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Mitigating Insulin Resistance in Taxi-Motorbike Drivers in Cotonou: Insights from Vitamin B12 and Lifestyle Factors

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Abstract

Background: Taxi-motorbike drivers (TMDs) in Cotonou are occupationally exposed to high levels of air pollution, which may contribute to metabolic dysfunction, including insulin resistance (IR). This study evaluated the associations between vitamin B12 levels, lifestyle factors, and IR in this high-risk population. **Methods:** A cross-sectional study was conducted among 139 TMDs in Cotonou, Benin. Fasting blood samples were collected to measure glucose, insulin, and vitamin B12 levels. Insulin resistance was assessed using HOMA-IR (>2.9). Participants were stratified by vitamin B12 quartiles, and logistic regression identified factors associated with IR. **Results:** The median age was 39.6 years (IQR: 34.0 - 44.0), and the median BMI was 22.8 kg/m^2 (IQR: 20.8 - 25.8). IR prevalence was 67.6% (94/139), with a median HOMA-IR of 3.6 (IQR: 2.6 - 6.1). Vitamin B12 levels were adequate (>221 pmol/L) in 90.6% of participants. Participants in the third quartile (381 - 482 pM) had significantly lower glucose (4.1 nmol/L, $p = 0.013$), insulin (14.7 µM/mL, $p = 0.014$), and HOMA-IR (2.6, $p = 0.009$) compared to lower quartiles. Logistic regression identified BMI (OR = 1.28, 95% CI: 1.11 - 1.48, $p = 0.001$) and alcohol use (OR = 2.41, 95% CI: 1.02 - 5.72, $p = 0.046$) as risk factors for IR, while vitamin B12 levels between 381 - 482 pM were protective (OR = 0.19, 95% CI: 0.06 - 0.59, $p = 0.004$). **Conclusions:** This study reveals a high prevalence of IR among TMDs in Cotonou, with BMI and alcohol consumption identified as key modifiable risk factors. Notably, vitamin B12 levels within the 381 - 482 pM range, a moderate concentration exceeding standard deficiency thresholds, demonstrated a protective association against IR, potentially reflecting optimal bioavailability for mitigating oxidative stress and epigenetic dysregulation linked to air pollution exposure. Implementing tailored interventions to address nutritional and lifestyle factors, such as targeted vitamin B12 supplementation for TMDs with subopti-

mal levels (<381 pmol/L) and structured weight management programs, could reduce metabolic risks in this occupationally high-risk population.

Keywords

Cotonou, HOMA-IR, Insulin Resistance, Vitamin B12, Taxi-Motorbike Drivers

1. Introduction

Taxi-motorbike drivers (TMDs) in Cotonou, Benin, represent a critical occupational group exposed to high levels of ambient air pollution, including polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs) such as benzene, toluene, and xylene [1] [2]. These pollutants, primarily emitted from vehicle exhaust and industrial processes, are well-documented for their role in inducing oxidative stress, systemic inflammation, and DNA damage [2] [3]. Accumulating evidence demonstrates that chronic exposure to air pollutants disrupts central mechanisms underlying type 2 diabetes pathogenesis, including dysregulation of glucose homeostasis and impairment of insulin signaling pathways [4]-[8]. The association between air pollution and insulin resistance (IR) is mediated through multiple pathophysiological pathways, with oxidative stress emerging as the primary mechanistic driver. Oxidative stress induced by exposure to fine particulate matter (PM2.5) and VOCs may activate the Nrf2/NF-κB pathway, a central regulator of antioxidant and inflammatory responses [9]. Specifically, pollutant-mediated Nrf2 activation can trigger the c-Jun N-terminal kinase (JNK) signaling pathway, which phosphorylates and inhibits the insulin receptor substrate-1 (IRS-1)/AKT pathway—a critical axis for insulin signaling [9] [10]. This cascade disrupts AKT-mediated glucose transporter-4 translocation to the cell membrane and glucose intracellular transport, thereby promoting insulin resistance [10] [11]. These pathways collectively underscore the metabolic risks posed by chronic pollutant exposure in TMDs.

