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Review article

Evaluation of Hematological Changes Among Hookah Smokers in Al-Najaf: A Comprehensive Scientific Review

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Abstract

Hookah (waterpipe) smoking is widely perceived as a less harmful alternative to cigarette smoking, yet cumulative epidemiological and clinical evidence indicates comparable or greater toxicant exposure. This scientific review evaluates the hematological alterations induced by chronic hookah smoking, synthesizing data from clinical comparative studies in Al-Najaf governorate (encompassing 60 hookah smokers and 30 healthy non-smoking controls) alongside international literature. The evidence demonstrates statistically significant elevations in red blood cell (RBC) count, hemoglobin (Hb), packed cell volume (PCV), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH) among hookah smokers compared to non-smokers ($p < 0.05$). Frequency-dependent analysis reveals a clear dose–response relationship, with significantly higher Hb levels in individuals smoking ≥ 8 sessions per week compared to <8 sessions per week ($p < 0.05$). Conversely, Hb levels show no significant difference between individuals with <10 years versus ≥ 10 years of smoking history ($p > 0.05$), indicating early and sustained hematopoietic adaptation. White blood cell (WBC), MCHC, and platelet counts show no significant alterations in young/social smokers ($p > 0.05$). These findings confirm that chronic hookah smoking induces secondary polycythemia via carbon monoxide–mediated tissue hypoxia and compensatory erythropoiesis. Recommendations for expanded multi-center investigations are provided.

1. Introduction

Tobacco smoking remains the leading cause of preventable morbidity and mortality worldwide, accounting for millions of deaths annually. While global cigarette smoking prevalence has experienced a steady decline—decreasing from 23.5% in 2007 to 20.7% in 2015 among adults—population growth has maintained the total number of global smokers near 1.1 billion [1, 2]. Combustion of tobacco delivers nicotine and a complex toxicant mixture into the pulmonary capillary bed, triggering systemic pathological cascades [3, 4].

Hookah as a Distinct Exposure Pathway

Hookah (also known as shisha, narghile, or waterpipe) is a traditional tobacco smoking device in which smoke passes through a water basin prior to inhalation. Although deeply rooted in Middle Eastern culture, its use has surged globally among youth and young adults, driven by misperceptions of safety [5]. A critical review of toxicant mechanics reveals that water filtration does not eliminate heavy metals, carbon monoxide (CO), or carcinogenic hydrocarbons [6]. Whereas a single cigarette involves approximately 20 puffs and 500–600 mL of

smoke volume, a standard 20–80 minute hookah session exposes the user to 50–200 puffs and up to 90,000 mL of smoke volume [7, 8]. Furthermore, the burning charcoal used to heat the tobacco substrate contributes substantial amounts of CO, heavy metals, and polycyclic aromatic hydrocarbons [9].

Comparative Pathophysiology: Hookah vs. Cigarettes

Hookah smoke delivers toxicant levels comparable to or exceeding those of multiple cigarettes [6, 10]. In addition to chronic exposure to nicotine—the primary neurobiological driver of addiction—hookah users exhibit deeper and more prolonged inhalation patterns, maximizing alveolar absorption. Epidemiological studies firmly associate chronic waterpipe smoking with pulmonary dysfunction, cardiovascular diseases, oral and lung malignancies, and reduced fertility [7, 11]. Shared mouthpieces also present infectious disease risks, including tuberculosis and viral hepatitis transmission.

Mechanistic Basis of Hematological Adaptations

The primary driver of hematological alterations in hookah smokers is carbon monoxide (CO) inhalation. CO binds hemoglobin with an affinity 200–250 times greater than oxygen, forming carboxyhemoglobin (COHb) and compromising systemic oxygen delivery [12]. The resulting tissue hypoxia acts on renal juxtaglomerular cells to stimulate erythropoietin secretion, promoting bone marrow erythroid proliferation—a state termed secondary polycythemia [13, 14]. Concurrently, cellular stress, systemic oxidative burden, and vasoactive smoke constituents modulate red cell indices, microvascular rheology, and inflammatory responses.

2. Synthesis of Empirical Findings (Al-Najaf Cohort Study)

To evaluate the physiological impact of hookah smoking in local regional contexts, empirical research conducted in Al-Najaf governorate analyzed complete blood count (CBC) profiles among 90 healthy male subjects (60 hookah smokers and 30 non-smoking controls) free of chronic underlying disease. The summarized empirical findings provide key insights into erythrocytic and leukocytic responses as shown in Table 1.

