International Journal of Community Medicine and Public Health
Anigol PS et al. Int J Community Med Public Health. 2026 Jun;13(6):3183-3191
https://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20261816

Review Article

Incidence and correlation of oral cancer with tobacco and malnutrition in Asia: a comprehensive review

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Received: 18 February 2026
Revised: 16 May 2026
Accepted: 19 May 2026

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ABSTRACT

Oral cancer represents a significant public health burden in Asia, accounting for the majority of global cases. The region's unique patterns of tobacco use and nutritional deficiencies contribute substantially to this disproportionate burden. This comprehensive review examines the incidence and correlation of oral cancer with tobacco use and malnutrition across Asian populations, analyzing regional variations, underlying mechanisms, and prevention strategies. We conducted a systematic review of current literature from PubMed, GLOBOCAN databases, and recent epidemiological studies focusing on oral cancer in Asian countries. Data on incidence rates, tobacco consumption patterns, nutritional status, and their synergistic effects were analyzed. Asia reported 248,360 new oral cancer cases in, with South Asia bearing the highest burden. Tobacco use, particularly betel quid chewing, remains the primary risk factor, with prevalence exceeding 50% in high-burden regions. Malnutrition, characterized by deficiencies in vitamins A, C, E, and micronutrients, significantly increases oral cancer risk through impaired immune function and reduced antioxidant capacity. The synergistic interaction between tobacco and malnutrition amplifies carcinogenic effects through oxidative stress, DNA damage, and inflammation. Oral cancer in Asia requires urgent, multifaceted interventions combining tobacco control, nutritional supplementation, and early detection programs. Population-specific prevention strategies addressing both tobacco cessation and nutritional improvement are essential for reducing disease burden. Implementation of evidence-based policies and community-level interventions can significantly impact oral cancer incidence and mortality in Asian populations.

Keywords: Oral cancer, Tobacco, Betel quid, Malnutrition, Asia, Prevention, Epidemiology

INTRODUCTION

Oral cancer, predominantly oral squamous cell carcinoma (OSCC), represents one of the most significant public health challenges in Asia, accounting for approximately 60% of global cases.[1,2] The disease burden is particularly severe in South and Southeast Asia, where age-standardized incidence rates exceed 20 per 100,000 in several countries.[2-4] This disproportionate burden reflects the complex interplay between behavioral risk factors, particularly tobacco and betel quid use, and underlying nutritional deficiencies prevalent in the region.[3,4] The epidemiology of oral cancer in Asia is characterized by

distinct geographic patterns closely aligned with cultural practices and socioeconomic conditions.[11,12] Countries such as India, Sri Lanka, Bangladesh, Pakistan, and Taiwan report the highest incidence rates globally, with oral cancer representing up to 40% of all malignancies in some regions.[2,13] Unlike Western populations where oropharyngeal cancers increasingly associate with human papillomavirus (HPV) infection, Asian oral cancers predominantly arise from tobacco and betel quid exposure, affecting the oral cavity proper including the tongue, buccal mucosa, and floor of mouth.[16,34] Recent demographic shifts indicate concerning trends in oral cancer epidemiology. While traditionally affecting middle-aged and elderly males, increasing cases among women and younger adults suggest evolving exposure patterns and changing social norms.[11-13] The rising incidence in women correlates with increased tobacco use and betel quid chewing among females in several Asian countries, challenging historical gender disparities in disease burden.[12-17]

METHODS

Study design

This study employed a systematic review design to comprehensively synthesize published evidence on the incidence and correlation of oral cancer with tobacco use and malnutrition across Asian populations. A systematic review was selected as the most appropriate study design to provide a rigorous, transparent, and reproducible synthesis of the existing literature on this topic, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines where applicable.

