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Habit-driven risk determinants of oral potentially malignant disorders: A multivariate analysis

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Abstract:

Oral potentially malignant disorders are strongly associated with tobacco and areca nut habits, but their specific risk determinants remain unclear. Therefore, it is of interest to assess the habit-related determinants of oral potentially malignant disorders in 700 tobacco and areca nut users. Oral mucosal lesions occurred in 535 users (76.43%) and in none of the 700 matched habit-negative controls. Tobacco pouch keratosis (20.37%) and oral submucous fibrosis (18.88%) were the most prevalent lesions. Independent predictors were mixed habits (aOR 5.31), smokeless tobacco (4.14), smoking (3.25), male gender (3.15) and frequency ≥ 6 /day (2.34). Thus, habit type and frequency were the dominant risk drivers, supporting cessation counselling and targeted screening.

Keywords: Oral potentially malignant disorders (OPMDs), tobacco, areca nut, oral submucous fibrosis, tobacco pouch keratosis, logistic regression, Ahmedabad, risk factors, habit frequency, Gujarat

Background:

India carries one of the highest oral cancer burdens globally, a pattern inseparably linked to the widespread use of tobacco in both smoked and smokeless forms and to areca nut consumption across social and cultural settings [1]. Within India, Gujarat occupies a distinctive epidemiological position: high tobacco cultivation and use, a diverse array of smokeless products incorporating areca nut derivatives and legally restricted alcohol availability - the last factor removing a confounding variable ubiquitous in most other Indian epidemiological studies [2]. Oral potentially malignant disorders (OPMDs) are defined as mucosal abnormalities associated with a statistically increased risk of oral cancer. The spectrum - encompassing leukoplakia, erythroplakia, oral submucous fibrosis (OSMF), tobacco pouch keratosis, smoker's palate, smoker's melanosis and quid-induced lichenoid reaction - reflects a direct tissue response to the chemical, thermal and mechanical properties of different tobacco and areca nut products [3]. Understanding which habit parameters most strongly drive OML occurrence determines when cessation counselling must be deployed and which patient subgroups demand the highest clinical vigilance. Published cross-sectional studies from India have largely reported lesion prevalence with descriptive statistics or bivariate associations [4]. Multivariate analyses quantifying the independent contributions of habit type, frequency and duration to OML risk are sparse and virtually absent from western India [5]. The last epidemiological study from Gujarat predates 2016 and no study from Ahmedabad - a city of over 8 million with a complex tobacco-use profile - has examined habit parameters as independent OPMD predictors. The present study addressed this gap. Using binary logistic regression applied to a hospital-

based cross-sectional dataset of 700 tobacco and areca nut users, we determined the independent habit-related risk determinants for OML occurrence, characterised habit-specific lesion profiles and mapped anatomical site predilections. The findings are intended to generate a clinically usable, regionally calibrated risk-stratification framework for oral cancer screening and prevention in Gujarat [6]. Therefore, it is of interest to evaluate the independent habit-related determinants of oral mucosal lesion occurrence among tobacco and areca nut users, to characterise habit-specific lesion profiles and anatomical site predilections and to generate a clinically usable, regionally calibrated risk-stratification framework for oral cancer screening and prevention in Gujarat.

Materials and Methods:**Study design and ethical approval:**

This hospital-based cross-sectional analytical study was conducted in the Department of Oral Medicine and Radiology, College of Dental Sciences and Research Centre, Ahmedabad, Gujarat, India, between 2021 and 2024. Ethical approval was granted by the Institutional Ethics Committee (Registration No. CDSRC/IEC/20210703/21). The study was conducted in accordance with the Declaration of Helsinki (2013) and the Indian Council of Medical Research National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017). Written informed consent, available in English and Gujarati, was obtained from all participants prior to enrolment.

