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Epidemiology, Pathogenicity and Treatment of Oral Parasitic Infections: A Review

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ABSTRACT

Oral parasitic diseases, such as those caused by *Entamoeba gingivalis* and *Trichomonas* tenax species are emergent in the sense that they are hitherto neglected oral pathologies occurring in some periodontal patients not responding to conventional therapy. Molecular diagnostics support higher prevalence rates then assumed before, hence increased interest in their epidemiology and clinical relevance. There is evidence that these protozoa are potentially involved in tissue damage, biofilm disruption and host immune response modification that increase disease severity. They are commonly – but not exclusively – found in combination with low oral hygiene standards, deep pockets and various systemic risk factors. Despite mechanical periodontal therapy being the mainstay of treatment adjunctive antiseptic and antiparasitic treatments have potential to improve oral parasitic burden. Prevention is based on strict oral hygiene, risk-factor management and periodic clinical follow-up. Although evidence is increasing, the pathogenic significance and therapeutic implications of these parasites need more research. This review strives to synthesize existing information about epidemiology, pathogenicity and treatment strategies in order to recommend more optimal diagnostic and therapeutic approaches.

INTRODUCTION

The predisposing factors to periodontal disease and other oral diseases continue to make it a significant global public-health issue due to its high prevalence, chronic nature, effect on quality of life, and associations with systemic disease. Etiologically, bacterial dysbiosis and host inflammatory reaction have been traditionally targeted (Bonner et al., 2018). However, growing molecular and epidemiological evidence has unearthed a stable yet least studied parasitic fraction of the oral microbiome—primarily protozoa such as *Entamoeba gingival* and *Trichomonas tenax*—that are often enriched in periodontal disease sites. New diagnostic tools (i.e. polymerase chain reaction, metagenomics) have significantly increased sensitivity and exhibited higher parasitic incidence rates in gingivitis and periodontitis, suggesting that these parasites are pathogenic not a part of the normal flora (Yaseen et al., 2021).

Entamoeba gingivalis and *T. tenax* are characterized by unique biologic characteristics as well as a distinct ecological niche in the oral cavity, some of which relate to pathogenicity. *Entamoeba gingivalis* —a pathogen closely related to E. histolytica—is ubiquitously present in periodontal disease (PD) pockets and can phagocytize host cells and bacteria; these behaviors are indicative of a potential contributory role in tissue damage and dysbiosis (Bonner et al., 2018). *Trichomonas tenax* (flagellate) produces proteolytic enzymes and adheres to epithelial and biofilm constituents; a systematic review meta-analysis found that T. tenax is more often detected at sites of disease than health and in vitro work points towards credible virulence mechanisms (Bisson et al., 2019). Statistically significant correlations between parasite presence and periodontal disease severity, socioeconomic indicators or

co-morbidities such as diabetes have been reported in various epidemiological studies in different geographical settings, yet causality has not been established (Yaseen et al., 2021; Bisson et al., 2019).

Recent methodological developments have altered the picture of oral parasuicida epzdemiology. By molecular typing, PCR-based prevalence studies (multilocus and subtyping) have been established, E. gingivalis lineages distinguished and co-infections with T.tenax are documented, whereas metagenomic surveys start to place the protozoa into the context of disturbed networks dominating periodontitis (Örsten et al., 2023). These methods also allow reanalysis of previous microscope-based data based estimates and demonstrate that culture-independent methods reveal dramatically higher frequency of carriage, particularly in patients with low oral hygienic levels, deep periodontal pockets or systemic risk factors. These combined molecular data not only reinforce the epidemiological association, but also raise mechanistic questions—whether parasite presence promotes dysbiosis and inflammation or whether an inflamed niche simply allows parasite expansion? (Yaseen et al., 2021). From this recent finding some therapeutic and clinical implications might come . Oral protozoa may enhance the destruction of periodontal tissues or maintain inflammation; combative antiparasitic interventions (rinses, systemic), enhanced sanitation of periodontal pockets, or inclusion of parasite testing in diagnostic panels may complement standard periodontal care (Bonner et al., 2018; Matthew et al., 2023).

Yet current intervention studies are few and far between, which systematic reviews identify as requiring randomised longitudinal trials to ascertain the extent that antiparasitic elimination changes clinical outcomes (Bisson et al., 2019). A limited number of studies also probed zoonotic reservoirs and the possibility of extra-oral spread (e.g., trichomonad identification in respiratory samples), indicative for wider One Health implications (Matthew et al., 2023).

In light of these findings, there is a need for an updated and focused review on the epidemiology, pathogenic mechanisms, diagnosis implications and treatment modalities for oral parasitic conditions. This review integrates molecular prevalence, mechanistic in vitro and ex vivo studies, systematic literature appraisals, and new therapeutic concepts published from 2015 until now to delineate gaps (in particular the lack of longitudinal and interventional clinical trials) and research priorities needed for a better understanding of causality and translation into diagnostics and treatments. By examination of relatively recent high-quality evidence, the focus of this review is to shift oral protozoa from the fringe to a contended element in periodontal knowledge and management.

