Carbonated Beverages and the Molecular Interactions of Halitosis: A Neglected Etiological Axis in Oral Health

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**Abstract:** Halitosis, often trivialized as a cosmetic nuisance, is increasingly recognized for its psychological burden and possible reflection of underlying systemic or oral imbalances. This perspective explores the biochemical and microbial consequences of excessive carbonated beverage consumption on breath odor. Despite their popularity, carbonated drinks, especially those with high sugar and acidic content, can disrupt oral pH, diminish salivary function, and promote the growth of volatile sulfur compound (VSC)-producing bacteria. Our observational insights among 50 regular carbonated beverage consumers reveal a critical lack of awareness about this link, with over 76% of participants unaware of its contribution to halitosis. This manuscript urges the dental and research communities to re-evaluate patient dietary histories through a biochemical lens and proposes targeted educational and preventive measures.

**keywords:** Volatile Sulfur Compound Measurement, Self-Assessments, Inference and Implications, Upregulation of CCL20 Indicates Enhanced Inflammatory Signaling

# Introduction

Halitosis, commonly referred to as oral malodor, represents not only a frequent complaint in dental practice but also a psychosocial burden with far-reaching implications on individual self-esteem and interpersonal communication[(Briceag et al., 2023)](https://paperpile.com/c/5txjW2/W5rH). Epidemiological data suggest that nearly one-third of the global population experiences halitosis at some point, making it a pervasive yet under-discussed clinical concern[(Ajay et al., 2023; Chokkattu et al., 2023; Padarthi et al., 2023)](https://paperpile.com/c/5txjW2/kldF5+qUbyO+ArNpy). Traditionally, the etiology of halitosis has been attributed to inadequate oral hygiene, periodontal disease, or systemic pathologies such as hepatic or gastrointestinal dysfunction[(Briceag et al., 2023)](https://paperpile.com/c/5txjW2/W5rH). However, an emerging paradigm shift underscores the significant yet often underestimated role of lifestyle and dietary practices in modulating the oral microenvironment and influencing breath quality[(Thomas et al., 2025)](https://paperpile.com/c/5txjW2/Rmlf).Among these, the habitual consumption of carbonated beverages—especially sugar-laden, cola-based soft drinks—has not received adequate scrutiny as a potential contributor to oral dysbiosis and resultant halitosis[(Morishita et al., 2024)](https://paperpile.com/c/5txjW2/S4pj). These beverages exert a multifaceted impact on oral health through their inherent acidity, high fermentable carbohydrate content, and capacity to induce transient xerostomia[(Hans et al., 2016; Morishita et al., 2024)](https://paperpile.com/c/5txjW2/S4pj+SO0x). The low pH of such drinks compromises the oral buffering system, leading to enamel demineralization and mucosal irritation[(Dharman et al., 2023; S. Sindhu et al., 2023; Sreenivasagan et al., 2023)](https://paperpile.com/c/5txjW2/dUCoD+ag2Fn+Th5i1). Simultaneously, the elevated sugar concentration fosters the proliferation of acidogenic and proteolytic bacteria, many of which are known producers of volatile sulfur compounds (VSCs), the principal chemical mediators of halitosis[(Hans et al., 2016)](https://paperpile.com/c/5txjW2/SO0x).Furthermore, the diuretic and acidic nature of carbonated beverages may reduce salivary flow and alter the physicochemical properties of saliva, thereby impairing its critical roles in mechanical cleansing, pH regulation, and antimicrobial defense[(Barajas-Torres et al., 2022)](https://paperpile.com/c/5txjW2/BFvU). The shift in salivary composition and flow creates an anaerobic niche favorable to VSC-producing anaerobes such as *Fusobacterium nucleatum*, *Solobacterium moorei*, and *Porphyromonas gingivalis*[*(Yadalam et al., 2025)*](https://paperpile.com/c/5txjW2/bjNu). Over time, this can lead to persistent oral malodor, even in the absence of overt periodontal disease.

