Original Article

Traffic-Related Carbon Monoxide Exposure, Carboxyhaemoglobin Levels, and Arterial Stiffness among Public Transport Workers in Dakar, Senegal: A Cross-Sectional Study

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Abstract

Introduction: Air pollution is a major public health concern, particularly in rapidly urbanizing African cities where vehicular traffic dominates. Road transport workers are at risk of chronic carbon monoxide (CO) exposure, but data on its health effects are limited. This study assessed CO exposure, carboxyhemoglobin (HbCO) levels, and their association with subclinical symptoms and arterial stiffness among public transport workers in Dakar, Senegal.

Methods: A cross-sectional study enrolled 76 publics transports workers. Sociodemographic, occupational, and smoking data were collected via questionnaire. Exhaled CO and HbCO levels were measured, and pulse wave velocity (PWV) was assessed. Logistic and linear regression analyses evaluated associations between HbCO and respiratory symptoms or PWV, adjusting for age, BMI, smoking, and employment duration.

Results: Participants were predominantly male, mean age 35.2 ± 8.3 years, BMI 24.8 ± 3.1 kg/m², working 16 ± 1.5 hours/day and 6.2 ± 0.8 days/week; 11.8% were current smokers. Mean exhaled CO was 3.72 ± 3.21 ppm, and mean HbCO was 1.26 ± 0.53%, with 26% exceeding 1.5%. HbCO strongly correlated with exhaled CO ($r = 0.68$, $p < 0.001$). Neurological (headache 23.7%) and musculoskeletal (back pain 21.1%) complaints were common. Respiratory symptoms occurred in 19.7% of participants, with higher prevalence in those with HbCO >1.5% (27% vs 8%, $p = 0.003$). PWV averaged 12.03 ± 4.10 m/s; participants with HbCO >1.5% had significantly higher PWV, and linear regression showed a positive association between HbCO and PWV ($\beta = 0.29$, 95% CI 0.02–0.56, $p = 0.034$).

Conclusion: Public transport workers in Dakar experience chronic CO exposure. Elevated HbCO was associated

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with increased respiratory symptoms and arterial stiffness, suggesting early subclinical cardiovascular and respiratory effects. These findings highlight the need for occupational health interventions and further research on air pollution impacts in sub-Saharan Africa.

Keys words: air pollution, carbon monoxide, carboxyhaemoglobin, cardiorespiratory function, occupational exposure, Dakar

Introduction

Air pollution is a major environmental and public health concern worldwide, causing an estimated 4.2 to 6.7 million premature deaths annually, mainly due to cardiovascular and respiratory diseases[1]. According to the World Health Organization (WHO), over 99% of the global population breathes air that exceeds recommended limits for fine particulate matter ($PM_{2.5}$), with the highest exposures observed in low- and middle-income countries, particularly in sub-Saharan Africa[2]. Urban areas in sub-Saharan Africa, including Dakar, Senegal, are particularly affected because of rapid urbanization, high traffic density, aging public transport fleets, and limited air quality monitoring[2].

Road transport is a primary source of air pollutants such as PM2.5, PM_{10}, nitrogen oxides (NO_2), and carbon monoxide (CO). CO binds hemoglobin with high affinity, forming carboxyhaemoglobin (HbCO), which reduces oxygen delivery and can lead to tissue hypoxia[3]. Chronic low-level exposure to CO has been associated with endothelial dysfunction, systemic inflammation, and increased arterial stiffness, early markers of cardiovascular risk[4].

Transport workers—including bus drivers, conductors, and ticket agents are particularly vulnerable due to prolonged occupational exposure in high-traffic environments. International studies have reported elevated exhaled CO and HbCO levels in similar occupational groups, with associated increases in arterial stiffness[5,6]. However, data from sub-Saharan Africa are extremely limited.

Arterial stiffness, assessed by pulse wave velocity (PWV), is a validated marker of vascular aging and subclinical cardiovascular risk. Previous studies show that exposure to traffic-related air pollution is linked to higher PWV, independently of traditional risk factors such as age, blood pressure, and smoking[5]. In Dakar, occupational exposure may be compounded by poor vehicle maintenance, informal transport systems, and prolonged working hours.

The rationale for this study is to address these knowledge gaps by assessing exhaled CO, HbCO, and PWV among public transport workers in Dakar. We hypothesize that chronic exposure to traffic-related air pollution is associated with elevated CO and HbCO levels, increased arterial stiffness, and higher prevalence of subclinical cardiorespiratory and neurological symptoms.

This study provides novel data for West Africa, integrating biomarkers of exposure with cardiovascular outcomes, and may inform occupational health surveillance and urban air quality interventions.

