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REVIEW ARTICLE

# Low levels of caries in aggressive periodontitis: A literature review



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## KEYWORDS

Dental caries;  
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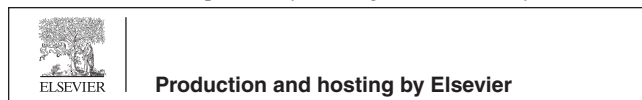
**Abstract** This article is a traditional literature review on caries levels in aggressive periodontitis. Aggressive periodontitis generally affects systemically healthy individuals aged < 30 years (older individuals can also be affected) and is characterized by a young age of onset, rapid rate of disease progression, and familial aggregation of cases. Dental caries is caused by the dissolution of enamel by acid-producing bacteria present in the plaque biofilm, especially when the biofilm reaches critical mass due to improper oral hygiene. The association between caries level and aggressive periodontitis has long been debated. Initial research indicated that caries levels were high in patients with aggressive periodontitis, but high-quality studies have consistently shown that caries and aggressive periodontitis are inversely related. A recent *in vitro* study showed that *Streptococcus mutans* was killed more readily in the saliva of patients with aggressive periodontitis and *Aggregatibacter actinomycetemcomitans* positivity than in patients with *A. actinomycetemcomitans* negativity. Other mechanisms possibly explaining the inverse relationship between caries and aggressive periodontitis in cases of Down's syndrome are also discussed in this literature review. The usefulness of caries level in the diagnosis of aggressive periodontitis in developing countries such as India, where the disease is diagnosed primarily on the basis of clinical and radiographic features and familial history is also discussed.

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## 1. Introduction

Aggressive periodontitis (AgP; formerly known as juvenile periodontitis) generally affects systemically healthy individuals aged <30 years (older individuals can also be affected) and is characterized by a young age of onset, rapid rate of disease progression, and familial aggregation of cases (Masamatti et al., 2012). AgP has been classified into localized and generalized types. Localized AgP usually affects first molars and incisors, resulting in deep periodontal pockets and advanced “arc-shaped” bone loss. The main organism implicated in localized AgP is *Aggregatibacter actinomycetemcomitans*, which colonizes the subgingival plaque biofilm. Generalized AgP usually involves more teeth and is caused by *Porphyromonas gingivalis* (Masamatti et al., 2012).

Dental caries is caused by the dissolution of enamel by acid-producing bacteria present in the plaque biofilm, especially when the biofilm reaches critical mass due to improper oral hygiene (Broadbent et al., 2013). Dental caries can occur in pits and fissures, on smooth surfaces (labial, facial/palatal, or lingual and proximal), and on root surfaces. *Streptococcus mutans* is a common Gram-positive bacterium that plays a significant role in dental caries.

The level of caries occurring in association with AgP has long been debated. Initial research indicated that caries levels were high in individuals with AgP (Albandar et al., 1996), but high-quality studies have consistently shown that caries and AgP are inversely related (Al-Habashneh et al., 2009; Al Omari et al., 2008; Bial and Mellonig, 1987; Fine et al., 1984). This article presents a review of the literature on caries levels in AgP.

### 1.1. Studies on caries levels in aggressive periodontitis (formerly known as juvenile periodontitis) versus periodontally healthy patients

As early as 1984, Fine et al. compared caries levels in patients with juvenile periodontitis (now known as AgP) with those in periodontally healthy individuals. They found proximal decay in significantly fewer (26%) patients with juvenile periodontitis than in control group subjects (91%  $p < 0.05$ ). Bial and Mellonig (1987) screened 49,380 male naval recruits and diagnosed juvenile periodontitis in 270 cases, most of which showed minimal or no radiographic evidence of caries. In contrast, Albandar et al. (1996) stated that caries levels were higher in patients with localized juvenile periodontitis, and included a high rate of caries among clinical features used to

identify early-onset periodontitis (EOP; later included in the category of AgP) at an early stage. They found significantly more surfaces with untreated caries or fillings in patients with generalized or incidental EOP than in subjects with no periodontitis (mean, 7.7 versus 5.8 surfaces  $p < 0.05$ ) (Albandar et al., 1996).

### 1.2. Studies on caries levels in aggressive periodontitis (formerly known as juvenile periodontitis) versus chronic periodontitis patients

Sioson et al. (2000) concluded that patients with AgP had significantly fewer proximal caries than did those with chronic periodontitis, and stated that an in-depth study of AgP cases could provide vital clues about caries and periodontal disease. Al Omari et al. (2008) found significantly less caries experience in patients with AgP ( $n = 210$ ) than in those with chronic periodontitis ( $n = 332$ )  $p < 0.001$ . Al-Habashneh et al. (2009) conducted a cross-sectional study to assess caries levels in various forms of periodontal disease, and concluded that decayed, missing, and filled teeth indices were significantly lower in patients with AgP than in those with chronic gingivitis or chronic periodontitis. Although only seven clinical studies on the topic of caries levels in aggressive periodontitis have been published, more research related to the inverse relationship of caries and periodontal disease has been published. Additional research has been done and is explained in further paragraphs.