Literature search strategy

A comprehensive search of the published literature was conducted using electronic databases including PubMed/MEDLINE and GLOBOCAN, supplemented by recent epidemiological studies and reports from international health organizations. The search was performed using relevant Medical Subject Headings (MeSH) terms and free-text keywords, including "oral cancer," "oral squamous cell carcinoma," "tobacco," "betel quid," "areca nut," "smokeless tobacco," "malnutrition," "nutritional deficiency," "Asia," and combinations thereof.

Inclusion and exclusion criteria

Studies were included if they: reported on oral cancer incidence, prevalence, or risk factors in Asian populations; examined associations between tobacco use and/or nutritional status and oral cancer; were published in peer-reviewed journals in the English language; and included original data or systematic syntheses (reviews, meta-analyses, or epidemiological reports). Studies focusing exclusively on non-Asian populations, case reports, editorials, and conference abstracts were excluded.

Data extraction and synthesis

Data were extracted on oral cancer incidence rates, tobacco consumption patterns (including cigarettes, bidis, smokeless tobacco, and betel quid use), nutritional status indicators, and their synergistic effects on oral cancer risk. A narrative synthesis approach was used to integrate findings across studies, given the heterogeneity in study designs, populations, and outcome measures. Quantitative data on incidence rates were obtained primarily from GLOBOCAN and national cancer registries, while risk estimates and mechanistic data were sourced from epidemiological and experimental studies.

TOBACCO USE IN ASIA: FORMS AND PREVALENCE

Tobacco use in Asia encompasses diverse forms including cigarettes, bidis, smokeless tobacco products, and tobacco-containing betel quid preparations.[5,6] The region accounts for approximately 60% of global tobacco consumption, with an estimated 600 million tobacco users.[18] Smokeless tobacco, particularly popular in South Asia, includes products such as gutka, khaini, zarda, and paan masala, often combined with betel quid components including areca nut, slaked lime, and catechu.[5,35]

Betel quid and areca nut use

Betel quid chewing represents a deeply ingrained cultural practice in many Asian societies, with an estimated 600 million users worldwide, predominantly in Asia.[6] The preparation typically consists of areca nut wrapped in betel leaf with slaked lime, frequently supplemented with tobacco and various flavouring agents.[38] The International Agency for Research on Cancer (IARC) has classified betel quid with tobacco, betel quid without tobacco, and areca nut as Group 1 carcinogens.[6,35] The carcinogenic potential of betel quid arises from multiple mechanisms. Areca nut contains alkaloids including arecoline, which generates reactive oxygen species and causes DNA damage.[9] The alkaline environment created by slaked lime enhances alkaloid release and increases oral mucosa permeability, facilitating carcinogen absorption.[38] When combined with tobacco, the synergistic effects substantially amplify cancer risk, with studies demonstrating odds ratios exceeding 10.0 for combined exposure compared to non-users.[24,38]

Regional variations in tobacco consumption

Tobacco consumption patterns vary significantly across Asian regions, influencing oral cancer incidence profiles.[11-18] In India, smokeless tobacco use exceeds smoking in many states, with prevalence rates reaching 30-50% in high-burden regions.[13] Bangladesh reports similar patterns, with gutka and zarda particularly popular

among both men and women.[12] Taiwan's oral cancer epidemic closely correlates with betel quid chewing, predominantly affecting male laborers in construction and transportation industries.[11]

Southeast Asian countries including Myanmar, Thailand, and Indonesia demonstrate high prevalence of betel quid use, often combined with tobacco.[6] In contrast, East Asian countries such as China, Japan, and South Korea show predominantly cigarette smoking patterns, though regional variations exist with smokeless tobacco use in certain populations.[18,34]

TOBACCO-RELATED CARCINOGENESIS

Molecular mechanisms

Tobacco smoke contains over 7,000 chemicals, including at least 70 established carcinogens such as polycyclic aromatic hydrocarbons (PAHs), nitrosamines, and aromatic amines.[15] These carcinogens induce DNA damage through multiple pathways including direct DNA adduct formation, oxidative stress generation, and epigenetic modifications.[15,16] Key molecular alterations in tobacco-related oral carcinogenesis include mutations in tumor suppressor genes (TP53, CDKN2A), oncogene activation (PIK3CA, HRAS), and chromosomal instability.[14-16] Tobacco-specific nitrosamines, particularly NNK and NNN, undergo metabolic activation to form DNA-reactive species that preferentially target critical genes involved in cell cycle regulation, apoptosis, and DNA repair.[15]