Methods

Study Design and Setting

A cross-sectional study was conducted among public transport workers in Dakar, Senegal, between October 2017 and April 2018. The study was carried out at two of the city's busiest bus stations: Petersen, located in the city center adjacent to Sandaga market and serving more than twenty bus lines with over one hundred buses, and Lat Dior, situated nearby and operating approximately fifteen minibus lines. These sites were chosen because of their central location and the high density of minibus traffic, which contributes substantially to urban air pollution.

The study population included public transport workers, specifically drivers and conductors operating minibus lines at Petersen and Lat Dior. Eligible participants were men aged 18–55 years, employed in the transport sector in Dakar for at least 12 months, and with a minimum of three years of continuous professional activity. Participation required written informed consent. Individuals with a history of chronic cardiopulmonary disease (e.g., asthma, chronic obstructive pulmonary disease, or heart failure) prior to employment in transportation were excluded. While female staff were considered, the workforce was overwhelmingly male; pregnancy was therefore defined as an additional exclusion criterion.

A total of 76 participants were consecutively recruited. Written informed consent was obtained from each worker after authorization from station managers. The study followed the ethical principles of the Declaration of Helsinki and received approval from the Ethics Committee of Université Cheikh Anta Diop (UCAD) (Approval No. 0349/2018/CER/UCAD).

Data Collection

Clinical examination

Each participant underwent a standardized clinical examination, including measurement of anthropometric parameters and vital signs. Height and weight were measured using a calibrated stadiometer and digital scale, and body mass index (BMI) was calculated as weight (kg)/height^2 (m^2). Blood pressure and heart rate were assessed with an automated sphygmomanometer (Omron HEM-907, Japan) after 5–15 minutes of rest in a seated position. Two readings were taken on the left arm and one on the right arm, with the mean value used for analysis. Hypertension was defined as systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg.

Questionnaire

A standardized interviewer-administered questionnaire, adapted from validated occupational exposure surveys[5,6], was used to collect information in four domains:

- **Sociodemographic characteristics:** age, sex, education level.
- **Occupational history:** years of employment, average daily working hours, number of working days per week.
- **Lifestyle factors:** smoking status (current, former, never), alcohol consumption, physical activity.
- **Medical history and symptoms:** respiratory complaints (cough, wheezing, dyspnea), neurological symptoms (headache, dizziness), musculoskeletal pain, gastrointestinal disorders, and other relevant conditions.

Exhaled Carbon Monoxide (CO) Measurement

Exhaled CO was assessed using the piCO+ Smokerlyzer® (Bedfont Scientific, UK). Participants were instructed to inhale deeply, hold their breath for 15 seconds, and exhale slowly into a disposable mouthpiece. Results were expressed in parts per million (ppm) and automatically converted into carboxyhemoglobin equivalent (HbCO%). Assessments of exhaled CO were conducted immediately after the participants completed their daily work shift, directly at the bus stations in order to capture real-time occupational exposure. Measurements were performed in a seated position, with up to two additional attempts if the initial test failed.

Arterial Stiffness Assessment

Arterial stiffness was evaluated using pulse wave velocity (PWV) with the pOpmètre®, a validated non-invasive device[7]. Infrared sensors were placed on the finger and toe to record pulse transit time. Participants rested for 10 minutes in the supine position before measurement. Three consecutive readings were obtained and averaged, with results expressed in meters per second (m/s).

Statistical Analysis

Data were entered in Microsoft Excel 2016 and analyzed using SPSS version 25.0 (IBM, USA) and GraphPad Prism 5.

Descriptive statistics: continuous variables were expressed as mean ± standard deviation (SD); categorical variables as frequencies and percentages.

Comparative analysis: Chi-square test was used for categorical variables, and Spearman correlation test assessed associations between continuous variables.

Risk estimation: Odds ratios (OR) with 95% confidence intervals (CI) were calculated to evaluate the association between smoking status and cardiovascular outcomes.

A p-value <0.05 was considered statistically significant.

Comparative analyses were performed by stratifying participants according to carboxyhemoglobin (HbCO) levels, using a threshold of 1.5% to distinguish normal from elevated values. This cut-off reflects reference ranges in non-smokers, with higher levels indicating significant environmental or occupational CO exposure[8,9]. Independent t-tests and chi-square tests were used to compare means and proportions.Associations between exposure and health outcomes: Logistic regression was used to calculate odds ratios (ORs) with 95% confidence intervals (CIs) for respiratory, neurological, musculoskeletal, and digestive symptoms according to CO and HbCO levels. Linear regression was used to assess the association between HbCO and PWV, adjusting for age, BMI, smoking status, and years of occupational exposure. Significance threshold: A two-sided p-value <0.05 was considered statistically significant.

