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Exhaled Breath Condensate as a Non-Invasive Biomarker of Oxidative Stress in Smokers: A Cross-Sectional Case–Control Study

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Abstract:

Introduction: smoking remains a major global health burden, driving disease through persistent oxidative stress and impaired antioxidant defenses. Traditional serum assays provide valuable information but are invasive and impractical for large-scale screening. Exhaled breath condensate (EBC) offers a simple, non-invasive alternative for assessing oxidative biomarkers in the airways.

Objectives: This study aimed to evaluate whether oxidative stress biomarkers measured in EBC reflect systemic oxidative changes in apparently healthy smokers and to determine their diagnostic performance.

Methods: A cross-sectional, case–control study was conducted among 60 adults (30 smokers, 30 non-smokers) aged 18–45 years. EBC was collected using a standardized condenser system, and both EBC and serum samples were analyzed for hydrogen peroxide (H_2O_2), 8-isoprostane, malondialdehyde (MDA), and total antioxidant capacity (TAC). Data were analyzed using t-tests, correlation analyses, and receiver-operating-characteristic (ROC) curves.

Results: Smokers showed markedly higher oxidative-stress biomarkers in both EBC and serum compared with non-smokers. Mean EBC H_2O_2 was 1.84 ± 0.42 µM in smokers versus 0.96 ± 0.28 µM in controls ($p < 0.001$), and EBC 8-isoprostane levels were 57.4 ± 14.3 pg/mL versus 33.1 ± 10.8 pg/mL ($p < 0.001$). TAC values were significantly lower in smokers across both matrices. EBC H_2O_2 correlated strongly with serum MDA ($r = 0.61$, $p < 0.01$), indicating concordant airway and systemic oxidative stress. ROC analysis showed high diagnostic accuracy for EBC H_2O_2 (AUC = 0.87, 95% CI 0.77–0.95).

Conclusions: EBC biomarkers, particularly H_2O_2 and 8-isoprostane, reliably mirror systemic oxidative stress in cigarette smokers. The strong correlation between EBC and serum markers supports EBC as a non-invasive, reproducible, and clinically relevant tool for early detection of smoking-related oxidative injury. Its simplicity and low cost make it especially promising for preventive health programs and research in low- and middle-income settings.

Keywords:

Exhaled breath condensate, oxidative stress, hydrogen peroxide, 8-isoprostane, smokers, biomarkers, total antioxidant capacity, non-invasive diagnostics.

Introduction

Cigarette smoking remains the leading cause of preventable disease and premature death worldwide (1). According to the Global Burden of Disease (GBD) 2021 report, tobacco use is responsible for more than eight million deaths each year, including about 1.3 million from exposure to second-hand smoke (2). Despite decades of public health efforts, nearly one in five adults globally continues to smoke, with the highest prevalence concentrated in low- and middle-income countries (LMICs), where healthcare systems often struggle to manage the burden of smoking-related disease (3). The biological harm caused by cigarette smoking is driven largely by oxidative stress, an imbalance between reactive oxygen species (ROS) and the body's antioxidant defenses. Excessive ROS generation leads to lipid peroxidation, DNA damage, and endothelial dysfunction, all of which play central roles in the development of chronic obstructive pulmonary disease (COPD), cardiovascular disease, and several types of cancer (4).

Cigarette smoke contains over 7000 reactive compounds, including free radicals and transition metals, which initiate oxidative injury and impair cellular repair mechanisms (5). Persistent oxidative stress in smokers is also associated with accelerated ageing, tissue injury, and increased susceptibility to infections (6).

Traditionally, biomarkers of oxidative stress such as malondialdehyde (MDA) and total antioxidant capacity (TAC) are measured in blood samples (7). While these assays are valuable, blood collection is invasive and unsuitable for large-scale screening, frequent monitoring, or use in preventive health programs (5). This limitation has led to growing interest in identifying non-invasive biomarkers that can reliably reflect systemic oxidative status (8).

