

Review

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Posted Date: 16 January 2026

doi: 10.20944/preprints202601.1222.v1

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Review

Oral Cellular Homeostasis and Occupational Wellbeing in Healthcare Professionals Under the Lens of Salivary, Immune, and Microbiome Mechanisms

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Highlights

- Positions oral cellular homeostasis as a biological interface linking occupational stress, immunity, and the oral microbiome.
- Integrates evidence from oral biology, nutrition, and occupational health.

Main findings

- Occupational stress is associated with dysregulation of salivary biomarkers and oral immune-microbial balance.

Implications of the main findings

- Oral biomarkers may support early detection of occupational strain.
- Integrated stress and diet-based interventions may enhance professional wellbeing.

Abstract

Background: Healthcare professionals experience continuous biological and psychosocial stressors that may disturb oral and systemic homeostasis. Alterations in salivary secretion, mucosal immunity, and microbiome composition reflect adaptive cellular responses to chronic occupational stress. Understanding these mechanisms may provide a biological framework for resilience and wellbeing in clinical everyday practice. Objective: To narratively review the evidence linking oral cellular and molecular mechanisms, -salivary biomarkers, epithelial and immune cell activity, and microbiome dynamics- with stress, fatigue, burnout, and wellbeing outcomes among healthcare professionals. Methods: A PRISMA-guided search of PubMed, Scopus, Web of Science, and Cochrane Oral Health identified studies investigating oral cellular or molecular parameters in relation to occupational stress or wellbeing indicators in healthcare settings. Eligible designs included observational, experimental, and interventional studies. Data were extracted using standardized forms, quality was appraised via ROBINS-I and the Newcastle-Ottawa scale, and results were synthesized thematically. Results: Evidence from 99 studies suggests that chronic occupational stress elevates salivary cortisol, oxidative stress markers, and pro-inflammatory cytokines (IL-6, TNF- α), while reducing protective salivary immunoglobulin A and microbiome diversity. Balanced oral immune and microbial profiles were associated with better psychological adaptation and lower fatigue indices. Conclusions: Oral cellular homeostasis offers a promising window into the biological underpinnings of occupational stress and resilience in healthcare professionals. Systematic integration of salivary and mucosal biomarkers into workplace wellbeing programs could enhance early detection of dysregulated stress physiology. Future interdisciplinary research should bridge oral biology, occupational medicine, and mental health to strengthen sustainable wellbeing strategies across the health workforce.

Keywords: epithelial-immune crosstalk; mucosal homeostasis; biopsychosocial models; oxidative stress; occupational stress response; occupational wellbeing indices; salivary diagnostics in workplace medicine; oral homeostasis; salivary biomarkers; immune regulation; microbiome; healthcare professionals; resilience

1. Introduction

The oral cavity constitutes a highly dynamic mucosal barrier where epithelial cells, saliva, immune mediators, and a diverse microbial community interact continuously to maintain tissue homeostasis [1,2]. The structural and functional complexity of the oral mucosa reflects its composite defense architecture meaning physical, biochemical, microbiological, and immunological layers that collectively regulate pathogen exclusion and immune tolerance [3,4]. Recent advances in mucosal immunology report that epithelial-immune crosstalk determines not only local oral health but also systemic inflammatory and neuroendocrine dynamics, highlighting the integrative nature of oral barrier biology [5,6]. The oral microbiome forms an essential component of this ecosystem, shaping immune maturation, barrier integrity, and inflammatory tone [7,8]. Dysbiosis within oral microbial communities has been linked to periodontal disease, mucosal inflammation, wound-healing dysregulation, and metabolic and neurocognitive outcomes, positioning oral microbial ecology as an important determinant of host systemic physiology [9,10]. Updated reviews have further shown that fluctuations in oral microbial composition interact with mucosal cytokine networks and epithelial signaling pathways, contributing to allostatic responses across the lifespan [11,12].

Complementing microbial mechanisms, saliva has emerged as a powerful, non-invasive diagnostic matrix that reflects both local oral status and systemic physiological states [13,14]. Salivary immunoglobulins, cytokines, proteomic signatures, and metabolites offer insight into mucosal immunity, inflammation, oxidative stress, and endocrine responses [15,16]. Contemporary analyses highlight saliva’s diagnostic potential in infection, oncology, metabolic disorders, and mucosal immunopathology, reinforcing its relevance for personalized health monitoring [17,18]. Indeed, recent immunobiology syntheses reveal that saliva integrates epithelial, microbial, and neuroendocrine signals, making it a sensitive biomarker matrix for stress-associated biological alterations [19,20].

Parallel to developments in oral biology, occupational wellbeing among healthcare professionals has become an urgent global concern, with high rates of burnout, chronic fatigue, circadian misalignment, depressive symptoms, and stress-related physiological dysregulation [21–23]. Systematic reviews consistently show that healthcare workers face elevated emotional and physical strain associated with workload pressure, moral distress, patient acuity, and disrupted work-rest cycles [24,25]. Importantly, biological studies increasingly demonstrate measurable physiological alterations, including immune suppression, cytokine activation, and neuroendocrine dysregulation, associated with occupational strain in healthcare environments [26,27]. Salivary biomarkers have been central in this research domain, with cortisol, SIgA, and pro-inflammatory cytokines widely used as indicators of occupational stress and allostatic load [28–30]. Methodological syntheses confirm that diurnal cortisol rhythms, cortisol awakening response, and stress-induced salivary cytokine fluctuations correlate strongly with occupational demands across nursing, emergency care, and shift-based medical work [31,32]. Evidence also suggests that mucosal immune components, including SIgA and epithelial-derived mediators, respond to acute and chronic stress exposure, further supporting the role of oral biomarkers in monitoring work-related physiological strain [14,33,34].

Despite these converging scientific trajectories, the interface between oral cellular homeostasis and occupational wellbeing in healthcare professionals remains substantially underexplored. Research in mucosal immunology has reported on the epithelial-immune-microbial signaling pathways but rarely considers psychosocial or occupational determinants as upstream modulators [1,5]. Conversely, occupational health studies employing salivary biomarkers often frame them as

peripheral stress indicators rather than components of an interconnected mucosal system shaped by microbial and epithelial dynamics [28,30]. This disciplinary separation obscures potential bidirectional pathways through which occupational stress could influence oral homeostasis and through which oral mucosal biology might mediate resilience or vulnerability to chronic strain [33]. Healthcare professionals represent a uniquely relevant population in this context: continuous exposure to biological aerosols, pathogens, altered circadian patterns, and emotional labor may create distinct pressures on mucosal barrier immunity and microbiome stability [26,27]. Yet the extent to which occupational exposures reshape oral immune or microbial equilibrium, or how oral biomarkers might inform early detection of burnout and stress-related dysregulation, remains largely uncharacterized.

Accordingly, this narrative review aims to synthesize the current knowledge on oral cellular homeostasis, including salivary, immune, and microbiome pathways, and its potential links to occupational wellbeing in healthcare professionals. Specifically, we (i) summarize advances in oral mucosal immunity and microbiome science relevant to systemic regulation; (ii) review the diagnostic and physiological roles of salivary biomarkers in occupational contexts; and (iii) integrate emerging findings into a conceptual framework for understanding how oral biological mechanisms may interact with occupational stress, resilience, and wellbeing in healthcare settings. Within this interdisciplinary context, the present review is guided by a central question: how oral cellular homeostasis, capturing salivary endocrine and immune biomarkers, epithelial barrier function, and oral microbiome dynamics, relates to occupational wellbeing, stress physiology, and burnout vulnerability in healthcare professionals? Specifically, we examine whether chronic occupational demands, circadian disruption, and emotional strain can modulate salivary cortisol, SIgA, cytokines, or microbial community structure, and whether such biological shifts parallel validated psychological measures of stress, fatigue, and wellbeing. We further consider the methodological constraints that influence the interpretation of salivary and mucosal biomarkers and explore whether oral immune-microbial signatures may serve as early indicators of resilience or susceptibility under sustained professional pressure. Ultimately, this review seeks to outline how neuroendocrine-mucosal cross-talk might mediate the biological imprint of occupational stress and how an integrated oral-occupational health framework could enhance monitoring, prevention, and wellbeing strategies within healthcare settings. The PICO approach we used is in healthcare professionals (P), exposure to oral cellular mechanisms, including salivary, immune, and microbiome pathways (I), that is examined in relation to occupational wellbeing outcomes such as stress physiology, burnout, and resilience (O), and compared with populations experiencing lower occupational strain where applicable (C).

2. Materials and Methods

This work was designed as a narrative review aiming to synthesize current evidence on the biological mechanisms of oral cellular homeostasis -salivary, epithelial-immune, and microbiome pathways- and their potential relationship to occupational wellbeing in healthcare professionals. A narrative review methodology was selected because the research question spans multiple heterogeneous domains (oral immunology, microbiome science, salivary diagnostics, occupational medicine, psychoneuroimmunology), for which available studies vary widely in design, outcomes, biomarkers, and conceptual frameworks [35]. The goal was to provide an integrative, theory-building overview rather than a quantitative meta-analysis [36].

2.1. Search Strategy and Information Sources

The selection of databases was guided by the interdisciplinary nature of the research question, which spans oral biology, immunology, microbiome science, occupational health, and mental wellbeing. PubMed/MEDLINE was chosen as the primary biomedical database due to its comprehensive coverage of oral immunology, salivary diagnostics, stress physiology, and occupational health research, as well as its structured indexing through Medical Subject Headings

(MeSH), which enabled precise mapping of core biological and psychosocial concepts. Scopus and Web of Science were included to ensure broader interdisciplinary coverage and citation tracking, capturing relevant studies published in dental science, public health, psychology, and systems biology journals that may not be indexed in PubMed alone. Cochrane Oral Health was additionally searched to identify high-quality evidence syntheses and intervention-focused studies relevant to oral health and preventive strategies.

Search queries were tailored to the indexing systems and search functionalities of each database. In PubMed/MEDLINE, both Medical Subject Headings (MeSH) and free-text keywords were employed to capture core concepts related to oral cellular homeostasis (including oral mucosa, oral immunity, saliva, and the oral microbiome), biological and stress-related biomarkers (such as cortisol, secretory IgA, cytokines, and metabolomics), and indicators of occupational wellbeing (including stress, burnout, fatigue, and healthcare professionals). Boolean operators (AND/OR) were applied to systematically combine conceptual domains while retaining thematic relevance. In Scopus and Web of Science, searches were conducted using keyword combinations applied to titles, abstracts, and author keywords in order to accommodate variations in terminology across disciplines. To ensure comprehensive coverage, the reference lists of all included articles and relevant narrative or systematic reviews were manually screened for additional eligible studies. Only peer-reviewed publications available in full English text were considered for inclusion.

2.2. Eligibility Criteria

Studies were eligible for inclusion if they met at least one of the following criteria: (1) examined oral epithelial, salivary, or microbiome mechanisms related to mucosal immunity or barrier homeostasis; (2) evaluated salivary biomarkers of stress, endocrine activity, immune activity, or inflammation; (3) assessed occupational stress, burnout, fatigue, psychological wellbeing, or physiological responses in healthcare professionals; (4) provided conceptual, mechanistic, or empirical insights bridging oral biology with systemic stress physiology. Overall, experimental, observational, interventional, and review studies were included. Exclusion criteria were: non-oral biomarkers without relevance to mucosal immunity, non-healthcare populations unless offering transferable biological mechanisms, animal studies without translational value, non-peer-reviewed documents, conference abstracts, and publications not available in English.