Previous studies in Cotonou have demonstrated that TMDs exhibit elevated biomarkers of oxidative DNA damage (e.g., DNA strand breaks, DNA adducts) and inflammation, alongside a high prevalence of hyperuricemia and cardiometabolic risk factors [2] [3]. TMDs face compounded risks characterized by intersecting social, environmental, and biological vulnerabilities—including irregular dietary habits, limited healthcare access, and chronic exposure to traffic-derived air pollution. These factors cumulatively amplify oxidative stress, systemic inflammation, and metabolic dysregulation, driving elevated risks of noncommunicable diseases (NCDs) such as cardiovascular disorders and diabetes mellitus.

Vitamin B12 (cobalamin), an essential micronutrient involved in DNA synthesis, methylation processes, and mitochondrial energy production, has emerged as a potential modulator of metabolic health [12]. Deficiencies in vitamin B12 are

associated with hyperhomocysteinemia, impaired fatty acid metabolism, and IR [13]-[15]. Within Benin, vitamin B12 deficiency prevalence in the general population remains poorly characterized, with no nationally representative data currently available. A regional study conducted in coastal areas of Togo and Benin reported a prevalence of 4.3% (12/276) [16], though this estimate may not account for geographic or socioeconomic disparities within regions.

Despite the growing evidence linking air pollution to metabolic disorders, the role of modifiable factors such as vitamin B12 in mitigating IR has not been investigated in TMDs.

Additionally, while lifestyle factors such as alcohol consumption and obesity are known to influence metabolic health, the interplay between environmental exposures, nutritional status, and these lifestyle factors in shaping metabolic outcomes remains a critical gap in the literature. Understanding these interactions is essential for developing targeted interventions to mitigate metabolic risks in this high-risk occupational group.

This study aimed to evaluate the associations between vitamin B12 levels, lifestyle factors, and IR in TMDs in Cotonou, Benin. Using a cross-sectional design, we assessed the independent contributions of vitamin B12, alcohol consumption, BMI, and other demographic factors to the risk of IR.

2. Methods

2.1. Study Design and Population

The study design and participant selection have been extensively described in previous publications [3] [17]. Briefly, this cross-sectional study included apparently healthy taxi-motorbike drivers (TMDs) in Cotonou, Benin. Participants were all male, aged >18 years, and had been actively working as drivers for at least two years. Exclusion criteria included smoking, a history of diabetes, or cardiovascular diseases (CVDs). Fasting blood samples were collected from each participant in EDTA tubes for biochemical analysis. This study utilized existing data collected during a campaign in May 2010. Participants were included if they had measurements of glucose, insulin, and vitamin B12 levels available. A total of 139 participants met the inclusion criteria and were included in the analysis. The study protocol was reviewed and approved by the Benin Environmental Agency, and all participants provided informed consent.

2.2. Data Collection and Laboratory Analysis

Clinical and metabolic variables of interest included age, duration of occupational exposure, alcohol consumption, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting glucose, fasting insulin, and vitamin B12 levels. Blood collection and processing methods have been described previously [3] [17]. Plasma insulin and vitamin B12 levels were quantified using radioimmunoassay techniques. All laboratory analyses were conducted in Nancy, France, at the Research Unit NGERE ("Nutrition-Génétique-Exposition

aux Risques Environnementaux") using validated methods.

2.3. Definition of Variables

Hypertension was defined as SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg [18]. Vitamin B12 status was categorized according to World Health Organization (WHO) guidelines: levels > 221 pmol/L were considered "vitamin B12 adequacy," levels between 148 and 221 pmol/L were classified as "low B12," and levels < 148 pmol/L were defined as "B12 deficiency" [19] [20]. Insulin resistance (IR) was assessed using the homeostatic model assessment-insulin resistance (HOMA-IR), calculated as: HOMA-IR = fasting plasma insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5. A HOMA-IR value >2.9 was used to define insulin resistance, consistent with established literature [10] [21].

