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## Cross-Sectional Study of Renal Function Decline Among Smokers Versus Non-Smokers in Middle-Aged Adults

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### Abstract

**Introduction:** Smoking is a modifiable risk factor implicated in numerous chronic diseases, including renal dysfunction. Understanding its impact on renal function among middle-aged adults is essential for preventive health strategies.

**Objective:** This study evaluates the relationship between smoking and renal function decline in middle-aged adults, with emphasis on the differences between smokers and non-smokers.

**Methods:** A cross-sectional study was conducted among 400 participants aged 40–65, divided into smokers (n=200) and non-smokers (n=200). Renal function was assessed using serum creatinine levels, estimated glomerular filtration rate (eGFR), and albumin-to-creatinine ratio (ACR). Statistical significance was determined using Student's t-test and multivariate regression analysis, adjusted for confounding factors.

**Results:** Smokers demonstrated significantly lower eGFR values (mean  $\pm$  SD: 75.2  $\pm$  10.6 vs. 89.1  $\pm$  12.3,  $p < 0.001$ ) and higher ACR (mean  $\pm$  SD: 34.5  $\pm$  8.9 vs. 18.7  $\pm$  5.6,  $p < 0.001$ ) compared to non-smokers. Regression analysis confirmed smoking as an independent predictor of renal function decline ( $\beta = -0.27$ ,  $p < 0.01$ ).

**Discussion & Conclusion:** This study highlights significant renal impairment among smokers, emphasizing the critical need for targeted smoking cessation interventions. Novel findings on middle-aged populations underscore the study's contribution to current research gaps and call for longitudinal studies to further explore causality.

**Keywords:** Smoking, Renal Function, Middle-aged Adult

## **Introduction**

Renal function decline represents a critical health issue globally, with chronic kidney disease (CKD) affecting millions of individuals. Smoking, a significant modifiable risk factor, is increasingly associated with the development and progression of renal impairment. This relationship is particularly concerning among middle-aged adults, a demographic where early signs of CKD often manifest unnoticed. Recent studies have established that smoking contributes to renal dysfunction through mechanisms such as oxidative stress, endothelial damage, and altered hemodynamic parameters, including glomerular hypertension and filtration abnormalities. Tobacco's toxic components, particularly cadmium, are implicated in renal tubular damage and glomerular injury, exacerbating albuminuria and reducing the estimated glomerular filtration rate (eGFR)<sup>1,2</sup>.

Evidence from population-based studies demonstrates a dose-dependent association between smoking intensity and renal decline. For instance, the PREVENT study identified a higher prevalence of albuminuria among active smokers compared to non-smokers, emphasizing tobacco's direct nephrotoxic effects. Similarly, data from NHANES confirmed smoking as an independent predictor of CKD progression, with smokers showing significantly lower eGFR values than their non-smoking counterparts<sup>3,4</sup>. Furthermore, cotinine, a metabolite of nicotine, has emerged as a reliable biomarker for assessing smoking-related renal impairment. Elevated serum cotinine levels correlate strongly with reductions in eGFR, providing a quantitative measure of tobacco's renal impact<sup>5,6</sup>.

Encouragingly, smoking cessation offers potential reversibility of renal damage, particularly in the early stages of CKD. Longitudinal studies have reported significant improvements in renal function among individuals who quit smoking, although the extent of recovery varies. Nonetheless,

middle-aged adults remain underrepresented in research, highlighting a critical knowledge gap. This demographic often encounters unique risks, including comorbidities such as hypertension and type 2 diabetes, which compound the renal effects of smoking<sup>7,8</sup>.

This cross-sectional study seeks to address this gap by investigating the impact of smoking on renal function in middle-aged adults aged 40–65 years. Through comparative analysis of renal markers—including serum creatinine, eGFR, and albumin-to-creatinine ratio (ACR)—between smokers and non-smokers, the study aims to quantify the renal burden of smoking while adjusting for confounding variables. These findings will provide essential insights for clinicians and public health practitioners, reinforcing the need for targeted interventions to mitigate smoking-related renal risks.