EPIDEMIOLOGY OF THE ORAL PARASITIC INFECTIONS

The epidemiology of the oral protozoan infections become increasingly important, as molecular and meta-analytic data demonstrate that E. gingivalis and T. tenax may be much more abundant in the subgingival and gingival location than previously thought. For E. gingivalis, a meta-analysis of 52 studies (7,596 individuals) reported an overall pooled prevalence of 37% (95% CI 29-46%) among populations globally. Prevalence differed by country— from approximately 3% in Portugal to 87% in Jordan. Molecular diagnosis provided detection rates (53%) greater than those obtained by direct microcopy (36%). More importantly, colonization was associated with a 4.34-times increased risk of presentation of an oral disease state (i.e., periodontitis) by the meta-analysis (Badri et al., 2021).

Concurrent evidence of T. tenax further suggests a significant global burden. Based on a systematic review and meta-analysis of 65 studies, the pooled prevalence was approximately 17% (95% CI 14-22%) and with an observed level between ~3% in Kenya and 56% in Chile. In summary, these data indicate that oral protozoa are not uncommon nor confined to low-income conditions but may constitute an under-recognized component of the oral microbiome (Stefański et al., 2021).

Cross-sectional PCR-dependent studies offer a more nuanced view on the relationship between parasitic colonization and oral health status and risk factors. For example, in Jordan 237 people were sampled with E. gingivalis detected in 88.9% of them for periodontitis patients, 84.9% for gingivites and only 47.9% in healthy cases; whilst T. tenax at these sites was found to be present at some time at levels of: (25.6%, 5,7% and3,2%,) respectively (Yaseen et al., 2021). E. gingivalis colonization was associated with an OR of 6.6 for periodontal disease, compared with non-disease controls; the presence of diabetes mellitus, a low income and no access to dental care were other correlates. In a Nigerian dental-clinic study population of 180 patients, overall protozoan prevalence (either species) was 40%; E. gingivalis constituted 15.6% and T. tenax ~13.3%. It was more prevalent in poor oral hygiene and old age (Ani et al., 2020).

Recent investigations in vulnerable populations also demonstrate the variability of risk. On the other hand, PLB shedding was detected in about 18.1 and 19.5% of nondisabled controls in western children with mental disability from west of Iran to 42.8 and 40.5% E. gingivalis and T tenax (microscopy and PCR respectively) vs.~18.1–19.5%. Independent risk factors were urban dwelling, lower level of parental education and tooth brushing less than optimal (Aciöz et al., 2025).

A variety of geographic, socioeconomic or behavioural and methodological factors accounts for the variation in prevalence. Effect of diagnostic method: PCR detection (molecular) generally gives a higher prevalence when compared to microscopy alone, indicating under-estimation in older studies (Badri et al., 2021).

Socioeconomic status including low income, poor access to dental treatment and oral hygiene, smoking and comorbidities (e.g., diabetes) are all seen as predominant correlates in studies (Yaseen et al., 2021). Moreover, zoonotic and companion-animal reservoirs are rising issues: a 2022 Polish study detected T. tenax in domestic animals (dogs, cats, horse) highlighting interspecies transmission and the widening of the parasite's ecological niche (Dybicz et al., 2018).

Although global burden data for oral parasitic infections are lacking as compared with other oral diseases, this wider epidemiological context is pertinent. For instance, the burden of periodontal disease was high worldwide (over 1 billion global prevalent cases in 2021 and significant regional variability). Due to the close relationship with periodontal disease, the three oral protozoans probably have some epidemiological importance. In fact, meta-analyses indicate colonization might represent disease severity rather than carrier status (Badri et al., 2021; Yaseen et al., 2021).

However, important gaps remain. The majority of prevalence studies are cross-sectional and do not allow conclusions to be drawn about causation or progression. Prospective longitudinal cohort studies of protozoan colonization over time are limited. Furthermore, geographical coverage is also unbalanced; studies often come from the Middle East, some African countries or developing countries and are less common in high-income areas. Standardised diagnostic terminology and full mouth sampling protocols are frequently absent, hindering comparison and meta-analysis (Moosazadeh et al., 2025). Furthermore, there are scarce data on sub-clinical carriage in healthy individuals and children, as well in immunodeficient populations. Finally, the mechanistic relevance of co-infection with both E. gingivalis and T. tenax, as well as interaction with bacterial biofilms and systemic diseases are not sufficiently explored under the light of etiology. In a recent scoping review it was highlighted T. tenax's zoonotic potential as well as its existence in animals, thus “integrating One Health" to epidemiology (Matthew et al., 2023).