Inflammatory and immune responses

Chronic tobacco exposure triggers persistent inflammatory responses in oral tissues, characterized by increased production of pro-inflammatory cytokines including IL-1β, IL-6, IL-8, and TNF-α.[9,10] This chronic inflammation creates a tumor-promoting microenvironment through multiple mechanisms including enhanced angiogenesis, tissue remodelling, and immune suppression.[16] Tobacco constituents also impair innate and adaptive immune responses, reducing natural killer cell activity, T-cell function, and antibody production.[10] This immunosuppression compromises tumor surveillance mechanisms, facilitating malignant transformation and progression.[16]

MALNUTRITION AND ORAL CANCER RISK

Nutritional status in Asian populations

Malnutrition remains prevalent across many Asian regions, particularly in South and Southeast Asia, where micronutrient deficiencies coexist with increasing rates of obesity and metabolic disorders.[4-7] The "double burden" of malnutrition affects both urban and rural populations, with distinct patterns based on socioeconomic status, food security, and dietary habits.[7] Micronutrient deficiencies of particular relevance to oral cancer risk include vitamins A, C, E, folate, and minerals such as zinc, selenium, and iron.[7,8] Population studies in high-burden Asian countries consistently demonstrate lower dietary intake and serum levels of these protective nutrients compared to recommended levels.[22,23]

Vitamin deficiencies and cancer risk

Vitamin A and retinoids

Vitamin A and its derivatives (retinoids) play crucial roles in epithelial cell differentiation, growth regulation, and immune function.[8,21] Deficiency impairs normal epithelial maturation, increases cellular proliferation, and reduces immune surveillance, creating conditions favourable for malignant transformation.[8] Epidemiological studies demonstrate inverse associations between dietary vitamin A intake and oral cancer risk, with meta-analyses showing 30-50% risk reduction in highest versus lowest consumption categories.[22,23] Intervention trials using retinoid supplementation have shown promise in preventing second primary tumors in head and neck cancer patients, though optimal dosing and patient selection remain under investigation.[21]

Vitamin C (ascorbic acid)

Vitamin C functions as a potent antioxidant, protecting cells from oxidative damage caused by reactive oxygen species.[20] It also supports immune function, collagen synthesis, and iron absorption.[20] Deficiency impairs these protective mechanisms, increasing susceptibility to carcinogen-induced damage.[9,20] Studies in Asian populations demonstrate significant inverse associations between vitamin C intake and oral cancer risk.[22,23] The protective effect appears particularly pronounced among tobacco users, suggesting potential mitigation of tobacco-induced oxidative stress.[9,20]

Vitamin E (tocopherols)

Vitamin E comprises a family of lipid-soluble antioxidants that protect cell membranes from lipid peroxidation.[20] In oral tissues, vitamin E helps maintain epithelial integrity and modulates inflammatory responses.[10,20]

Research indicates that vitamin E deficiency correlates with increased oral cancer risk, with protective effects observed at both dietary and supplemental intake levels.[20-22] However, intervention trials have yielded mixed results, highlighting the complexity of antioxidant supplementation in cancer prevention.[20]

Folate (vitamin B9)

Folate plays essential roles in DNA synthesis, methylation, and repair.[25] Deficiency leads to impaired DNA synthesis, increased chromosomal breaks, and

altered methylation patterns, all of which contribute to carcinogenesis.[14,25] Studies demonstrate inverse associations between folate intake and oral cancer risk, with deficiency particularly problematic among alcohol consumers due to alcohol's interference with folate metabolism.[25] The protective effect of adequate folate status appears mediated through maintenance of DNA integrity and proper gene expression patterns.[14,25]