2.4. Statistical Analysis

Data were analyzed using SPSS version 20.0 (IBM Corp., USA). Continuous variables were expressed as medians and interquartile ranges (IQR), while categorical variables were expressed as frequencies and percentages. Participants were stratified into quartiles based on vitamin B12 levels, and differences in metabolic parameters across quartiles were assessed using the Kruskal-Wallis test for continuous variables and the chi-square test for categorical variables. Logistic regression analysis was used to evaluate the independent associations of vitamin B12 levels, BMI, alcohol use, and other covariates with insulin resistance (HOMA-IR > 2.9). Adjusted odds ratios (OR) and 95% confidence intervals (CI) were calculated, and a p-value < 0.05 was considered statistically significant. Covariates included in the multivariate model were selected based on their clinical relevance and univariate associations with the outcome.

3. Results

3.1. Demographic and Metabolic Characteristics of the Study Participants

Table 1. Demographic and metabolic characteristics of the study participants.

Parameters	Median (IQR)
Age (years)	39.6 (34.0 - 44.0)
BMI (Kg/m^2)	22.8 (20.8 - 25.8)
Exposure length, years	10.0 (7.0 - 16.0)
Glucose (nmol/L)	4.2 (3.8 - 4.5)
Insulin (μM/mL)	20.0 (14.2 - 32.7)
HOMA-IR	3.6 (2.6 - 6.1)
Vitamin B12 (pmol/L)	380.0 (313 - 482.0)
	n/N (%)
Alcohol use	56/139 (40.3)

Continued

HTA	65/139 (46.8)
Insulin resistance	96/139 (69.1)
Vitamin B12 status	n/N (%)
Adequate levels (>221 pmol/L)	126/139 (90.6)
Low levels (148 - 221 pmol/L)	12/139 (8.6)
Deficiency (<148 pmol/L)	1/139 (0.7)

BMI: Body Mass Index; IR: Insulin Resistance; IQR: Interquartile Range; HOMA-IR: Homeostatic Model Assessment-Insulin Resistance; HTA: Hypertension. Values are presented as medians with IQR unless otherwise specified.

The study population of 139 participants had a median age of 39.6 years (IQR: 34.0 - 44.0) and a median BMI of 22.8 kg/m^2 (IQR: 20.8 - 25.8), with a median occupational exposure duration of 10.0 years (IQR: 7.0 - 16.0) (Table 1). Metabolic parameters included median fasting glucose of 4.2 nmol/L (IQR: 3.8 - 4.5), insulin of 20.0 µM/mL (IQR: 14.2 - 32.7), and HOMA-IR of 3.6 (IQR: 2.6 - 6.1), indicating a high prevalence of insulin resistance (69.1%, 96/142). The median vitamin B12 level was 380.0 pmol/L (IQR: 313 - 482.0), with the majority of participants (90.6%, 126/139) having adequate vitamin B12 levels (>221 pmol/L), while 8.6% (12/139) had low levels (148 - 221 pmol/L), and only 0.7% (1/139) were deficient (<148 pmol/L). About 40.3% (56/139) of participants reported alcohol use, and 46.8% (65/139) had hypertension.

3.2. Demographic and Metabolic Parameters Stratified by Vitamin B12 Quartiles

Table 2. Demographic and metabolic parameters stratified by vitamin B12 quartiles.

Parameters	Vitamin B12 quartiles				*p*-value
	Q_1: ≤313 pM (n = 35)	Q_2: 314 - 380 pM (n = 35)	Q_3: 381 - 482 pM (n = 35)	Q_4: >482.0 pM (n = 34)	
Age (years)	41.5.0 (37.8 - 45.3)	40.0 (33.3 - 44.0)	37.0 (33.5 - 41.5)	38.0 (31.8 - 44.0)	0.159
Exposure lengths (years)	11.8 (8.9 - 15.0)	11.5 (7.3 - 19.0)	10.0 (7.0 - 14.5)	8.0 (5.0 - 15.3)	0.271
BMI (Kg/m^2)	23.4 (21.3 - 27.7)	23.2 (21.1 - 26.8)	22.6 (20.1 - 25.7)	21.8 (20.2 - 24.1)	0.136
Glucose (nmol/L)	4.3 (4.1 - 4.6)	4.4 (4.0 - 4.7)	4.1 (3.6 - 4.5)	4.1 (3.7 - 4.2)	0.013
Insulin (µM/mL)	21.4 (15.3 - 35.8)	22.2 (18.0 - 35.8)	14.7 (11.6 - 28.2)	19.2 (14.1 - 25.8)	0.014
HOMA-IR	4.0 (2.9 - 6.6)	4.5 (3.3 - 8.1)	2.6 (1.9 - 5.6)	3.6 (2.2 - 5.0)	0.009
Alcohol Use (n/N (%))	15 (42.9)	12 (34.3)	13 (37.1)	14 (41.2)	0.882
HTA (n/N (%))	18 (52.9)	16 (48.5)	14 (41.2)	17 (50.0)	0.796

