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**Research Article**

# Association Between Tobacco Chewing Habits and Leukoplakia in Low-Income Urban Communities

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**Abstract:** Background: Chewable tobacco remains endemic in many South-Asian low-income urban communities and is strongly implicated in the pathogenesis of oral potentially malignant disorders, particularly leukoplakia. This community-based cross-sectional study quantified the association between specific tobacco-chewing behaviours and oral leukoplakia in disadvantaged city neighbourhoods. Methods: Between March 2022 and December 2023, 1,050 adults (aged 18–70) residing in three low-income urban clusters underwent door-to-door screening and oral clinical examination. Detailed habit histories (product type, frequency, duration, placement site, concomitant areca nut use) were recorded. A stratified random sub-sample (n = 420) underwent confirmatory brush cytology/biopsy when indicated. Sample size was computed in Epi Info™ to detect an odds ratio (OR) of 1.8 for leukoplakia among habitual chewers versus non-chewers with 90% power and 95% confidence. Results: Overall prevalence of leukoplakia was 9.8% (103/1,050). Current chewable tobacco use prevalence was 41.6%. Adjusted logistic regression showed strong associations between daily chewing ≥3 times/day (adjusted OR 3.9; 95% CI 2.6–5.7; $p < 0.001$), habit duration ≥10 years (aOR 2.8; 95% CI 1.9–4.1; $p < 0.001$), and use of unprocessed/unregulated products (naswar/khaini) (aOR 4.5; 95% CI 3.0–6.8; $p < 0.001$) with leukoplakia. Concurrent areca nut use conferred additive risk (aOR 2.3; 95% CI 1.5–3.4). Lesions were most commonly located at buccal mucosa and commissures; dysplasia on biopsy was present

in 18% of sampled lesions. Conclusion: In low-income urban communities, high frequency, long duration and use of unregulated chewable tobacco products are independently associated with markedly increased odds of oral leukoplakia. Public-health interventions should prioritise targeted cessation, product regulation and community screening to reduce premalignant burden. **Keywords:** tobacco chewing, leukoplakia, smokeless tobacco, low-income urban, oral premalignant lesions.

**Introduction:** Oral leukoplakia is the commonest clinically recognised oral potentially malignant disorder (OPMD), manifesting as white plaques that cannot be scraped off and that have variable propensity for dysplastic transformation. In South-Asia, where various forms of smokeless tobacco and betel-quid are widely practised, leukoplakia remains a major precursor lesion for oral squamous-cell carcinoma and contributes substantially to regional cancer burden. Recent community surveys and hospital-based series document a persistently elevated prevalence of OPMDs in populations with high smokeless-tobacco exposure, highlighting persistent public-health gaps.1-3

Smokeless tobacco products vary considerably by composition and mode of use — ranging from industrially processed commercial formulations to unregulated local products (for example naswar, khaini, gutkha and paan with tobacco) — and this heterogeneity affects mucosal exposure to nitrosamines, alkaloids and local irritants. Product-specific differences, along with frequency, duration, intraoral placement and co-exposure to areca nut and slaked lime, determine mucosal contact time and carcinogen dose, thus modulating risk of leukoplakia and malignant progression. Epidemiological analyses suggest that unprocessed or artisanal products often confer greater relative risk than standardised commercial products because of higher concentrations of carcinogenic constituents and prolonged mucosal retention.4-7

Socio-economic determinants strongly shape tobacco chewing patterns. Low-income urban communities frequently face higher prevalence of smokeless tobacco use driven by affordability, social norms, limited access to cessation services, and the informal availability of cheap local products. These structural factors compound biological risk and create pockets of elevated OPMD prevalence within cities. Recent community interventions and prevalence studies emphasise that

municipal and outreach-level efforts are required to identify and manage early lesions in such settings. 8-11

Although the causal link between smokeless tobacco and leukoplakia is well established, there remain important gaps in contemporary, community-level evidence characterising the relative contributions of product type, frequency and cumulative exposure in low-income urban populations. Many prior investigations were clinic-based or small in scale, and few have integrated cytopathology to corroborate clinical diagnosis in community screenings. Moreover, quantification of additive risk from areca nut — often chewed together with tobacco in the region — is essential because the combination may have supra-additive carcinogenic effects.12-13

To address these gaps, the present study conducted a large door-to-door survey in three socio-economically disadvantaged urban clusters, combining systematic clinical screening, detailed habitography and targeted histopathological confirmation. The aims were to: (1) estimate the prevalence of oral leukoplakia in adults living in low-income urban communities; (2) quantify associations between specific tobacco-chewing behaviours (product type, frequency, duration, placement) and the presence of leukoplakia; and (3) assess the incremental risk contributed by concurrent areca nut use. The working hypothesis posited that higher frequency, longer duration and use of unregulated tobacco products would be independently associated with substantially elevated odds of leukoplakia after adjustment for age, sex, alcohol use and oral hygiene.

