akinyemi 2018). The positive correlation with years of driving further confirms a dose-response relationship, indicating that cumulative occupational exposure progressively impairs biological defense mechanisms.

Public Health Implications

Commercial drivers function as a critical "sentinel population" for urban air pollution. Because they spend extensive hours immersed in traffic, their biological profiles reflect the broader environmental health hazards confronting the entire urban populace.

Limitations

While robust in its biochemical and clinical findings, this study has limitations. Its cross-sectional design restricts causal inferences, and personal exposure tracking was not utilized in place of ambient motor park monitoring.

VII. CONCLUSION AND RECOMMENDATIONS

Conclusion

Commercial bus drivers in Benin Metropolis suffer from elevated oxidative stress, systemic inflammation, and respiratory symptoms driven by heavy traffic-related air pollution. Protecting this vulnerable workforce is an essential step toward achieving sustainable urban health.

Recommendations

- Policy Interventions: The Federal Road Safety Corps (FRSC) and Vehicle Inspection Officers (VIO) should rigorously enforce vehicular emission standards and implement policies to phase out commercial vehicles older than 15 years.
- Occupational Health Services: Transport unions and health authorities should establish mandatory annual health screenings for drivers, incorporating blood pressure evaluations, PEFR assessments, and oxidative stress panels.
- Environmental Strategies: City planners should expand efficient public transport systems to reduce vehicle congestion and install green vegetative barriers (roadside trees) around major motor parks.
- Individual Measures: Drivers should be encouraged to utilize appropriate particulate face masks, minimize vehicle idling times, and incorporate dietary antioxidants to mitigate personal exposure risks.

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