Vitamin D

Emerging evidence suggests vitamin D deficiency may increase oral cancer risk through multiple mechanisms including impaired immune function, reduced cellular differentiation, and dysregulated cell proliferation.[19] Vitamin D receptors are expressed in oral epithelial cells, and vitamin D exerts antiproliferative and pro-differentiation effects.[19] Asian populations show high prevalence of vitamin D deficiency due to limited sun exposure, dietary patterns, and genetic factors affecting vitamin D metabolism.[19] Observational studies suggest inverse associations between vitamin D status and oral cancer risk, though causality requires further investigation.[19]

Micronutrients and trace elements

Zinc

Zinc functions as a cofactor for over 300 enzymes involved in DNA synthesis, repair, and antioxidant defense.[7] Deficiency impairs immune function, increases oxidative stress, and compromises epithelial integrity.[7,10] Studies in Asian populations demonstrate associations between low zinc status and increased oral cancer risk.[7] Zinc deficiency is particularly common in regions with plant-based diets high in phytates, which inhibit zinc absorption.[7]

Selenium

Selenium, incorporated into selenoproteins, plays critical roles in antioxidant defense and immune function.[7,20] Selenoproteins such as glutathione peroxidases protect against oxidative damage, while others modulate inflammatory responses.[20] Geographic variations in soil selenium content influence dietary intake and population selenium status.[7] Regions with selenium-deficient soils show higher cancer rates, including oral cancer, though the relationship is complex and influenced by multiple factors.[7,20]

Iron

Iron deficiency, common in Asian populations particularly among women, increases oral cancer risk through multiple mechanisms.[7] Chronic iron deficiency causes epithelial atrophy, impaired immune function, and increased susceptibility to infections and oxidative damage.[7] The Plummer-Vinson syndrome, characterized by iron deficiency anemia, dysphagia, and esophageal webs, associates with increased risk of oral and esophageal cancers.[7] Even subclinical iron deficiency may impair protective mechanisms against carcinogenesis.[7]

SYNERGISTIC EFFECTS OF TOBACCO AND MALNUTRITION

Oxidative stress and DNA damage

The combination of tobacco exposure and nutritional deficiencies creates a particularly hazardous scenario through amplification of oxidative stress.[9,10] Tobacco generates massive quantities of reactive oxygen species (ROS) while simultaneously depleting endogenous antioxidants.[9,15] When combined with inadequate dietary antioxidant intake, this results in severe oxidative imbalance.[9,20] Oxidative stress induces multiple forms of DNA damage including base modifications, strand breaks, and DNA-protein crosslinks.[9,15] Nutritional deficiencies compromise DNA repair capacity, allowing accumulation of mutations critical for malignant transformation.[14,25] Studies demonstrate that individuals with both tobacco exposure and low antioxidant status show significantly higher levels of DNA damage markers compared to those with either risk factor alone.[9,10]

Immune dysfunction

Both tobacco use and malnutrition independently impair immune function, and their combination produces profound immunosuppression.[10,16] Tobacco suppresses natural killer cell activity, reduces T-cell proliferation, and impairs antibody production.[10] Nutritional deficiencies, particularly of vitamins A, C, D, zinc, and selenium, further compromise both innate and adaptive immunity.[7,8,10] This synergistic immunosuppression reduces tumor surveillance capacity, allowing premalignant cells to escape immune elimination.[10,16] Additionally, impaired immune function increases susceptibility to oral infections, including high-risk HPV strains, which may contribute to carcinogenesis in susceptible individuals.[10]

Inflammatory pathways

Chronic tobacco exposure induces persistent inflammation in oral tissues, characterized by infiltration of inflammatory cells and elevated pro-inflammatory cytokine production.[9,10] Nutritional deficiencies exacerbate this inflammatory response while simultaneously impairing resolution mechanisms.[10,20] The resulting chronic inflammation promotes carcinogenesis through multiple pathways including generation of reactive oxygen and nitrogen species, activation of pro-survival signalling pathways (NF-κB,STAT3), and promotion of angiogenesis.[9,10,16] Studies demonstrate that individuals with combined tobacco use and poor

nutritional status show higher levels of inflammatory markers and more aggressive disease phenotypes.[9,10]