**Methodology:** A community-based cross-sectional study with nested diagnostic confirmation at Ziauddin University, Karachi. Clusters were selected purposively to represent diverse patterns of informal economy, high population density and documented high prevalence of smokeless tobacco retail outlets. All adults aged 18–70 years permanently resident in selected households were eligible; pregnant women and those with severe systemic illness were excluded. Door-to-door enumeration identified 1,050 consenting participants who completed an interviewer-administered structured questionnaire covering socio-demographics, detailed tobacco and areca-nut habit history (product name, daily frequency, years of use, intraoral placement, concurrent smoking or alcohol), oral hygiene practices and past oral disease. The minimum required sample size was computed using Epi Info StatCalc for an unmatched cross-sectional design: assuming an anticipated leukoplakia prevalence of 8% among non-chewers and 14% among chewers (OR

≈1.8), 90% power, 95% confidence and 5% precision produced a sample requirement of 1,002; recruitment was set at 1,050 to allow for non-response. Oral examinations were performed by calibrated dental surgeons using standard lighting and disposable tongue depressors; lesions consistent with leukoplakia were photographed, mapped (anatomic site) and classified by morphology (homogeneous vs non-homogeneous). A stratified random sub-sample of 420 participants — oversampling those with suspected lesions and heavy-use chewers — underwent brush cytology and, where indicated and consented, excisional/incisional biopsy for histopathological confirmation and grading of dysplasia. All cytology/biopsy specimens were processed in a central histopathology laboratory with blinded assessment by senior oral pathologists. Data were double-entered and analysed in SPSS v26. Prevalence estimates used survey weights to account for cluster sampling. Bivariate comparisons employed chi-square tests and t-tests as appropriate. Multivariable logistic regression modelled odds of clinically detected leukoplakia, including covariates age, sex, education, alcohol use, oral hygiene, daily chewing frequency (≤1, 2, ≥3 times/day), duration (<5, 5–9, ≥10 years), product type (commercial processed, naswar/khaini/unprocessed, betel quid with tobacco), intraoral placement site (buccal, labial sulcus, gingivobuccal complex), and concurrent areca nut use; adjusted odds ratios (aOR) and 95% confidence intervals (CI) were reported. Statistical significance was set at $p < 0.05$. Ethical approval was obtained from the institutional review board and verbal and written informed consent was secured; participants with suspicious lesions were offered free referral and treatment.

**Results:** A total of 1,050 participants completed screening (response rate 94.6%); mean age was 39.8 ± 12.6 years, 54.3% were female and 72.1% reported monthly household income below the national median. Current chewable tobacco use was reported by 437 participants (41.6%); 284 (27.0%) used processed commercial products (packaged gutkha/pan masala), 116 (11.0%) used naswar/khaini (unprocessed local mixtures), and 37 (3.5%) chewed paan with tobacco. Areca nut was used by 328 (31.2%) participants, often in combination with tobacco.

**Table 1. Demographic and habit characteristics (n = 1,050)**

| Characteristic | Overall n (%) or mean ± SD |
|---|---|
| Age (years) | 39.8 ± 12.6 |

| Characteristic | Overall n (%) or mean ± SD |
|---|---|
| Female sex | 570 (54.3%) |
| Low household income | 757 (72.1%) |
| Current chewable tobacco use | 437 (41.6%) |
| Daily chewing ≥3 times/day | 198 (18.9%) |
| Habit duration ≥10 years | 246 (23.4%) |
| Concurrent areca nut use | 328 (31.2%) |

Table 1 summarises population characteristics and habit distributions; high prevalence of habitual chewing and prolonged durations were observed.

Clinically suspected leukoplakia was identified in 103 participants (crude prevalence 9.8%); the buccal mucosa/commissure was the most frequent site (61%), followed by gingivobuccal sulcus (22%) and labial mucosa (9%). Of the 420 participants who underwent cytology/biopsy, 94 clinically suspected lesions were sampled and histopathology confirmed leukoplakia in 78/94 (83.0%), with dysplasia (mild to moderate) present in 14/78 (17.9%).

**Table 2. Leukoplakia prevalence by tobacco habit (n = 1,050)**

| Habit category | Leukoplakia n (%) | Crude prevalence (%) |
|---|---|---|
| Non-chewers (n = 613) | 21 (3.4) | 3.4 |
| Chewers overall (n = 437) | 82 (18.8) | 18.8 |
| Processed product users (n = 284) | 32 (11.3) | 11.3 |
| Naswar/khaini users (n = 116) | 34 (29.3) | 29.3 |
| Daily ≥3 times/day (n = 198) | 51 (25.8) | 25.8 |
| Duration ≥10 years (n = 246) | 46 (18.7) | 18.7 |
| Chewing + areca nut (n = 328) | 48 (14.6) | 14.6 |

Table 2 shows markedly higher leukoplakia prevalence among chewers versus non-chewers, with particularly high prevalence in naswar/khaini users and frequent daily chewers.

In multivariable logistic regression adjusted for confounders, significant independent predictors of leukoplakia included daily chewing frequency ≥3 times/day (aOR 3.9; 95% CI 2.6–5.7; p < 0.001), habit duration ≥10 years (aOR 2.8; 95% CI 1.9–4.1; p < 0.001), use of unprocessed naswar/khaini (aOR 4.5; 95% CI 3.0–6.8; p < 0.001), and concurrent areca nut use (aOR 2.3; 95% CI 1.5–3.4; p < 0.001). Female sex and poor oral hygiene were also associated with higher odds but were attenuated after adjustment.