2.3. Study Selection Process

Two reviewers independently screened titles and abstracts for relevance. Full texts were retrieved for all studies meeting or potentially meeting the inclusion criteria. Disagreements were resolved through discussion and consensus. Although narrative in design, the review followed PRISMA principles for transparency in search and selection [37]. The heterogeneity of study designs, biomarkers, and occupational outcomes precluded quantitative meta-analysis.

PRISMA Flow Description

The literature search across PubMed/MEDLINE, Scopus, Web of Science, and Cochrane Oral Health initially yielded 1,247 records. After removal of 312 duplicate records, 935 unique articles remained for title and abstract screening. During the initial screening phase, 739 records were excluded because they did not address oral biology, salivary or mucosal mechanisms, occupational stress, or healthcare-related populations. The remaining 196 articles were assessed in full text for eligibility. Of these, 97 full-text articles were excluded for the following reasons: lack of relevance to oral cellular mechanisms ($n = 41$), focus on non-healthcare populations without transferable biological mechanisms ($n = 28$), absence of occupational or wellbeing-related outcomes ($n = 19$), or insufficient methodological or conceptual contribution ($n = 9$). A total of 99 studies met the inclusion criteria and were incorporated into the qualitative synthesis. These studies encompassed mechanistic immunology and microbiome research, salivary biomarker investigations, occupational health and

wellbeing studies, and selected interventional trials relevant to stress biology and resilience in healthcare professionals. Given the heterogeneity of study designs, populations, biomarkers, and outcome measures, no quantitative meta-analysis was performed.

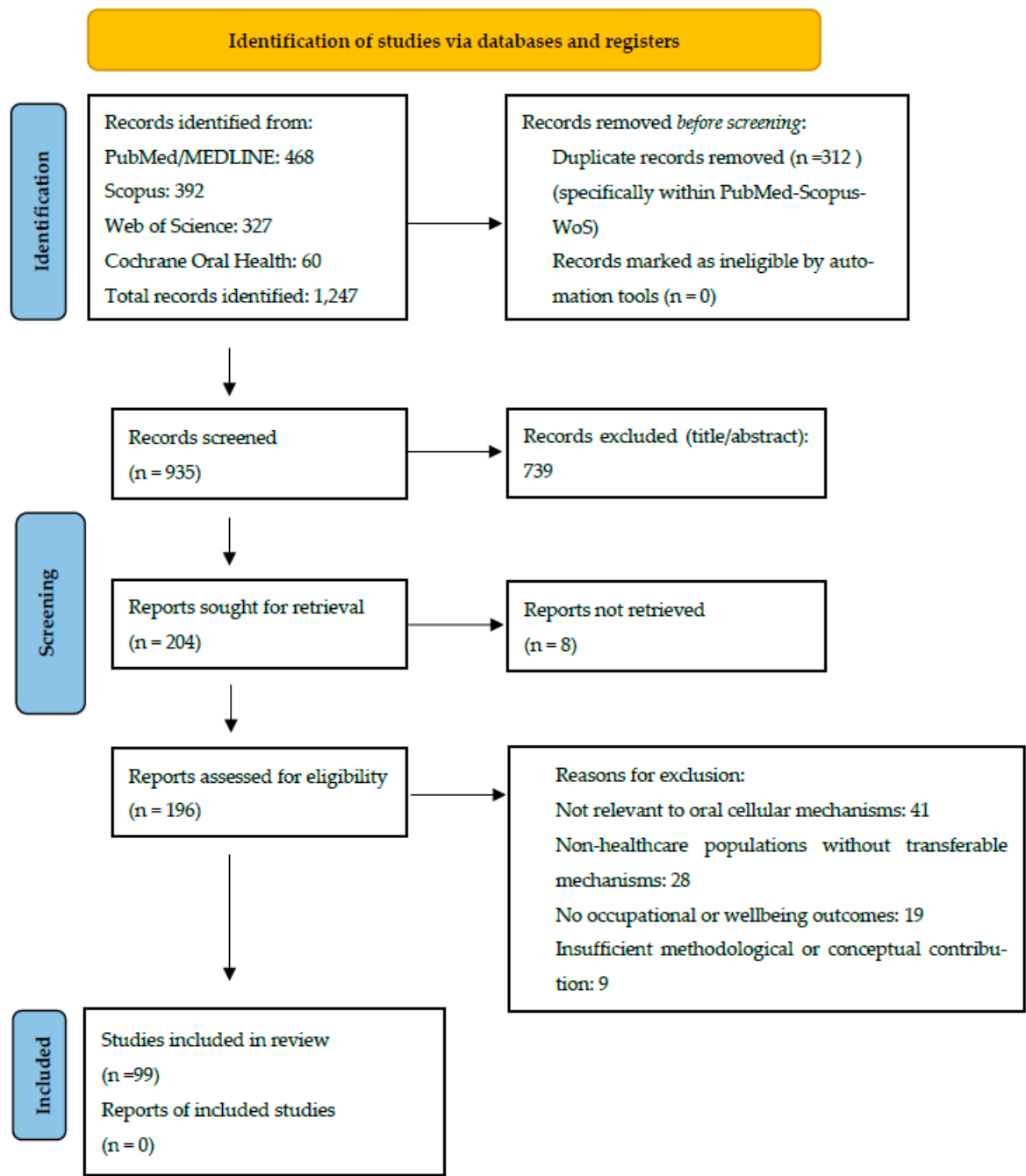


Figure 1. Prisma flow chart of the study.

2.4. Data Extraction and Thematic Organization

Data extraction focused on: (i) study design; (ii) population characteristics; (iii) oral biomarkers assessed (salivary cortisol, SIgA, cytokines, microbiome profiles, epithelial immune markers); (iv) occupational or psychological outcomes (burnout, stress, fatigue, circadian disruption); and (v) mechanistic insights linking oral biology to systemic physiology. Thematic synthesis was applied, grouping emerging evidence into three major domains: (1) oral mucosal immunity and epithelial barrier regulation; (2) oral microbiome composition and its interaction with immunological and

neuroendocrine pathways; (3) salivary biomarkers as indicators of stress physiology and occupational load in healthcare professionals.

Cross-domain integration was then performed to construct a conceptual model describing potential bidirectional pathways between oral cellular homeostasis and occupational wellbeing. The model derived out of this search is to be seen in Figure 2.

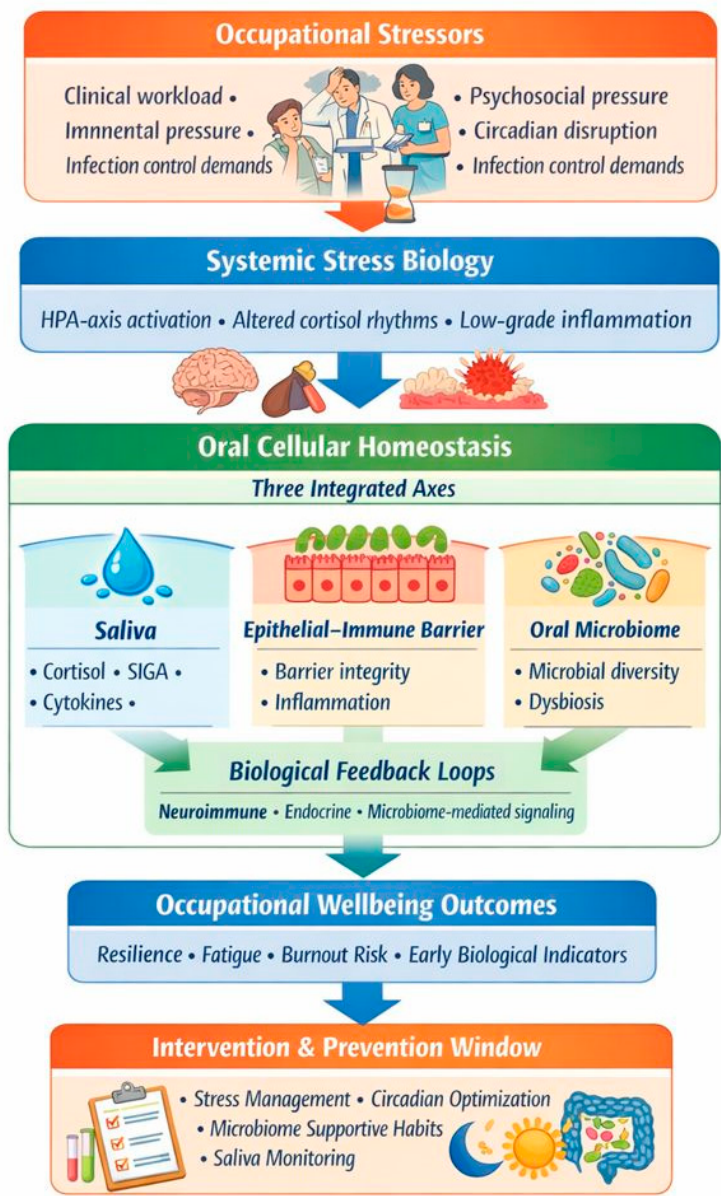


Figure 2. Occupational stress activates systemic stress biology, which interacts bidirectionally with oral salivary, epithelial-immune, and microbiome mechanisms. Disruption of oral cellular homeostasis may reflect and modulate occupational wellbeing, positioning the oral cavity as a potential interface for early detection and preventive intervention in healthcare professionals.

2.5. Quality Considerations and Methodological Limitations

Given the narrative design of this review and the substantial heterogeneity of the included literature, the uniform application of formal risk-of-bias assessment tools (such as ROBINS-I, the Newcastle-Ottawa Scale, or RoB 2) was not considered appropriate across all study types. Instead, methodological quality was evaluated qualitatively, with attention to key features relevant to the research question, including sample size adequacy, validity and specificity of oral and salivary

biomarkers, conditions and timing of saliva collection, analytical and laboratory techniques, control for diurnal and contextual variation, and the operationalization and measurement of occupational stressors. Particular consideration was given to methodological transparency and consistency in defining biological and psychosocial outcomes. Variability in biomarker assays, microbiome sequencing platforms, bioinformatic pipelines, and psychological assessment instruments was recognized as a major source of methodological heterogeneity, limiting direct comparability across studies [38]. These methodological differences were explicitly taken into account during the synthesis process and informed the relative weighting and interpretation of findings within and across thematic domains. Rather than serving as grounds for exclusion, such limitations were treated as critical contextual factors shaping the strength and transferability of the available evidence.

2.6. Review Scope and Conceptual Orientation

This review does not aim to present an exhaustive systematic catalogue of all relevant studies; rather, it integrates high-quality and mechanistically informative evidence to outline how oral immune-microbial-endocrine dynamics may intersect with occupational stress biology. The approach reflects the inherently interdisciplinary nature of the topic and seeks to advance conceptual understanding, generate hypotheses, and identify gaps requiring targeted empirical investigation.