Results

Participant characteristics

A total of 76 public transport workers were enrolled in the study. The mean age was 35.17 ± 8.28 years (range 21–54), and the majority were male (sex ratio: 1.8) The mean body mass index (BMI) was 24.8 ± 3.1 kg/m². Participants reported working an average of 16 ± 1.5 hours per day and 6.2 ± 0.8 days per week. Among the participants, 11.8% were current smokers, while 5.3% were former smokers. No participants reported chronic cardiovascular or respiratory diseases prior to the study.

Table 1. Demographic and occupational characteristics of study participants (n = 76)

Characteristic	Value
Age, years	35.17 ± 8.28 (range: 21–54)
Sex ratio (male/female)	1.8 (male predominant)
Body mass index (BMI), kg/m²	24.8 ± 3.1
Working hours/day	16 ± 1.5
Working days/week	6.2 ± 0.8
Current smokers, n (%)	9 (11.8%)
Former smokers, n (%)	4 (5.3%)
Chronic cardiovascular or respiratory disease	None reported

Exhaled CO and HbCO as biomarkers of CO exposure in participants

The mean exhaled carbon monoxide (CO) concentration was 3.72 ± 3.21 ppm (range: 0.5–12 ppm), while the mean carboxyhemoglobin (HbCO) level was 1.26 ± 0.53% (range: 0.3–2.8%). Based on established reference thresholds, 74% of participants exhibited HbCO levels ≤1.5%, consistent with normal background exposure, whereas 26% showed elevated HbCO levels (>1.5%), suggestive of increased environmental or occupational CO exposure. HbCO levels were strongly correlated with exhaled CO ($r = 0.68$, $p < 0.001$), supporting the reliability and concordance of these two biomarkers in assessing CO exposure.

Table 2. Carbon monoxide exposure and carboxyhemoglobin (HbCO) levels (n = 76)

Biomarker	Mean ± SD	Range	Stratification
Exhaled CO (ppm)	3.72 ± 3.21	0.5–12	–
HbCO (%)	1.26 ± 0.53	0.3–2.8	≤1.5%: 56 (74%) >1.5%: 20 (26%)
Correlation exhaled CO – HbCO	r = 0.68	p < 0.001	–

Prevalence of subclinical symptoms and association with HbCO levels

Study participants reported a range of subclinical symptoms across multiple systems, with neurological (headache 23.7%) and musculoskeletal (back pain 21.1%) complaints being most common. Respiratory symptoms, including chronic cough (19.7%), dyspnea on exertion (13.2%), and wheezing (7.9%), were also observed, suggesting early airway irritation. Notably, participants with HbCO > 1.5% had a significantly higher prevalence of respiratory symptoms than those with HbCO ≤ 1.5% (27% vs 8%, $p = 0.003$), indicating a potential dose-response relationship. Digestive symptoms (epigastric discomfort 9.2%, nausea 6.6%) and joint stiffness (14.5%) were less frequent, possibly reflecting systemic effects of CO exposure or occupational factors. Participants with HbCO > 1.5% had a higher prevalence of respiratory symptoms compared to those with HbCO ≤1.5% (27% vs 8%, p = 0.003).

Symptom category	Symptom	Prevalence (%)
Respiratory	Chronic cough	19.7
	Dyspnea on exertion	13.2
	Wheezing	7.9
Neurological	Headache	23.7
	Dizziness	15.8
Digestive	Epigastric discomfort	9.2
	Nausea	6.6
Musculoskeletal	Back pain	21.1
	Joint stiffness	14.5

Association between carboxyhemoglobin levels and arterial stiffness

PWV measurements revealed a mean value of 12.03 ± 4.10 m/s. Participants with HbCO >1.5% exhibited a slightly higher mean PWV (14.0 ± 2.1 m/s) compared to those with HbCO ≤1.5% (11.7 ± 1.8 m/s), and this difference was significant (p = 0.04). Linear regression adjusting for age, BMI, smoking, and duration of employment showed a positive association between HbCO and PWV (β = 0.29, 95% CI 0.02–0.56, p = 0.034).

