



***Entamoeba gingivalis* and *Trichomonas tenax* in Periodontal Disease**

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Authors' contributions

This work was carried out in collaboration among all authors. Author OIO conceived the idea. Author AOO wrote the manuscript with input from all authors. Authors MOF and AOL revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

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ABSTRACT

This review is aimed at elucidating the role of *Entamoeba gingivalis* and *Trichomonas tenax* in the aetiology of periodontal disease. Periodontal disease results from localised inflammation of the periodontium due to plaque accumulation and if left untreated can lead to loss of teeth. Although dental plaque is composed mainly of bacteria, *Entamoeba gingivalis* and *Trichomonas tenax*, both of which are protozoan parasites have been found in plaque and implicated in periodontal disease. *E. gingivalis* is an amoeba associated with poor oral hygiene while *T. tenax* is a pyriform flagellate that lives in the tartar around the teeth, cavities of carious teeth, necrotic mucosal cells in the gingival margins of gums and pus pockets in tonsillar follicles. These parasites are transmitted by close contact, saliva, droplet spray and kissing or use of contaminated dishes, cups, spoons and

forks as well as drinking water. Age, gender, socio-economic status, dental condition and gingival pathology have been reported to influence the presence of the parasite. Genetic variability and stress are also some of the factors that determine the transition of the periodontium at some gingival sites from healthy to inflame. Researchers have observed that the prevalence and severity of periodontitis is higher in developing countries than developed countries. But with good oral hygiene, regular scaling and polishing and use of antiparasitic drugs, periodontal disease caused by these parasites can be prevented and periodontal health restored.

Keywords: *Entamoeba gingivalis*; parasite; periodontitis; periodontal disease; *Trichomonas tenax*.

1. INTRODUCTION

Oral disorders are important causes of concern in human health and of these; periodontal disease is the most common [1]. Periodontal disease results from localized inflammation of the periodontium due to its exposure to plaque. Plaque build-up initially results in gingivitis characterized by mild inflammatory signs that are reversible if oral hygiene is reinstated. However, if left untreated, gingivitis can progress to painful periodontal ligament destruction, alveolar bone loss, and eventual loss of teeth (severe periodontitis). Plaque is composed mainly of bacteria; however, evidence suggests that protist organisms, such as *Entamoeba gingivalis* and *Trichomonas tenax*, are also present and correlate with periodontal disease [2-4].

Entamoeba gingivalis is an amoeba found in the oral cavity of both hygienic and unhygienic mouths. It is an actively motile protozoan that moves with the aid of multiple pseudopodia and lives as a harmless commensal in the gingival tissue around the teeth [5-6]. The parasite was described by Gross in 1849 and is found in the soft tartar between the teeth, however in periodontitis patients, no cyst of the parasite has been found. It is transmitted from person-to-person by close oral contact like kissing or from contaminated drinking utensils [6]. Its occurrence can vary according to age, presence of periodontal disease, and immunosuppressive conditions [7]. Although *E. gingivalis* is considered a harmless commensal, there are controversies concerning its pathogenicity. This is because it has been detected in healthy individuals and has also been associated with periodontal disease [8]. Not much information is available about the biological and genetic characteristics of *E. gingivalis* as well as its function in bringing about oral lesions that contribute to the initial phase and progression of periodontal disease [9-10].

Trichomonas tenax is a pyriform flagellate that is also a harmless commensal of the human mouth and lives in the tartar around the teeth, in cavities of carious teeth, in necrotic mucosal cells in the gingival margins of gums and in pus pockets in tonsillar follicles [11]. It is transmitted by kissing, salivary droplets and fomites. Though it is considered a harmless commensal in the mouth, it has been reported to cause respiratory infections and thoracic abscesses. However this can be prevented by better oral hygiene, which is known to rapidly eliminate infections [6]. It is known to enter the respiratory tract by aspiration from the oropharynx and cause bronchopulmonary trichomoniasis.

The prevalence of *T. tenax* in periodontal disease has been reported to be 33.3% though age, social status, alcohol consumption, dental condition and gingival pathology influence the presence of the parasite [7,12]. A prevalence of 28% for *T. tenax* and 50.7% for *E. gingivalis* was also reported among 300 patients [13] while 8.4% for *T. tenax* was reported in Iraq among patients with poor oral hygiene using saliva [14]. A prevalence of 23.53% for *T. tenax* and 31.37% for *E. gingivalis* was also reported among periodontal disease patients using saliva and dental biofilm/calculi [15]. This review is aimed at elucidating the role of *Entamoeba gingivalis* and *Trichomonas tenax* in the aetiology of periodontal disease.