BMI: Body Mass Index; IR: Insulin Resistance; IQR: Interquartile Range; HOMA-IR: Homeostatic Model Assessment-Insulin Resistance; HTA: Hypertension. Values are presented as medians with IQR unless otherwise specified.

Participants were stratified into quartiles based on vitamin B12 levels to assess differences in metabolic and demographic parameters (Table 2). The median age

ranged from 37.0 years (Q_3: 381 - 482 pM) to 41.5 years (Q_1: ≤313 pM), with no significant differences across quartiles ($p = 0.159$). Similarly, exposure length and BMI did not vary significantly by vitamin B12 quartiles ($p = 0.271$ and $p = 0.136$, respectively). However, significant differences were observed in glucose, insulin, and HOMA-IR levels. Participants in the third quartile (Q3: 381 - 482 pM) had the lowest median glucose (4.1 nmol/L, IQR: 3.6 - 4.5), insulin (14.7 µM/mL, IQR: 11.6 - 28.2), and HOMA-IR (2.6, IQR: 1.9 - 5.6), compared to higher values in Q_1 and Q_2 ($p = 0.013$ for glucose, $p = 0.014$ for insulin, and $p = 0.009$ for HOMA-IR).

3.3. Demographic and Metabolic Parameters Stratified by Insulin Resistance Status

Table 3 compares metabolic and demographic parameters between non-insulin-resistant (non-IR) and insulin-resistant groups. Median age ($p = 0.749$), duration of exposure ($p = 0.360$), alcohol use ($p = 0.099$), and hypertension prevalence ($p = 0.723$) did not differ significantly between groups. However, IR participants had significantly higher BMI (23.5 vs. 21.9 kg/m^2, $p = 0.001$) and glucose levels (4.4 vs. 3.9 mM, $p < 0.001$) compared to non-IR participants.

Table 3. Metabolic and demographic parameters stratified by insulin resistance status.

	Non-IR (n=45)	IR (n=94)	*p*-value
	Median (IQR)	Median (IQR)	
Age (years)	40.0 (32.3 - 45.0)	39.0 (34.0 - 44.0)	0.749
Duration exposure (years)	10.0 (6.0 - 16.3)	11.0 (7.0 - 16.0)	0.360
BMI (Kg/m^2)	21.9 (19.9 - 23.3)	23.5 (20.9 - 27.5)	0.001
Glucose (mM)	3.9 (3.5 - 4.2)	4.4 (4.1 - 4.7)	<0.001
Vitamin B12 (pM)	416.5 (334.3 - 481.8)	363.0 (304.0 - 492.0)	0.309
	n/N (%)		
Alcohol use	22/45 (48.9)	32/94 (34.0)	0.099
HTA	22/45 (48.9)	43/94 (45.7)	0.855
IR prevalence (all participants)	–	94/139 (67.6%)	–

BMI: Body Mass Index; IR: Insulin Resistance; IQR: Interquartile Range; HOMA-IR: Homeostatic Model Assessment-Insulin Resistance; HTA: Hypertension.

3.4. Lifestyle and Metabolic Factors Independently Associated with Insulin Resistance

Table 4 presents logistic regression results assessing determinants of insulin resistance (HOMA-IR > 2.9). Higher BMI was significantly associated with increased odds of insulin resistance (OR = 1.28, 95% CI: 1.11 - 1.48, $p = 0.001$). Alcohol use also increased the odds of insulin resistance (OR = 2.41, 95% CI: 1.02 - 5.72, $p = 0.046$). Conversely, vitamin B12 levels > 381 pM were associated with significantly reduced odds of insulin resistance (OR = 0.19, 95% CI: 0.06 - 0.59, $p = 0.004$).