Sample size calculation:

Sample size was estimated using the Charan and Biswas (2013) formula [12], applying a prior prevalence estimate of $p = 0.70$ [13], a 95% confidence level ($Z_{\alpha/2} = 1.96$) and an acceptable margin of error of 5%, yielding a minimum requirement of 323 participants per group. Seven hundred habit-positive participants and 700 age-sex-matched habit-negative controls were enrolled (total $N = 1400$).

Eligibility criteria:

Inclusion criteria (habit group):

Age ≥ 15 years; current daily or near-daily use of any form of tobacco (smoked or smokeless), areca nut, or any combination thereof, for a minimum of six months; willingness to provide written informed consent.

Exclusion criteria:

Age < 15 years; habit duration < 6 months; history of alcohol consumption (excluded due to legal restrictions and consequent self-reporting unreliability in Gujarat [14]); oral lesions with confirmed non-tobacco aetiology (*e.g.*, lichen planus, pemphigus); refusal or inability to consent.

Data collection:

Demographic data (age, gender, occupation) and habit history were recorded using a modified World Health Organization Oral Health Assessment Form for Adults (2013) [15]. Habit group was categorised as: (i) smoking tobacco (cigarettes, bidis, hookah, and chillum); (ii) smokeless tobacco (khaini, mawa, dry snuff and tobacco-containing paan); (iii) areca nut only; or (iv) mixed habits (any combination). Frequency was categorised as ≤ 5 , 6-10, 11-15, or > 15 uses per day. Duration was categorised as ≤ 5 , 6-10, 11-15, or > 15 years.

Clinical oral examination:

All participants underwent a systematic oral mucosal examination under standardised illumination using a dental chair light, plane mouth mirror and probe. Examinations were performed by trained postgraduate students under continuous faculty supervision. Lesions were identified and mapped to seven anatomical sites: labial mucosa, buccal mucosa, palate, tongue and floor of mouth, alveolar mucosa, retromolar pad area and vestibule. Lesion classification followed Shafer's Textbook of Oral Pathology (9th edition, 2020) [16], Sarode *et al.* (2011) [17] and Burket's Oral Medicine (13th edition, 2021) [18]. Participants with clinically suspicious lesions were referred for specialist evaluation and biopsy.

Statistical analysis:

Data were entered and double-verified in Microsoft Excel and analysed using SPSS version 26.0. Descriptive statistics were computed for all categorical variables. Bivariate associations were assessed using chi-square tests; $p < 0.05$ was considered statistically significant (NS = not significant; $*p < 0.05$; $p < 0.001$). Binary logistic regression (enter method) was performed with OML presence (yes/no) as the dependent variable and habit type, gender, frequency category (≤ 5 vs ≥ 6 /day, dichotomised)

and duration category (≤ 5 vs ≥ 6 years, dichotomised) as covariates. Goodness of fit was assessed with the Hosmer-Lemeshow test. Results are expressed as adjusted odds ratios (aOR) with 95% confidence intervals (95% CI).

Results:

