E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.48047/ijprt/16.02.844

Research Article

Association Between Tobacco Use, Oral Health Status, and Cardiovascular Risk Factors in Adults

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Abstract

Background: Tobacco consumption represents a primary preventable cause of global morbidity and mortality, contributing significantly to both oral mucosal/periodontal degradation and systemic cardiovascular pathology. While the individual links connecting tobacco use to periodontal disease and atherogenesis are documented, the integrated relationship between tobacco modalities, standardized oral health metrics, and clinical cardiovascular risk profiles in a unified adult cohort requires comprehensive characterization.

Objective: To evaluate the association between different modalities of tobacco use, oral health status (periodontal health and tooth loss), and systemic cardiovascular risk factors among adult patients.

Methods: This hospital-based cross-sectional study evaluated 160 adult participants (aged 25 to 65 years) attending outpatient medicine and dental clinics at Rai Medical College. Participants were categorized based on self-reported tobacco history into non-tobacco users (n = 50), smoked tobacco users (n = 55), and smokeless tobacco users (n = 55). Oral health status was assessed via standardized dental examination, recording the Community Periodontal Index for Treatment

Needs (CPITN), Clinical Attachment Loss (CAL in mm), Simplified Oral Hygiene Index (OHI-S), and total missing teeth. Systemic cardiovascular risk factors—including resting blood pressure, fasting plasma glucose, lipid profile parameters (total cholesterol, triglycerides, LDL, HDL), and calculated 10-year Framingham Risk Scores (FRS)—were compiled. Bivariate comparisons, Spearman correlations, and multivariable logistic regression analyses were conducted.

Results: The mean age of the total cohort was 46.8 +/- 10.4 years (62.5 percent male). Severe periodontal disease (CPITN Code 4) was significantly more prevalent in smoked tobacco users (45.5 percent) and smokeless tobacco users (40.0 percent) compared to non-tobacco users (12.0 percent, $p < 0.001$). Mean Clinical Attachment Loss was greatest among smoked tobacco users (4.8 +/- 1.4 mm) followed by smokeless tobacco users (4.2 +/- 1.2 mm) and non-users (2.1 +/- 0.8 mm, $p < 0.001$). Systolic blood pressure, resting heart rate, serum triglycerides, and total cholesterol/HDL ratios were significantly elevated in both tobacco groups relative to non-users ($p < 0.001$). High Framingham Cardiovascular Risk (FRS > 20 percent) was identified in 38.2 percent of smoked tobacco users, 30.9 percent of smokeless tobacco users, and 8.0 percent of non-users ($p < 0.001$). Multivariable logistic regression revealed that severe periodontal disease (adjusted OR = 3.12, 95 percent CI: 1.42 to 6.85, $p = 0.004$) and current smoked tobacco use (adjusted OR = 4.28, 95 percent CI: 1.85 to 9.92, $p = 0.001$) were independent predictors of high 10-year cardiovascular risk.

Conclusion: Tobacco use, irrespective of delivery modality, is strongly associated with advanced periodontal breakdown and an adverse cardiovascular risk profile. The significant independent correlation between severe oral health degradation and elevated systemic cardiovascular risk underscores the necessity of integrated oral-systemic health interventions and tobacco cessation programs in primary healthcare settings.

Keywords: Tobacco use; Periodontal disease; Oral health; Cardiovascular risk; Framingham Risk Score; Hypertension; Dyslipidemia; Systemic inflammation.

1. Introduction

Tobacco use remains one of the greatest global public health threats, accounting for over 8 million deaths annually worldwide. Tobacco consumption manifests in multiple forms, including smoked tobacco (cigarettes, cigars, bidi, waterpipe/shisha) and smokeless tobacco (chewing tobacco, gutka, paan masala, naswar, snuff). Both delivery modalities introduce a complex mixture of

chemical carcinogens, heavy metals, nicotine, and reactive oxygen species into the oral cavity and systemic circulation.[1-3]

The oral cavity serves as the primary portal of entry for tobacco exposure. Tobacco combustion products and smokeless compounds induce localized tissue hypoxia, microvascular constriction, suppression of neutrophil chemotaxis, and altered subgingival microbial ecosystems. Consequently, tobacco users experience accelerated periodontal tissue destruction, manifested as deeper periodontal probing depths, advanced clinical attachment loss, alveolar bone resorption, and premature tooth loss. Furthermore, chronic mucosal irritation from smokeless tobacco additives promotes localized oral epithelial dysplastic changes. [4-6]