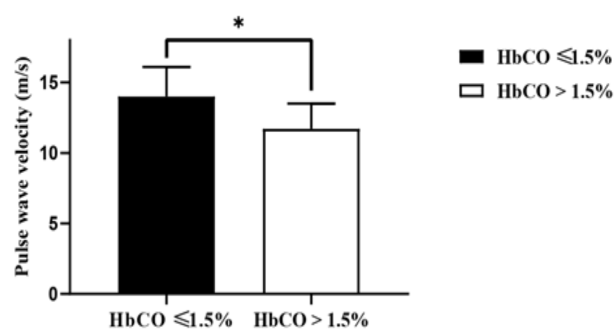

Table 1. Multivariable linear regression showing factors associated with PWV

Independent variables	β coefficient	95% Confidence Interval	p-value
HbCO (%)	0.29	0.02 – 0.56	0.034*
Age (years)	0.15	−0.01 – 0.31	0.067
BMI (kg/m²)	0.08	−0.05 – 0.21	0.214
Smoking (continuous, pack-years)	0.11	−0.03 – 0.25	0.118
Duration of employment (years)	0.10	−0.04 – 0.24	0.151

Discussion

This study evaluated exposure to carbon monoxide (CO) and its health effects among public transport workers in Dakar, Senegal, highlighting significant clinical and toxicological implications even at low-level exposures. The mean exhaled CO concentration in the study population was 3.72 ± 3.21 ppm, and the mean carboxyhemoglobin (HbCO) level was 1.26 ± 0.53%, consistent with values reported in similar occupational settings. For example, bus drivers in São Paulo, Brazil, demonstrated exhaled CO levels of 3–5 ppm and HbCO levels between 1–2%[2]. The strong correlation between exhaled CO and HbCO (r = 0.68, p < 0.001) supports the reliability of these non-invasive biomarkers for assessing CO exposure in this population.

Although the observed CO and HbCO levels are below acute toxic thresholds (>25 ppm or >10% HbCO), chronic low-level exposure can have deleterious effects. Even subclinical concentrations may induce oxidative stress, endothelial dysfunction, and vascular remodeling, ultimately contributing to arterial stiffness and elevated cardiovascular risk[4,7]. The study showed that participants with HbCO >1.5% exhibited a higher prevalence of respiratory symptoms compared to those with lower HbCO levels (27% vs 8%, p = 0.003), suggesting a dose-response relationship. Factors such as duration of exposure, individual susceptibility, and metabolic clearance of CO likely influence symptom manifestation. Prior research has demonstrated that genetic polymorphisms affecting hemoglobin affinity, antioxidant capacity, or nitric oxide bioavailability can modulate physiological responses to CO[10], which may explain variability in symptom expression.

The PWV of 12.03 ± 14.88 m/s indicates early vascular stiffness even in this relatively young cohort. Higher HbCO levels were positively associated with increased PWV after adjustment for age, body mass index, smoking status, and duration of employment ($\beta = 0.29$, $p = 0.034$), consistent with prior studies showing that chronic CO exposure can impair endothelial function, reduce nitric oxide bioavailability, and promote arterial remodeling[4]. PWV is a robust predictor of cardiovascular events independent of traditional risk factors, and early detection in occupational cohorts is critical for preventive interventions[7]. Mechanistically, CO exposure may contribute to arterial stiffness through hypoxia-induced vascular remodeling, oxidative stress, inflammation, and autonomic dysregulation. CO binds hemoglobin with high affinity, reducing tissue oxygenation; chronic tissue hypoxia can stimulate vascular smooth muscle proliferation and extracellular matrix deposition, increasing arterial rigidity[11,12]. Additionally, CO-induced reactive oxygen species activate pro-inflammatory pathways such as NF-κB, promoting endothelial dysfunction[12].

Subclinical symptoms were prevalent, with neurological complaints (headache 23.7%, dizziness 15.8%), musculoskeletal pain (back pain 21.1%), and respiratory symptoms (chronic cough 19.7%, dyspnea 13.2%). Smoking status did not significantly affect symptom prevalence, likely due to the low proportion of smokers (11.8%). These findings suggest that occupational exposure to traffic-related air pollution is a primary contributor to health effects. Neurological and musculoskeletal symptoms may reflect combined effects of CO-induced hypoxia, prolonged sitting, and exposure to vehicle vibrations[11]. Similar findings have been reported in populations exposed to urban traffic emissions, where chronic low-level CO exposure is linked to fatigue, headache, and impaired cognitive function[12].

Comparisons with studies from Europe and Asia, where baseline air pollution levels are lower, highlight the heightened vulnerability of workers in West Africa, where aging vehicle fleets and lack of emission control amplify exposure[11]. This underscores the need for targeted occupational health interventions, including routine monitoring of CO and HbCO, improved vehicle maintenance, ventilation of workplaces, and provision of personal protective equipment, to reduce exposure and protect worker health.