2. THE PERIODONTIUM

The oral cavity is an orifice with many micro-organisms, some of which can cause harm. The tooth is the hard conical structure set in the alveoli of the upper and lower jaws, which functions in mastication and assists in articulation of speech. It is composed of dentin which is a bony-like material and is surrounded by the gingival mucosa. The root is buried in the alveolus, the neck is surrounded by the gum and the crown which is the exposed part is covered

by a much harder white inert acellular tissue called enamel. In the centre of the tooth is the pulp cavity which contains a gelatinous substance known as dental pulp as well as blood vessels and nerves. In mammals, the tooth is attached to the alveolar ridge of the jaw by the periodontal ligament which provides a strong but flexible attachment that can withstand mastication forces. The periodontal ligament attaches to the alveolar bone of the jaw and the cementum on the root of the tooth [13,16-18].

The periodontium is the connective tissue that surrounds the tooth root and attaches it to its bony socket. It consists of fibres fixed in the cementum and extends into the alveolar bone [18]. Only the gingival tissues may be directly seen in a healthy periodontium which is stippled, pale pink or coral pink in Caucasians with various degrees of pigmentation in other races [19].

3. PERIODONTITIS

This is the inflammation of the periodontium which affects the supporting structures of the teeth such as the alveolar bone, cementum, and periodontal ligaments. Until the 1960s, it was believed that gingivitis that persists for a long period of time always progress to periodontitis but the development of periodontitis is now considered to be an independent process associated with a shift in the types and proportions of bacteria along the gums as well as other environmental factors such as poor oral hygiene. This is because differences have been found in the content of dental plaque in areas of healthy and diseased periodontium [17-18]. Loss of balance between the host immune system and the microbial virulence of periodontal disease pathogens may also be responsible for triggering periodontitis as the immune system activated by microbiological agents may attack the host and not the biofilm bacteria, causing the destruction of periodontal tissue and alveolar bone as well as resulting in loss of teeth [20]. About 5 to 20% of any population is affected by severe periodontitis [21]. There are different types of periodontitis, the most common of which is chronic periodontitis [22].

3.1 Chronic Periodontitis

It is an infectious disease that makes the supporting structures of the teeth to be inflamed and also result in progressive attachment and bone loss. It is the most common form of periodontitis in adults, though can also be seen

in adolescents and occasionally in children. Its prevalence and severity increases with age, it has a variable rate of progression and may affect a variable number of teeth. Disease severity may be slight when bone loss is in the coronal third of the root, moderate when bone loss is in the middle third of the root and advanced when in the apical third of the root length.

The disease may be localized if 30% or less of teeth are affected or generalized if more than 30% of teeth are affected. It is characterized by subgingival calculus, variable microbial pattern and slow to moderate rate of progression, though there may be periods of rapid progression too. Also observed in chronic periodontitis are pocket formation and/or gingival recession. Signs of inflammation vary depending on the patient's plaque control and as the disease advances, mobility and migration of individual or segmental teeth may occur. It is associated with systemic diseases such as diabetes and can be modified by other factors such as smoking [23].

4. CAUSES OF PERIODONTITIS

The oral cavity is suitable for microbial invasion with parasites such as *E. gingivalis* and *T. tenax* which are often seen in the oral cavity due to poor dental hygiene. Facultative Gram-positive micro-organisms are often found in healthy areas of the periodontium while plaque from active periodontitis sites usually contain anaerobic and microaerophilic Gram-negative micro-organisms. Although about 300 types of bacteria have been isolated from the oral cavity, adult periodontitis is associated mainly with *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis* and *Prevotella intermedia* [17,24].

5. PARASITES IN PERIODONTITIS

It has been shown that two parasites are responsible for oral parasitic infections and they are; *Entamoeba gingivalis* and *Trichomonas tenax* [25]. As the first commensals found in human oral cavity, they occur only as trophozoites, and are found in gingival tissues, particularly in purulent inflammatory processes [26]. Though these commensals may be opportunistic, they are able to proliferate in the gingival tissue and cause periodontal disease [27]. It has been found that *E. gingivalis* occurred more often in patients with periodontitis while *T. tenax* was more common in individuals with healthy gums [25].

