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Beyond the Numbers: Inflammation as the Missing Link in Decayed, Missing, Filled Teeth–Based Buccal Mucosa Cancer Risk Models

TO THE EDITOR:

George et al[1] have examined the association between the Decayed, Missing, Filled Teeth (DMFT) index—a widely used metric for assessing overall dental health—and the risk of buccal mucosa cancer (BMC) in a large Indian cohort. This study included 1,673 cases of BMC that were histologically confirmed, along with 1,601 frequency-matched controls, providing a robust framework for analysis. The study results showed an increased risk of decayed (odds ratio [OR] >2 decayed teeth, 1.53) and missing teeth (OR >2 missing teeth, 2.59) and high DMFT scores (OR >3 DMFT score, 2.07). Protective effect was observed for teeth filling (OR >2 teeth-fillings, 0.59) and denture use (OR ever *v* never used dentures, 0.63).

Extensive research by George et al[1] provides valuable epidemiologic insights into the intricate relationship between oral health status and oral cancer risk, especially in regions characterized by high tobacco consumption and limited access to comprehensive dental care services. However, we believe that a nuanced interpretation of the DMFT index as a risk factor is crucial. There should be a clear distinction between structural indicators of dental health—such as decayed, missing, and filled teeth—and the more complex underlying biological processes that may influence disease development. This critical reflection is essential for understanding the multifaceted nature of oral health and its impact on overall health outcomes.

First, the assumption that higher DMFT scores directly indicate an increased risk for BMC, without a comprehensive examination of the underlying biological mechanisms that connect the two, may reflect a conceptual deficiency. Specifically, it remains unclear whether the observed risk is attributable to the physical absence of teeth or to the chronic inflammation that occurs before tooth loss and dental decay. Tooth loss itself is merely a consequence, not a causative factor. It can result from trauma, prosthetic interventions, or socioeconomic influences and does not inherently signify an active disease process. By contrast, long-term mucosal inflammation—stemming from periodontitis, deep caries, or persistent local irritation—represents a biologically plausible catalyst for carcinogenesis, capable of inflicting DNA damage through inflammatory cytokines, reactive oxygen species, and microbial imbalance.[2-5] The observed protective effect of dental fillings noted in the study lends credence to this perspective: early and proactive restorative dental care almost certainly plays a role in alleviating chronic inflammatory states and curbing bacterial overgrowth, thereby potentially diminishing the risk of malignant transformation in the surrounding tissues. However, in the absence of data on inflammatory markers, microbial profiles, or lesion locations, it remains uncertain whether DMFT serves as a marker for chronic inflammation or merely as a fundamental structural indicator.

Second, although the DMFT index incorporates metrics for decayed and missing teeth, it does not directly assess periodontitis, which is arguably the most critical oral inflammatory condition associated with both tooth loss and cancer risk. Periodontitis is a progressive disease characterized by immune-mediated destruction of the periodontal ligament and alveolar bone. Often chronic and asymptomatic, it frequently goes unnoticed by patients. In contrast to dental caries, which result from localized acid production, periodontitis arises from a complex interplay of microbial biofilms, host immune responses, and systemic inflammation.[6-8] Numerous epidemiologic studies have identified an independent link between periodontitis and oral, pancreatic, and GI cancers.[9] In this study, the absence of clinical indicators of periodontal health—such as probing depth, clinical attachment loss, or radiographic bone loss—represents a missed opportunity to establish a more specific and biologically relevant connection to BMC. Furthermore, while the study categorizes risk according to tobacco and alcohol consumption, it overlooks local inflammatory factors such as oral hygiene practices, frequency of professional dental cleanings, or plaque levels. Incorporating these elements could help elucidate the considerable variation observed in DMFT and provide more actionable targets for public health initiatives.

In conclusion, we commend the authors for emphasizing the significance of oral health as a critical yet frequently neglected component of cancer prevention. However, future research should incorporate clinical inflammatory markers, distinguish between active and past dental diseases, and treat periodontitis as a distinct and potentially more informative variable rather than merely counting teeth. Achieving a deeper mechanistic understanding of how oral pathology contributes to mucosal carcinogenesis will be essential for progressing from correlation to causation. Finally, we would like to extend our heartfelt gratitude to the authors for allowing us the opportunity to share our insights and perspectives on this vital subject within the realms of head and neck and dental oncology.

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