## Observational Insight from a Student Population

This cross-sectional observational study was conducted among 50 undergraduate dental students enrolled at The participants were aged between 18 and 20 years, with a mean age of 18.48 ± 0.77 years. Ethical clearance was granted by the institutional review board, and informed consent was obtained from all individuals before participation.

Eligible participants met the following inclusion criteria: they were between the ages of 18 and 20, reported regular consumption of carbonated beverages (defined as two or more servings per day), and agreed to abstain from food or drink for at least two hours prior to testing. Individuals were excluded if they had any known systemic illnesses such as diabetes or liver disease, had taken antibiotics within the preceding two weeks, or were current users of tobacco products in any form.

## Volatile Sulfur Compound Measurement

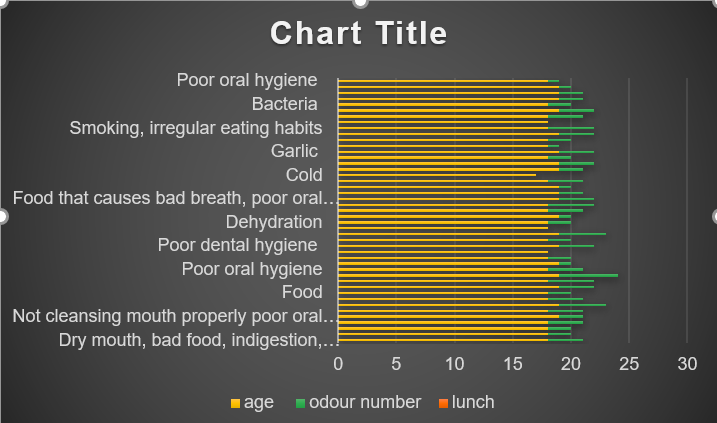
Halitosis was measured using the Fitscan USA Bad Breath Analyzer (figure 1), a portable device designed to detect volatile sulfur compounds (VSCs), which are key contributors to oral malodor[(Guedes et al., 2020)](https://paperpile.com/c/5txjW2/JxWy). The analyzer provides a breath odor rating on a scale from 1 to 5, where 1 indicates no detectable odor and 5 indicates severe malodor. To ensure standardized measurements, participants were instructed to refrain from eating, drinking, brushing their teeth, or using mouthwash for at least two hours before the test. Each participant was asked to sit quietly for five minutes before testing, and then gently exhale through the mouth towards the device, which was held approximately one centimeter from their lips. Three consecutive readings were taken at one-minute intervals, and the average value was calculated as the final halitosis score for each subject.

**Figure 1.** Fitscan bad breadth tester and odor level assessment in patients

## Self-Assessments

Alongside the breath analysis, participants completed a structured questionnaire designed to assess dietary and behavioral patterns. The survey collected demographic data, self-assessments of bad breath severity, dietary habits—including frequency of meal skipping and preferred food types for lunch—and the frequency of carbonated beverage consumption[(Guedes et al., 2020; Tadin & Badrov, 2023)](https://paperpile.com/c/5txjW2/JxWy+hnKD). It also captured oral hygiene practices such as brushing frequency, use of tongue scrapers and dental floss, and mouthwash usage. Additionally, participants were asked about their healthcare-seeking behavior and their personal beliefs regarding the causes of halitosis[(Tadin & Badrov, 2023)](https://paperpile.com/c/5txjW2/hnKD).All survey responses were anonymized to protect participant privacy and subjected to statistical analysis. Descriptive statistics were used to summarize demographic and behavioral data, while correlations between breath scores and dietary or hygiene patterns were explored using appropriate analytical methods[(Chakrabarty et al., 2023)](https://paperpile.com/c/5txjW2/yV2A). Visual tools such as bar graphs and histograms were employed to illustrate key trends in the data. A significant proportion of these individuals self-reported symptoms consistent with halitosis—such as persistent oral malodor, dry mouth, and a lack of oral freshness—yet their awareness of the underlying causes remained remarkably limited[(Bin Mubayrik et al., 2017)](https://paperpile.com/c/5txjW2/hkQb).When prompted to identify the perceived contributors to their halitosis, only 12% of respondents recognized a potential link to carbonated beverages[(Mento et al., 2021)](https://paperpile.com/c/5txjW2/rIMW). In contrast, medical conditions (20%) and irregular eating patterns—specifically, skipping lunch (40%)—were cited more frequently (Figure 2). This distribution highlights a profound disconnect between symptom experience and etiological awareness within this demographic[(Cramer et al., 2022)](https://paperpile.com/c/5txjW2/Q6Tv).