**Table 3. Multivariable logistic regression for presence of leukoplakia (n = 1,050)**

| Predictor | Adjusted OR (95% CI) | p-value |
|---|---|---|
| Daily chewing ≥3 times/day | 3.9 (2.6–5.7) | <0.001 |
| Habit duration ≥10 years | 2.8 (1.9–4.1) | <0.001 |
| Naswar/khaini (vs non-chewer) | 4.5 (3.0–6.8) | <0.001 |
| Processed product (vs non-chewer) | 1.9 (1.2–3.0) | 0.006 |
| Concurrent areca nut use | 2.3 (1.5–3.4) | <0.001 |
| Female sex | 1.4 (0.98–1.9) | 0.07 |
| Poor oral hygiene | 1.5 (1.0–2.2) | 0.04 |

Table 3 indicates robust, statistically significant associations between high-intensity chewing habits, unregulated product use and leukoplakia.

Brief explanatory notes: Table 1 provides participant and habit descriptors; Table 2 presents unadjusted lesion prevalence by habit type showing highest rates among users of unprocessed products and frequent chewers; Table 3 presents adjusted effect estimates confirming independent associations and dose–response relationships.

**Discussion:** This large community-based investigation demonstrates a clear and clinically important association between chewable tobacco habits and oral leukoplakia in low-income urban communities. The overall leukoplakia prevalence of 9.8% is consistent with recent regional surveys of OPMDs conducted in South-Asian urban populations and underscores the persistent burden of premalignant oral disease in tobacco-endemic settings. Community screening data of

similar design have reported comparable prevalences, particularly where smokeless products are widely available and unregulated. 14-16

A striking finding is the heterogeneity of risk by product type. Users of unprocessed naswar/khaini experienced markedly higher prevalence and adjusted odds of leukoplakia than users of processed commercial products. This aligns with mechanistic and compositional analyses showing that artisanal products often contain higher levels of tobacco-specific nitrosamines and abrasive particulates, and they are frequently held in the vestibule for prolonged periods, increasing mucosal exposure. These product-specific differentials highlight the need for regulatory attention to local formulations rather than treating smokeless tobacco as a homogeneous exposure.17-18

Dose–response relationships were evident: daily frequency ≥3 times and duration ≥10 years conferred substantially increased risk. This temporal and intensity gradient supports a biological plausibility model where cumulative mucosal insult escalates the likelihood of keratinocyte dysregulation and premalignant transformation. Earlier community interventions have similarly documented a graded increase in OPMD risk with cumulative exposure metrics, reinforcing cessation advocacy that stresses reduction as well as cessation. 19-20

Concurrent areca nut use independently amplified risk in this cohort, consistent with evidence that arecoline and other alkaloids potentiate fibrogenic and genotoxic pathways. The apparent supra-additive effect when areca nut and tobacco are co-used aligns with prior epidemiologic reports and laboratory data and has critical implications because mixed-habit practices are common in the region. Public-health messaging must therefore address combined exposures and not only tobacco in isolation.

The histopathological confirmation rate among sampled lesions (83%) and the presence of dysplasia in nearly 18% of confirmed leukoplakia cases demonstrate the clinical significance of lesions detected through community screening. These findings argue for embedding diagnostic confirmation and referral pathways into screening programs to ensure early therapeutic intervention and surveillance for malignant transformation. Mobile diagnostic clinics and task-sharing with trained primary-care providers could operationalise this approach in resource-limited settings.

Several strengths underpin the study's credibility: a large, community-derived sample with high response rate; structured and calibrated oral examination; targeted histopathological validation; and rigorous multivariable adjustment for confounders. Nonetheless, limitations merit acknowledgement. The cross-sectional design precludes direct inference of temporal causality; recall bias in self-reported habit histories is possible although mitigated by detailed probing and corroborative questions; and the stratified sub-sampling for biopsy, while pragmatic and ethically justified, may under-ascertain dysplasia in unbiopsied lesions. Despite these caveats, the magnitude and consistency of associations support a causal interpretation in line with Bradford-Hill criteria.

Policy and practice implications are immediate. Interventions should prioritise (1) targeted cessation support in low-income urban clusters with outreach tailored to product types and cultural practices; (2) regulatory measures addressing production, sale and composition of naswar/khaini and similar unprocessed products; (3) community screening coupled with streamlined pathways for biopsy and treatment; and (4) integrated behaviour-change campaigns that include areca-nut cessation. Operational research is warranted to determine the most cost-effective mix of screening frequency, diagnostic algorithms and cessation modalities in these settings.

**Conclusion:** In disadvantaged urban communities, high-frequency and long-duration chewable tobacco use — particularly of unregulated naswar/khaini — and concurrent areca-nut exposure are associated with substantially increased odds of oral leukoplakia. Community screening linked with cessation programs and product regulation should be prioritised to reduce premalignant oral disease burden and downstream cancer risk.

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