Of the 700 participants in the habit group, 637 (91.0%) were male and 63 (9.0%) were female. The largest age cohort was 45-54 years (152 participants, 21.71%), followed by 25-34 years (140, 20.00%) and 35-44 years (132, 18.86%). The youngest age cohort (15-24 years) was the least represented (88, 12.57%). Smokeless tobacco was the most prevalent habit (335 participants, 47.86%), followed by mixed habits (186, 26.57%), smoking tobacco (118, 16.86%) and areca nut alone (61, 8.71%). The gender-habit association was highly significant ($p = 0.002$): no female participant reported smoking tobacco; 38.10% of women used areca nut exclusively compared to only 5.81% of men, while smokeless tobacco was prevalent in both sexes (male 47.41%; female 52.38%). Oral mucosal lesions were detected in 535 of 700 habit-positive participants (76.43%). No oral mucosal lesion attributable to tobacco, areca nut, or mixed habits was identified in any of the 700 age-sex-matched controls without harmful habits, confirming the habit-specific aetiology of all documented OMLs ($p < 0.001$). Among the 535 OML-positive participants, the distribution was: > 1 simultaneous lesion (212, 39.63%), tobacco pouch keratosis (109, 20.37%), oral submucous fibrosis (101, 18.88%), smoker's palate (38, 7.10%), quid-induced lichenoid reaction (22, 4.11%), oral cancer (22, 4.11%), leukoplakia (16, 2.99%), smoker's melanosis (11, 2.06%) and erythroplakia (4, 0.75%). Gender-wise Distribution of Oral Mucosal Lesions (Table 1) smoker's palate, smoker's melanosis, leukoplakia and erythroplakia occurred exclusively in male participants (100% in each case; $p < 0.001$ for all). Oral submucous fibrosis was predominantly male (78/101, 77.23%; $p = 0.002$), as was tobacco pouch keratosis (103/109, 94.50%; $p < 0.001$). Overall, 494 of 535 (92.34%) OML-positive participants were male ($p < 0.001$). Habit-wise Distribution of Oral Mucosal Lesions (Table 2) habit type was strongly associated with lesion specificity (overall $p = 0.041$). Smoker's palate (76.32%), smoker's melanosis (100%), leukoplakia (81.25%) and erythroplakia (75.00%) were confined to the smoking tobacco group; none of these lesions occurred in smokeless tobacco or areca nut-only users. Tobacco pouch keratosis was overwhelmingly smokeless-tobacco-associated (98/109, 89.91%) and oral submucous fibrosis was distributed across smokeless tobacco (62/101, 61.39%), areca nut (26/101, 25.74%) and mixed habit (13/101, 12.87%) groups. Oral cancer occurred predominantly in smokeless tobacco users (13/22, 59.09%) and mixed habit users (7/22, 31.82%). Participants with mixed habits accounted for 54.72% of all multiple simultaneous lesion cases Frequency-wise Distribution of Oral Mucosal Lesions (Table 3) frequency of habit use was significantly associated with OML occurrence ($p = 0.008$). Most OML-positive participants reported a frequency of ≤ 5 uses per day (341, 63.74%), with a secondary cluster at 6-10/day (141, 26.36%). Even at the lowest frequency band, 63.74% of participants harboured lesions, indicating the absence of a safe lower

threshold. Oral submucous fibrosis (81.19%) and tobacco pouch keratosis (80.73%) were concentrated in the ≤ 5 /day band, while smoker's melanosis (72.73%) and leukoplakia (87.50%) were disproportionately concentrated at 6-10/day. Duration-wise Distribution of Oral Mucosal Lesions (**Table 4**) Overall habit duration did not significantly predict OML occurrence ($p=0.316$). Leukoplakia (87.50%), oral cancer (63.64%) and smoker's melanosis (54.55%) were concentrated in the 6-10 year duration band. Erythroplakia was entirely confined to the 11-15 year band (100%). Oral submucous fibrosis (48.51%) and tobacco pouch keratosis (46.79%) occurred predominantly within ≤ 5 years of habit onset, demonstrating that short-term smokeless tobacco and areca nut exposure is sufficient to initiate fibrotic and keratotic OPMD development. Site-wise Distribution of Oral Mucosal Lesions A total of 867 lesion sites was mapped across 535 participants (mean 1.62 sites per OML-positive participant). The buccal mucosa was the most frequently involved site (356/867, 41.00%), followed by the vestibule (146, 17.00%), palate (105, 12.11%), retromolar pad area (101, 11.89%), tongue and floor of mouth (67, 8.00%), labial mucosa (55, 6.00%) and alveolar mucosa (37, 4.00%). Site-lesion specificity was significant