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One promising approach is the analysis of exhaled breath condensate (EBC), which captures volatile and non-volatile molecules from the airway lining fluid by cooling exhaled air. EBC provides a simple and repeatable means of sampling airway biochemistry without discomfort or risk to participants. Biomarkers such as hydrogen peroxide (H_2O_2) and 8-isoprostane, both indicators of oxidative stress, have been successfully measured in EBC and correlated with disease activity in asthma and COPD (9). However, there is still limited evidence on whether EBC biomarkers can detect oxidative stress in apparently healthy smokers and how well they correlate with conventional serum markers. This study aimed to determine whether EBC biomarkers reflect oxidative stress in smokers and to assess their relationship with serum oxidative markers. We hypothesized that smokers would exhibit elevated oxidative stress biomarkers in EBC, consistent with systemic changes, thereby supporting the use of EBC as a non-invasive and clinically relevant tool for assessing smoking-related oxidative injury.

Methods

Study design and participants

This cross-sectional, case–control study was carried out between January and June 2025 at the Advanced Biochemistry Research Unit, College of Medicine, University of Kerbala. A convenience sample of eligible adults aged 18–45 years was recruited consecutively until the planned sample size (n = 60) was achieved. The smoker group included individuals who reported smoking at least ten cigarettes per day for three or more consecutive years. exposure was quantified in pack-years (packs/day × years). Age- and sex-matched non-smokers with no history of tobacco use served as controls.

Participants were excluded if they had chronic respiratory diseases such as asthma or COPD, recent respiratory infections, antioxidant supplementation, or systemic corticosteroid therapy within the preceding three months. All participants provided written informed consent before enrollment.

Procedures

Each participant completed a brief structured interview, followed by collection of both exhaled breath condensate (EBC) and venous blood samples under standardized conditions. Participants were instructed to refrain from smoking, eating, or drinking

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(except water) for at least two hours prior to sampling to minimize short-term biochemical variation.

EBC collection

EBC was obtained using a standardized condenser system (RTube™, Respiratory Research Inc., USA). Participants breathed tidally through a mouthpiece for 15 minutes while wearing a nose clip at ambient temperature (22 ± 2 °C, 50 ± 10 % relative humidity). Samples were immediately aliquoted and handled under chilled conditions (0–4 °C) to minimize degradation.

Sample handling and storage

When ultra-low freezers (−70 °C to −80 °C) were available, aliquots were stored immediately. In facilities lacking such equipment, samples were kept on wet ice and analyzed the same day or stored at −20 °C for no longer than 7 days. Samples were limited to one freeze–thaw cycle. For H_2O_2 assays, samples were stabilized at the point of collection by mixing with ferrous-oxidation–xylenol-orange (FOX) reagent. Samples requiring extended storage were transported on dry ice to the central laboratory at the University of Kerbala. For quality control, the time-to-freeze, storage duration, and number of freeze–thaw cycles were documented for every specimen.

Biochemical analysis

EBC biomarkers:

Exhaled breath condensate (EBC) and serum samples were analyzed for key oxidative stress and antioxidant biomarkers. Hydrogen peroxide (H_2O_2) concentrations were quantified using the ferrous oxidation–xylenol orange (FOX) assay, a colorimetric method based on the oxidation of ferrous (Fe^{2+}) to ferric (Fe^{3+}) ions by peroxides, forming a complex with xylenol orange measurable at 560 nm (10).

8-isoprostane levels were determined using a competitive enzyme-linked immunosorbent assay (ELISA) kit (Cayman Chemical, Ann Arbor, MI, USA) according to the manufacturer’ s instructions (11).

Total antioxidant capacity (TAC) was assessed by a Trolox-equivalent colorimetric assay, which evaluates the ability of antioxidants in the sample to reduce ferric ions, with results expressed as millimoles of Trolox equivalents per liter (11).

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Serum biomarkers:

Venous blood samples were centrifuged, and serum was analyzed for TAC using the same colorimetric method and for malondialdehyde (MDA) by the thiobarbituric acid reactive substances (TBARS) assay, which detects the pink chromogen produced by MDA–TBA complex formation (12).