3. Results

Across the final body of evidence included in this review, studies were organized into seven major thematic domains reflecting the core components of oral cellular homeostasis and their intersections with occupational wellbeing: (i) oral mucosal immunity and epithelial barrier regulation, (ii) oral microbiome composition and its systemic physiological links, (iii) integrated evidence on oral microbiome, mucosal immunity, saliva, and systemic-stress biology, (iv) occupational Stress, wellbeing & biomarkers, (v) dietary, nutritional, and food-related modulators of oral cellular homeostasis, and (vi) interventions targeting stress biology, oral homeostasis, and occupational wellbeing. This thematic categorization allowed for the synthesis of highly heterogeneous research designs, ranging from mechanistic immunology and microbiome analyses to cross-sectional wellbeing studies and randomized controlled interventions, while preserving the mechanistic coherence necessary for interpreting how oral biological pathways may mirror or modulate occupational stress physiology. Table 1 presents the key articles mapped to each domain, summarizing study characteristics, methodological approaches, and principal findings relevant to the conceptual model of oral homeostasis as a potential biomarker and resilience interface in healthcare professionals.

Table 1. Key articles grouped by thematic domains relevant to oral cellular homeostasis and occupational wellbeing.

A. Oral mucosal immunity & epithelial barrier biology			
Thematic domain	Authors & year	Study type / methods	Key findings / relevance
Oral mucosal barrier mechanisms	[1] Lin et al., 2021	Mechanistic immunology review	Microbe-epithelial-immune crosstalk; foundation of oral homeostasis.
Physical & immune barriers of oral mucosa	[3] Şenel, 2021	Comprehensive immunity review	Defines physical, microbial, immune layers of the oral barrier.
Oral vs gastrointestinal immune niches	[5] Suárez et al., 2021	Comparative immunology review	Highlights uniqueness of oral immune niche vs gut.

$\gamma\delta$ T cells in oral diseases	[6] Wei et al., 2024	Immunology review	Shows $\gamma\delta$ T cell roles in epithelial surveillance under stress.
Sensory-neuroimmune interactions	[7] Wang et al., 2024	Neuroimmune review	Demonstrates endocrine-neural modulation of mucosal immunity.
Oral innate immunity & epithelial cells	[34] Nittayananta, 2026	Immunology chapter	Maps epithelial innate mechanisms; core for homeostasis definition.
Oral mucosal barrier & systemic health	[39] Xin & Lei, 2026	Narrative review; mechanistic synthesis	Describes the oral mucosal barrier as a dynamic immune-epithelial interface regulating microbial balance, inflammation, and systemic signaling.

B. Oral microbiome composition, stability & systemic links

Lifespan mucosal-microbial interactions	[7] Iliev et al., 2025	Conceptual immunology review	Microbiota shapes mucosal immunity across lifespan.
Periodontitis microbiome biomarkers	[8] Radu et al., 2025	Narrative review	Links microbiome & salivary biomarkers to inflammation.
Hormonal effects on oral microbiome	[11] Rus et al., 2025	Clinical/experimental review	Shows endocrine modulation of oral microbiome; relates to stress.
Microbiome & wound healing	[12] Simon-Soro et al., 2025	Human biomarker study	Microbial shifts correlate with inflammatory wound healing responses.
Social capital-oral health-aging	[10] Liang & Gomaa, 2023	Cohort multi-omics	Oral health, cognition & aging interconnected biologically.
Tooth loss & cognitive decline	[9] Galindo-Moreno et al., 2022	Systematic review	Oral health linked to neurocognitive outcomes.
Gut-oral microbiome & periodontitis	[40] Yu et al., 2025	Meta-analysis of Mendelian randomization studies	Supports a causal role of gut microbiota composition in periodontitis risk, strengthening oral-systemic microbiome links.
Microbiome & viral-associated disease	[41] Zhang et al., 2025	Systematic review & meta-analysis	Demonstrates microbiome alterations in viral infection-associated inflammatory diseases, highlighting immune-microbial interactions.
Endodontic infections & microbiome	[42] Liu et al., 2025	Systematic review & meta-analysis (NGS-based)	Identifies Enterococcus as a persistent endodontic pathogen, emphasizing microbial resilience under inflammatory stress.
Prosthodontics & oral microbiome	[43] Madhan Kumar et al., 2025	Systematic review & meta-analysis	Shows dentures significantly alter oral microbiome composition and inflammatory profiles.
Immune signaling & barrier traversal	[44] Kulkarni et al., 2025	Meta-analysis of RNA-seq data	Identifies interferon-induced genes facilitating barrier

Nutritional modulation of immunity	[45] Lu et al., 2024	Randomized intervention study	crossing, relevant to mucosal immune vulnerability. L-glutamine supplementation enhances mucosal immunity and hormonal balance under physical stress.
Mucosal vaccination & immunity	[46] Flitter et al., 2025	Phase 2 placebo-controlled trial	Oral vaccination induces mucosal immunity and reduces viral shedding, validating oral immune responsiveness.
Oral mucosa & respiratory immunity	[47] Wang et al., 2024	Narrative review	Highlights oral microecological control of virus-related inflammatory responses.
Antiviral immunity in oral cavity	[48] Hickman & Moutsopoulos, 2025	Narrative review	Positions the oral cavity as an active antiviral immune site with systemic relevance.
Saliva-microbiome interactions	[49] Heller et al., 2025	Narrative review	Describes saliva as an active regulator of microbial stability and host defense.
Salivary metabolomics	[15] Zhao et al., 2025	Narrative review	Establishes saliva metabolomics as a non-invasive tool linking oral and systemic disease.
Microbial metabolites & immunity	[50] Schütz et al., 2025	Mechanistic review	Shows microbiome-derived metabolites modulate host immune responses and inflammation.
Oral epithelial innate immunity	[34] Nittayananta, 2026	Narrative review	Details epithelial cells as immune sentinels maintaining oral barrier homeostasis.
Saliva & systemic diagnostics	[13] Surdu et al., 2025	Narrative review	Supports saliva as a diagnostic medium for systemic and inflammatory diseases.
Oral immune responses	[14] Matsuoka et al., 2025	Experimental & narrative synthesis	Describes natural and induced immune mechanisms in saliva and oral tissues.
Micronutrients & emotional regulation	[51] Katta et al., 2024	Randomized controlled trial	Shows orally absorbed micronutrients reduce emotion dysregulation, linking nutrition and neurobiology.
Periodontitis & metabolomics	[52] Albahri et al., 2025	Observational metabolomics study	Links salivary metabolic profiles with chronic periodontal inflammation.
Saliva & cancer immunotherapy	[53] Nejat Dehkordi et al., 2025	Narrative review	Highlights salivary biomarkers as predictors of immunotherapy response.
Environment, stress & microbiome	[54] Shibata et al., 2025	Randomized controlled trial	Demonstrates environmental enrichment alters cortisol levels and microbiome composition.
Salivary transcriptomics	[55] Barnes et al., 2025	Methodological review	Positions saliva transcriptomics as a window into systemic gene regulation.

Saliva in precision dosing	[56] Xu et al., 2025	Methodological review	Validates saliva as a matrix for therapeutic drug monitoring.
Salivaomics in cancer	[57] Saravanan et al., 2025	Narrative review	Integrates proteomic, metabolomic, and transcriptomic saliva data for disease monitoring.
Salivary proteome & immunity	[18] Carneiro et al., 2026	Narrative review	Describes salivary proteins as mediators of immune defense and infection control.
Wound healing & microbiome	[58] Santamaria et al., 2025	Observational study	Links microbiome profiles and inflammatory biomarkers with oral wound healing outcomes.
Hormone diagnostics via saliva	[17] Ferrari et al., 2025	Narrative review	Establishes saliva as a reliable matrix for endocrine and stress hormone assessment.
Pediatric stress biomarkers	[59] Main et al., 2025	Narrative review	Identifies salivary markers as indicators of stress in dental settings.
Periodontal biomarkers	[60] Alavi et al., 2025	Narrative review	Positions salivary biomarkers as tools for early detection and precision dentistry.
Salivary diagnostics overview	[16] Albagieh et al., 2025	Narrative review	Summarizes applications, benefits, and limitations of salivary diagnostics.
C. Integrated evidence on oral microbiome, mucosal immunity, saliva, and systemic-stress biology			
Nutritional modulation of microbiome & hormones	[61] Carter et al., 2025	Randomized controlled trial	Demonstrates that specific dietary oligosaccharides can simultaneously remodel the microbiome, circulating hormones, and metabolic profiles, supporting diet-driven regulation of immune–endocrine homeostasis.
Immune amplification therapies	[62] Steffin et al., 2025	Translational clinical study (CAR-T)	Illustrates how cytokine-enhanced immune signaling can reshape tumor-immune dynamics, highlighting systemic consequences of immune modulation relevant to mucosal immunity frameworks.
Anti-inflammatory dietary interventions	[63] Pardiñas López et al., 2025	Triple-blind randomized clinical trial	Provides clinical evidence that natural dietary agents exert antimicrobial and anti-inflammatory effects against periodontal pathogens, reinforcing nutrition as a modulator of oral inflammation.
Epithelial cytokine signaling	[64] McSorley & Hodge, 2025	Mechanistic parasitology review	Identifies epithelial-derived cytokines as central regulators of mucosal immune balance,

			relevant to stress- and infection-induced barrier modulation.
Antigen presentation & mucosal tolerance	[65] Hoelting et al., 2025	Mechanistic immunology review	Establishes antigen-presenting cells as key arbiters of tolerance versus inflammation at mucosal surfaces.
Metabolic syndrome & oral permeability	[66] Nehaoua et al., 2026	Narrative review	Links metabolic dysregulation with oral hyperpermeability and microbiome-driven inflammation, supporting oral-systemic disease integration.
Viral infection & mucosal immunity	[67] Shacklett, 2025	Narrative review	Highlights vulnerability and plasticity of mucosal immunity under acute viral stress.
Nanotechnology & immune modulation	[68] Jung & Son, 2025	Experimental/narrative review	Shows how nanoparticle-based strategies modulate mucosal immunity and microbiome interactions.
Anti-inflammatory cytokine regulation	[69] Branchett et al., 2024	Mechanistic immunology review	Positions IL-10 as a central regulator limiting mucosal inflammation across tissues.
Salivary cortisol & stress biology	[30] Dong et al., 2024	Systematic review	Defines methodological standards and limitations for using salivary cortisol as a stress biomarker.
Salivary exosomes	[70] Yu et al., 2024	Narrative review	Identifies salivary exosomes as emerging diagnostic and signaling mediators in oral disease.
Vaccine delivery via mucosa	[71] Laa et al., 2025	Narrative review	Describes protein nanocages as innovative platforms for mucosal immune activation.
T cell-epithelial crosstalk	[72] Bordoni & Fazio, 2025	Mechanistic genetics review	Explains how epithelial-T cell signaling regulates mucosal immune equilibrium.
Microbiota-dendritic cell interactions	[73] Letz et al., 2025	Mechanistic immunopathology review	Demonstrates microbiota-driven shaping of dendritic cell responses in chronic inflammation.
Flavonoids & oxidative stress	[74] Jomova et al., 2025	Mechanistic review	Links dietary flavonoids to redox balance and inflammatory control.
Polyphenols & inflammatory injury	[75] Wang L. et al., 2025	Experimental pharmacology study	Shows polyphenols mitigate inflammation and oxidative damage via cytokine modulation.
Oxidative stress & immune dysfunction	[76] Luo et al., 2025	Mechanistic immunology review	Establishes oxidative stress as a driver of immune-mediated tissue damage.
Vascular inflammation	[77] Shao et al., 2024	Mechanistic cell biology review	Demonstrates oxidative stress-induced microenvironmental disruption in chronic disease.

