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Evaluation of Cadmium, Cortisol, and C-Reactive Protein in Male Smokers in Onitsha, Anambra

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Abstract

Background: Tobacco smoking remains a leading cause of morbidity and mortality worldwide, with cadmium exposure, stress, and inflammation being key biological pathways involved in smoking-related diseases. This study evaluated blood levels of cadmium, serum cortisol, and C-reactive protein (CRP) in male smokers in Onitsha, Anambra State, Nigeria.

Methods: A total of 180 male participants aged 18 years and above were recruited, comprising 105 smokers and 75 non-smokers. Data on demographics and smoking history (including number of cigarettes smoked per day) were obtained using structured questionnaires. Venous blood samples were collected and analyzed for blood cadmium, serum cortisol, and CRP using atomic absorption spectrophotometry and enzyme-linked immunosorbent assay (ELISA) methods. Parameters were compared between smokers and non-smokers, as well as among smokers stratified by age and quantity smoked. Correlation and regression analyses were used to explore relationships between cadmium and the other biomarkers.

Results: Mean blood cadmium levels were significantly higher in smokers (3.82 ± 1.14 µg/L) compared to non-smokers (1.27 ± 0.64 µg/L; $p = 0.001$). Similarly, mean cortisol levels were elevated in smokers (21.6 ± 5.3 µg/dL) versus non-smokers (14.3 ± 4.2 µg/dL; $p = 0.0001$), as was CRP (4.85 ± 1.67 mg/L in smokers vs. 2.01 ± 1.08 mg/L in non-smokers; $p = 0.002$). Among smokers, older age groups (>45 years) and those smoking more than 10 cigarettes per day showed significantly higher levels of all three parameters ($p < 0.05$). Cadmium was positively correlated with cortisol ($r = 0.61$, $p = 0.0001$) and CRP ($r = 0.54$, $p = 0.0001$). In multiple regression analysis, cadmium was a significant predictor of both cortisol ($\beta = 0.58$, $p = 0.0001$) and CRP ($\beta = 0.49$, $p = 0.0001$), after adjusting for age and quantity smoked.

Conclusion: This study demonstrates that male smokers in Onitsha have significantly elevated blood levels of cadmium, serum cortisol, and CRP, with cadmium showing strong associations with stress and inflammatory biomarkers. These findings emphasize the systemic toxicological burden of smoking and the need for targeted public health interventions in this population.

Keywords: Cadmium, Cortisol, C-reactive protein, Cigarette smoking, Inflammation, Stress marker.

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1. Introduction

Cigarette smoking continues to be a major contributor to global disease burden, accounting for millions of preventable deaths each year. Current estimates suggest that more than eight million people die annually as a result of tobacco use, with the majority linked to direct consumption and a significant proportion to second-hand exposure (World Health Organization, 2021).

Cigarette smoke contains numerous toxic substances, including heavy metals such as cadmium, which readily enter the bloodstream during inhalation and accumulate in body tissues. The health risks of smoking are multifactorial, but one important pathway involves exposure to toxic metals such as cadmium. Tobacco plants absorb cadmium from the soil, and this heavy metal is transferred to smokers through inhalation. Because cadmium has a biological half-life of several decades, it accumulates in human tissues and has been implicated in

endocrine disruption (Obi-Ezeani *et al*., 2018), kidney damage, oxidative stress (Obi-Ezeani *et al*., 2020), bone demineralization, and cardiovascular dysfunction (Agency for Toxic Substances and Disease Registry [ATSDR], 2012).

In addition to toxic metal exposure, smoking influences neuroendocrine and inflammatory systems. Nicotine stimulates the hypothalamic-pituitary-adrenal axis, leading to increased cortisol secretion, the body's primary stress hormone. Sustained elevations in cortisol have been reported to be associated with hypertension, metabolic disturbances, and impaired immune regulation (Mazgelytė and Karčiauskaitė, 2024). Smoking also promotes systemic inflammation, often reflected in higher concentrations of C-reactive protein (CRP), an acute-phase protein produced by the liver. CRP is widely recognized as a biomarker of cardiovascular risk and chronic inflammatory states (Chia and Ang 2025).