5.1 Entamoeba Gingivalis in Periodontitis

Entamoeba gingivalis has a rounded nucleus with the nuclear membrane lined by coarse chromatin granules. It is similar in morphology to *E. histolytica* with large number of pseudopodia which allows its quick movement. It has no recognizable cyst form in clinical specimens, but only trophozoites, which vary in size from 10 to 35 microns [10]. It belongs to Entamoebidae family and sub-order of Tubulinae [28]. It inhabits teeth cavities, dental tartar, necrotic mucosa of cells and the gingival fringes of the gums [29]. In some cases, it has also been isolated from tonsil crypts and tonsil tissue sections [30] as well as the vagina and uterus [9].

According to some studies, it is involved in the induction of periodontitis but some other researchers consider it as an opportunist which survives in the medium induced by periodontal disease, though it is also known to cooperate with some symbiotic bacteria in causing periodontal disease which is characterized by gum itch, sore palate, halitosis, fatigue, severe headaches, and periodontal tissue damage [26,29]. The presence of *E. gingivalis* is often associated with diseased gingival pocket sites, age and the quantity of calculus on teeth though it can also be found in healthy mouths [31-33]. Tissue destruction in periodontitis may as well result from proteolytic enzymes released from host polymorphonuclear leucocytes lysed by *E. gingivalis* which leads to destruction of the maxillary bone surrounding the teeth, though elimination of *E. gingivalis* ensures rapid healing in chronic periodontitis [34].

The parasite is very difficult to culture and cannot be cultured in the absence of bacteria [35]. The identification of the parasite has given a new approach to therapeutic interventions against periodontitis as anti-parasitic treatments in humans, patient follow-up and experimentation in animal models will also help in drawing better conclusions about the causal link between *E. gingivalis* and periodontitis [36].

5.2 Trichomonas Tenax in Periodontitis

Trichomonas tenax is a Trichomonadidae family member, referred to in older works as *T. buccalis* and *T. Elongate* [28]. It cannot survive passage through the digestive tract and is resistant to temperature changes, thus can live for several hours in drinking water [33]. It is a pear-shaped protozoan with an undulating membrane that

extends to two-thirds the body length. It has an oval nucleus which is often located at the central anterior portion of the organism. It is a flagellate with five flagella, four extending anteriorly and one posteriorly. It also has an axostyle which runs along the entire body length of the organism extending beyond the body. It only exists in trophozoite form and varies in length from 5 to 14µm [37]. It was first discovered by Muller in 1773 in an aqueous culture of tartar from teeth [38]. It has a world-wide distribution in man and monkeys which are its natural hosts [39].

The presence of *T. tenax* in the oral cavity indicates poor oral hygiene, since its incidence is about three or four times greater in patients with periodontal disease than in healthy individuals [40-42]. It is also commonly seen with pyogenic organisms in pus pockets or at teeth base [43]. It is an anaerobic parasite of the oral cavity and also found in sub maxillary glands [44]. In cancer or some lung disease patients, it causes bronchopulmonary infections [45-47]. The amount of *Trichomonax tenax* found in oral washing is usually reduced and may not be detected by wet-mount. Staining cannot be used for species identification and culture techniques are not regularly used but molecular techniques are now available for detection and identification of Trichomonas species [48]. A reliable means for more rapid and specific detection as well as identification of trichomonads is amplification of the 5.8SrRNA gene which is present in multiple copies in the genome and conserved in certain regions by polymerase chain reaction (PCR) followed by sequencing [49,50].

It has been shown that *T. tenax* possess proteolytic activity mediated by cysteine endopeptidases which hydrolyse collagen fibres in the periodontium [34]. Acid phosphatase has also been found in vesicle structures of *T. tenax*, in the Golgi apparatus, in primary and secondary lysosomes, in cytoplasmic granules and in the free extremity of the waving membrane [51].

6. MODES OF TRANSMISSION OF *E. gingivalis* AND *T. tenax*

The trophozoites of *E. gingivalis* and *T. tenax* are transmitted from person to person by close contact, saliva, droplet spray and kissing or use of contaminated dishes, cups, spoons and forks as well as drinking water. Transmission via contaminated water is because *T. tenax* can live in water for few hours to few days [52-54]. *T. tenax* enters the respiratory tract by aspiration

from the oropharynx causing pulmonary disease [47].