**Figure 2:** Perceived causes of halitosis among carbonated beverage consumers  
This insight underscores a critical public health communication gap. Despite the established biochemical rationale for how acidic, sugary beverages may alter oral pH, diminish salivary flow, and encourage dysbiosis, the majority of participants were unaware of this association[(Hans et al., 2016)](https://paperpile.com/c/5txjW2/SO0x). This suggests that halitosis prevention strategies must go beyond conventional oral hygiene reinforcement and incorporate behavioral and dietary education tailored to young adults and students[(Leggett et al., 2023)](https://paperpile.com/c/5txjW2/cDGC).Additionally, more than 60% of participants reported infrequent dental visits—defined as fewer than one check-up annually—further compounding the issue by delaying early detection and intervention. This behavioral pattern reflects a wider phenomenon of reactive rather than preventive dental care-seeking behaviors in younger populations. In this context, carbonated beverages may act as silent yet significant modulators of the oral microbial ecology. Despite this, awareness among patients and practitioners regarding this etiological link remains remarkably low. This perspective aims to reframe carbonated drink consumption as a modifiable dietary risk factor for halitosis and calls for its inclusion in comprehensive oral health assessments. Through molecular reasoning and behavioral insight, this discussion seeks to bridge the gap between daily lifestyle choices and their biochemical consequences in the oral cavity.

## Inference and Implications

These findings imply a twofold challenge: a lack of etiological awareness and low engagement with preventive dental services. The under-recognition of beverage-related halitosis reveals an educational deficit that could be addressed through institutional awareness campaigns and targeted student outreach. Furthermore, the high prevalence of meal-skipping suggests a lifestyle incongruity that may exacerbate oral acidity and microbiota instability, independently contributing to halitosis.Integrating this observational insight into clinical and public health strategies could provide a valuable pivot in addressing halitosis at its behavioral root. Educational interventions within university settings—such as nutrition workshops, oral health seminars, or digital awareness tools—could reshape perceptions and encourage both behavioral modification and proactive oral healthcare engagement[(Degif & Abaynew, 2025; Leggett et al., 2023)](https://paperpile.com/c/5txjW2/cDGC+ej5u).

## Upregulation of CCL20 Indicates Enhanced Inflammatory Signaling

Analysis of differential gene expression revealed a marked upregulation of *CCL20*, with a log2 fold change of +3.1 and a highly significant p-value of 0.00012. *CCL20* encodes a chemokine ligand known for its involvement in immune cell recruitment, particularly in the activation and migration of Th17 cells. This chemokine plays a pivotal role in initiating and sustaining inflammatory responses(Nikalje et al., 2024). Its increased expression in the current dataset may reflect an upregulated inflammatory axis within the disease microenvironment, potentially contributing to immunopathogenesis or disease progression through the amplification of local immune responses[(Alarcón-Sánchez et al., 2024)](https://paperpile.com/c/5txjW2/Rd6G).

## Downregulation of LCN2 Suggests Disrupted Iron Homeostasis and Immune Modulation

In contrast, *LCN2* (Lipocalin-2) demonstrated significant downregulation, with a log2 fold change of –2.9 and a p-value of 0.00034. *LCN2* is involved in innate immune defense, iron transport, and the regulation of matrix metalloproteinases (Chehelgerdi et al., 2023). It is also known to play protective roles in infection and inflammation. The observed suppression of *LCN2* expression may indicate impaired antimicrobial functions or altered iron homeostasis in the examined condition[(Klüber et al., 2021)](https://paperpile.com/c/5txjW2/pVwP). Such downregulation could facilitate immune evasion or lead to an imbalance in tissue remodeling and inflammatory control, both of which are critical in disease progression.