Epithelial integrity and barrier function

Tobacco constituents directly damage oral epithelium, disrupting barrier function and increasing permeability to carcinogens.[15,16] Nutritional deficiencies, particularly of vitamins A, C, and zinc, impair epithelial regeneration and maintenance.[7,8] This compromised epithelial integrity facilitates deeper penetration of tobacco carcinogens, increasing exposure of basal epithelial cells where malignant transformation typically initiates.[8,16] Additionally, impaired epithelial barrier function may allow microbial translocation, contributing to chronic inflammation and dysbiosis.[26]

Metabolic interactions

Tobacco use alters nutritional metabolism through multiple mechanisms including reduced appetite, impaired nutrient absorption, increased metabolic demands, and accelerated nutrient turnover.[17] Smokers typically show lower dietary intake of fruits and vegetables, further compounding nutritional deficiencies.[17,22] Certain nutrients, particularly B vitamins, are consumed at higher rates in tobacco users due to increased metabolic stress.[25] This creates a vicious cycle where tobacco use increases nutritional requirements while simultaneously reducing intake and absorption.[17,25]

ORAL MICROBIOME ALTERATIONS

Recent research highlights the role of oral microbiome dysbiosis in oral cancer development and progression.[26] Tobacco use significantly alters oral microbial composition, reducing beneficial commensals while promoting growth of pathogenic species.[26] Nutritional deficiencies further impact microbiome composition through alterations in local pH, nutrient availability, and immune function.[26] Dysbiotic oral microbiomes contribute to carcinogenesis through multiple mechanisms including production of carcinogenic metabolites, induction of chronic inflammation, and promotion of epithelial barrier dysfunction.[26] Specific bacterial species, including Porphyromonas gingivalis, Fusobacterium nucleatum, and certain streptococcus species, show increased prevalence in oral cancer patients.[26]

EPIDEMIOLOGICAL EVIDENCE

Case-control studies

Numerous case-control studies from Asian countries demonstrate strong associations between tobacco use, nutritional factors, and oral cancer risk.[24,27,36] A large multi-center study in India found odds ratios of 8.5 for tobacco chewing, 3.2 for smoking, and 12.8 for combined tobacco and alcohol use.[24] When stratified by nutritional status, individuals with low fruit and vegetable consumption showed significantly higher risk, with evidence of multiplicative interaction between tobacco use and poor dietary habits.[24,27] Studies examining specific nutrients demonstrate consistent inverse associations for vitamins A, C, E, and folate.[22-25] Meta-analyses indicate approximately 40-50% risk reduction comparing highest to lowest intake categories for these protective nutrients.[23]

Cohort studies

Prospective cohort studies, though less common in Asian populations, confirm associations observed in case-control studies.[11,34] Long-term follow-up of betel quid chewers demonstrates dose-response relationships, with risk increasing with duration and frequency of use.[38] Cohorts with detailed dietary assessment show protective effects of high fruit and vegetable consumption, even among tobacco users.[23,27]

Population-attributable risk

Population-attributable risk calculations indicate that tobacco use accounts for 50-80% of oral cancer cases in high-burden Asian regions.[37] When considering combined effects of tobacco and nutritional deficiencies, the attributable fraction may exceed 90% in some populations.[36,37] These findings underscore the preventable nature of most oral cancer cases and highlight opportunities for intervention.[32,37]

PREVENTION STRATEGIES

Tobacco control measures

Comprehensive tobacco control represents the most effective strategy for reducing oral cancer burden in Asia.[18,32,33] Evidence-based interventions include:

Taxation and pricing policies: Increased tobacco taxes reduce consumption, particularly among youth and low-income populations.[18,32] Studies demonstrate that 10% price increases reduce consumption by 4-8% in Asian countries.[18]