All assays were performed in duplicate; intra-assay CV < 8 %, inter-assay CV < 10 %.

Outcomes

The primary outcome was the difference in EBC H_2O_2 concentrations between smokers and non-smokers. Secondary outcomes included differences in EBC 8-isoprostane and TAC, serum MDA and TAC, and the strength of correlations between EBC and serum biomarkers.

Statistical analysis

A minimum of 30 participants per group was estimated to provide 80 % power to detect a between-group difference of 0.3 µM in EBC H_2O_2 at an alpha level of 0.05, based on pilot data (n=10). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) as appropriate. Normality was assessed using the Shapiro–Wilk test. Between-group comparisons were performed using Student's t-test or Mann–Whitney U test. Associations between EBC and serum biomarkers were evaluated with Pearson correlation coefficients. Diagnostic performance was examined using receiver-operating-characteristic (ROC) curve analysis, and the Youden Index was used to determine the optimal cutoff. Ninety-five-percent confidence intervals were reported throughout. All analyses were conducted using SPSS version 26 (IBM Corp., USA). Statistical significance was defined as $p < 0.05$. Sensitivity analyses excluding samples stored beyond seven days at −20 °C were performed to confirm robustness.

Ethical approval

The study protocol was approved by the Research Ethics Committee of the College of Medicine, University of Kerbala (Approval No. 102/2025). Informed consent was obtained from all participants prior to enrollment, and data confidentiality was maintained throughout the study.

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Results

Participants

A total of 60 participants were enrolled: 30 current smokers (≥10 cigarettes per day for at least three years) and 30 age- and sex-matched non-smokers. The mean age was 32.1 ± 5.8 years for smokers and 31.4 ± 6.2 years for controls. Both groups were 70 % male (Table 1). All participants completed sample collection and laboratory analyses without missing data or assay failure. None of the participants reported chronic respiratory illness or recent antioxidant supplementation.

Table 1. Baseline characteristics of participants. Age and sex distributions were comparable between groups (p > 0.05).

Characteristic	*Smokers (n = 30)*	*Non-smokers (n = 30)*
Age, years (mean ± SD)	32.1 ± 5.8	31.4 ± 6.2
Male sex, n (%)	21 (70%)	21 (70%)

EBC biomarkers

Smokers exhibited markedly higher oxidative-stress markers in exhaled breath condensate. Mean H_2O_2 concentration was 1.84 ± 0.42 µM versus 0.96 ± 0.28 µM in controls (mean difference 0.88 µM [95 % CI 0.69–1.06]; $p < 0.001$). 8-Isoprostane levels were 57.4 ± 14.3 pg/mL vs 33.1 ± 10.8 pg/mL (mean diff 24.3 pg/mL [95 % CI 17.6–31.0]; $p < 0.001$). Antioxidant capacity was significantly lower among smokers (0.47 ± 0.12 mmol Trolox eq/L vs 0.71 ± 0.15; $p < 0.001$). Figure 1 shows the upward shift in EBC H_2O_2 distributions.