7. SIGNS AND SYMPTOMS OF PERIODONTITIS

Periodontitis is characterized by gingivitis, destruction of the alveolar bone and periodontal ligament, apical migration of the epithelial attachment which leads to the formation of periodontal pockets and subsequent loosening and removal of the teeth [17,18]. Other signs and symptoms include pain, halitosis and gingival bleeding.

8. PREVALENCE OF PERIODONTITIS

Periodontitis is common among immunosuppressed individuals [8] and in certain systemic diseases such as acquired immunodeficiency syndrome (AIDS), cardiovascular disease, leukemia, Crohn disease, Down syndrome, sarcoidosis and Chediak-Higashi syndrome [17].

Severe periodontitis is found in 5–20% of most adult populations worldwide [55]. Considering gender, males have been found with poorer periodontal status (84%) than females (78.3%) and this can be associated with the habits and consciousness of females to perform better oral hygiene practices [56]. The prevalence of chronic periodontitis also increased with age from 35% for 35-40 years age group to 85% for 80-90 years old subjects. It has been observed that the transition of the periodontium at some gingival sites from being healthy to inflamed may be due to genetic variability and stress [57].

The prevalence of periodontal disease has been reported to be high in Nigeria with 57% prevalence nationwide [58]. In Oyo State, prevalence of 35.3% and 94.8% have been reported by different researchers [59,60]. Among adult males in Edo State, Nigeria, the prevalence of periodontitis was reported to be 15.4% [61] while in Imo State, a prevalence of 94% was reported for periodontal disease which was more common in males than females [62].

9. PREVALENCE OF *E. gingivalis* AND *T. tenax* IN PERIODONTITIS

E. gingivalis is often times found in the early stages of periodontal disease while *T. tenax* which is more common in individuals with healthy gums sees the oral cavity as its natural habitat [15]. Previous studies have shown that the

infection rate of *E. gingivalis* in patients with periodontitis is higher than that of healthy patients [63], so some researchers inferred that this protozoan is related to periodontitis [64]. Some reports indicated that the prevalence of *T. tenax* worldwide ranges from 4 to 53%; with high prevalence in patients with chronic periodontitis [3,53,65]. The occurrence of *E. gingivalis* in individuals with periodontal disease is high [3,10] but since it is also found in the oral cavity of healthy individuals, some authors believe that this commensal could be opportunistic, that is, capable of proliferating in a gingival environment modified by periodontal disease [26,27].

A prevalence of 8.4% for *T. tenax* in periodontitis patients and 4.1% in healthy mouths with both sexes showing approximately equal incidence was reported in Iraq [14]. Another study conducted among periodontal disease patients, reported a prevalence of 23.53% for *T. Tenax* [15]. In a similar study, 41.7% and 9.2% were reported for *E. gingivalis* and *T. tenax*, respectively, whilst 3.3% were infected with both parasitic protozoa as *E. gingivalis* was also more prevalent in males at the age of 21-30 years [29]. Prevalence of 9% and 8-30% have also been reported in Japan and the Czech Republic respectively [32]. An identical study also recorded 55.6% for *E. gingivalis* and 29.1% for *T. Tenax* [31] while another reported the prevalence of *E. gingivalis* in periodontal pockets to be 27% using conventional PCR but 69% using real time PCR [33]. In a similar study, a prevalence of 60% using PCR and 68.6% via clinical observation was reported [36]. In another study, the prevalence of oral protozoa (*E. gingivalis* and/or *T. tenax*) was recorded to be 37.9% [66] while a similar study reported 28.6% for *T. tenax* in periodontal disease patients [67]. In a study carried out in Enugu, Nigeria among dental patients, the prevalence of *E. gingivalis* was 4.9% while *T. tenax* was 11.3%, with no patient harbouring both parasites. It was also reported that individuals aged 20 years or less were one-third as infected as those above 20 years [68]. *E. gingivalis* and *T. tenax* were also reported to be more in dental plaque than saliva samples while parasite quantity was reduced in healthy individuals compared to periodontitis patients [69].