Concurrently, tobacco use is a well-established major independent risk factor for cardiovascular disease (CVD), including coronary artery disease, myocardial infarction, hypertension, stroke, and peripheral arterial disease. Nicotine triggers sympathetic nervous system activation, elevating resting heart rate, blood pressure, and myocardial oxygen demand. Systemically absorbed combustion byproducts drive endothelial dysfunction, intravascular oxidative stress, systemic systemic inflammatory cascades, and pro-thrombotic platelet aggregation, accelerating atherogenesis. [7-9]

In recent years, the bidirectional paradigm connecting oral inflammation to systemic vascular disease has gained significant attention. Chronic periodontitis acts as a persistent low-grade systemic inflammatory stressor. Periodontopathic bacteria (such as Porphyromonas gingivalis) and locally produced pro-inflammatory cytokines (IL-1 beta, IL-6, TNF-alpha) enter the bloodstream, driving hepatic secretion of acute-phase reactants such as C-reactive protein (CRP). This persistent low-grade systemic bacteremia and cytokine load accelerates arterial stiffness, endothelial injury, and plaque instability. [10]

Although the individual links between tobacco and oral health, as well as tobacco and cardiovascular risk, are established, there is a need to systematically evaluate how different modalities of tobacco use interrelate with standardized oral health indices and clinical cardiovascular risk profiles within a single, prospective observational framework.

Therefore, this cross-sectional study was undertaken to investigate the associations between tobacco use patterns, quantitative oral health metrics, and systemic cardiovascular risk factors in an adult population.

2. Rationale of the Study

Tobacco users frequently present with co-existing, severe oral mucosal/periodontal disease and unmanaged systemic cardiovascular risk factors. In many healthcare settings, oral health and cardiovascular care operate in isolation. Evaluating the trilateral relationship between specific tobacco modalities (smoked vs. smokeless), standardized oral health indices (CPITN, CAL, OHI-S), and clinical cardiovascular risk metrics (FRS, lipid parameters, blood pressure) provides crucial evidence for medical and dental practitioners to implement integrated risk screening, cross-disciplinary referrals, and targeted tobacco cessation interventions.

3. Objectives

Primary Objective

To assess the association between tobacco use modalities (smoked and smokeless tobacco), oral health status (Community Periodontal Index for Treatment Needs, Clinical Attachment Loss), and 10-year Framingham Cardiovascular Risk Scores in adult patients.

Secondary Objectives

1. To compare periodontal parameters (CPITN, CAL, OHI-S) and missing tooth counts across non-tobacco users, smoked tobacco users, and smokeless tobacco users.
2. To evaluate differences in hemodynamic variables (blood pressure, heart rate) and lipid profiles (cholesterol, triglycerides, LDL, HDL) among tobacco user groups.
3. To analyze bivariate correlations between quantitative periodontal destruction metrics and clinical cardiovascular risk markers.
4. To establish a multivariable logistic regression model determining independent predictors of high 10-year cardiovascular risk.

4. Materials and Methods

4.1 Study Design and Setting

A hospital-based cross-sectional observational study was conducted at Rai Medical college.

4.2 Study Duration

The study was conducted over an eight-month period, encompassing participant screening, standardized oral health examination, clinical cardiovascular risk parameter recording, blood sample collection, laboratory processing, and statistical modeling.

4.3 Study Population

Adult male and female patients aged 25 to 65 years attending the outpatient medical and dental clinics.

4.4 Sample Size

A sample size of 160 adult participants was enrolled. The sample size was calculated using G-Power software for one-way Analysis of Variance (ANOVA) and Chi-square tests across three comparative groups, assuming a medium effect size (f = 0.28), an alpha error level of 0.05, and a statistical power of 85 percent.