Inflammation & tumorigenesis	[78] Wang M. et al., 2025	Narrative review	Frames inflammation–oxidative stress axis as a universal oncogenic driver.
Oxidative stress in cancer	[79] Tomaziu-Todosia Anton et al., 2025	Narrative review	Reviews antioxidant strategies targeting inflammatory carcinogenesis.
Infection, inflammation & fertility	[80] Potiris et al., 2025	Narrative review	Highlights systemic consequences of chronic inflammation and oxidative stress.
Inflammation & mental health	[81] Teixeira et al., 2025	Narrative review	Positions inflammation as a biological substrate of late-life depression.
Anxiety & aging	[82] Johnco et al., 2024	Clinical psychiatry review	Connects stress-related disorders with biological aging processes.
Oxidative stress biomarkers	[83] Aravapally et al., 2025	Methodological review	Discusses advanced strategies for quantifying oxidative stress in complex systems.
Gingival immune regulation	[84] Hsu et al., 2025	Narrative review	Describes immune and tissue dynamics in experimental gingivitis.

D. Occupational Stress, wellbeing & biomarkers

Stress, anxiety, depression, resilience & spiritual wellbeing in students	[85] Mangoulia et al., 2025	Cross-sectional observational study	Reports high psychological burden among dental & nursing students post-pandemic; highlights resilience & hope as protective factors; relevant to occupational wellbeing trajectories.
Quality of life & wellbeing in academic dental personnel	[86] Antoniadou, Mangoulia & Myrianthefts, 2023	Cross-sectional study comparing academic staff & service quality	Demonstrates associations between professional wellbeing, QoL parameters, and perceived quality of academic services; supports link between occupational strain and performance outcomes.
Circadian dysregulation & burnout	[22] Ungurianu & Marina, 2025	Systematic review	Burnout affects circadian biology; linked to cortisol.
Immune & mucosal function under stress	[23] Zhang et al., 2025	Immunology review	Chronic stress disrupts systemic and mucosal immunity.
Burnout, depression & suicide in HCWs	[24] Nguyen et al., 2025	Scoping review	High burden of psychological distress in healthcare.
Stress-reduction therapies	[25] Meneses Damasceno et al., 2025	Integrative review	Interventions improve physiological stress markers.
Tele-yoga for HCWs	[26] Naveen et al., 2024	Randomized controlled trial	Improves cortisol and immune markers.
Night-shift worker intervention	[27] Robinson et al., 2025	Randomized crossover trial	Improves salivary cortisol and metabolic responses.

Salivary biomarkers in occupational medicine	[28] Koh & Koh, 2007	Landmark review	Classic reference establishing saliva as biomarker matrix.
Salivary cortisol methodology	[30] Dong et al., 2024	Systematic methodological review	Timing, diurnal variation, sampling, assay accuracy.
Cortisol Awakening Response	[31] Stalder et al., 2025	Endocrine review	CAR as reliable HPA-axis stress indicator.
Environmental richness & cortisol	[32] Shibata et al., 2025	Randomized trial	Environment modulates cortisol & microbiota. Demonstrates associations between salivary biomarkers and occupational fatigue, supporting saliva as a non-invasive indicator of work-related physiological strain.
Salivary biomarkers & occupational fatigue	[87] Mahdavi et al., 2025	Observational study; salivary biomarker assessment	
E. Dietary, nutritional, and food-related modulators of oral cellular homeostasis			
			Diet is a key determinant of oral microbiota composition; fiber-rich diets support microbial diversity and immune balance, whereas high-sugar patterns promote dysbiosis and inflammation. Links nutrition to oral and periodontal health via immune modulation, oxidative stress regulation, and microbiome-mediated effects on epithelial resilience and inflammation. Higher dietary inflammatory potential is associated with altered oral and gut microbiota and increased systemic inflammation. Identifies SCFAs as metabolic mediators linking diet to immune regulation, epithelial energy metabolism, and microbial stability. Examines oral-gut microbiome crosstalk and the role of diet in shaping microbial ecology and immune signaling. High-fiber, anti-inflammatory diets protect against periodontal inflammation via SCFA production and immune modulation. Dietary composition shapes oral microbiome structure, inflammatory signaling, and disease susceptibility.
Diet-Oral Microbiota Interaction	[88] García HC et al., 2025	Systematic review	
Nutrition and Oral-Periodontal Health	[89] Curca FR et al., 2026	Narrative review	
Dietary Inflammatory Potential and Microbiome	[90] Liu J et al., 2025	Observational study with microbiome profiling	
SCFAs and Microbiome-Host Crosstalk	[91] Nireeksha et al., 2025	Narrative mechanistic review	
Oral-Gut Microbiome Axis	[92] Parveen S et al., 2024	Narrative review	
Dietary Patterns and Periodontitis	[93] Mao J.-S. et al., 2025	Narrative review	
Diet and Oral Microbiome	[94] Santonocito S et al., 2025	Book chapter / narrative synthesis	
F. Interventions targeting stress biology, oral homeostasis, and occupational wellbeing			

Oral health coaching & dietary interventions	[95] Antoniadou & Varzakas, 2020	Narrative review; coaching models	Structured oral health and diet coaching improves adherence, self-management, and preventive oral health behaviors in independent elderly populations. Highlights diet as a modulator of oral and systemic inflammation, microbiome balance, and chronic disease risk.
Diet, oral health, and systemic links	[96] Antoniadou & Varzakas, 2021	Narrative review	Integrates probiotic-based strategies with patient coaching to support caries prevention and oral microbial balance. Demonstrates the role of microbiome-targeted functional foods in metabolic regulation and inflammatory control, with implications for oral-systemic health.
Probiotics, prebiotics, and caries prevention	[97] Amargianitakis et al., 2021	Narrative review	Presents dietary strategies as tools for reducing systemic and oral disease burden via immune and microbiome modulation. Digital interventions significantly reduce stress, anxiety, and depression, supporting scalable occupational wellbeing strategies.
Functional foods, gut microbiome, and metabolic health	[98] Bezirtzoglou et al., 2021	Systematic review	Shows broader wellbeing benefits of digital mental health interventions beyond depressive symptom reduction. Identifies unmet psychosocial and emotional needs contributing to chronic stress and burnout in caregiving roles.
Dietary interventions for disease prevention	[99] Antoniadou & Varzakas, 2024	Narrative review	Frames moral injury as a distinct stressor requiring targeted preventive and therapeutic interventions. Tele-yoga reduced burnout and improved immune markers in healthcare workers, linking stress reduction with biological outcomes.
Digital lifestyle interventions & mental health	[100] Brinsley et al., 2025	Systematic review & meta-analysis	Summarizes effectiveness of non-pharmacological stress-
Digital interventions beyond depression	[101] Linardon et al., 2025	Meta-analysis	
Caregiver burden and psychosocial stress	[102] Gholami et al., 2025	Scoping review	
Moral injury prevention	[103] Williamson et al., 2025	Narrative review	
Mind-body interventions & immune markers	[104] Naveen et al., 2024	Pilot randomized controlled trial	
Non-pharmacological stress interventions	[25] Meneses Damasceno & Pimentel, 2025	Integrative review	

		reduction strategies in healthcare workers.
Breathwork and stress resilience [105] Little, 2025	Narrative review	Breath-based interventions enhance stress resilience and emotional regulation through autonomic modulation.
Lifestyle, circadian disruption, and metabolic health [27] Robinson et al., 2025	Randomized crossover trial	Lifestyle intervention improved metabolic and mental health outcomes in female night-shift healthcare workers.

3.1. Oral Mucosal Immunity and Epithelial Barrier Responses

Across the selected literature, oral mucosal immunity emerges as a tightly regulated, multilayered system in which epithelial integrity, innate immune surveillance, and resident microbiota operate as a single functional unit [34]. Mechanistic analyses demonstrate that homeostasis depends on continuous bidirectional communication between epithelial cells, commensal microorganisms, and immune mediators, a process mediated by cytokines, antimicrobial peptides, and pattern-recognition receptors that calibrate tolerance and inflammation in real time [1]. The oral mucosa is described not merely as a physical barrier but as a metabolically active immune organ whose structural and immunological layers respond rapidly to environmental disturbances, microbial pressures, and endocrine changes [15,34,46,48]. Detailed immunological mapping confirms that the oral barrier consists of an epithelial-microbial-immune triad capable of immediate adaptation via tight-junction remodeling, mucin secretion, and activation of innate effectors such as defensins and secretory IgA [3,39]. Comparative mucosal analyses reveal that, unlike the gastrointestinal tract, the oral mucosa is exposed to higher microbial diversity, rapid antigenic turnover, and continuous mechanical and chemical stress, requiring a more vigilant innate immune posture and faster epithelial renewal [5]. Central to this defense are specialized lymphocyte subsets, most notably $\gamma\delta$ T cells, which contribute to epithelial repair, microbial containment, and immune tolerance, and which have been shown to be sensitive to neuroendocrine modulators and stress-related dysregulation [6]. Parallel advances in neuroimmune biology show that sensory innervation of the oral mucosa is intimately connected with immune activation: neuropeptides, sympathetic mediators, and glucocorticoids influence epithelial cytokine expression, dendritic cell priming, and the balance between pro- and anti-inflammatory responses [7]. Under conditions of sustained stress, these neuroimmune pathways can induce transient barrier weakening, alter antimicrobial output, and shift inflammatory thresholds [1,7]. Further immunological work maps how epithelial cells act as both structural and immune sentinels, initiating rapid signaling through Toll-like receptors, inflammasome components, and chemokine networks that guide leukocyte recruitment and microbial containment [34]. When epithelial and immune homeostasis is perturbed, whether through microbial imbalance, endocrine fluctuation, or chronic systemic stress, the mucosa demonstrates measurable shifts in permeability, cytokine gradients, oxidative stress, and immune-cell composition [1,3]. These findings indicate overall that the oral mucosa is a dynamic neuroimmune barrier whose cellular homeostasis is exquisitely sensitive to systemic physiological stressors, including those commonly observed in healthcare work environments. This establishes a strong mechanistic basis for considering epithelial-immune-microbial interactions as potential early indicators of occupational strain and resilience [39]. As seen in Figure 3, the oral mucosa functions as a dynamic epithelial-immune-microbial interface regulated by continuous bidirectional signaling among epithelial cells, innate and adaptive immune mechanisms, resident microbiota, and neuroendocrine inputs. Systemic and occupational stressors modulate these interactions through endocrine and neural pathways, leading to measurable changes in barrier integrity, immune signaling, and inflammatory balance [7,11,12]. This model illustrates the mechanistic basis by which oral cellular homeostasis may act as a sensitive biological interface for stress-related dysregulation.