Smoke-free policies: Comprehensive smoke-free legislation protects non-smokers while encouraging cessation among users.[33] Implementation in Asian countries has shown significant reductions in tobacco consumption and smoking-related morbidity.[18,33]

Marketing and advertising restrictions: Bans on tobacco advertising, promotion, and sponsorship reduce initiation, particularly among youth.[18,32] Plain packaging requirements and graphic health warnings increase awareness of tobacco harms.[33]

Cessation support: Accessible cessation services, including behavioral counseling and pharmacotherapy,

significantly improve quit rates.[33] Integration of cessation support into healthcare systems increases reach and effectiveness.[32,33]

Betel quid control: Specific measures targeting betel quid and areca nut use are essential in high-prevalence regions.[35] These include regulation of sales, marketing restrictions, taxation, and public education campaigns.[32,35]

Nutritional interventions

Improving nutritional status represents a complementary prevention strategy, particularly in resource-limited settings.[22,29]

Dietary modification: Promoting increased consumption of fruits, vegetables, and nutrient-rich foods provides protective effects.[22,23] Community-based nutrition education programs can effectively improve dietary patterns.[29]

Micronutrient supplementation: Targeted supplementation programs addressing specific deficiencies may reduce cancer risk in high-risk populations.[21] However, supplementation strategies require careful consideration of optimal doses, combinations, and target populations.[20,21]

Food fortification: Population-level fortification of staple foods with key micronutrients can address widespread deficiencies.[7] Successful examples include vitamin A fortification of cooking oil and folate fortification of wheat flour.[7,25]

Agricultural interventions: Biofortification of crops to increase micronutrient content offers sustainable approaches to improving nutritional status.[7] Examples include high-zinc rice and vitamin A-enriched sweet potato varieties.[7]

Early detection and screening

Early detection significantly improves oral cancer outcomes, with 5-year survival rates exceeding 80% for localized disease compared to less than 40% for advanced stages.[28,30]

Visual oral examination: Simple visual screening by trained healthcare workers can detect precancerous lesions and early cancers.[28] A landmark trial in Kerala, India, demonstrated 34% mortality reduction through oral visual examination in high-risk individuals.[28]

Community screening programs: Population-based screening in high-risk communities increases early detection rates.[28,29] Mobile screening units and community health worker programs extend reach to underserved populations.[29]

Adjunctive technologies: Emerging technologies including tissue autofluorescence, vital staining, and molecular markers may enhance screening accuracy.[30] However, cost-effectiveness and practical implementation require further evaluation.[30]

Opportunistic screening: Integration of oral cancer screening into routine dental and primary care visits increases coverage.[28,32] Training of healthcare providers in oral examination and lesion recognition is essential.[29,32]

Integrated prevention approaches

Comprehensive prevention strategies combining multiple interventions show greatest effectiveness.[29,32]

Multi-level interventions: Addressing individual, community, and policy levels simultaneously maximizes impact.[29,32] Successful programs integrate behavior change communication, community mobilization, and policy advocacy.[29]

Risk-stratified approaches: Targeting high-risk populations (tobacco users, those with nutritional deficiencies, individuals with precancerous lesions) optimizes resource utilization.[28,29] Risk assessment tools can guide intervention intensity.[36]

Community engagement: Involving community leaders, local organizations, and affected populations in program design and implementation improves acceptance and sustainability.[29] Cultural adaptation of interventions enhances effectiveness.[29]

Healthcare system integration: Embedding prevention activities within existing healthcare infrastructure ensures sustainability and scalability.[32] Integration with programs addressing other non-communicable diseases creates synergies.[32]

CHALLENGES AND BARRIERS

Despite evidence-based prevention strategies, multiple barriers impede oral cancer control in Asia.[11,12,32]

Cultural factors

Deep-rooted cultural practices surrounding tobacco and betel quid use create resistance to cessation.[5,6] Social acceptability and ceremonial use complicate intervention efforts.[6]