($p=0.039$). Smoker's palate was exclusively palatal (100%); erythroplakia exclusively buccal (100%); tobacco pouch keratosis predominantly vestibular (76.00%); OSMF primarily buccal (45.09%) with additional retromolar pad involvement (35.72%); and oral cancer most commonly affected the buccal mucosa (54.55%) and tongue/floor of mouth (13.64%). Binary Logistic Regression: Independent Predictors of OML Occurrence (**Table 5**). Binary logistic regression identified habit type, male gender, habit frequency and habit duration as independent predictors of OML occurrence. The Hosmer-Lemeshow goodness-of-fit test was non-significant ($p=0.312$), confirming adequate model fit. Nagelkerke $R^2 = 0.38$; overall classification accuracy = 79.4%. Among habit types (reference: areca nut only), mixed habits conferred the highest adjusted odds (aOR 5.31; 95% CI 3.44-8.20; $p<0.001$), followed by smokeless tobacco (aOR 4.14; 95% CI 2.69-6.37; $p<0.001$) and smoking tobacco (aOR 3.25; 95% CI 1.97-5.37; $p<0.001$). Male gender independently increased odds (aOR 3.15; 95% CI 1.98-5.02; $p<0.001$). Habit frequency ≥ 6 /day was a stronger predictor (aOR 2.34; 95% CI 1.56-3.51; $p<0.001$) than habit duration ≥ 6 years (aOR 1.89; 95% CI 1.28-2.79; $p=0.002$).

Table 1: Gender-wise distribution of oral mucosal lesions among habit-positive participants

Oral Mucosal Lesion	Male n (%)	Female n (%)	Grand Total n (%)	p-value
Smoker's palate	38 (100.00)	0 (0.00)	38 (7.10)	<0.001
Smoker's melanosis	11 (100.00)	0 (0.00)	11 (2.06)	<0.001
Leukoplakia	16 (100.00)	0 (0.00)	16 (2.99)	<0.001
Erythroplakia	4 (100.00)	0 (0.00)	4 (0.75)	<0.001
Oral submucous fibrosis	78 (77.23)	23 (22.77)	101 (18.88)	0.002*
Tobacco pouch keratosis	103 (94.50)	6 (5.50)	109 (20.37)	<0.001
Quid-induced lichenoid reaction	20 (90.91)	2 (9.09)	22 (4.11)	<0.001
Oral cancer	19 (86.36)	3 (13.64)	22 (4.11)	<0.001
>1 Simultaneous lesion	205 (96.70)	7 (3.30)	212 (39.63)	<0.001
Grand Total	494 (92.34)	41 (7.66)	535 (100)	<0.001

* $p<0.05$ significant; $p<0.001$ highly significant; Chi-square test

Table 2: Habit-wise distribution of oral mucosal lesions

	Smoking n (%)	Smokeless n(%)	Areca Nut n(%)	Mixed n(%)	Total	p-value
Smoker's palate	29 (76.32)	0 (0.00)	0 (0.00)	9 (23.68)	38	0.004*
Smoker's melanosis	11 (100.00)	0 (0.00)	0 (0.00)	0 (0.00)	11	<0.001
Leukoplakia	13 (81.25)	0 (0.00)	0 (0.00)	3 (18.75)	16	<0.001
Erythroplakia	3 (75.00)	0 (0.00)	0 (0.00)	1 (25.00)	4	0.001*
Oral submucous fibrosis	0 (0.00)	62 (61.39)	26 (25.74)	13 (12.87)	101	0.032*
Tobacco pouch keratosis	0 (0.00)	98 (89.91)	0 (0.00)	11 (10.09)	109	<0.001
Quid-induced lichenoid reaction	0 (0.00)	18 (81.82)	1 (4.55)	3 (13.64)	22	<0.001
Oral cancer	1 (4.55)	13 (59.09)	1 (4.55)	7 (31.82)	22	0.038*
>1 Simultaneous lesion	46 (21.70)	47 (22.17)	3 (1.42)	116 (54.72)	212	0.023*
Grand Total	103 (19.25)	238 (44.49)	31 (5.79)	163 (30.47)	535	0.041*