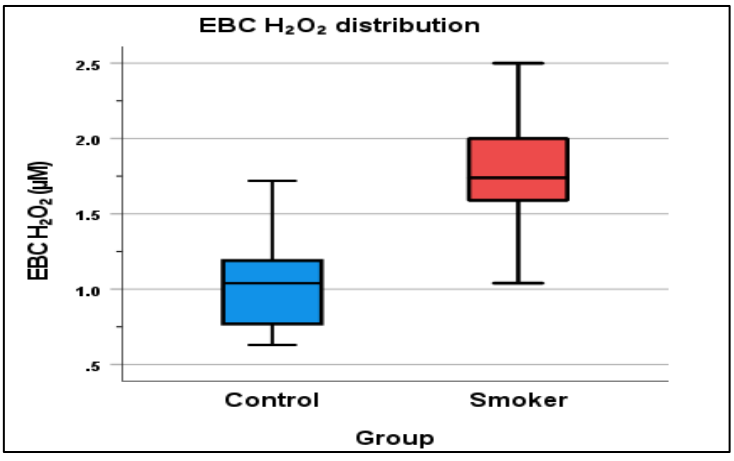

Figure 1: Distribution of exhaled breath condensate hydrogen peroxide (H_2O_2) concentrations in smokers and non-smokers. Box-and-whisker plots illustrate significantly higher exhaled H_2O_2 concentrations among smokers compared with age- and sex-matched non-smokers (mean ± SD: 1.84 ± 0.42 µM vs 0.96 ± 0.28 µM; $p < 0.001$). The central line denotes the median, boxes the interquartile range, and whiskers the total range. Elevated H_2O_2 levels in smokers indicate enhanced airway oxidative stress associated with cigarette smoke exposure.

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Serum biomarkers

Consistent patterns were observed in serum. MDA levels were 3.12 ± 0.56 µmol/L vs 1.89 ± 0.47 µmol/L (mean diff 1.23 µmol/L [95 % CI 0.95–1.50]; $p < 0.001$). Serum TAC was 0.93 ± 0.21 mmol/L vs 1.28 ± 0.24 mmol/L (mean diff –0.35 [95 % CI –0.47 to –0.23]; $p < 0.001$).

Correlations between EBC and serum biomarkers.

EBC H_2O_2 correlated positively with serum MDA ($r = 0.61$ [95 % CI 0.38–0.77], $p < 0.01$), and EBC TAC with serum TAC ($r = 0.55$ [95 % CI 0.31–0.73], $p < 0.01$). These relationships remained significant after excluding samples stored > 7 days at −20 °C. These findings suggest that oxidative stress detected in the airways mirrors systemic oxidative imbalance (Figure 2).

Table 2. Oxidative stress biomarkers in smokers and non-smokers. Values are mean ± SD. All differences between smokers and non-smokers were statistically significant ($p < 0.001$).

Biomarker	*Smokers (n = 30)*	*Non-smokers (n = 30)*	*p-value*
EBC H_2O_2 (µM)	1.84 ± 0.42	0.96 ± 0.28	<0.001
EBC 8-Isoprostane (pg/mL)	57.4 ± 14.3	33.1 ± 10.8	<0.001
EBC TAC (mmol Trolox eq/L)	0.47 ± 0.12	0.71 ± 0.15	<0.001
Serum MDA (µmol/L)	3.12 ± 0.56	1.89 ± 0.47	<0.001
Serum TAC (mmol/L)	0.93 ± 0.21	1.28 ± 0.24	<0.001