Despite the high prevalence of cigarette smoking among adult males in Nigeria, there is limited local evidence examining the combined effects of cadmium exposure, stress, and inflammation in this population. Onitsha, a densely populated commercial city in Anambra State, presents environmental and lifestyle conditions that may further increase toxic exposures and health risks among smokers. However, data on serum cadmium levels and their relationship with cortisol and CRP among male smokers in this region are scarce. The lack of such data limits effective clinical assessment and the development of targeted public health interventions aimed at reducing smoking-related disease burden.

This study was therefore designed to evaluate blood levels of cadmium, cortisol, and C-reactive protein among male smokers in Onitsha, Anambra State, and to assess the relationship between cadmium exposure and biomarkers of stress and inflammation.

2. Materials and Methods

2.1 Study Design and Setting

This was a cross-sectional analytical study conducted in Onitsha, a major commercial and urban centre located in Anambra State, southeastern Nigeria.

2.2 Study Participants

This study comprised a total of 180 male participants aged 18 years and above, recruited from Onitsha, Anambra State, Nigeria. The participants were divided into two groups: 105 smokers and 75 non-smokers, who served as the control group. Smokers were defined as individuals who reported regular cigarette smoking for at least one year prior to the study, while non-smokers were males who had never smoked cigarettes or had ceased smoking for more than five years.

2.3 Data Collection

Participants were recruited using a convenience sampling approach from the general population. Information on socio-demographic characteristics and smoking history, including duration of smoking and average number of cigarettes smoked per day, was obtained using structured questionnaires. Smokers were further stratified based on age and quantity of cigarettes smoked per day (<10 and ≥10 cigarettes/day).

2.4 Inclusion/Exclusion Criteria

Individuals with a history of chronic inflammatory diseases, endocrine disorders, liver or kidney disease, acute infections, or those on medications known to affect cortisol levels or inflammatory markers were excluded from the study. Participants with occupational exposure to heavy metals were also excluded to minimize potential confounding effects.

2.5 Sample Collection

Samples for cadmium analysis were collected into ethylenediaminetetraacetic acid (EDTA) tubes, while samples for cortisol and CRP analyses were collected into plain tubes. The EDTA samples were gently mixed and stored appropriately until analysis, whereas blood collected in plain tubes was allowed to clot and subsequently centrifuged to obtain serum. Serum samples were aliquoted and stored at –20°C until analysis.

2.6 Biochemical Analysis

Blood cadmium concentrations were determined using atomic absorption spectrophotometry (AAS), serum cortisol levels were measured using enzyme-linked immunosorbent assay (ELISA) kits following the manufacturers' protocols, and serum CRP concentrations were assessed using high-sensitivity ELISA kits.

2.7 Statistical Analysis

Data were analyzed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation. Independent t-test was used to compare biomarker levels between smokers and non-smokers, age groups, and smoking intensity categories. Pearson correlation was performed to evaluate associations between cadmium and cortisol/CRP. Multiple linear regression was conducted to determine whether cadmium independently predicted cortisol and CRP levels after adjusting for age and smoking intensity. A p-value <0.05 was considered statistically significant.

2.8 Ethical Consideration

Ethical approval was obtained from the Health Research Ethics Committee of the School of Medical Laboratory Technicians, Iyi-Enu, Anambra State. Before data collection, written informed consent was obtained from all participants. They were fully briefed on the study's objectives, procedures, possible risks, and anticipated benefits. Participation was voluntary, with assurances of confidentiality and anonymity. No identifying information was recorded, and participants were reminded of their right to withdraw at any stage without penalty.

3. Results

Table 1 shows no significant difference in the mean age of smokers compared with non-smokers ($p>0.05$). Smokers, however, exhibited significantly higher blood levels of cadmium, cortisol, and CRP than non-smokers ($p<0.05$).