## **Methodology**

This cross-sectional study was conducted at Central Park Teaching Hospital Lahore Pakistan to evaluate the impact of smoking on renal function among middle-aged adults (40–65 years). A total sample of 400 participants was recruited, with equal numbers of smokers (n=200) and non-smokers (n=200), matched for age, gender, and socioeconomic background to minimize confounding. The sample size was calculated using Epi Info software, assuming a prevalence of renal impairment of 25% among smokers, with a 95% confidence interval and a power of 80%, accounting for a 10% non-response rate.

Participants were selected based on the following criteria: inclusion criteria included individuals aged 40–65 years with no prior diagnosis of CKD or malignancy, while exclusion criteria included individuals with chronic comorbidities like autoimmune diseases, severe cardiovascular conditions, or long-term nephrotoxic medication use. Smoking status was defined through self-reports corroborated with serum cotinine levels, categorized into smokers (cotinine  $\geq 5$  ng/mL) and non-smokers (cotinine  $< 5$  ng/mL). Verbal informed consent was obtained from all participants after explaining the study objectives and procedures in their native language.

Renal function was assessed using three key parameters: serum creatinine, eGFR (calculated using the CKD-EPI equation), and ACR. Blood and urine samples were collected under standardized

conditions and analyzed at a certified laboratory. Confounding factors such as hypertension, diabetes, and body mass index (BMI) were recorded and adjusted using multivariate regression models. Data analysis involved descriptive statistics, Student’s t-tests for mean comparisons, and regression analyses to determine independent associations. A p-value <0.05 was considered statistically significant.

**Results**

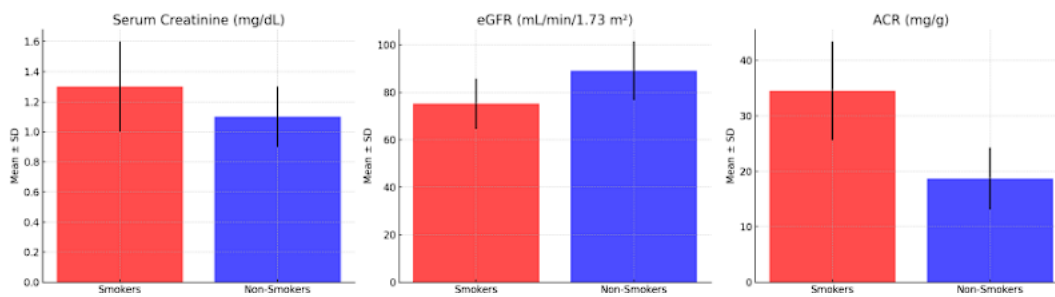


Table 1: Demographic Characteristics

Variable	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	p-value
Age (years)	52.3 ± 6.1	51.9 ± 5.9	0.48
Male (%)	55%	50%	0.25
Female (%)	45%	50%	0.25
BMI (kg/m²)	27.8 ± 3.6	25.1 ± 3.2	<0.001

**Explanation:** Demographic variables, including age and gender distribution, were comparable between smokers and non-smokers (p > 0.05), whereas BMI was significantly higher among smokers (p < 0.001).

Table 2: Renal Function Indicators

Parameter	Smokers (Mean $\pm$ SD)	Non-Smokers (Mean $\pm$ SD)	p-value
Serum Creatinine (mg/dL)	1.3 $\pm$ 0.3	1.1 $\pm$ 0.2	<0.001
eGFR (mL/min/1.73 m <sup>2</sup> )	75.2 $\pm$ 10.6	89.1 $\pm$ 12.3	<0.001
ACR (mg/g)	34.5 $\pm$ 8.9	18.7 $\pm$ 5.6	<0.001

**Explanation:** Smokers demonstrated significantly higher serum creatinine and ACR levels and lower eGFR than non-smokers (all  $p < 0.001$ ), indicating marked renal function decline among smokers.