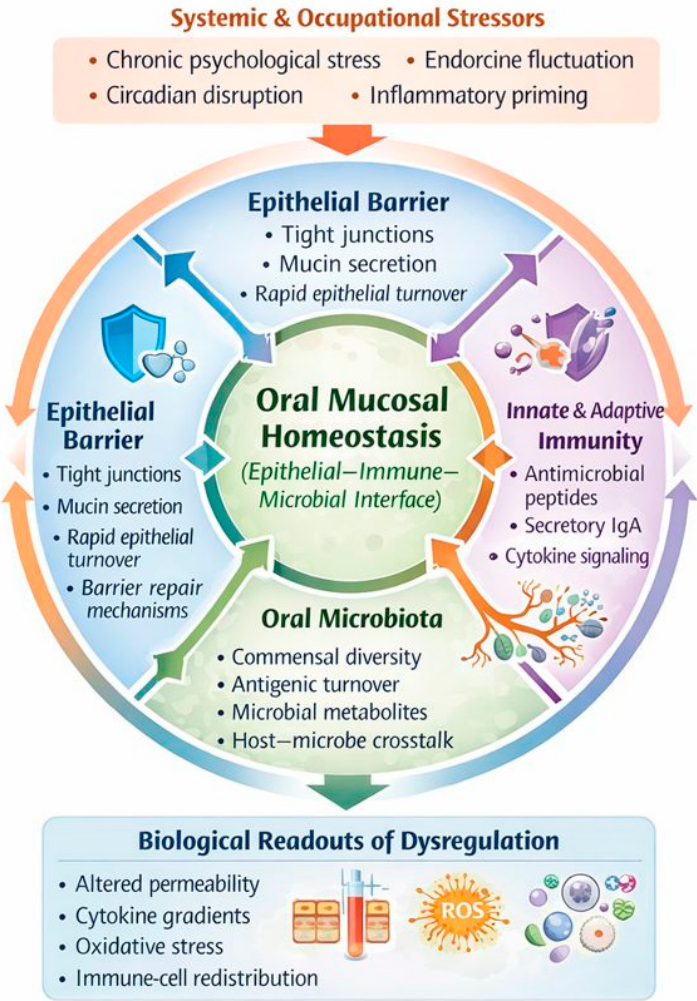


Figure 3. Integrated model of oral mucosal immunity as a neuroimmune barrier system.

3.2. Oral Microbiome Composition and Oral-Systemic Biological Interactions

Across the contemporary literature, oral cellular homeostasis emerges as the product of tightly interconnected mucosal-microbial-immune interactions that operate dynamically across the lifespan and respond sensitively to systemic, hormonal, environmental, and psychosocial influences [28,33]. Conceptual immunology frameworks describe the mucosal surface as an adaptive immune ecosystem in which commensal microbiota actively shape immune maturation, tolerance, and inflammatory thresholds from early life to aging, rather than acting as passive colonizers [7,14]. This perspective is reinforced by multi-omics and longitudinal evidence linking oral health trajectories with cognitive aging and social determinants, suggesting that oral biological integrity reflects cumulative biological and environmental exposures over time [9,10]. Within this framework, periodontitis-related research provides a concrete disease model illustrating how microbial imbalance, immune dysregulation, and systemic signaling intersect [8]. Narrative and biomarker-focused reviews consistently demonstrate that alterations in the oral microbiome are mirrored by shifts in salivary inflammatory and metabolic markers, supporting saliva as a functional readout of ongoing host-microbe conflict [8,60]. Importantly, causal inference approaches strengthen this association: Mendelian randomization analyses suggest that gut microbiota composition may exert a direct causal influence on periodontitis risk, highlighting bidirectional oral-gut microbial crosstalk rather than isolated local pathology [40]. Parallel systematic analyses of endodontic and prosthodontic conditions further show that clinical interventions and anatomical changes, such as root canal infection or denture use, reshape microbial ecology toward more inflammation-prone

configurations, emphasizing the sensitivity of oral ecosystems to both biological and mechanical stressors [9,42,43].

Hormonal and immune signaling represent critical modulatory layers within this system. Clinical and experimental syntheses demonstrate that endocrine environments, particularly stress- and sex-hormone fluctuations, reshape oral microbial composition and immune tone, providing a mechanistic bridge between psychosocial stress, HPA-axis activity, and oral dysbiosis [11,17]. At the cellular level, epithelial cells are increasingly recognized as active immune sentinels that integrate microbial signals, cytokine gradients, and neuroendocrine inputs to regulate barrier integrity and inflammatory responses [34]. Transcriptomic and proteomic studies of saliva extend this view, showing that salivary RNA and protein profiles capture both local immune activity and systemic physiological states, effectively positioning saliva as a multi-layered biosensor rather than a passive secretion [18,20]. The functional relevance of these interactions becomes particularly evident in contexts of immune challenge and environmental stress. Studies of viral infection, vaccination, and inflammatory disease demonstrate that oral mucosal immunity plays a decisive role in shaping systemic immune responses, viral susceptibility, and inflammatory resolution, with saliva-mediated mechanisms contributing to immune defense and pathogen control [46–48]. Microbiome-derived metabolites, including indole derivatives and short-chain fatty acids, further modulate immune signaling and inflammatory balance, reinforcing the concept that microbial metabolism constitutes a key regulatory layer of host immunity [50].

Finally, emerging interventional and environmental studies highlight the plasticity of oral biological systems. Nutritional interventions, micronutrient supplementation, and enriched environments have been shown to modulate salivary cortisol, immune markers, microbiome composition, and even emotional regulation, illustrating that oral cellular homeostasis responds measurably to modifiable behavioral and environmental inputs [45,51,54]. In sum, these findings converge on a unified model in which oral biology reflects and potentially mediates, the biological imprint of systemic stress, immune challenge, and lifestyle factors, supporting its relevance as both a mechanistic hub and a translational target in occupational and preventive health research.

3.3. Salivary Biomarkers of Endocrine, Immune, and Metabolic Activity

The evidence base on saliva converges on a central premise: saliva is a biologically active, information-dense matrix that integrates endocrine, immune, microbial, and metabolic signals at the oral mucosal interface and therefore functions as both a readout and a regulator of mucosal homeostasis [13,39]. Narrative syntheses emphasize that salivary composition captures local oral processes while remaining sensitive to systemic physiological states, which makes saliva particularly suitable for repeated, low-burden monitoring in clinical and occupational contexts where dynamic fluctuations are expected [13,16–18]. Mechanistically, saliva contains a layered repertoire of immune mediators (e.g., sIgA, cytokines, antimicrobial peptides), endocrine markers (e.g., cortisol), and molecular cargo (metabolites, proteins, nucleic acids, and extracellular vesicles), enabling multi-scale profiling of stress biology, immune tone, and disease susceptibility [14,17,18,40]. Importantly, methodological work suggests that salivary biomarker interpretability depends on sampling conditions, diurnal timing, assay characteristics, and contextual exposures, which are critical when translating saliva-based measures into occupational health monitoring [16,30].

From an immunological standpoint, saliva functions as a frontline effector of mucosal immunity: natural and inducible responses in the oral cavity rely on sIgA, cytokine signaling, and innate antimicrobial factors that collectively regulate microbial colonization and support epithelial integrity under continuous antigenic pressure [14,84]. Proteomic evidence further indicates that salivary proteins participate directly in host-microbe regulation by modulating adhesion, biofilm dynamics, and pathogen neutralization, linking molecular composition to ecological stability [18,49]. This ecological role is reinforced by microbiology-focused reviews showing that saliva is not merely a passive medium but actively stabilizes the oral microbiome through nutrient provisioning and selective antimicrobial pressure, thereby shaping community resilience and inflammatory thresholds

[39,49,50]. In parallel, salivary metabolomics expands the diagnostic scope beyond single analytes toward systems-level signatures: metabolomic patterns reflect inflammatory and metabolic states bridging oral and systemic disease processes, while also offering potential for risk stratification and monitoring [15,52–54]. Transcriptomic approaches similarly position the salivary transcriptome as an accessible window into local and systemic gene expression programs relevant to immune activation and stress responses [55].

Emerging salivary “next-generation” biomarkers strengthen this integrative model. Reviews of salivary exosomes highlight their role as diagnostic and signaling mediators in oral disease, expanding saliva’s utility from biochemical readouts to intercellular communication platforms [70]. At the endocrine level, saliva-based hormone diagnostics consolidate the rationale for cortisol monitoring in stress-related contexts, supporting the linkage between HPA-axis dynamics and mucosal immune modulation [17,30]. Collectively, these lines of evidence support the interpretation that salivary immune-endocrine-omic profiles can capture early biological dysregulation and recovery dynamics, potentially complementing subjective measures and enabling translational monitoring frameworks in high-demand healthcare environments [13,16,39]. Further, endocrine signaling adds a second regulatory layer. Diagnostic reviews centered on hormone detection highlight saliva as a matrix for measuring free (biologically active) fractions of steroid hormones, particularly cortisol, with good correlation to serum levels and strong practical advantages for field and occupational studies [17]. Landmark work in occupational and environmental medicine established salivary cortisol as a core indicator of HPA-axis activation and stress-related physiological burden, supporting its use in shift-work, healthcare, and high-demand professions [28]. More recent methodological syntheses refined this picture, emphasizing that diurnal patterns, the cortisol awakening response, sampling timing, and assay characteristics critically influence interpretability, and must therefore be carefully standardized in research linking occupational conditions to salivary readouts [30,31]. Together, these findings position salivary cortisol and related hormones as central tools for quantifying the endocrine dimension of occupational stress biology.

Metabolomic and transcriptomic studies extend the scope of salivary biomarkers beyond individual molecules toward system-level signatures. Metabolomics work demonstrates that salivary metabolic profiles track systemic metabolic states, inflammation, and disease risk, offering a multiplex, hypothesis-generating view of physiological perturbations [15]. Parallel transcriptomic approaches show that salivary RNA expression patterns can mirror local and systemic gene regulation, including stress- and immune-related pathways, suggesting that saliva can function as a minimally invasive “liquid biopsy” of both mucosal and systemic responses [46,48,55–59]. These high-dimensional approaches are particularly relevant to occupational settings, where chronic low-grade stress may produce subtle, multi-parameter shifts rather than large changes in any single biomarker [18,58,62].

Importantly, several reviews and empirical studies converge on the idea that the diagnostic utility of saliva depends not only on what is measured, but on how, when, and in whom it is measured [59,60]. Narrative syntheses mention the impact of collection methods, circadian timing, flow rate, stimulation, storage, and analytical platform on biomarker reliability, stressing the need for strong protocol design in both research and applied monitoring [13,63–66]. Trials that manipulate environmental or behavioral conditions illustrate this point concretely: interventions such as tele-yoga, lifestyle modification for night-shift workers, or enriched environments have demonstrated measurable changes in salivary cortisol, inflammatory markers, and microbiota-related parameters, linking modifiable workplace or lifestyle factors to salivary biomarker trajectories [26,27,32]. These findings reinforce saliva’s responsiveness to psychosocial and occupational exposures [78–83].

Taken together, the available evidence portrays saliva as a multi-layered biosensor that captures endocrine activation, immune balance, microbial interaction, and metabolic status within a single, non-invasive biological matrix [39]. Hormonal dynamics, mucosal immune activity, microbiome-derived signals, and high-dimensional molecular profiles converge to produce measurable salivary biomarker patterns. The interpretability of these patterns depends on sampling conditions, circadian

timing, and analytical methodology. This framework highlights saliva’s value for capturing the biological imprint of occupational stress and for monitoring physiological responses to workplace and lifestyle interventions (Figure 4). For the purposes of occupational wellbeing in healthcare professionals, this suggests that carefully selected and standardized salivary panels, combining cortisol dynamics, SIgA, cytokine profiles, and possibly metabolomic or transcriptomic signatures, could provide a sensitive window into the biological imprint of chronic work-related stress, as well as into the efficacy of resilience-building interventions [76–78].