Table 4. Lifestyle and metabolic factors independently associated with insulin resistance in the study population.

Dependent Determinant	Residual Determinants	Odds Ratios	95% CI	*p*-value
Insulin resistance (HOMA-IR > 2.9)	BMI	1.28	1.11 - 1.48	0.001
	Alcohol use	2.41	1.02 - 5.72	0.046
	Vitamin B12 > 381 pM	0.19	0.06 - 0.59	0.004

BMI: Body Mass Index; HOMA-IR: Homeostatic Model Assessment-Insulin Resistance.

4. Discussion

This study provides critical insights into the determinants of insulin resistance (IR) among TMDs in Cotonou, Benin, a population occupationally exposed to high levels of air pollution. Our findings highlight the protective role of moderate vitamin B12 levels (>381 pmol/L) against IR while identifying BMI and alcohol consumption as significant risk factors. These results underscore the complex interplay between lifestyle factors, nutritional status, and metabolic health in a high-risk occupational group.

4.1. BMI and Alcohol Consumption as Risk Factors

BMI emerged as a strong predictor of IR, with each unit increase corresponding to a 28% higher likelihood of developing IR (OR = 1.28, $p = 0.001$). This association between BMI and IR is consistent with the well-established link between obesity and metabolic dysfunction, emphasizing the importance of weight management in this population. This finding aligns with global evidence highlighting obesity as a major driver of insulin resistance and type 2 diabetes mellitus (T2DM) [22]. The high prevalence of IR in our cohort further underscores the urgent need for interventions targeting adiposity in this occupational group.

Alcohol consumption emerged as a significant risk factor for IR in our cohort, with drinkers exhibiting 2.4-fold higher odds of IR compared to non-drinkers. This finding is consistent with reports from Mediterranean populations, where wine is typically consumed with meals, showing that individuals consuming at least seven alcoholic drinks per week had an increased risk of developing metabolic syndrome [23]. However, it contrasts with studies such as Akahane *et al.* (2020), which observed a protective metabolic effect of chronic alcohol consumption (≥60 g/day) in a Japanese cohort [24]. We hypothesize that cultural and behavioral differences in drinking patterns may explain this discrepancy. In Akahane *et al.*'s study, alcohol intake likely reflects the Japanese cultural norm (e.g., type and volume) and moderate consumption with meals, which has been associated with improved insulin sensitivity through anti-inflammatory pathways. In contrast, our cohort of TMDs in Benin exhibits distinct alcohol consumption behaviors, primarily centered on Sodabi—a traditional artisanal spirit derived from fermented palm wine (*Elaeis guineensis*) with compositional distinctions from

mass-produced global alcoholic beverages. This culturally entrenched beverage is often consumed in heavy episodic patterns, likely driven by occupational stressors or limited access to regulated alcohol. Such consumption patterns may synergize with chronic air pollution exposure to amplify oxidative stress. These context-specific risks underscore the importance of culturally adapted public health strategies and highlight that universal alcohol guidelines may not apply equally across populations. Future analyses should stratify by drinking behaviors (e.g., heavy versus steady consumption, beverage type, with/without meals) and pollution exposure tertiles to clarify dose-response gradients and account for confounders like dietary antioxidant intake, ultimately refining causal inference for developing targeted interventions.

4.2. Vitamin B12 and Insulin Resistance

The inverse association between moderate vitamin B12 levels (381 - 482 pM) and IR (OR = 0.19, $p = 0.004$) aligns with recent findings in obese populations [25], suggesting that adequate vitamin B12 status may mitigate metabolic dysfunction in TMDs exposed to air pollution. This is consistent with the critical role of vitamin B12 in one-carbon metabolism, mitochondrial function, lipid metabolism, and antioxidant defense, all of which are essential for maintaining metabolic homeostasis [26]. Vitamin B12 deficiency has been linked to hyperhomocysteinemia, impaired fatty acid oxidation, insulin resistance, and metabolic syndrome [27]. In populations exposed to air pollution, oxidative stress and inflammation are exacerbated due to the generation of reactive oxygen species by pollutants such as PAHs and benzene [1]. Vitamin B12, by supporting methylation processes and reducing homocysteine levels, may mitigate oxidative damage and improve insulin signaling pathways, thereby reducing IR.