PATHOGENICITY OF THE ORAL PARASITIC INFECTIONS

It has becoming evident that a group of oral protozoan parasites, including E. gingivalis and T. tenax, have pathogenicity which is thought to disprove the previous belief in their nature as nonpathogens or commensals of the human mouth . E. gingivalis was previously recognized as non-pathogenic and only fed on bacteria and detritus in the gingival pocket (Aciöz et al., 2025). However, new studies in molecular and cell biology show that this amoeba can invade the gingival tissues, phagocyte live host cells by trogocytosis and induce a strong inflammatory gene response and may also participate in tissue destruction in periodontal disease. For instance, in epithelial cells infected with E. gingivalis transcriptome study found a 430-fold increased expression of TNF and a 359-fold increase of IL-8 and as well as enrichment of “chemokines and inflammatory molecules in myeloid cells” genes (Bao et al., 2021).

In particular, E. gingivalis has been demonstrated to invade the breached epithelial barriers, home to gingival connective tissues and phagocytize host epithelial cells and immune cells—phenomena resembling the pathogen Entamoeba histolytica (Rosenfeld et al., 2025). Clinically, the protozoan has been found in 77% of inflamed periodontal sites compared to 22% of health controls, and an in vitro exposure study with gingival fibroblasts resulted in up-regulation of MMP13 (extracellular matrix-degrading collagenase) by a factor of 11 providing evidence for tissue destruction. This mechanistic data indicate that the parasite not only survives within the dysbiotic, inflamed environment of periodontitis but could initiate pathological processes—breakdown of barrier function, host-cell lysis, pro-inflammatory cytokine production, and matrix destruction (Bao et al., 2021).

Regarding T. tenax, the flagellated protozoan which is commonly detected in periodontal pockets and dental calculus of individuals with inadequate oral hygiene or periodontal disease (Bracamonte-Wolf et al., 2019). A systematic review and meta-analysis reported a global pooled prevalence of about 17% (95% CI 14-22%) and found that the presence of T. tenax was associated with disease severity (Yaseen et al., 2021). In vitro mechanistic work published in 2023 showed that T. tenax causes cytotoxicity to gingival epithelial cells and disrupts tight-junction, adherens junction, and desmosomal proteins downregulates even at low multiplicity of infection with concomitant up regulation of IL-6 (Hong et al., 2023). These findings suggest that T. tenax is not a silent bystander, but might contribute to epithelial barrier breakdown and activation of host inflammatory responses, which could exacerbate loss of periodontal tissues.

The scenario becomes more complex from the zoonotic perspective as well: for example, a study in 2022 found T. tenax from domestic animals (dogs, cats and horse) in Poland with gingivitis and dental calculus which may indicate potential cross-species transmission and broader distribution of this species on the ecology niche. Whether such zoonotic transmission occurs, it remains even less well studied, but this raises the possibility that T. tenax might operate beyond human oral conditions and introduce new reservoirs of infection or reinfection (Ito et al., 2023).

It is suggested that of Trichomonas and Entamoeba correlate with bad clinical features (i.e., deeper pocketing, more bleeding/on islands) and their pathogenic effect seen biochemically provides biological plausibility. However, causality is still to be established and there are a number of caveats. For one, a lot of the work is still associative or in vitro; longitudinal studies showing that removing these protozoa improves outcomes do not exist. Second, diagnostic descriptions and sampling strategies of the studies are quite heterogeneous, hampering comparisons. Thirdly, host factors (immunity, systemic disease and oral hygiene), are expected to influence pathogenicity. For instance, while E. gingivalis was detected in HIV positive cases, the presence of the organism, among them, did have a direct relationship with the extent of immune deficiency (Coleman et al., 1998).

From a mechanistic perspective, the pathogenic pathways of these parasites seem to involve: cytotoxic (lysis or trogocytosis) of host cells, damage to epithelial barrier junctions, up-regulation of pro-inflammatory cytokines (IL-8, TNF and IL-6), activation of matrix metallomproteinases and tissue degrading pathways, and the redirection of microbial biofilm in favor of the parasite. Trogocytosis of live epithelial cells and ingestion of cell fragments by E. gingivalis was also observed via scanning electron

microscopy and transcriptome analysis (Bao et al., 2021). Barrier defects and cytokine induction were observed in epithelial lines, both of gingival and pulmonary origin, for T. tenax (Hong et al., 2023). Taken together, these strategies are indicative that these protozoan parasites may function as either faculatative or associate pathogens in periodontitis rather than inert individuals.