4.5 Inclusion Criteria

1. Adults aged 25 to 65 years.
2. Willingness to undergo standardized oral clinical examination and blood sampling.
3. Categorization into one of three defined tobacco exposure groups:
    - Non-tobacco users: No history of tobacco use or quit > 5 years prior.
    - Smoked tobacco users: Current daily smoking (cigarettes, bidi, waterpipe) for >= 2 years.
    - Smokeless tobacco users: Current daily use of smokeless products (chewing tobacco, gutka, paan with tobacco, naswar) for >= 2 years.
4. Provision of written informed consent.

4.6 Exclusion Criteria

1. Dual tobacco users (simultaneous current daily use of both smoked and smokeless tobacco) to ensure clean group stratification.
2. Prior history of documented major cardiovascular events (myocardial infarction, stroke, heart failure, coronary revascularization) to allow accurate primary risk estimation.
3. History of systemic immunosuppressive therapy, active chemotherapy, or systemic antibiotic/scaling therapy within the preceding 3 months.
4. Pregnancy or active lactation.
5. Completely edentulous patients lacking scorable teeth for periodontal indices.

4.7 Assessment of Tobacco Exposure

Detailed self-reported tobacco history was recorded using a structured questionnaire. Metrics included duration of habit (years), daily frequency of use, type of product consumed, and cumulative package-years (for smokers) or daily dose equivalents (for smokeless users).

4.8 Clinical Oral Health Examination

Oral examinations were performed by two calibrated dental surgeons using a WHO periodontal probe, mouth mirror, and standardized lighting. Recorded metrics included:

- Community Periodontal Index for Treatment Needs (CPITN): Scored from 0 to 4 (0 = Healthy; 1 = Bleeding on probing; 2 = Calculus present; 3 = Shallow pockets 4–5 mm; 4 = Deep pockets >= 6 mm).
- Clinical Attachment Loss (CAL in mm): Measured from the cemento-enamel junction (CEJ) to the base of the periodontal pocket across six sites per tooth.
- Simplified Oral Hygiene Index (OHI-S): Sum of Debris Index (DI-S) and Calculus Index (CI-S), scored 0 to 6.
- Total Tooth Loss: Number of missing teeth (excluding third molars).

4.9 Cardiovascular Risk Profile Assessment

- Hemodynamic Parameters: Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured after 10 minutes of quiet rest using a calibrated digital sphygmomanometer. Resting heart rate (bpm) was recorded.
- Laboratory Biochemistry: Venous blood samples were collected after a 12-hour overnight fast. Measured parameters included Fasting Plasma Glucose (FPG), Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), and Low-Density Lipoprotein Cholesterol (LDL-C) measured via automated enzymatic chemistry analyzers.
- Framingham Risk Score (FRS): Calculated for each participant using age, sex, TC, HDL-C, SBP, antihypertensive medication status, smoking status, and diabetes status. FRS was categorized into Low Risk (< 10 percent), Moderate Risk (10 to 20 percent), and High Risk (> 20 percent 10-year risk of hard coronary events).

4.10 Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were assessed for normality using the Kolmogorov-Smirnov test and expressed as mean +/- standard deviation. Categorical variables were presented as absolute numbers and percentages.

Inter-group comparisons among non-tobacco users, smoked tobacco users, and smokeless tobacco users were conducted using One-Way ANOVA followed by Tukey's post-hoc test for continuous parameters, and Pearson's Chi-square test for categorical variables. Spearman rank correlation coefficients assessed linear relationships between oral health indices (CAL, OHI-S, missing teeth) and cardiovascular parameters (FRS, SBP, lipid ratios). Multivariable binary logistic regression was constructed to evaluate independent predictors of High 10-Year Cardiovascular Risk (FRS > 20 percent). Statistical significance was defined as a two-sided $p < 0.05$.

5. Results

5.1 Baseline Demographic and Clinical Characteristics

The study cohort comprised 160 adults (mean age 46.8 +/- 10.4 years; 100 males [62.5 percent], 60 females [37.5 percent]). The cohort was stratified into non-tobacco users (n = 50), smoked

tobacco users (n = 55), and smokeless tobacco users (n = 55). Age and gender distribution across the three groups were comparable (p > 0.05).

Table 1: Baseline demographic characteristics of participants stratified by tobacco exposure.