Clinically, even subclinical elevations of HbCO may increase cardiovascular risk by promoting endothelial dysfunction and arterial stiffness, which are early markers of cardiovascular disease. Toxicologically, chronic low-level CO exposure can induce subtle hypoxic stress and inflammation, potentially exacerbating pre-existing metabolic or cardiovascular vulnerabilities. Recognition of these early effects allows for timely preventive strategies before overt disease develops, highlighting the importance of occupational surveillance programs in high-exposure urban settings.

Several limitations should be acknowledged. The cross-sectional design precludes causal inference, and longitudinal follow-up studies are warranted to assess the temporal progression of arterial stiffness and related clinical outcomes. The relatively small sample size ($n = 76$) may have limited statistical power, particularly for subgroup analyses. Symptom data were self-reported, introducing potential recall bias, although the use of structured questionnaires helped mitigate this risk. The study did not include personal monitoring of $PM_{2.5}$ or NO_x, and CO was used as a proxy for traffic-related air pollution. Additionally, other occupational exposures common in transport work such as heat, whole-body vibrations, and noise were not measured and could act as potential confounders. Finally, the predominantly male cohort may limit the generalizability of findings to female transport workers.

Despite these limitations, this study provides novel evidence of subclinical cardiovascular and respiratory effects associated with occupational exposure to urban traffic in Dakar, addressing a critical knowledge gap in sub-Saharan Africa. Future research should use longitudinal designs to track arterial stiffness and cardiovascular events. Personal monitoring of $PM_{2.5}$, NO_x, carbon monoxide, and volatile organic compounds (VOCs) gaseous pollutants from traffic, fuel, and urban sources along with endothelial biomarkers such as nitrites/nitrates or eNOS activity, would clarify whether vascular effects are driven by nitric oxide depletion or oxidative stress. Genetic and metabolic factors influencing susceptibility should also be considered. Finally, intervention studies such as reduced working hours, improved vehicle maintenance, and protective equipment are needed to evaluate strategies for mitigating occupational health risks.

Conclusion

Occupational exposure to traffic-related air pollution among Dakar public transport workers is associated with measurable CO biomarkers and early signs of vascular dysfunction. A substantial proportion of participants reported respiratory, neurological, musculoskeletal, and digestive symptoms, indicating widespread subclinical health effects. Higher HbCO levels were linked to increased arterial stiffness, suggesting potential long-term cardiovascular risk. These findings highlight the clinical and toxicological significance of chronic CO exposure and emphasize the need for occupational health monitoring, preventive strategies, and effective urban air quality policies.

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Conflict of Interest Disclosure

The authors declare no conflicts of interest related to this study.

References

1. World Health Organization. *Air pollution*. WHO; 2021. PMID: 34948829.
2. World Health Organization (WHO).*Ambient (outdoor) air pollution*. Key facts. Geneva: World Health Organization; Updated October 24, 2024.Available at: https://www.who.int/news-room/fact-sheets/detail/ambient-(outdoor)-air-pollution.
3. Rose J, et al. Carbon monoxide exposure: clinical and toxicological aspects. *Toxicol Rev*. 2017;36(1):25–33. PMID: 28347155.
4. Brook RD, Rajagopalan S, Pope CA 3rd, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*. 2010;121(21):2331–2378. PMID: 20123637.
5. Chuang KJ, Yan YH, Chiu SY, Cheng TJ. Effects of air pollution on blood pressure and heart rate variability in elderly adults. *Environ Health Perspect*. 2010;118(6):860–866. PMID: 19890153.
6. Andrade CF, et al. Occupational exposure to traffic-related air pollution and cardiovascular risk among bus drivers in Brazil. *Environ Res*. 2017;154:218–225. PMID: 28668249.
7. Van Bortel LM, Laurent S, Boutouyrie P, et al. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *Artery Res*. 2012;6(2):1–26. PMID: 22234987.
8. Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med*. 1998;339:1603–1608.
9. World Health Organization. WHO guidelines for indoor air quality: selected pollutants. Geneva: WHO; 2010
10. Amegah AK, Agyei-Mensah S. Urban air pollution and health in sub-Saharan Africa: a critical review. *Arch Environ Occup Health*. 2017;72:41–54.
11. Lungu C, Ganeshan R, Naidu BV, et al. Musculoskeletal and neurological effects of chronic traffic-related exposure in transport workers. *Occup Environ Med*. 2017;74:561–568. PMID: 28423312.
12. Zhang Z, Chen Y, Zhao J, et al. Cognitive function and low-level CO exposure in urban commuters. *Int J Environ Res Public Health*. 2019;16:1204. PMID: 31064357