* $p<0.05$; $p<0.001$; Chi-square test

Table 3: Frequency-wise distribution of oral mucosal lesions

OML	≤ 5 /day n(%)	6-10/day n(%)	11-15/day n(%)	>15/day n(%)	Total	p-value
Smoker's palate	28 (73.68)	9 (23.68)	0 (0.00)	1 (2.63)	38	<0.001
Smoker's melanosis	2 (18.18)	8 (72.73)	0 (0.00)	1 (9.09)	11	<0.001
Leukoplakia	2 (12.50)	14 (87.50)	0 (0.00)	0 (0.00)	16	<0.001
Erythroplakia	0 (0.00)	3 (75.00)	1 (25.00)	0 (0.00)	4	<0.001
Oral submucous fibrosis	82 (81.19)	15 (14.85)	2 (1.98)	2 (1.98)	101	<0.001
Tobacco pouch keratosis	88 (80.73)	16 (14.68)	3 (2.75)	2 (1.83)	109	<0.001
Quid-induced lichenoid reaction	15 (68.18)	7 (31.82)	0 (0.00)	0 (0.00)	22	<0.001
Oral cancer	8 (36.36)	10 (45.45)	4 (18.18)	0 (0.00)	22	0.005*
>1 Simultaneous lesion	116 (54.72)	59 (27.83)	9 (4.25)	28 (13.21)	212	0.44*
Grand Total	341 (63.74)	141 (26.36)	19 (3.55)	34 (6.36)	535	0.008*

* $p<0.05$; $p<0.001$; Chi-square test.

Table 4: Duration-wise distribution of oral mucosal lesions

OML	≤5yr n(%)	6-10yr n(%)	11-15yr n(%)	>15yr n(%)	Total	p-value
Smoker's palate	17 (44.74)	8 (21.05)	4 (10.53)	9 (23.68)	38	0.043*
Smoker's melanosis	2 (18.18)	6 (54.55)	2 (18.18)	1 (9.09)	11	0.021*
Leukoplakia	0 (0.00)	14 (87.50)	1 (6.25)	1 (6.25)	16	<0.001
Erythroplakia	0 (0.00)	0 (0.00)	4 (100.00)	0 (0.00)	4	<0.001
Oral submucous fibrosis	49 (48.51)	29 (28.71)	13 (12.87)	10 (9.90)	101	0.258 NS
Tobacco pouch keratosis	51 (46.79)	18 (16.51)	11 (10.09)	29 (26.61)	109	0.038*
Quid-induced lichenoid reaction	6 (27.27)	5 (22.73)	4 (18.18)	7 (31.82)	22	0.248 NS
Oral cancer	2 (9.09)	14 (63.64)	5 (22.73)	1 (4.55)	22	<0.001
>1 Simultaneous lesion	49 (23.11)	51 (24.06)	23 (10.85)	89 (41.98)	212	0.178 NS
Grand Total	176 (32.90)	145 (27.10)	67 (12.52)	147 (27.48)	535	0.316 NS

NS=not significant; *p<0.05; p<0.001. Chi-square test

Table 5: Binary logistic regression: independent predictors of oral mucosal lesion occurrence (n=700)

Predictor Variable	B	Adj. OR	95% CI Lower	95% CI Upper	p-value
Habit: Smokeless tobacco (vs areca nut)	1.42	4.14	2.69	6.37	<0.001
Habit: Smoking tobacco (vs areca nut)	1.18	3.25	1.97	5.37	<0.001
Habit: Mixed habits (vs areca nut)	1.67	5.31	3.44	8.20	<0.001
Gender: Male (vs Female)	1.15	3.15	1.98	5.02	<0.001
Frequency: ≥6/day (vs ≤5/day)	0.85	2.34	1.56	3.51	<0.001
Duration: ≥6 years (vs ≤5 years)	0.64	1.89	1.28	2.79	0.002*
Constant	-1.87	—	—	—	<0.001

Reference categories: habit type = areca nut only; gender = female; frequency = ≤5/day; duration = ≤5 years.
*p<0.05; p<0.001; Nagelkerke R²=0.38. Hosmer-Lemeshow test p=0.312. Model correctly classified 79.4% of cases.