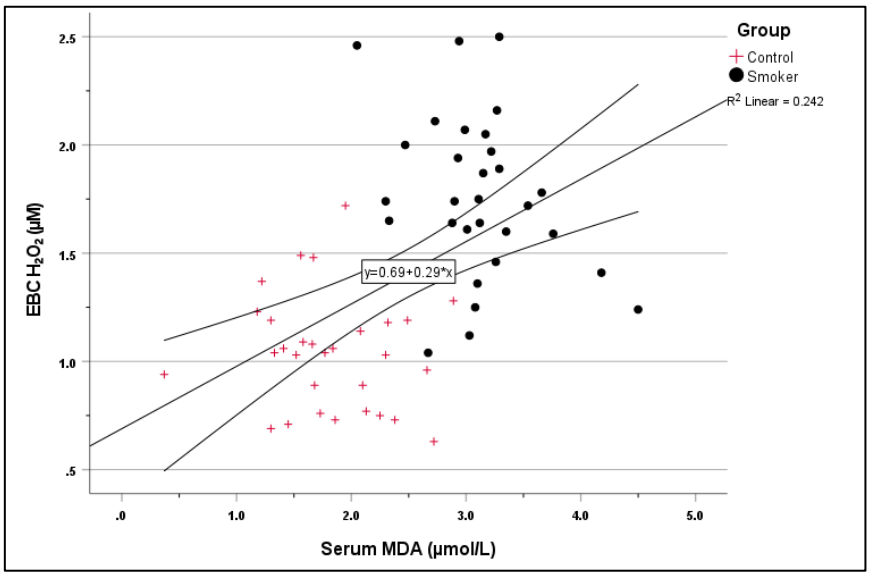

Figure 2: Scatterplot showing the correlation between exhaled breath condensate hydrogen peroxide (H_2O_2) and serum malondialdehyde (MDA). Smokers (black circles) and non-smokers (red crosses) are displayed. The straight black line represents the fitted linear regression trend, confirming a positive association between airway and systemic oxidative stress.

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Diagnostic performance

Receiver-operating-characteristic (ROC) analysis demonstrated strong discriminatory power of EBC H_2O_2 for differentiating smokers from non-smokers (Figure 3). The area under the curve (AUC) was 0.87 (95 % CI, 0.77–0.95), indicating high diagnostic accuracy. At an optimal cutoff value of 1.2 µM, sensitivity was 82 % and specificity 78 %, based on the Youden Index. Sensitivity analyses limited to samples stored ≤7 days at −20 °C yielded comparable results, confirming the robustness of findings despite variations in storage conditions.

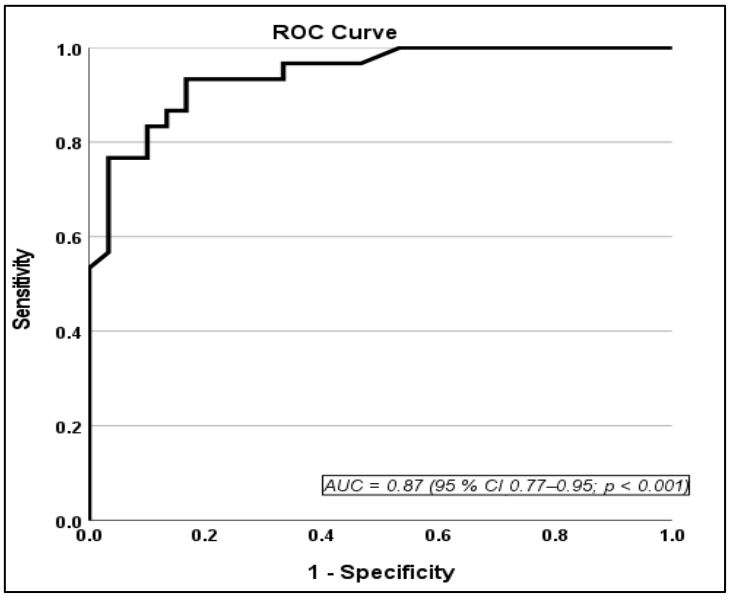

Figure 3: Receiver operating characteristic (ROC) curve for exhaled breath condensate hydrogen peroxide (H_2O_2) in distinguishing smokers from non-smokers. The area under the curve (AUC) was 0.87, indicating strong discriminatory performance.

Summary of findings

1. Smokers exhibited elevated oxidative stress biomarkers in both EBC and serum (H_2O_2, 8-isoprostane, MDA).
2. Antioxidant capacity was significantly reduced in smokers (EBC and serum TAC).
3. Strong positive correlations were observed between EBC and serum markers.
4. EBC H_2O_2 showed high diagnostic accuracy (AUC = 0.87) for distinguishing smokers from non-smokers.

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Discussion

In this case–control study, we found that smokers demonstrated significantly higher levels of oxidative stress in exhaled breath condensate (EBC) compared with non-smokers, as shown by elevated hydrogen peroxide (H_2O_2) and 8-isoprostane concentrations. At the same time, antioxidant capacity in EBC was markedly lower among smokers. These findings were consistent with serum results, where smokers exhibited higher malondialdehyde (MDA) levels and reduced total antioxidant capacity (TAC). Together, these results confirm that cigarette smoking induces measurable oxidative stress both locally in the airways and systemically.