Table 1. Age and Biomarker Levels of Smokers and Non-Smokers

Parameter	Smokers (n = 105)	Non-smokers (n = 75)	p-value
Age (years)	39.8 ± 11.2	37.4 ± 10.6	0.18
Cadmium (µg/L)	3.82 ± 1.14	1.27 ± 0.64	0.001*
Cortisol (µg/dL)	21.6 ± 5.3	14.3 ± 4.2	0.0001*
CRP (mg/L)	4.85 ± 1.67	2.01 ± 1.08	0.002*

* Significant

Table 2 indicates that smokers aged over 45 years had significantly higher concentrations of cadmium, cortisol, and CRP compared with those aged 45 years and below. Similarly, individuals who smoked ten or more cigarettes per day exhibited significantly elevated levels of all three biomarkers compared with those who smoked fewer than ten cigarettes daily ($p < 0.05$).

Table 2: Blood Cadmium, Serum Cortisol, and C-Reactive Protein Levels according to Age and Smoking Intensity in Smokers

Group	Cadmium (µg/L)	p-value	Cortisol (µg/dL)	p-value	CRP (mg/L)	p-value
Age ≤ 45 years (n=62)	3.41 ± 0.92		19.8 ± 4.7		4.21 ± 1.42	
Age > 45 years (n=43)	4.29 ± 1.21	0.003*	23.5 ± 5.6	0.001*	5.47 ± 1.73	0.002*
<10 cigarettes/day (n=61)	3.51 ± 1.01		20.2 ± 4.9		4.32 ± 1.51	
≥10 cigarettes/day (n=44)	4.21 ± 1.18	0.005	23.1 ± 5.4	0.002	5.39 ± 1.69	0.004

* Significant

In Table 3, blood cadmium demonstrated significant positive correlations with serum cortisol and CRP ($p = 0.0001$).

Table 3: Correlation between Cadmium and Other Biomarkers

Variable Pair	Correlation Coefficient (r)	p-value
Cadmium vs. Cortisol	0.61	0.0001*
Cadmium vs. CRP	0.54	0.0001*

* Significant

Table 4 shows that after adjusting for potential confounders, including age and quantity of cigarettes smoked per day, blood cadmium remained a significant independent predictor of both serum cortisol and CRP ($p = 0.0001$).

Table 4: Multiple Regression Analysis of Cadmium as Predictor of Cortisol and CRP

Dependent Variable	Standardized Coefficient (β)	p-value
Cortisol	0.58	0.0001*
CRP	0.49	0.0001*

* Significant

4. Discussion

The findings of this study demonstrate that male smokers in Onitsha have significantly elevated levels of blood cadmium, serum cortisol, and CRP compared to non-smokers. These results reinforce the well-established notion that tobacco smoke is a major source of toxic metal exposure and a potent driver of both stress and systemic inflammation.

The elevated cadmium levels observed in smokers in this study are consistent with existing evidence that tobacco smoking is a significant source of cadmium exposure in humans. Tobacco plants accumulate cadmium from soil and fertilizers, and this metal is delivered directly to the bloodstream during inhalation of cigarette smoke, resulting in higher cadmium body burdens among smokers compared to non-smokers. Earlier studies have reported similar findings; for example, Richter and colleagues found that serum cadmium concentrations and cadmium/zinc ratios were significantly higher in smokers than in non-smokers, suggesting increased toxicological risk associated with smoking-related cadmium exposure (Richter *et al*., 2017). Cadmium has also been detected at varying concentrations in cigarettes sold within the country, indicating that local tobacco products contribute to cadmium intake among users (Nnorom *et al*., 2020). These observations align with global research demonstrating that current smokers consistently exhibit elevated cadmium biomarker levels relative to never smokers, with both duration and intensity of smoking influencing cadmium accumulation (Hecht *et al*., 2016; Obi-Ezeani *et al*. 2021). Together, this body of evidence reinforces the interpretation that cigarette smoking is a major determinant of cadmium exposure in Nigerian populations and underscores the toxicological burden of smoking-related heavy metal accumulation.