Discussion:

The present study shows that habit type is the strongest independent predictor of oral mucosal lesion (OML) occurrence, with mixed habits conferring the highest risk, followed by smokeless tobacco, smoking tobacco and areca nut alone. This hierarchy is biologically plausible because concurrent exposure to multiple carcinogens and fibrogenic agents amplifies oxidative stress, chronic inflammation, epithelial dysplasia and extracellular matrix remodeling. Recent evidence has shown that combined tobacco and areca nut exposure produces synergistic molecular alterations involving transforming growth factor-β (TGF-β), reactive oxygen species, epithelial-mesenchymal transition and aberrant collagen metabolism, thereby accelerating both potentially malignant disorders and oral carcinogenesis [7, 8]. The overall OML prevalence of 76.43% among habit users in the present study exceeds that reported in several contemporary Indian investigations. Although regional variation in habit patterns, socioeconomic status and healthcare access partly explains these differences, the remarkably high prevalence observed here is likely attributable to the predominance of smokeless tobacco and mixed-habit users, who constituted nearly three-fourths of the study population. Contemporary epidemiological studies consistently demonstrate that smokeless tobacco products, particularly those containing areca nut, generate a greater burden of oral potentially malignant disorders than smoked tobacco alone because of prolonged mucosal contact with carcinogenic nitrosamines and areca alkaloids [9, 10]. The predominance of tobacco pouch keratosis and oral submucous fibrosis (OSMF) in this cohort reflects the habit profile of the study population and is consistent with current evidence from South Asian populations. OSMF remains one of the most prevalent oral potentially malignant disorders in regions where areca nut consumption is common. Molecular studies have demonstrated that arecoline stimulates fibroblast proliferation, collagen synthesis and reduced collagen degradation through activation of TGF-β signaling, connective

tissue growth factor and lysyl oxidase pathways, ultimately producing progressive fibrosis and restricted mouth opening [11]. The high frequency of tobacco pouch keratosis similarly reflects repeated placement of smokeless tobacco in the labial vestibule, resulting in localized epithelial hyperkeratosis and chronic mucosal injury. An important observation in the present study is that nearly half of OSMF cases developed within five years of habit initiation. This finding supports the growing understanding that molecular alterations begin long before advanced clinical fibrosis becomes evident. Experimental and translational studies indicate that areca nut exposure rapidly induces oxidative DNA damage, inflammatory cytokine release, epithelial atrophy and fibroblast activation, initiating irreversible tissue remodeling even during relatively short exposure periods [8, 11]. Considering the well-documented malignant transformation potential of OSMF, estimated to range between approximately 4% and 13% depending on follow-up duration and study population, these findings reinforce recommendations for early screening and immediate cessation counselling in all areca nut users [12]. The duration-wise distribution of lesions observed in this study also reflects distinct pathogenic mechanisms. OSMF and tobacco pouch keratosis predominated during the early years of exposure, whereas leukoplakia, smoker's melanosis, oral cancer and erythroplakia appeared predominantly after longer habit duration. This temporal progression is consistent with current concepts of oral carcinogenesis, in which chronic exposure to tobacco-specific nitrosamines, polycyclic aromatic hydrocarbons and reactive oxygen species results in cumulative genetic and epigenetic alterations involving TP53, CDKN2A, NOTCH1, EGFR and multiple signaling pathways before malignant transformation becomes clinically evident [13]. The confinement of erythroplakia to long-duration users is particularly noteworthy because erythroplakia possesses one of the highest rates of epithelial dysplasia and malignant transformation among oral potentially malignant disorders. A particularly important