The strong positive correlation between EBC H_2O_2 and serum MDA further supports this link, indicating that EBC biomarkers can reflect systemic oxidative changes. This relationship likely reflects shared oxidative mechanisms across respiratory and circulatory systems, such as lipid peroxidation and free radical generation triggered by cigarette smoke (13). Our findings are in line with previous studies showing increased oxidative burden and impaired antioxidant defenses in smokers, (14, 15) though most prior work relied solely on serum or bronchoalveolar lavage samples. The ability to capture this oxidative imbalance using a simple, non-invasive method like EBC adds significant value to both research and clinical practice.

Comparison with previous studies

Previous studies have reported elevated EBC oxidative markers in patients with asthma, COPD, and other inflammatory airway diseases (16, 17). However, there has been limited exploration of these markers in apparently healthy smokers, particularly in the absence of diagnosed disease. Our study extends the existing evidence by demonstrating that even young, asymptomatic smokers exhibit oxidative stress in the airway lining fluid, suggesting early subclinical changes that precede clinical disease. The strong correlation between EBC and serum markers supports the concept that airway oxidative stress may mirror systemic processes and could serve as an accessible indicator of overall oxidative burden.

Strengths and novelty

A key strength of this study is the dual assessment of both local (EBC) and systemic (serum) biomarkers within the same participants. This parallel approach provides a more complete picture of oxidative balance and supports the biological relevance of EBC measurements. To our knowledge, this is among the first controlled studies conducted in apparently healthy smokers from a low- and middle-income

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setting, a population often underrepresented in biomarker research. Moreover, by standardizing collection and storage conditions, we demonstrated that reliable EBC biomarker assessment is feasible even in laboratories with limited access to ultra-low freezing facilities.

Limitations

Several limitations should be acknowledged. The sample size was modest, which may limit generalizability and precluded subgroup analysis by smoking intensity or duration. Although we performed sensitivity analyses, short-term storage at −20 °C could have introduced some variability in biomarker stability (18). The cross-sectional design also limits causal inference; we cannot determine whether the observed oxidative stress directly contributes to disease development. Future longitudinal studies are needed to evaluate whether EBC biomarkers can predict the progression or reversibility of oxidative stress following smoking cessation.

Clinical and public health implications

EBC sampling is simple, safe, and well tolerated, requiring minimal training or equipment. Demonstrating that EBC biomarkers mirror systemic oxidative stress suggests that this method could be used for early risk assessment, large-scale screening, and monitoring of smoking cessation or antioxidant interventions. In low- and middle-income countries, where access to advanced diagnostics is limited but smoking prevalence remains high, EBC analysis may represent a cost-effective, scalable tool for evaluating tobacco-related harm and guiding preventive strategies.

Conclusion and Future Directions

This study demonstrates that exhaled breath condensate (EBC) biomarkers, particularly hydrogen peroxide (H_2O_2) and 8-isoprostane, can reliably reflect oxidative stress in cigarette smokers. The strong correlations between EBC and serum biomarkers support the idea that oxidative stress in the airways parallels systemic oxidative imbalance. Smokers also exhibited reduced antioxidant capacity, reinforcing the concept that long-term cigarette exposure overwhelms both local and systemic defense mechanisms.

EBC analysis offers a simple, repeatable, and non-invasive approach for assessing oxidative stress, making it especially valuable in preventive medicine and public health programs. Its low cost and ease of use make it suitable for large-scale screening,

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monitoring of smoking cessation, and early detection of oxidative injury in high-risk populations.

While the study provides strong cross-sectional evidence, future work should focus on longitudinal designs to determine whether EBC biomarkers can predict disease progression or recovery following cessation. Further standardization of EBC collection and storage protocols will also help improve reproducibility across laboratories and establish clinical reference values.

In conclusion, EBC biomarkers hold promise as accessible and clinically meaningful indicators of oxidative stress in smokers. Their application could help bridge the gap between laboratory research and real-world screening, ultimately contributing to earlier intervention and reduced tobacco-related harm.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest that could have influenced the conduct or findings of this study.

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