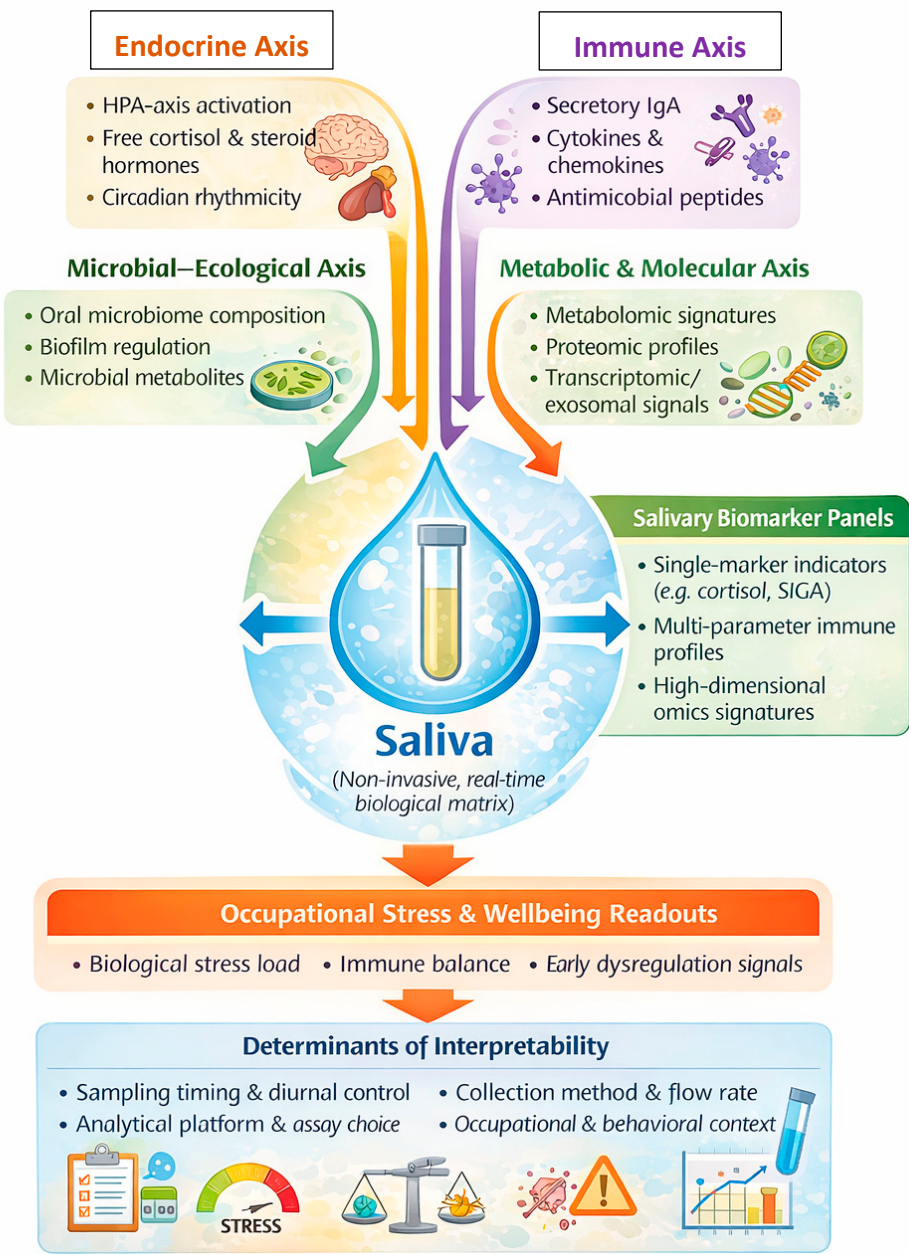


Figure 4. Saliva as an integrative biosensor of systemic and local physiological activity.

3.4. Occupational Wellbeing in Healthcare and Its Intersection with Oral Biology

The literature on occupational wellbeing consistently portrays healthcare environments as high-strain systems in which chronic psychological load, organizational pressures, and moral distress accumulate into measurable biological dysregulation [22,41,85,86]. Scoping and systematic reviews document high rates of burnout, depression, anxiety, and suicidal ideation among healthcare professionals, with nurses, physicians, and other frontline staff showing persistent vulnerability

across settings and countries [22,87–95]. Beyond psychological outcomes, these reviews emphasize that occupational distress is biologically embodied through alterations in circadian rhythms, hypothalamic-pituitary-adrenal (HPA) axis activity, immune function, and systemic inflammation, placing healthcare workers at long-term risk for cardiometabolic and neuropsychiatric morbidity [22].

Within this broader context, emerging work in dentistry and academic health settings provides more fine-grained insight into how stress is experienced and modulated. Cross-sectional data from Greek dentistry and nursing students indicate substantial burdens of stress, anxiety, and depression two years after the COVID-19 pandemic, but also highlight resilience, hope, and spiritual wellbeing as potential protective resources that may buffer the impact of academic responsibilities [85]. Similarly, comparative work on academic dental and nursing personnel shows that indices of quality of life and wellbeing are closely related to perceived quality of services and academic functioning, suggesting that staff wellbeing is not an isolated “soft” outcome but interwoven with educational performance and service delivery [86]. Together, such studies position oral health professionals and related academic staff as a meaningful subgroup within the broader healthcare workforce, facing both typical healthcare stressors and profession-specific demands (clinical precision, infection control, patient-facing communication, and teaching responsibilities). Interventional and integrative studies further delineate the modifiable components of occupational stress biology. An integrative review of non-pharmacological interventions for healthcare workers reports that structured stress-reduction strategies, such as mindfulness, relaxation practices, and behavioral programs, can yield improvements not only in self-reported wellbeing but also in physiological markers of stress and immune function [25]. Furthermore, a pilot randomized controlled trial on tele-yoga for healthcare workers on COVID-19 duty demonstrated reductions in burnout and improvements in mental health indices, accompanied by favorable shifts in immune markers, underlining the feasibility of remote, biologically meaningful interventions in high-demand clinical contexts [26]. Similarly, a randomized crossover lifestyle intervention in female night-shift healthcare workers reported benefits in metabolic and mental health parameters, with concomitant changes in salivary cortisol and related biomarkers, directly linking occupational scheduling, behavioral modification, and measurable stress physiology [27]. Such findings collectively indicate that occupational distress is not a fixed trait but a modifiable state, with interventions capable of recalibrating both psychological and biological endpoints.

Salivary and mucosal biomarkers sit at the intersection of these phenomena. Landmark work on salivary biomarkers in occupational and environmental medicine positioned saliva, particularly salivary cortisol, as a central non-invasive tool for assessing acute and chronic stress load in workplace settings [28]. More recent methodological syntheses refine this paradigm by articulating how sampling protocols, diurnal timing, and assay characteristics critically shape the interpretation of salivary cortisol and related markers in biobehavioral research [30]. The cortisol awakening response (CAR), in particular, has been identified as a sensitive, relatively stable index of HPA-axis activation under chronic stress, further reinforcing saliva’s utility for tracking occupational strain [31]. Randomized trials manipulating environmental richness in early life provide additional proof-of-principle that contextual factors can alter both salivary cortisol profiles and microbiota-related parameters, illustrating the plasticity of stress-related biomarkers in response to environmental modulation [32].

When viewed alongside the evidence on oral mucosal immunity (Section 3.1), microbiome dynamics (Section 3.2), and the complex biomarker landscape of saliva (Section 3.3), these occupational studies suggest a coherent, though still underdeveloped, picture. Healthcare workers, including dental professionals and academic staff-experience chronic psychosocial stress that is biologically inscribed through endocrine, immune, and metabolic pathways, many of which can be accessed via oral biomarkers [22,96,97]. At present, most occupational studies treat saliva primarily as a convenient stress readout [87,98,99]. However, the underlying science of oral epithelial barriers, immune responses, and microbial ecosystems indicates that the oral cavity itself may be an active

participant in stress adaptation and vulnerability [100]. This creates a conceptual bridge between occupational wellbeing and oral cellular homeostasis, supporting the hypothesis that dentists and other healthcare professionals could be monitored and perhaps protected through integrated panels of salivary, immune, and microbiome indicators that capture both psychological load and mucosal resilience over time.

3.5. Dietary, Nutritional, and Food-Related Modulators of Oral Cellular Homeostasis

Dietary and nutritional factors constitute critical upstream modulators of oral cellular homeostasis, exerting their effects through interconnected immune, microbial, and metabolic pathways that closely parallel mechanisms described in intestinal and systemic biology [88,89]. Systematic and narrative syntheses demonstrate that diet shapes oral epithelial integrity, mucosal immune tone, and microbial ecology by regulating substrate availability, inflammatory signaling cascades, and redox balance within the oral environment [88,89,92]. Diets enriched in fermentable fibers, polyphenols, and bioactive micronutrients favor the production of microbial-derived metabolites, most notably short-chain fatty acids (SCFAs), which exert anti-inflammatory, barrier-stabilizing, and immunoregulatory effects, whereas highly processed, sugar-rich dietary patterns are consistently associated with dysbiosis, oxidative stress, and pro-inflammatory signaling in oral tissues [89–93]. Emerging evidence further indicates that dietary inflammatory potential is associated with distinct oral and gut microbiota profiles and systemic inflammatory markers, supporting the concept that diet-driven inflammation influences host-microbiome interactions across mucosal sites [90]. Mechanistic analyses highlight SCFAs as key mediators linking dietary substrates to epithelial energy metabolism, immune modulation, and microbial stability in both the oral cavity and the gastrointestinal tract [91,93]. Within this framework, prebiotics, probiotics, postbiotics, and synbiotics represent complementary nutritional strategies capable of reshaping microbial composition, strengthening epithelial defense mechanisms, and calibrating innate and adaptive immune responses [88,89]. Although much of the mechanistic evidence originates from gastrointestinal models, converging data support the transferability of these pathways to the oral cavity, where constant microbial exposure and rapid epithelial turnover render oral tissues particularly responsive to dietary inputs [91–93]. Collectively, these findings position diet not merely as a background lifestyle factor but as an active biological regulator of oral cellular homeostasis, with implications for inflammation control, immune balance, and stress-related vulnerability in healthcare populations [94]. Building on these diet-microbiome-immune interactions, postbiotics have emerged as a promising class of bioactive compounds through which nutritional modulation may exert targeted effects on oral cellular homeostasis.

Postbiotics

Postbiotics appear as non-viable microbial cells or live microorganisms, structural components, or metabolites comprising diverse bioactive compounds such as short-chain fatty acids (SCFAs), exopolysaccharides (EPS), bacteriocins, antioxidant enzymes, surface layer proteins, and bacterial lysates. They use pattern recognition receptors and epigenetic modifications to enhance epithelial barrier function, regulate innate and adaptive immune responses, and modulate host gene expression [106]. Obesity, immune modulation, and mental health are the outcomes of their health-promoting potential, despite reduced viability and antibiotic resistance transfer issues [107,108].