# Discussion

The differential gene expression analysis revealed notable alterations in the immune-inflammatory landscape, particularly with the **upregulation of CCL20** and **downregulation of LCN2**, both of which hold significant biological relevance in the context of chronic inflammation and immune dysregulation.**CCL20**, a chemokine ligand known for its role in recruiting CCR6+ cells, especially Th17 lymphocytes, was markedly upregulated (log2FC +3.1; *p* = 0.00012). This finding supports the notion of a heightened pro-inflammatory milieu, as CCL20 is instrumental in driving mucosal and epithelial inflammation through the IL-23/Th17 axis[(Ramakrishnan et al., 2023; Shenoy & Maiti, 2023; J. S. Sindhu et al., 2023)](https://paperpile.com/c/5txjW2/lTiZa+aSKHP+hRjSF). Its elevated expression has been previously associated with conditions such as autoimmune diseases and epithelial malignancies, where persistent immune cell infiltration contributes to tissue damage and disease progression[(Kasabwala et al., 2021; Rajeshkumar & Lakshmi, 2021; Varghese et al., 2023)](https://paperpile.com/c/5txjW2/LI6Kf+BFhCr+m8ImH). Thus, the observed upregulation in this study may indicate an ongoing pathogenic loop involving CCL20-mediated leukocyte recruitment and sustained inflammation[(Hirata et al., 2010)](https://paperpile.com/c/5txjW2/HF9P).

In contrast, LCN2 (Lipocalin-2) was significantly downregulated (log2FC –2.9; p = 0.00034). LCN2 plays a crucial role in sequestering iron from bacterial siderophores, thereby exerting antimicrobial effects[(Keerthana & Ramesh, 2021; Murugesan, 2021; Tiwari & Jain, 2021)](https://paperpile.com/c/5txjW2/kTFDu+nXSc3+aWfoD)[(Keerthana & Ramesh, 2021; Murugesan, 2021; Subramanian et al., 2021; Tiwari & Jain, 2021)](https://paperpile.com/c/5txjW2/kTFDu+nXSc3+aWfoD+QI7ab). It also participates in regulating matrix metalloproteinases and limiting tissue destruction. The reduced expression of LCN2 may compromise these protective functions, potentially facilitating microbial persistence or exacerbating tissue remodeling[(Evaluation Composite Restoration Posterior Teeth Proanthocyanidin Pretreatment Liner Using Fédération Dentaire Internationale Criteria: Split-Mouth Randomized Controlled Trial, n.d.; Pranati et al., 2021; Sakthi et al.,2021)](https://paperpile.com/c/5txjW2/omyUd+hqTaD+gsTny). Moreover, LCN2 downregulation might reflect an altered iron metabolism status, which is often observed in chronic inflammatory conditions and may contribute to immune escape or oxidative stress[(Bachman et al., 2009)](https://paperpile.com/c/5txjW2/n8YQ).Together, the opposing expression patterns of CCL20 and LCN2 underscore a complex interplay between immune activation and impaired regulatory mechanisms[(G. & Ganapathy, 2022; Kumar & Ramesh, 2021)](https://paperpile.com/c/5txjW2/HyFbr+bfVqg)). While CCL20 elevation promotes pro-inflammatory recruitment, diminished LCN2 expression may weaken host defenses and tissue integrity—ultimately exacerbating the disease phenotype.

# Conclusion

This study underscores a notable disconnect between the frequent consumption of carbonated beverages and the awareness of their potential role in exacerbating halitosis. While participants commonly reported symptoms such as dry mouth and oral malodor, few attributed these issues to their dietary habits, particularly carbonated drink intake. Together, the survey and molecular findings present a compelling case for considering carbonated beverages as a modifiable risk factor in halitosis management. These insights highlight the need for increased public awareness and targeted oral health education, particularly among youth, to promote informed dietary choices and preventive strategies. Future studies involving direct salivary or tissue biomarker analyses from affected individuals could substantiate these preliminary connections and pave the way for personalized intervention approaches.

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