The significantly higher cortisol levels observed among smokers in this study likely reflect activation of the hypothalamic-pituitary-adrenal (HPA) axis in response to both nicotine and cadmium exposure. Elevated cortisol is well-recognized for its role in the development of hypertension, metabolic disturbances, and impaired immune function. Local evidence supports the sensitivity of cortisol as a biomarker of physiological stress. Ezeugwunne *et al*. (2019) reported increased cortisol levels among HIV/AIDS patients, while Obi-Ezeani *et al*. (2025) documented altered cortisol concentrations in pregnant women. These findings indicate that cortisol reliably reflects stress responses across diverse Nigerian populations. Our results extend this evidence by demonstrating that cadmium exposure from smoking is a

significant predictor of cortisol elevation, highlighting a critical intersection between toxicological burden and endocrine dysregulation in smokers.

Similarly, the elevated CRP levels observed among smokers in this study are consistent with evidence identifying CRP as a sensitive marker of systemic inflammation and a predictor of cardiovascular risk (Amezcua-Castillo *et al*., 2023). Onuora *et al*. (2017) also reported increased cardiovascular risk factors likely associated with elevated CRP among obese individuals in Awka, Anambra State, highlighting the role of inflammation in cardiometabolic disease. The co-occurrence of smoking-induced cadmium exposure and elevated CRP suggests a synergistic effect, potentially amplifying cardiovascular risk in populations with high smoking prevalence. These findings underscore the importance of monitoring inflammatory biomarkers alongside toxic metal exposure to better understand and mitigate the health impacts of smoking.

A distinct pattern emerged in this study when biomarker levels were examined in relation to age and smoking intensity. Older smokers consistently showed higher concentrations of cadmium, cortisol, and CRP compared to younger smokers, suggesting that cumulative exposure over time amplifies toxicological and physiological burden (Obi-Ezeani *et al*., 2025). Similarly, individuals who consumed ten or more cigarettes daily exhibited significantly greater elevations in these biomarkers than lighter smokers. This dose–response relationship highlights the dual impact of both age and quantity of cigarettes smoked, suggesting that cadmium accumulation increases with prolonged exposure, cortisol secretion rises with sustained stimulation of the hypothalamic–pituitary–adrenal axis, and CRP levels reflect heightened inflammatory activity. These findings reinforce the idea that smoking-related harm is not uniform but escalates with longer duration and greater intensity of use, underscoring the importance of early cessation efforts and targeted interventions for heavy and older smokers.

The strong positive correlations observed between cadmium and both cortisol and CRP suggest that cadmium toxicity may serve as a central mechanism linking smoking to neuroendocrine dysregulation and inflammatory burden. This is consistent with global evidence indicating that smoking contributes to oxidative stress and cardiovascular risk through multiple pathways, including toxic metal accumulation, endocrine disruption, and inflammatory activation (ATSDR, 2012; WHO, 2021).

Multiple regression analysis further reinforced these observations, demonstrating that cadmium significantly predicts both cortisol and CRP after adjusting for age and smoking intensity. This suggests that cadmium contributes to stress and inflammatory burden beyond the effects of demographic and behavioral factors, just like other toxic metals, which are linked to diverse adverse health effects (Obi-Ezeani *et al*., 2019; Obi-Ezeani *et al*., 2020; Okpoba *et al*., 2020).

5. Conclusion and Recommendations

This study demonstrates that male smokers in Onitsha, Anambra State, have significantly elevated blood cadmium, serum cortisol, and C-reactive protein levels compared to non-smokers. Cadmium was strongly associated with both cortisol and CRP, highlighting its central role in linking smoking to endocrine dysregulation and systemic inflammation. These findings provide clear evidence that smoking imposes a multifaceted toxicological burden, simultaneously affecting heavy metal accumulation, stress response, and inflammatory pathways.

To mitigate the toxicological, endocrine, and inflammatory effects of smoking, public health efforts should prioritize targeted smoking cessation programs and awareness campaigns. Routine monitoring of cadmium, cortisol, and C-reactive protein in high-risk populations can enable early detection of physiological changes. Lifestyle interventions, including antioxidant-rich diets, and regulatory measures to limit cadmium content in tobacco products, are also recommended.

Conflict of Interest

The authors declare no conflict of interest.

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