finding of the present study is that habit frequency demonstrated a stronger independent association with OML occurrence than habit duration. This observation suggests that repeated daily exposure of the oral mucosa to carcinogenic substances may be more critical than total years of use. Recurrent placement of smokeless tobacco exposes the mucosa to sustained concentrations of nicotine-derived nitrosamines, areca alkaloids, heavy metals and reactive oxygen species, promoting continuous epithelial injury and impaired mucosal repair [14]. From a clinical perspective, these findings emphasize that even relatively recent users with high daily consumption should be considered at substantial risk and should receive immediate cessation interventions and regular oral examination. The observed gender differences primarily reflect differences in habit patterns rather than biological susceptibility. Smoking-associated lesions, including smoker's palate and smoker's melanosis, occurred almost exclusively among men because smoking tobacco was uncommon among women in this cohort. Female participants predominantly consumed smokeless tobacco and areca nut products, resulting in greater representation of OSMF and tobacco pouch keratosis. Similar gender-specific patterns have been documented in recent Indian epidemiological studies, highlighting the need for prevention strategies tailored to local patterns of tobacco and areca nut use rather than focusing exclusively on smoking [15]. The site-specific distribution identified in this study also has practical implications. Buccal mucosa and labial vestibule constituted the principal sites affected by smokeless tobacco-related lesions, whereas palatal lesions remained associated with smoking habits. These findings correspond closely with the placement of tobacco products and the duration of direct mucosal contact, supporting recommendations that systematic examination of the bilateral buccal mucosa, vestibular sulcus, retromolar region and palate should form the core of opportunistic oral cancer screening in primary dental care [16, 17]. The present study has several limitations. Its cross-sectional hospital-based design precludes causal inference and may overrepresent symptomatic individuals seeking treatment. Self-reported habit history is susceptible to recall bias, particularly regarding duration and daily frequency of exposure. The exclusion of alcohol use limits direct comparison with studies from regions where alcohol acts synergistically with tobacco. Finally, histopathological confirmation was not available for every lesion, although standardized clinical diagnostic criteria were applied consistently by calibrated examiners. Despite these limitations, the large sample size, inclusion of a habit-free control group, detailed lesion characterization and multivariable analysis strengthen the validity of the findings and provide clinically relevant evidence for targeted oral cancer prevention and early detection strategies.

Conclusion:

In this cohort of 700 tobacco and areca nut users in Ahmedabad, 76.43% presented with oral mucosal lesions, prevalence confirmed as habit-specific by its complete absence in matched controls. Habit type and frequency of use were the strongest

independent predictors of OML occurrence, with mixed habits and smokeless tobacco carrying the greatest risk and supporting immediate rather than deferred cessation counselling. These habit- and site-specific lesion patterns provide a regionally calibrated framework for risk-stratified oral cancer screening in Gujarat.

Ethical approval:

This study was approved by the Institutional Ethics Committee of the College of Dental Sciences and Research Centre, Ahmedabad, Gujarat, India (Registration No. CDSRC/IEC/20210703/21). The study was conducted in conformity with the Declaration of Helsinki (2013) and the Indian Council of Medical Research National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017).

Informed consent:

Written informed consent was obtained from all participants prior to enrolment. A bilingual patient information sheet and consent form was provided in English and Gujarati. Participation was entirely voluntary; participants retained the right to withdraw at any stage without impact on their clinical care. For participants unable to read or write, witnessed thumb impression consent was obtained in the vernacular language.

Author contributions:

NJ: Study conception and design, data collection and entry, statistical analysis, manuscript preparation and revision. All other authors contributed to study supervision, clinical examination, academic guidance, data collection and critical manuscript review. All authors read and approved the final manuscript.

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The authors declare no conflicts of interest with respect to the research, authorship, or publication of this article.

Data availability statement:

The de-identified dataset supporting the findings of this study is available from the corresponding author upon reasonable request and subject to institutional data-sharing protocols.

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