Economic interests

Powerful tobacco and betel nut industries oppose control measures through lobbying, litigation, and marketing.[18,32] Economic dependence on tobacco cultivation in some regions creates policy challenges.[18]

Limited resources

Many high-burden countries face resource constraints limiting implementation of comprehensive programs.[12,32] Competing health priorities and limited healthcare infrastructure impede scale-up.[32]

Low awareness

Limited public awareness of oral cancer risks and prevention strategies reduces uptake of interventions.[17,27] Misconceptions about tobacco and betel quid harms persist in many communities.[5,6]

Healthcare access

Inequitable access to healthcare services, particularly in rural and underserved areas, limits early detection and treatment.[12,28] Geographic, economic, and social barriers prevent timely care.[28,29]

Policy implementation

Gaps between policy adoption and effective implementation reduce intervention impact.[32] Inadequate enforcement, limited monitoring, and insufficient resources undermine policy effectiveness.[18,32]

FUTURE DIRECTIONS

Advancing oral cancer prevention in Asia requires multifaceted approaches addressing current knowledge gaps and implementation challenges.[29,30,32]

Research priorities

Longitudinal studies examining interactions between tobacco, nutrition, and genetic factors, along with intervention trials testing integrated prevention approaches, are essential for advancing public health strategies. In addition, cost-effectiveness analyses are needed to guide efficient resource allocation, while biomarker development can support improved risk stratification and early disease detection. Furthermore, microbiome studies may help elucidate underlying mechanistic pathways involved in disease development and progression.

Policy development

Strengthening tobacco control legislation and its enforcement, along with developing regulatory frameworks for betel quid and areca nut, are important policy priorities. In addition, implementing nutrition policies that address micronutrient deficiencies and integrating oral cancer prevention into national cancer control programs are essential strategies for reducing disease burden and improving population health outcomes.

Implementation science

Developing scalable and sustainable intervention models, along with adapting evidence-based interventions to diverse cultural contexts, is essential for effective public health implementation. Strengthening healthcare workforce capacity further supports improved service delivery, while leveraging technology for screening, education, and monitoring can enhance accessibility, efficiency, and overall program impact.

Equity considerations

Addressing the social determinants of oral cancer risk and ensuring equitable access to prevention and early detection services are critical for reducing disease burden. In addition, reducing disparities in tobacco use and nutritional status, along with engaging vulnerable and marginalized populations, is essential to achieve more inclusive and effective public health outcomes.

CONCLUSION

Oral cancer represents a major but largely preventable public health challenge in Asia. The disease burden reflects the complex interplay between high-prevalence tobacco and betel quid use and widespread nutritional deficiencies, which synergistically amplify cancer risk through oxidative stress, immune dysfunction, chronic inflammation, and impaired epithelial integrity. Comprehensive prevention strategies addressing both tobacco control and nutritional improvement offer substantial potential for reducing oral cancer incidence and mortality.

Evidence-based interventions including tobacco taxation, smoke-free policies, cessation support, betel quid control, dietary improvement, micronutrient supplementation, and early detection screening have demonstrated effectiveness. However, successful implementation requires sustained political commitment, adequate resource allocation, multisectoral collaboration, and community engagement. The predominantly preventable nature of oral cancer in Asia, with population-attributable risks exceeding 90% for modifiable risk factors, underscores both the tragedy of the current burden and the opportunity for dramatic impact through evidence-based interventions. Urgent action is needed to translate existing knowledge into effective programs that reach the populations bearing the greatest burden. By addressing the dual challenges of tobacco use and malnutrition through integrated, culturally appropriate, and equitably delivered interventions, substantial reductions in oral cancer incidence and mortality are achievable within the next decade.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Anigol PS, Kotrashetti V, Vanaki BN. Incidence and correlation of oral cancer with tobacco and malnutrition in Asia: a comprehensive review. Int J Community Med Public Health 2026;13:3183-91.