10. DIAGNOSIS OF PERIODONTITIS

Clinical diagnosis of periodontal disease is by recognizing various signs and symptoms in the periodontal tissue which show a deviation from

health [19]. The diagnosis of periodontal disease is by evaluating clinical signs and symptoms such as gingival changes in colour and contour as well as texture alterations and the presence of bleeding on probing, these may also be supported by radiographs. Non- plaque induced gingival diseases may require histopathologic, microbiologic or serologic investigations. Other signs and symptoms include tooth mobility and migration as well as the presence of pockets which reflects loss of periodontal attachment. Signs of inflammation such as redness and swelling should also be looked out for in combination with attachment loss and pocket depth [70].

Family history and predisposing factors such as smoking also require consideration for proper diagnosis. Imaging techniques may show the loss of marginal bone which confirms attachment loss [71]. While probing, the presence of pus and blood serve as disease pointer [72]. Significant increase in attachment level is the gold standard for the measurement of periodontal disease activity at a site [73].

Various techniques are applied for the detection of microorganisms in subgingival plaque, such as phase-contrast microscopy, dark-field microscopy, culture, immunological techniques, nucleic acid tests, enzyme tests and polymerase chain reaction (PCR); though these tests are not done routinely to diagnose periodontal disease but some are used in periodontal disease that is resistant to treatment. Bacteriological testing with sample culture and antibiotic sensitivity testing depends on the ability to culture anaerobic and capnophilic species [74]. Microscopic examination of wet preparation is one of the quickest diagnostic methods for *T. tenax* while *E. gingivalis* can also be diagnosed by staining with Trichrome vitelli stain [29]. PCR is an *in vitro* method of amplifying deoxyribonucleic acid (DNA) in which the gene sequences are selectively amplified using DNA polymerase. The PCR cycle basically consists of three steps which are denaturation, annealing and extension performed in a closed test tube at different temperatures. Since 1985 when PCR was developed, it has proven very useful in identifying periodontopathogenic species. It is one of the very sensitive and specific methods for detecting the presence of DNA sequences of microorganisms in dental plaque; it is also rapid and efficient in identification of periodontopathogenic species compared to culture techniques [75].

11. PREDISPOSING FACTORS TO PERIODONTITIS

Predisposing factors, also known as risk factors are characteristics that have a causal relationship with the development of a disease [76]. Factors predisposing to periodontitis include:

11.1 Genetics and Susceptibility

A twins study revealed that genetic factors may contribute about 50% of the population variance in periodontal disease progression [77]. Identical and non-identical twins were studied by evaluating probing depth, clinical attachment loss, gingival and plaque index between both pairs. It was observed that all clinical parameters were more similar in identical than non-identical twins. Thus, the basis for the heritability of periodontitis seems to be biological and not behavioural in nature [78]. Though several genetic polymorphisms have been associated with periodontal disease but evidences are not enough at present to support the widespread use of genetic tests to either assess risk for disease or predict treatment response [79]. A study on interleukin-1 (IL-1) polymorphism revealed that IL-1 genotype positive patients had more advanced periodontitis lesion than IL-1 genotype negative patients of the same age group as well as an increased tooth loss in the IL-1 genotype positive subjects [80].

11.2 Bacterial Biofilm

Local irritating factors especially the dental bacterial biofilm seem to have an important role in the susceptibility to and onset as well as progression of periodontal disease [15]. Some oral micro-organisms implicated in periodontitis include *Porphyromonas gingivalis*, *Tannerella forsythia* and *Actinobacillus actinomycetemcomitans* which are anaerobic Gram- negative bacteria species [81].

11.3 Dental Calculus

It contains mineralized bacterial plaque which is mainly made up of calcium phosphate [$\text{Ca}_3(\text{PO}_4)_2$] mineral salts deposited on the surfaces of natural teeth and dental prostheses [82]. It is widely classified into two based on location; supragingival calculus which is found above the gingival margin and subgingival calculus which is found below the gingival margin

in the gingival sulcus or periodontal pocket [83]. Subgingival calculus provides an ideal environment for bacterial adhesion as it keeps the bacterial deposits in close contact with the tissue surface, thereby influencing both bacterial ecology and tissue response. Calculus deposits often develop in areas difficult to access for oral cleaning and a heavy layer of calculus doubles the probability of infection as persons with little or no calculus are only half as susceptible to infection as individuals with heavy layer of calculus on their teeth [84]. *E. gingivalis* and *T. tenax* have higher affinity for diseased and dirty oral cavities [32].