Variable	Non-Tobacco Users (n = 50)	Smoked Tobacco Users (n = 55)	Smokeless Tobacco Users (n = 55)	Total Cohort (n = 160)	p-value
Age (years)	45.2 +/- 11.2	47.8 +/- 9.8	47.2 +/- 10.1	46.8 +/- 10.4	0.412
Male Sex, n (%)	28 (56.0)	38 (69.1)	34 (61.8)	100 (62.5)	0.384
Female Sex, n (%)	22 (44.0)	17 (30.9)	21 (38.2)	60 (37.5)	0.384
Body Mass Index (kg/m squared)	26.2 +/- 3.8	25.4 +/- 4.1	26.8 +/- 4.2	26.1 +/- 4.0	0.218
Mean Duration of Tobacco Use (years)	Not Applicable	16.4 +/- 7.2	14.8 +/- 6.8	Not Applicable	0.234

5.2 Comparison of Oral Health Parameters

Oral health parameters demonstrated marked deterioration in both tobacco user groups compared to non-tobacco users (p < 0.001). Severe periodontal destruction (CPITN Code 4) was present in 45.5 percent of smoked tobacco users and 40.0 percent of smokeless tobacco users, compared to 12.0 percent of non-users (p < 0.001). Clinical Attachment Loss was highest among smoked tobacco users (4.8 +/- 1.4 mm), followed by smokeless tobacco users (4.2 +/- 1.2 mm) and non-users (2.1 +/- 0.8 mm, p < 0.001).

Table 2: Comparison of oral health metrics across tobacco exposure groups.

Oral Health Parameter	Non-Tobacco Users (n = 50)	Smoked Tobacco Users (n = 55)	Smokeless Tobacco Users (n = 55)	p-value
CPITN Code Distribution, n (%)				<0.001
- Code 0 to 2 (Healthy / Calculus)	24 (48.0)	6 (10.9)	8 (14.5)	
- Code 3 (Shallow Pockets 4–5 mm)	20 (40.0)	24 (43.6)	25 (45.5)	
- Code 4 (Deep Pockets >= 6 mm)	6 (12.0)	25 (45.5)	22 (40.0)	
Clinical Attachment Loss (mm)	2.1 +/- 0.8	4.8 +/- 1.4	4.2 +/- 1.2	<0.001
Simplified Oral Hygiene Index (OHI-S)	1.8 +/- 0.6	3.6 +/- 0.9	3.2 +/- 0.8	<0.001
Total Missing Teeth (n)	2.2 +/- 1.4	5.8 +/- 2.6	4.9 +/- 2.2	<0.001

5.3 Systemic Cardiovascular Risk Factors and Framingham Risk Scores

Hemodynamic measurements, lipid profiles, and Framingham Risk Scores revealed significantly elevated cardiovascular risk profiles in tobacco users ($p < 0.001$). Systolic blood pressure, resting heart rate, serum triglycerides, and TC/HDL ratios were significantly higher in both smoked and smokeless tobacco groups compared to non-users. High 10-year Framingham Cardiovascular Risk (FRS > 20 percent) was identified in 38.2 percent of smokers, 30.9 percent of smokeless users, and 8.0 percent of non-users ($p < 0.001$).

Table 3: Comparison of cardiovascular risk factors and lipid profiles among groups.

Cardiovascular Risk Parameter	Non-Tobacco Users (n = 50)	Smoked Tobacco Users (n = 55)	Smokeless Tobacco Users (n = 55)	p-value
Systolic Blood Pressure (mmHg)	122.4 +/- 11.2	136.8 +/- 15.4	134.2 +/- 14.2	<0.001
Diastolic Blood Pressure (mmHg)	78.2 +/- 7.4	86.4 +/- 9.2	85.1 +/- 8.8	<0.001
Resting Heart Rate (bpm)	72.4 +/- 6.8	82.6 +/- 8.4	79.8 +/- 7.8	<0.001
Fasting Plasma Glucose (mg/dL)	98.4 +/- 14.2	114.2 +/- 26.8	110.8 +/- 22.4	0.002
Total Cholesterol (mg/dL)	178.4 +/- 28.2	212.6 +/- 38.4	204.2 +/- 34.6	<0.001
Triglycerides (mg/dL)	132.6 +/- 32.4	188.4 +/- 52.6	176.2 +/- 48.2	<0.001
HDL Cholesterol (mg/dL)	48.2 +/- 8.4	38.6 +/- 7.2	40.2 +/- 7.6	<0.001
LDL Cholesterol (mg/dL)	103.6 +/- 22.4	136.2 +/- 31.8	128.8 +/- 28.6	<0.001
Framingham Risk Category, n (%)				<0.001
- Low Risk (< 10 percent)	32 (64.0)	12 (21.8)	16 (29.1)	
- Moderate Risk (10 to 20 percent)	14 (28.0)	22 (40.0)	22 (40.0)	
- High Risk (> 20 percent)	4 (8.0)	21 (38.2)	17 (30.9)	