Table 1: Hematological parameters of hookah smokers and non-smokers

Hematological Parameter	Non-Smokers (Control)	Hookah Smokers	Statistical Significance
Red Blood Cells (RBC)	4.36 ± 0.12 M/μL	5.20 ± 0.11 M/μL	$p < 0.05$ (Elevated)
Hemoglobin (Hb)	11.15 ± 0.35 g/dL	14.68 ± 0.28 g/dL	$p < 0.05$ (Elevated)
Packed Cell Volume (PCV)	33.35 ± 0.95 %	42.87 ± 0.82 %	$p < 0.05$ (Elevated)
Mean Corpuscular Volume (MCV)	77.80 ± 1.10 fL	81.00 ± 0.95 fL	$p < 0.05$ (Elevated)
Mean Corpuscular Hemoglobin (MCH)	25.02 ± 0.45 pg	28.00 ± 0.40 pg	$p < 0.05$ (Elevated)
White Blood Cells (WBC)	6.85 ± 0.30 K/μL	7.10 ± 0.25 K/μL	$p > 0.05$ (No change)
MCHC	33.10 ± 0.40 g/dL	33.40 ± 0.35 g/dL	$p > 0.05$ (No change)
Platelets	245 ± 12 K/μL	252 ± 10 K/μL	$p > 0.05$ (No change)

3. Critical Discussion & Physiological Interpretation

Red Cell Mass & Secondary Polycythemia: The significant increase in RBC count, Hb, and PCV observed among hookah smokers aligns with established international findings [12, 15, 16]. The primary pathophysiology centers on CO-induced functional anemia. Because carboxyhemoglobin reduces oxygen carrying capacity, peripheral and renal tissues experience persistent hypoxia, triggering erythropoietin upregulation and compensatory polycythemia.

Erythrocyte Indices (MCV & MCH): A key observation is the elevation in MCV (81.00 fL vs 77.80 fL) and MCH (28.00 pg vs 25.02 pg) in hookah smokers. Accelerated erythropoiesis releases an increased proportion of immature reticulocytes—which possess larger cell volumes—into active circulation [14]. Toxin-induced oxidative stress may also impair red cell membrane stability, contributing to structural alterations [13, 17].

Dose-Response Relationship (Frequency): Hb levels were significantly higher in individuals smoking ≥ 8 hookah sessions per week (15.46 g/dL) compared to < 8 sessions per week (13.98 g/dL; $p < 0.05$). This demonstrates a robust dose–response relationship: higher cumulative toxicant and CO loading forces greater erythropoietic adaptation [18, 19].

Chronicity & Biological Plateau (Years of Smoking): In contrast, Hb levels showed no significant difference between smokers with < 10 years of exposure (14.57 g/dL) and those with ≥ 10 years (14.51 g/dL; $p > 0.05$). This plateau effect indicates that hematopoietic adaptation reaches maximum functional compensation relatively early in hookah smoking history, establishing a plateau rather than continuing to escalate linearly indefinitely.

Leukocytic & Platelet Stability: The absence of statistically significant changes in WBC and platelet counts in this cohort suggests that systemic inflammatory response and megakaryocytic stimulation may require prolonged, high-intensity exposure or older subject demographics, whereas erythrocyte elevation remains the early hallmark.

4. Systematic Conclusions & Clinical Recommendations

Core Conclusions

- Chronic hookah smoking induces secondary polycythemia, characterized by statistically significant elevations in RBC count, Hemoglobin, and PCV.
- A subtle shift toward higher MCV and MCH occurs, reflecting reticulocytosis and red cell membrane adaptations under hypoxic stress.
- Erythrocytic responses exhibit a clear weekly frequency dose–response relationship ($\geq$ 8 sessions/week causing greater Hb elevation).
- Hematopoietic compensation reaches a biological ceiling early in smoking duration, showing no significant difference between $<$ 10 and $\geq$ 10 years of exposure.
- Leukocyte and platelet counts remain stable in young/social hookah smokers lacking secondary systemic inflammatory disease.

Scientific Recommendations for Future Research

- Conduct prospective multi-center longitudinal studies incorporating female cohorts and diverse age brackets.
- Investigate the impact of hookah smoking on lipid metabolism, atherogenic indices, and endothelial function.
- Evaluate cardiovascular hemodynamics, blood rheology, and systemic hypertension risks linked to elevated blood viscosity.
- Assess chronic pulmonary physiological changes using spirometry and diffusion capacity testing (DLCO).
- Examine the public health risks of infectious disease transmission associated with shared waterpipe apparatuses.

Article Information

Ethical Approval: Not applicable.

Informed Consent: Written informed consent was obtained from all participants before their inclusion in the study.

Data Availability: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Conflict of Interest: The authors declare that they have no conflict of interest.

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