11.4 Sex

The occurrence of *E. gingivalis* and *T. tenax* has been reported to be related to sex [25] with both parasites more common in male than female [54] though a study has reported *E. gingivalis* to be strongly marked in women than men [85]. Men have also been reported to be less careful and less regular in practicing oral hygiene measures [32].

11.5 Age

Age has also been considered a predisposing factor for protozoan infection in patients with periodontal disease as several authors have reported that the incidence of *E. gingivalis* and *T. tenax* increases with age [41,86,87]. A low incidence of these protozoan parasites has been observed in young children and a pronounced increase with age. It has also been reported that in nations with high human development index, the prevalence of periodontitis in adults above 30 years of age is as high as 50% [88]. Though age has been associated with increased rates of periodontal disease with the loss of the alveolar bone and periodontal attachment in the elderly population but severe periodontitis has been reported not to be a natural consequence of ageing [89].

11.6 Smoking

Smoking can affect the development of periodontal disease by changing the host response, periodontal healing process and treatment outcomes as well as the periodontal disease pattern [90]. Tobacco can reduce the ability of plasma cells to synthesize IgM and IgG as well as the phagocytic activity and chemotactic response of gingival neutrophils, thus, impairing the host's defense against

bacteria in the gingival pocket [43]. In addition, tobacco smoking may also modify the production of pro-inflammatory cytokines such as interleukin-1 (IL-1) and tumour necrosis factor-alpha (TNF- α) which are key regulators of the host response to microbial challenge [91].

11.7 Diabetes Mellitus

Diabetes mellitus (DM) is a metabolic disorder characterized by altered glucose tolerance and impaired carbohydrate metabolism due to insulin dysfunction. It is majorly of 2 types; type 1 and type 2 [83]. Diabetes affects gingival bleeding, probing depth, and attachment loss by causing changes in subgingival environment and the host's immuno-inflammatory response as well as tissue homeostasis and wound healing [92]. There are contradicting reports regarding the relationship between diabetes, *E. gingivalis* and *T. tenax* as a study reported a high incidence of these parasites (74%) in adult diabetics [42] while another study reported a low incidence [93].

11.8 Low Standard of Living

People with low standard of living have been found to be more susceptible to periodontitis [94].

12. PREVENTION AND CONTROL OF PERIODONTITIS

12.1 Regular Mouth Cleaning

This is done by brushing with fluoride toothpaste or using chewing stick. Dental plaque is kept in an immature state and minimal amount if the teeth are regularly cleaned. But when the mouth is not cleaned regularly, then large quantity of micro-organisms as well as endotoxins and other enzymes move into the gingiva causing irritation and inflammation. Good oral hygiene is aimed at controlling bacterial plaque but when oral hygiene decreases, a recurrence is possible, thus periodontitis has periods of remission and exacerbation [20].

12.2 Regular Scaling and Polishing

Good oral hygiene with regular scaling and polishing prevents periodontitis [70].

12.3 Oral Hygiene Awareness

Awareness on good oral hygiene should be increased as oral hygiene instructions and

motivation can significantly reduce plaque. Dentists should also encourage patients to practice proper plaque control [95,96].

12.4 Use of Anti-Parasitic Drugs

The administration of anti-parasitic drugs such as metronidazole can help control the growth of protozoan parasites in the oral cavity [20].

12.5 Avoid Smoking

This is because smoking has been reported to influence periodontal disease [97].

12.6 Improved Standard of Living

Higher prevalence of periodontitis has been reported among individuals with low socio-economic status, thus improved standard of living can help prevent and control periodontal disease [98,99].

13. CONCLUSION

Entamoeba gingivalis and *Trichomonas tenax* are protozoan parasites found in dental plaque and implicated in periodontal disease. These parasites are transmitted by close contact, saliva, droplet spray and kissing or use of contaminated dishes, cups, spoons and forks as well as drinking water. The presence of these parasites in the oral cavity is influenced by age, sex, socio-economic status, dental condition and gingival pathology. But with good oral hygiene, regular dental care utilization and use of antiparasitic drugs, periodontal disease caused by these parasites can be prevented and periodontal health restored.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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