TREATMENT STRATEGIES

Therapeutic regimens for oral protozoa infestations are still an evolving topic in view of the fact that most clinical guidelines are based on bacterial dysbiosis and mechanical periodontal therapy than use of specific anti-protozoan medication. However, as more mechanistic and epidemiologic evidence emerges suggesting potential contributory roles for E. gingivalis and T. tenax in periodontal disease (Rachmawati et al., 2023), efforts to develop treatment modalities that target these parasites should be considered further. As such, the treatment of oral parasitic infections must be approached with a tripartite strategy: optimization of conventional periodontal therapy (mechanical debridement, oral hygiene), adjunctive antiparasitic and/or antiseptic therapies, and prophylactic/maintenance protocols to control recolonization and cross-infection (Matthew et al., 2023).

Firstly, mechanical periodontal therapy is a cornerstone of PD treatment and has been suggested to decrease parasitic loads. In a study of 46 patients with moderate-severe chronic periodontitis, nonsurgical periodontal treatment (scaling and root planning plus oral hygiene instruction) led to a significantly lower prevalence of E. gingivalis in saliva ($p = 0.007$) and plaque ($p = 0.027$), as well as T. tenax in saliva ($p = 0.030$) but not in plaque [(Saliva p=0.0069); (Plaque p=91 3)]. The logical deduction is that by diminishing the tissue niche amenable to protozoan colonization (pocket reduction, calculus/biofilm removal and resolution of inflammation)], one also reduces parasite load (Maybodi et al.,2016).

Second, adjunctive pharmacologic and antiseptic approaches against protozoa should be included in the discussion although the body of evidence is scant. Supporting veterinary data are available for T. tenax: in dogs and cats with dental plaque, the use of metronidazole eradicated oral Trichomonas infections and dramatically improved gingivitis/halitosis (Hosaka et al., 2017). This hints at the fact that anti-protosoals (like metronidazole) may be beneficial, although we don't have direct human trials. For E. gingivalis a recent in vitro study revealed that exposure of the amoeba to amoxicillin and metronidazole caused the formation of cyst-like structures inside the amoebae, which may provide enhanced resistance against therapy (Becker et al., 2023). The fact that C4B encystation state exists renders necessary drugs that can penetrate (or bypass) cyst-forms. Furthermore, antiseptic mouthwashes (e.g. chlorhexidine) are frequently mentioned in non-refereed publications as adjuncts for the control of oral protozoa, but high-quality trials do not exist. Taken together, until definitive clinical trials of specific antiparasitic therapies in humans are conducted, the clear companion to physical debridement is an antiseptic/antiprotozoal agent directed at reducing residual protozoan burden (Rosenfeld et al., 2024).

Third, a preventive and maintenance outline is important to reduce the recolonization of oral protozoa and cross-infection, because such organisms live in niches of poor hygiene (e.g., deep pockets and systemic comorbidity settings) (Yaseen et al., 2021). This encompasses daily strict oral hygiene (tooth brushing, interdental cleaning, and antimicrobial rinses), professional maintenance therapy at regular intervals, reduction of risk factors (smoking cessation, control of diabetes) and screening at least in high-risk patients/groups (immunocompromised patients or those with orthodontic appliances). Some recent pilot studies with probiotic therapy and dietary intervention in periodontal diseases demonstrated that altering the microenvironment may reduce protozoa parasites indirectly by re-establishing the balance of microbiota (CDC et al., 2025).

However, many research and clinical needs are still required. There are no universal therapeutic regimes for E. gingivalis and T. tenax in humans according to research, similarly human random tests have not been done. It matters if diagnostic heterogeneity (microscopy vs PCR), sampling variability (saliva vs plaque vs pocket) and longitudinal data are missing. The ability of E. gingivalis to encyst (Becker et al., 2023) suggests that failure in resistance or relapse is possible unless all life forms are targeted. Moreover, the zoonotic potential of T. tenax (Matthew et al., 2023). questions cross-reservoir reinfection, which could demand community based rather than individual public health attention. Studies should compare the addition of antiprotozoal /antiseptic adjuncts to mechanical therapy with mechanical therapy alone, conduct long-term follow-up for recolonization and evaluate treatment of confection (protozoa + bacteria) as well as intervention cost-effectiveness and safety in the oral environment.

CONCLUSION

An oral parasitic infestation namely by organisms like Entamoeba gingivalis and Trichomonas tenax has also been thought to be contributory toward periodontal inflammation and tissue destruction. Treatment at the moment is largely based on mechanical periodontal therapy, which efficiently reduces the parasitic load and ameliorates clinical signs. Use of adjunctive antiseptic or antiparasitic agents may provide additional benefit, but strong human data are lacking. Continued oral hygiene and maintenance of gingival health as well as management of systemic and behavioral risk factors are important in long-term prevention. In general, standardised diagnostic approaches as well with thoroughly planned clinical studies are desperately needed for defining unambiguous treatment recommendations and the real pathogenic relevance of these oral parasites.

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