5.4 Correlation Analysis

Spearman rank correlation analysis revealed significant positive correlations between Clinical Attachment Loss and Framingham Risk Score ($r = 0.48$, $p < 0.001$), Systolic Blood Pressure ($r = 0.42$, $p < 0.001$), and TC/HDL Ratio ($r = 0.38$, $p < 0.001$). Total missing teeth also correlated positively with FRS ($r = 0.41$, $p < 0.001$).

Table 4: Bivariate correlation matrix between oral health metrics and cardiovascular variables.

Parameter Pair	Spearman Correlation (r)	p-value
Clinical Attachment Loss vs. Framingham Risk Score	0.48	<0.001
Clinical Attachment Loss vs. Systolic Blood Pressure	0.42	<0.001
Clinical Attachment Loss vs. TC / HDL Ratio	0.38	<0.001
OHI-S Index vs. Triglycerides	0.35	<0.001
Total Missing Teeth vs. Framingham Risk Score	0.41	<0.001
Duration of Tobacco Use vs. Clinical Attachment Loss	0.52	<0.001

5.5 Multivariable Logistic Regression Analysis

In multivariable binary logistic regression modeling for High 10-Year Cardiovascular Risk (FRS > 20 percent), current smoked tobacco use (adjusted OR = 4.28, 95 percent CI: 1.85 to 9.92, p = 0.001), severe periodontal disease (CPITN Code 4; adjusted OR = 3.12, 95 percent CI: 1.42 to 6.85, p = 0.004), current smokeless tobacco use (adjusted OR = 2.94, 95 percent CI: 1.22 to 7.08, p = 0.016), and total missing teeth >= 5 (adjusted OR = 2.45, 95 percent CI: 1.12 to 5.36, p = 0.024) were confirmed as independent predictors.

Table 5: Multivariable logistic regression modeling predictors of high 10-year cardiovascular risk.

Predictor Variable	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Smoked Tobacco Use (vs. Non-Users)	6.88 (2.18–21.68)	<0.001	4.28 (1.85–9.92)	0.001
Smokeless Tobacco Use (vs. Non-Users)	5.08 (1.58–16.32)	0.006	2.94 (1.22–7.08)	0.016
Severe Periodontal Disease (CPITN Code 4)	5.24 (2.35–11.68)	<0.001	3.12 (1.42–6.85)	0.004
Total Missing Teeth >= 5	3.82 (1.82–8.02)	<0.001	2.45 (1.12–5.36)	0.024
Simplified Oral Hygiene Index > 3.0	3.14 (1.52–6.48)	0.002	1.82 (0.84–3.94)	0.128

6. Discussion

This cross-sectional study evaluated the trilateral association between tobacco consumption modalities, quantitative oral health metrics, and systemic cardiovascular risk profiles in an adult population. The primary findings confirm that both smoked and smokeless tobacco consumption are strongly associated with advanced periodontal breakdown, accelerated tooth loss, and an adverse clinical cardiovascular profile, with severe periodontal disease serving as an independent predictor of high 10-year cardiovascular risk. [11-13]

6.1 Impact of Tobacco Modalities on Oral Health Status

The destructive impact of tobacco on periodontal tissue is driven by distinct localized and systemic mechanisms. In smoked tobacco users, thermal radiation and combustion products cause microvascular vasoconstriction, reducing gingival blood flow and blunting the early inflammatory bleeding response. However, subgingival tissue destruction progresses silently, leading to deep periodontal pockets (CPITN Code 4), extensive clinical attachment loss (mean 4.8 mm), and premature tooth loss. [14-16]

Smokeless tobacco users similarly exhibit severe localized periodontal attachment loss (mean 4.2 mm) and elevated OHI-S scores. Direct placement of smokeless tobacco products against buccal/labial mucosal surfaces introduces high concentrations of nicotine, tobacco-specific nitrosamines (TSNAs), and alkaline additives. This causes localized gingival recession, mucosal hyperkeratosis, and alveolar bone destruction at the placement site.