A growing body of evidence indicates that postbiotics exert antioxidant, anti-inflammatory, immunomodulatory, and metabolic effects that are relevant to both systemic and oral health, supporting their role as biologically active mediators of diet-microbiome-host interactions [95,96,109,110]. Key postbiotic components, including short-chain fatty acids (SCFAs), polysaccharides, proteins, and bacterial cell wall fragments, contribute to microbial balance and protection against immune-mediated and inflammatory disorders by modulating epithelial function and immune signaling pathways [97,111,112]. In particular, *Lactobacillus*-derived postbiotics have demonstrated promising effects on glucose regulation, obesity-related metabolic dysregulation, and intestinal homeostasis, highlighting their relevance in the context of metabolic-immune crosstalk

[98,113,114]. Complementary to postbiotics, prebiotics-defined as non-digestible substrates such as inulin and oligofructose-selectively stimulate beneficial microbial taxa, enhance SCFA production, and promote metabolically favorable shifts in bile acid and fatty acid metabolism, thereby reducing the risk of metabolic and inflammatory disorders [99,115,116]. Further, synbiotics, which combine probiotics and prebiotics, further optimize microbial survival and functionality, yielding synergistic effects on microbial stability and host immune responses [99]. Conclusively, the integrated use of prebiotics, probiotics, postbiotics, and synbiotics represents a complementary and biologically coherent strategy for modulating the microbiota and its downstream immune and metabolic effects, with emerging relevance for oral cellular homeostasis and disease prevention, although further mechanistic and long-term clinical studies are warranted [117,118].

Moreover, exopolysaccharides (EPSs) are high-molecular weight carbohydrate polymers produced by a range of commensal and probiotic microorganisms and are increasingly recognized as bioactive mediators of host-microbe interactions across mucosal surfaces [119]. Accumulating evidence demonstrates that EPSs exert a broad spectrum of health-promoting effects, including inhibition of pathogenic adhesion to epithelial surfaces, reinforcement of mucosal barrier integrity, and modulation of innate and adaptive immune responses [120–122]. Structurally, EPSs are classified into homopolysaccharides (HoPSs), composed of repeating units of a single monosaccharide, and heteropolysaccharides (HePSs), consisting of complex repeating units of different monosaccharides; this structural diversity is closely linked to functional specificity in epithelial interaction and immune modulation [123,124]. Recent mechanistic studies indicate that EPSs can interact directly with epithelial cells to enhance tight-junction protein expression and mucin production, thereby strengthening barrier function and contributing to mucosal homeostasis [122,125]. In parallel, EPSs have been shown to modulate immune signaling pathways by attenuating pro-inflammatory cytokine production and promoting regulatory immune responses, including effects on dendritic cell activation and T-cell polarization [126,127]. Beyond immunoregulation, anticancer properties of EPSs have been increasingly reported, including inhibition of tumor cell proliferation, induction of apoptosis, and modulation of oxidative stress pathways, with EPSs derived from *Lactobacillus* species receiving particular attention due to their consistent bioactivity and favorable safety profile [128–130]. Collectively, contemporary evidence supports EPSs as multifunctional microbial metabolites that contribute to mucosal barrier stability, immune balance, and epithelial resilience, with emerging relevance for oral cellular homeostasis within diet-microbiome-host interaction frameworks.

Further, SCFAs are absorbed and transported through the bloodstream to peripheral tissues, exerting systemic effects [131] playing critical roles in gut homeostasis, playing key roles as anti-inflammatory and immunomodulatory mediators and acting as a bridge between microbiota and host immunity [132]. In intestinal epithelial cells (IECs), acetyl-CoA is the metabolic product of SCFAs via the TCA cycle, offering energy production and immune cell function [133,134].

Additionally, bacteriocins are secreted by Gram-positive and Gram-negative bacteria, with either narrow or broad inhibitory spectra, and are ribosomally synthesized, proteinaceous antimicrobial peptides [135]. They protect themselves via immunity proteins and efflux pumps by producing strains [136]. Pore formation in bacterial membranes is their main action, leading to lysis, while reducing resistance development [137]. Primary bacteriocin producers are LABS-including *Pediococcus*, *Leuconostoc*, *Lactococcus*, *Enterococcus*, *Streptococcus*, *Lactobacillus*, and *Bifidobacterium* [138].

Mechanisms of action of postbiotics and probiotics

Postbiotics and probiotics through multiple mechanisms engaging innate and adaptive immune responses exert notable immunomodulatory effects. Most specifically probiotics interact with TLRs (Toll-like receptors), which are pivotal components of the host's initial defense against pathogenic microorganisms. Modulation of innate immune pathways is carried out by probiotics through TLR signaling. For instance, recognition of probiotic ligands and often formation of heterodimers such as

TLR2/TLR6 is done by TLR2, inducing tolerogenic IL-10 secretion and mediation of anti-inflammatory effects [139,140].

Key intracellular signaling cascades, including nuclear factor-kappa B (NF-κB) and mitogen-activated protein kinases (MAPKs) are affected by probiotics thereby facilitating communication with the host immune system. Hence, this affects the production of anti-inflammatory cytokines and chemokines, ultimately contributing to maintaining immune homeostasis [141]. Similarly, postbiotics, particularly butyrate and propionate, exhibit substantial immunomodulatory effects. Butyrate enhances the diversity of regulatory T cells (Tregs), while propionate facilitates the expansion of peripheral Tregs within the intestinal mucosa. Colonic health is promoted by reducing luminal pH, stimulating epithelial cell proliferation, and enhancing colonic blood flow [119,142]. Their role is key in gluconeogenesis, serving as an essential energy source for colonocytes and intestinal epithelial cells. Finally, their contribution to cellular homeostasis by promotion of apoptosis in colon cancer cells, thereby supporting metabolic function and colorectal health is noteworthy [143].

In Figure 5 we show the interconnected pathways through which postbiotics and probiotics influence mucosal and systemic physiology. Postbiotics, including short-chain fatty acids (SCFAs), exopolysaccharides (EPSs), bacteriocins, and bacterial structural components, exert their effects predominantly via metabolic signaling and epigenetic regulation, whereas probiotics act through live microbial interactions with host epithelial and immune cells, primarily via pattern-recognition receptors such as Toll-like receptors (TLRs). These upstream signals converge on key intracellular pathways, including NF-κB and MAPK signaling, leading to modulation of innate and adaptive immune responses, promotion of regulatory T-cell (Treg) activity, and attenuation of pro-inflammatory cytokine production. Downstream outcomes include reinforcement of epithelial barrier integrity, stabilization of microbial ecosystems, regulation of metabolic function, and protection against chronic inflammation. Collectively, the figure conceptualizes how diet-microbiome-host interactions mediated by postbiotics and probiotics support oral cellular homeostasis and broader systemic resilience.

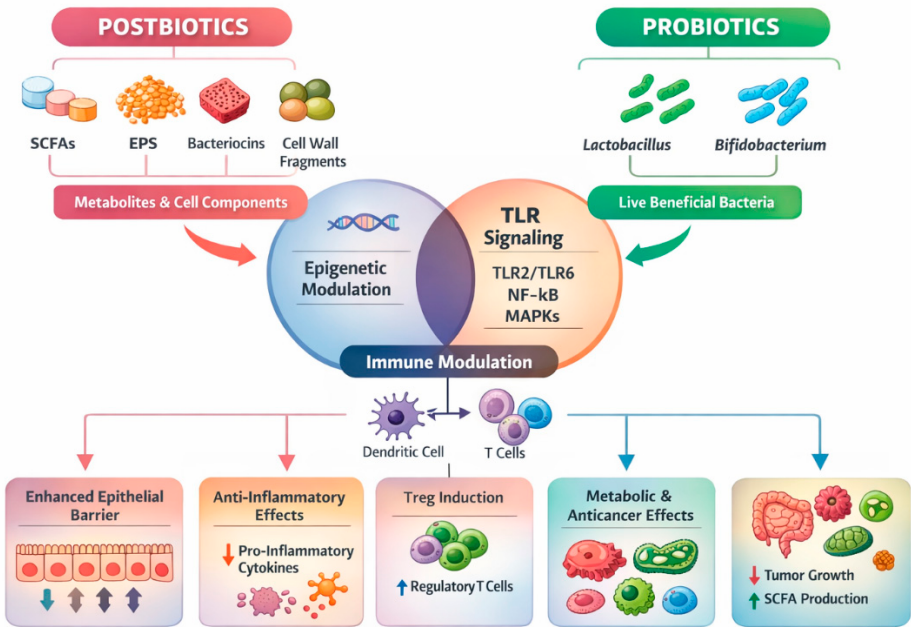


Figure 5. Mechanisms of action of postbiotics and probiotics on mucosal and systemic homeostasis.

3.6. Interventions Targeting Stress Biology, Oral Homeostasis, and Occupational Wellbeing

Interventions relevant to occupational wellbeing increasingly operate across two complementary levels: (i) modulating stress physiology and mental health outcomes through scalable behavioral and digital strategies, and (ii) strengthening oral-systemic health through nutrition-focused approaches and structured coaching models that translate biological mechanisms into daily practice [99,100]. Digital lifestyle interventions show measurable benefits across stress, anxiety, depression, and broader wellbeing outcomes, supporting their role as scalable tools for prevention and early intervention in occupational contexts [100,101]. In parallel, non-pharmacological interventions used in healthcare environments, including mind-body and behavioral approaches, demonstrate clinically meaningful improvements in stress-related outcomes and support a multi-component approach to workforce resilience [25,105]. Evidence from an interventional trial in healthcare workers further suggests that structured mind-body programs such as tele-yoga can improve burnout-related outcomes while also influencing immune markers, reinforcing the biological plausibility of integrated stress-immune interventions in high-demand clinical settings [104]. Lifestyle programs targeting vulnerable occupational groups such as night-shift healthcare workers also indicate improvements in metabolic and mental health outcomes, aligning with the broader view that circadian and behavioral optimization can reduce allostatic load and improve wellbeing [144].

From the oral-systemic health perspective, nutrition-based interventions and diet-oriented health models provide a coherent framework for linking dietary modulation, microbiome balance, and inflammatory control with oral health outcomes, which may indirectly support occupational resilience by reducing chronic inflammatory burden [95,96,99,145]. Reviews focusing on functional foods, gut microbiome modulation, and metabolic health further emphasize that dietary strategies can shape host-microbe interactions relevant to inflammation and systemic regulation, particularly in conditions characterized by metabolic vulnerability such as diabetes [98]. Within dentistry, probiotic- and prebiotic-oriented approaches have been positioned not only as biological adjuncts but also as behaviorally mediated interventions that benefit from structured patient coaching, supporting sustained adherence and prevention-oriented oral health behaviors [97]. Coaching models for independent older adults further illustrate how effective intervention depends on implementation design, goal setting, tailored education, motivational support, and continuous feedback, rather than informational exposure alone, an approach that is transferable to occupational wellbeing programs in healthcare [95]. Finally, interventions targeting occupational wellbeing must also account for contextual and psychosocial determinants that shape stress trajectories, including caregiver burden and moral injury, which are increasingly recognized as high-impact drivers of distress in healthcare and allied settings [102,103]. Taken together, the evidence supports a layered intervention paradigm in which digital and behavioral stress-reduction tools, structured mind-body programs, circadian and lifestyle optimization, and nutrition- and coaching-based oral health strategies act synergistically to improve psychological outcomes while potentially influencing biological pathways linked to immune balance and systemic inflammation [25,99,100].