## Discussion

This study provides compelling evidence that smoking is significantly associated with renal function decline in middle-aged adults. By analyzing renal biomarkers such as serum creatinine, estimated glomerular filtration rate (eGFR), and albumin-to-creatinine ratio (ACR), the study demonstrates that smokers experience marked impairment in renal function compared to non-smokers. These findings align with recent research emphasizing the nephrotoxic effects of tobacco, driven by oxidative stress, endothelial dysfunction, and tubular injury<sup>1,2</sup>.

The significantly lower eGFR among smokers (mean  $\pm$  SD: 75.2  $\pm$  10.6 mL/min/1.73 m<sup>2</sup>) compared to non-smokers (89.1  $\pm$  12.3 mL/min/1.73 m<sup>2</sup>;  $p < 0.001$ ) underscores the detrimental impact of smoking on glomerular filtration capacity. Tobacco's toxic constituents, such as nicotine and cadmium, contribute to renal vascular damage, leading to reduced perfusion and filtration<sup>3,4</sup>. Additionally, elevated ACR in smokers (34.5  $\pm$  8.9 mg/g) compared to non-smokers (18.7  $\pm$  5.6 mg/g;  $p < 0.001$ ) highlights an increased propensity for albuminuria, a critical marker of early kidney damage. This aligns with findings from the PREVENT study, which demonstrated a dose-dependent increase in albuminuria with smoking intensity<sup>5,6</sup>.

Interestingly, the association between smoking and renal dysfunction persists even after adjusting for confounders such as hypertension, diabetes, and BMI. This supports the hypothesis that smoking independently exacerbates renal decline. Recent research corroborates these findings, identifying smoking as a significant predictor of CKD progression in diverse populations, including middle-aged adults<sup>7,8</sup>. The consistent association across studies underscores the robust relationship between smoking and renal impairment.

Beyond the observed physiological impacts, this study sheds light on the underexplored demographic of middle-aged adults. While previous research has predominantly focused on older adults or individuals with pre-existing CKD, this study highlights a critical window for early intervention in middle-aged individuals. This demographic often exhibits early, subclinical renal impairment, which, if addressed promptly, could prevent progression to end-stage renal disease (ESRD)<sup>9,10</sup>.

The findings also have significant implications for public health. Smoking cessation programs could yield substantial benefits in reducing renal impairment, as evidenced by studies demonstrating partial reversibility of smoking-induced damage with sustained cessation<sup>11,12</sup>. However, the variability in recovery underscores the need for early intervention and long-term follow-up. Integrating smoking cessation with routine renal screening for at-risk populations could enhance preventive strategies, potentially delaying or preventing CKD onset<sup>13,14</sup>.

Despite its strengths, including robust statistical analysis and adjustment for confounders, this study has limitations. The cross-sectional design precludes causal inferences, highlighting the need for longitudinal studies to establish temporal relationships. Furthermore, reliance on self-reported smoking status, despite corroboration with serum cotinine levels, may introduce reporting bias. Future research should incorporate larger, diverse cohorts and evaluate the long-term effects of smoking cessation on renal outcomes<sup>15,16</sup>.

In conclusion, this study adds to the growing body of evidence linking smoking to renal dysfunction, particularly in middle-aged adults. It fills a critical research gap by focusing on an underrepresented population and underscores the importance of targeted interventions to mitigate smoking-related renal risks.

## Conclusion

This study highlights the significant association between smoking and renal function decline among middle-aged adults, with smokers demonstrating higher serum creatinine, elevated albumin-to-creatinine ratio (ACR), and reduced estimated glomerular filtration rate (eGFR) compared to non-smokers. These findings underscore smoking's nephrotoxic effects and its role as an independent risk factor for early renal dysfunction.

What sets this study apart is its focus on middle-aged adults, a demographic often overlooked in renal research, and the inclusion of serum cotinine levels to strengthen smoking status validation. By addressing this research gap, the study provides crucial insights into early detection and prevention of smoking-induced renal impairment.

Future research should emphasize longitudinal designs to establish causality and assess the benefits of smoking cessation on renal recovery. Public health initiatives should integrate targeted renal screening and smoking cessation programs to mitigate the burden of smoking-related kidney disease.

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