6.2 Tobacco Use and Cardiovascular Risk Dynamics

Both smoked and smokeless tobacco modalities were associated with adverse cardiovascular risk profiles. Nicotine absorption, regardless of route, stimulates adrenal catecholamine release, increasing resting heart rate, peripheral vascular resistance, and blood pressure. In our cohort, systolic blood pressure was significantly higher in smokers (136.8 mmHg) and smokeless users (134.2 mmHg) compared to non-users (122.4 mmHg). [17-18]

Furthermore, tobacco consumption drives atherogenic dyslipidemia. Chronic nicotine and free radical exposure impairs lipoprotein lipase activity, elevating serum triglycerides and LDL cholesterol while depressing protective HDL cholesterol levels. The resulting elevation in the TC/HDL ratio, combined with smoking status and blood pressure, contributed to the significantly

higher prevalence of High Framingham Risk (FRS > 20 percent) in tobacco users (38.2 percent in smokers, 30.9 percent in smokeless users). [19-20]

6.3 The Oral-Cardiovascular Inter-Connection

A critical finding of this study is the independent association between severe periodontal disease (CPITN Code 4) and elevated 10-year cardiovascular risk (adjusted OR = 3.12). Chronic periodontitis provides a persistent ulcerated epithelial surface within periodontal pockets, enabling periodontopathic bacterial endotoxins and localized cytokines (IL-1, IL-6, TNF-alpha) to enter systemic circulation.

This systemic low-grade inflammatory burden stimulates hepatic production of C-reactive protein and fibrinogen, inducing systemic endothelial dysfunction, promoting LDL oxidation, and accelerating atheromatous plaque vulnerability. Thus, severe periodontal breakdown acts not merely as a localized oral pathology, but as an active systemic inflammatory contributor to cardiovascular risk.

6.4 Clinical and Public Health Implications

These findings highlight the necessity of breaking down traditional silos between dental and medical care. Dental practitioners evaluating tobacco users with severe periodontal disease should routinely measure blood pressure and screen for cardiovascular risk factors, referring high-risk patients for medical evaluation. Conversely, primary care physicians managing patients with hypertension, dyslipidemia, or tobacco dependence should incorporate basic oral health screening and encourage routine periodontal care as part of primary cardiovascular prevention.

6.5 Limitations

Several study limitations should be noted. First, the cross-sectional design precludes establishing direct temporal causality between periodontal disease progression and cardiovascular events. Second, systemic inflammatory biomarkers (such as high-sensitivity CRP or IL-6) were not measured in this primary clinical cohort. Third, dietary habits and physical activity levels were not fully controlled for in multivariable models.

7. Conclusion

Tobacco use, whether in smoked or smokeless form, is strongly associated with severe periodontal tissue destruction, increased tooth loss, and an adverse cardiovascular risk profile. Severe

periodontal disease and tobacco consumption independently predict high 10-year cardiovascular risk. Integrating routine periodontal screening and structured tobacco cessation programs into both dental and primary medical care workflows is essential to mitigate oral and systemic cardiovascular disease burdens.

8. Recommendations

1. Implement routine cardiovascular screening (blood pressure measurement, lipid profile assessment) for all tobacco users presenting with severe periodontal disease in dental clinics.
2. Incorporate basic oral health evaluations and dental referral pathways into primary care medical clinics managing patients with tobacco dependence and cardiovascular risk factors.
3. Provide comprehensive tobacco cessation counseling addressing both smoked and smokeless tobacco modalities in medical and dental settings.
4. Educate public health professionals and patients regarding the oral-systemic link connecting periodontal inflammation to cardiovascular disease.
5. Conduct prospective longitudinal cohort studies to evaluate whether professional periodontal therapy and sustained tobacco cessation collectively reduce systemic inflammatory biomarkers and long-term cardiovascular event rates.

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