3.7. Conceptual Funnel of Occupational Stress, Oral Cellular Homeostasis, and Professional Wellbeing

Figure 6 illustrates the sequential pathway through which occupational exposures shape systemic stress physiology, disrupt oral cellular homeostasis (salivary, epithelial-immune, and microbiome mechanisms), and ultimately influence professional wellbeing outcomes. The current inverted-pyramid form highlights that intervention points appear last, positioned at the narrowest section of the model, reflecting the empirical reality that most healthcare systems respond to stress after dysregulation has already occurred—that is, only when burnout, fatigue, or biological strain becomes clinically visible [22,24]. However, the model is intentionally designed so that the pyramid can be conceptually reversed. When flipped, the intervention layer moves to the top, becoming the entry point rather than the final response. In this orientation, proactive measures, such as stress-modulating strategies, circadian alignment, microbiome-supportive behaviors, and saliva-based

biomarker monitoring, act upstream to stabilize the systemic stress response, protect oral mucosal and microbial homeostasis, and prevent downstream impairment of wellbeing [25,27,28,31]. At the end, viewing the diagram in this reversible way emphasizes a key argument of this review: oral cellular mechanisms are not merely passive endpoints of occupational strain but early, sensitive interfaces where dysregulation can be detected and potentially corrected, before professional wellbeing deteriorates. Transforming the model from a reactive to a preventive scaffold aligns with broader calls for integrative occupational health strategies that combine biological, psychological, and organizational approaches to strengthen the resilience of healthcare professionals, including dentists [85,86].

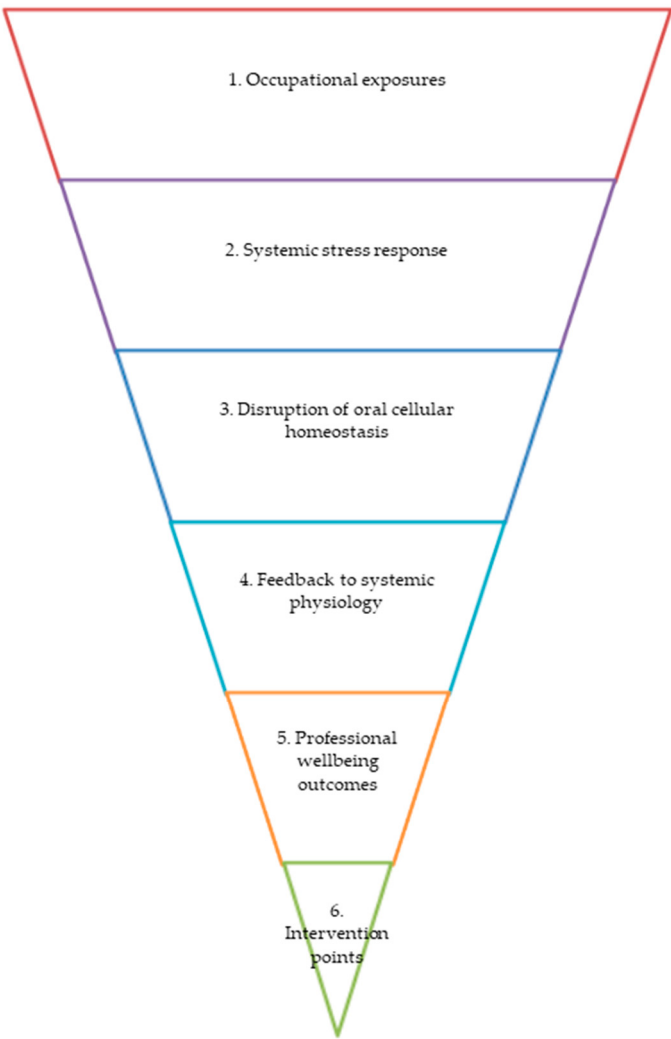


Figure 6. Conceptual pathway linking occupational stress to oral cellular homeostasis and professional wellbeing.

It represents: 1. Occupational exposures (Clinical workload, psychosocial stressors, time pressure, circadian disruption, infection control demands, and interpersonal demands in healthcare settings.), 2. Systemic stress response (Activation of the HPA axis with altered cortisol rhythms (including changes in the cortisol awakening response), sympathetic overactivation, low-grade systemic inflammation, and disturbed circadian alignment.), 3. Disruption of oral cellular homeostasis (a. Salivary System: Changes in salivary cortisol, secretory IgA, cytokines (IL-6, TNF- α), antimicrobial peptides, and metabolic/proteomic signatures; reduced buffering and antimicrobial capacity. b. Epithelial-Immune Barrier: Altered tight junction integrity, impaired mucosal repair, increased inflammatory signaling, and compromised innate immune responses. c. Oral Microbiome:

Reduced microbial diversity, dysbiosis, shifts toward pro-inflammatory or stress-responsive taxa, altered metabolite production (e.g., SCFAs, indole derivatives), and weakened ecological stability.), 4. Feedback to systemic physiology (Amplification of systemic inflammation, altered neuroimmune communication, impaired stress recovery, microbiome-gut-brain axis signaling, and endocrine-mucosal crosstalk contributing to chronic allostatic load.), 5. Professional wellbeing outcomes (Increased fatigue, burnout, emotional exhaustion, anxiety, depressive symptoms, cognitive overload, reduced resilience, reduced occupational engagement, and increased susceptibility to oral and systemic disease.)

3.8. Risk of Bias

Studies included in this review exhibit several methodological limitations that require cautious interpretation without diminishing the value of their findings. A substantial proportion of the microbiome, mucosal immunity, and occupational wellbeing literature remains cross-sectional, restricting the ability to determine directionality or causal pathways among stress physiology, oral cellular mechanisms, and wellbeing outcomes [7,11]. Microbiome studies vary in sequencing methods, sampling protocols, and analytical pipelines, which can introduce between-study heterogeneity and limit direct comparability. Also, research involving salivary biomarkers often shows inconsistencies in sampling timing and diurnal control, reflecting known challenges in biobehavioral research and contributing to potential measurement variability [28,30]. Further, occupational wellbeing studies, including those involving dental students, academic personnel, and healthcare workers, frequently rely on validated self-report instruments, which, while widely accepted, may introduce elements of recall bias and subjective variability [24,85,86]. Moreover, interventional studies report more structured designs, yet sample sizes and follow-up durations remain modest, limiting conclusions about long-term physiological effects [26,27]. Despite these limitations, the overall convergence of evidence across diverse methodologies supports the broader conceptual links explored in this review and highlights the need for stronger longitudinal and mechanistic research rather than diminishing the current findings.

4. Discussion

This review integrates evidence across oral biology, occupational health, and behavioral science to support the concept that oral cellular homeostasis operates as a sensitive biological interface of occupational stress and resilience in healthcare professionals, with dentistry representing a high-exposure occupational model [25,85,86,145,146]. Across these domains, convergent findings indicate that chronic occupational strain is biologically embodied through hypothalamic-pituitary-adrenal (HPA) axis activation, mucosal immune modulation, epithelial barrier alterations, and microbiome shifts, all of which are detectable in saliva and related oral biofluids [17,66,147,148]. Importantly, these biological signals exhibit dynamic temporal variability and may precede, or remain undetected by, self-report instruments, positioning saliva-based biomarkers as complementary, real-time indicators of physiological load in occupational settings [16,100,101].

As it was revealed, biological plausibility is supported by a coherent mechanistic cascade in which occupational stress activates the HPA axis, elevating cortisol and sympathetic tone, with downstream suppression of mucosal immunity and disruption of epithelial integrity [30,34]. Reduced secretory IgA levels, altered cytokine profiles, and impaired tight-junction regulation compromise epithelial turnover and barrier function, thereby reshaping microbial ecology within the oral cavity [14,17,49]. Resultant dysbiosis and low-grade inflammation can, in turn, feed back into systemic stress pathways via neuroimmune and metabolic signaling, reinforcing a bidirectional amplification loop between stress biology and mucosal dysfunction [53,66,75,81]. Within this framework, diet-derived modulators, particularly postbiotics such as short-chain fatty acids, exopolysaccharides, and bacteriocins, emerge as biologically credible intervention levers capable of stabilizing epithelial barriers, recalibrating immune signaling, and supporting microbial homeostasis [97–99]. Experimental and clinical nutrition studies further indicate that such microbial metabolites

intersect immune, endocrine, and metabolic pathways, positioning nutritional strategies as modulators of both stress physiology and oral cellular resilience [45,61,74].

Additionally, integration with psychological and resilience-oriented models strengthens the interpretation of these biological findings. From a resilience perspective, stable salivary IgA patterns, preserved epithelial integrity, and balanced microbiome profiles are associated with adaptive stress recovery and reduced susceptibility to burnout and emotional dysregulation [26,100]. This supports a biopsychosocial model in which biological robustness at the mucosal interface contributes actively to emotional regulation, occupational sustainability, and mental well-being, rather than functioning solely as a passive marker of distress [101,103,144].

Key limitations of the current evidence base include the predominance of cross-sectional designs, modest sample sizes, heterogeneity in salivary sampling protocols and microbiome analytics, and a limited number of longitudinal or integrated multi-omics studies [15,16,55]. Critically, most intervention studies do not employ oral biomarkers as primary outcomes, constraining causal inference and delaying translational implementation in occupational health frameworks [25,27]. These limitations report on the need for standardized saliva collection protocols (including timing and diurnal control), harmonized analytical pipelines, and integrative study designs that combine biological, psychological, and occupational metrics to advance precision prevention strategies in high-stress healthcare professions [13,99,145].

Implications for future research and occupational health practice are both substantive and actionable. First, the deployment of salivary biomarker panels, including cortisol dynamics, secretory IgA, inflammatory cytokines, and targeted omics approaches, should be prioritized as early-warning indicators within occupational health surveillance frameworks, given their sensitivity to dynamic stress-related physiological changes [15,17,85,86,97,145–148]. Second, microbiome-informed monitoring should be incorporated for high-risk professional groups, as accumulating evidence links occupational strain with shifts in oral microbial ecology and mucosal immune balance [39,49,149–153].

Third, future interventions should adopt hybrid, systems-level designs that integrate stress-management strategies (e.g., mindfulness-based approaches, circadian alignment), targeted dietary and postbiotic interventions, and organizational or workload redesign, with outcomes evaluated against both biological (salivary, microbiome, immune) and psychological endpoints [26,99,145]. Finally, these findings support a shift towards a preventive, upstream occupational health model, moving beyond reactive burnout management to proactive stabilization of stress biology, mucosal immunity, and oral cellular homeostasis [22,27,96–99]. Collectively, this body of evidence repositions the oral cavity from a passive biomarker reservoir to an active biological interface within occupational stress physiology, offering feasible pathways for early detection, prevention, and resilience-building in healthcare workforces [13,14].

5. Conclusions

This narrative review highlights oral cellular homeostasis as a dynamic and sensitive interface linking occupational stress biology with immune, microbial, and epithelial regulation. Evidence suggests that salivary biomarkers and oral microbiome patterns capture real-time physiological responses to occupational strain, offering advantages over self-report measures alone. Dietary and behavioral modulators, particularly postbiotics and stress-management interventions, emerge as promising, biologically plausible strategies to support mucosal resilience and wellbeing. Integrating oral biomarker monitoring with preventive occupational health frameworks may enable earlier detection of risk, targeted interventions, and more sustainable resilience strategies for healthcare professionals.

Author Contributions: Conceptualization, M.A. and T.V.; methodology, M.A. and T.V.; software, M.A. and T.V.; validation, M.A. and T.V.; formal analysis, M.A. and T.V.; investigation, M.A. and T.V.; resources, M.A. and T.V.; data curation, M.A. and T.V.; writing-original draft preparation, M.A. and T.V.; writing—review and editing,

M.A. and T.V.; visualization, M.A. and T.V.; supervision, M.A. and T.V.; project administration, M.A. and T.V.; funding acquisition, M.A. and T.V.; All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created.

Conflicts of Interest: The authors declare no conflicts of interest.

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