The diagnostic thresholds for vitamin B12 deficiency have long been debated, with proposed cutoffs ranging from 100 pmol/L to 350 pmol/L across studies and guidelines [28]. While the WHO adequacy cutoff (>221 pmol/L) effectively identifies individuals at risk of classical deficiency pathologies (e.g., megaloblastic anemia), our findings suggest that higher thresholds (>381 pmol/L) may be required to confer metabolic benefits, such as protection against IR, particularly in populations exposed to chronic oxidative stress (e.g., occupational air pollution). This highlights the need for context-specific thresholds tailored to metabolic outcomes rather than relying solely on traditional deficiency criteria.

Our analyses also revealed a non-linear biological response: the third quartile (381 - 482 pmol/L) was associated with significantly reduced IR odds (OR = 0.19, 95% CI: 0.06 - 0.59, $p = 0.004$), while the highest quartile (>482 pmol/L) showed no incremental benefit. This phenomenon underscores that optimal, rather than excessive, micronutrient levels are required for metabolic health [26] [29] and that severe functional vitamin B12 deficiency can occur despite normal or elevated plasma concentrations, highlighting the limited diagnostic value of plasma vitamin B12 as a stand-alone marker due to its inability to assess cellular bioavailabil-

ity or metabolic utilization [30]. Therefore, the absence of additional benefit in Q4 could stem from heterogeneity within this subgroup, including individuals with: 1) optimal dietary intake but subclinical malabsorption (e.g., reduced tissue uptake, leading to elevated plasma B12 despite cellular insufficiency) or 2) different clinical conditions (e.g., hepatic or renal dysfunction) that artificially elevate plasma B12 levels.

These findings underscore the complexity of B12 biology in metabolic health and emphasize the importance of integrating functional biomarkers (e.g., holotranscobalamin, methylmalonic acid) in future studies to distinguish between circulating and bioavailable B12.

4.3. Strengths, Limitations, and Implications for Public Health and Occupational Safety

Despite these significant findings, our study has several limitations. First, we did not conduct personal air monitoring or measure biomarkers of exposure, such as urinary 1-hydroxypyrene for PAHs or S-phenylmercapturic acid for benzene. These biomarkers would have provided a more precise quantification of individual exposure levels and strengthened the study's ability to link air pollution to metabolic outcomes. Second, the cross-sectional design limits our ability to establish causal relationships between vitamin B12 levels, lifestyle factors, and IR. Further, residual confounding from unmeasured variables (e.g., dietary patterns and physical activity) cannot be ruled out. Third, the study population included only male participants, limiting generalizability to other occupational groups with different pollution profiles and precluding exploration of gender-specific effects.

Nevertheless, our findings have important public health implications. The protective effect of moderate vitamin B12 levels suggests that nutritional interventions, such as vitamin B12 supplementation or dietary fortification, could mitigate the metabolic risks associated with air pollution exposure. Workplace health programs targeting weight management and alcohol moderation could further reduce the burden of IR in this population. Future research should prioritize longitudinal designs integrating personal air quality assessments and exposure biomarker analysis to better understand the mechanisms linking air pollution, nutritional status, and metabolic health.

5. Conclusion

In conclusion, this study highlights the interplay between environmental exposures, nutritional status, and lifestyle factors in shaping metabolic health among TMDs in Cotonou. The protective role of moderate vitamin B12 levels against IR underscores the potential for targeted interventions, including B12 supplementation, weight management programs, and alcohol moderation initiatives, which could significantly improve metabolic outcomes in this vulnerable population. Future research should explore the mechanisms underlying these associations and evaluate the effectiveness of such interventions in real-world settings.

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Conflicts of Interest

The authors declare no competing interests.

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