## 2. Discussion

### 2.1. Studies on caries levels in patients with Down's syndrome

A lower prevalence of caries, most notably interproximal lesions, has also been documented in patients with Down syndrome (Barnett et al., 1986). A severe form of periodontal disease develops in many of these patients, due primarily to defects in immune function (Barnett et al., 1986). Cogulu et al. (2006) found significantly lower  $p < 0.05$  caries indices in children with than in those without Down syndrome, but no difference in salivary *S. mutans* levels of their association with dental caries. Arbitrarily primed polymerase chain reaction typing revealed that all *S. mutans* profiles differed between children with Down syndrome and the control group, leading the authors to suggest that these profiles underlie the low prevalence of caries in individuals with Down syndrome (Cogulu et al., 2006).

A recent *in vitro* study showed that *S. mutans* was killed more readily in the saliva of patients with aggressive peri-

odontitis and *A. actinomycetemcomitans* positivity than in patients with *A. actinomycetemcomitans* negativity (Fine et al., 2007). This finding supports the concept that a naturally occurring, genetically variable salivary factor can influence oral microflora and later susceptibility to a particular disease (Fine et al., 2007). However, the bacteria in this *in vitro* study were planktonic, and they may behave differently in a biofilm environment. Further studies are needed to test this behavior (Rudney and Staikov, 2002).

To investigate the mechanism underlying the inverse relationship between caries and AgP, Vellyyagounder et al. (2003) examined whether a lysine/arginine polymorphism exists at position 29 in the highly charged N-terminal region of human lactoferrin. Several possible mechanisms may explain the contribution of the observed functional difference between variants containing lysine human milk lactoferrin-K (hLf-K) and arginine human milk lactoferrin-R (hLf-R) to the pathogenesis of localized AgP (Vellyyagounder et al., 2003). This form of AgP is associated with high levels of the Gram-negative bacterium *A. actinomycetemcomitans* in the periodontal pocket. The oral microbiota of subjects harboring one or two hLf-K alleles may contain lower levels of *S. mutans*, which causes caries by colonizing interproximal surfaces, due to the increased antibacterial activity of hLf-K against Gram-positive bacteria (Vellyyagounder et al., 2003). This alteration in the oral microflora could account for the decreased incidence of proximal caries in patients with localized AgP in comparison with control subjects. Increased transcriptional activation activity of hLf-K may also contribute to an altered microenvironment that favors *A. actinomycetemcomitans* colonization.

### 3. Lower caries level as a clinical feature in the diagnosis of AgP

The clinical distinction between chronic periodontitis and AgP, especially the generalized form, is not clear (Armitage and Cullinan, 2010). Although distinction of these diseases may not be significant from a treatment perspective, it is essential from a research perspective to enable a complete understanding of their etiology and pathogenesis (Armitage and Cullinan, 2010). Darby et al. (2005) found that the majority 79.7% of dental practitioners in Victoria, Australia, felt confident in the diagnosis of gingivitis and initial periodontitis, but only 61.9% felt confident in diagnosing AgP and early-onset periodontitis. Thus, some confusion exists among clinicians, especially general dental practitioners, regarding the diagnosis of AgP. In contrast to Albandar et al. (1997) argument that a high caries rate characterized early-stage EOP, now classified as AgP, a growing body of literature suggests that a low caries level is a useful clinical feature for the identification of AgP. This readily identified clinical sign could be used to diagnose AgP, especially in developing countries such as India, where the disease is diagnosed primarily on the basis of clinical and radiographic features and familial history.

### 4. Conclusion

Research now is quite clear that caries levels in cases of aggressive periodontitis are low. This finding can be used as an additional feature in diagnosis of cases of aggressive periodontitis,

especially in countries where advanced microbiological tests are not available during diagnosis of cases. Further studies in the future will decipher the reasons for the existence of this unique inverse relationship not only in planktonic conditions but also intraorally in an biofilm environment.

### Conflict of interest

The author has no conflict of interest to declare.

### References

- Albandar, J.M., Brown, L.J., L oe, H., 1996. Dental caries and tooth loss in adolescents with early-onset periodontitis. *J. Periodontol.* 67 (10), 960–967.
- Albandar, J.M., Brown, L.J., L oe, H., 1997. Clinical features of early-onset periodontitis. *J. Am. Dent. Assoc.* 128 (10), 1393–1399.
- Al-Habashneh, R., Al-Omari, M.A., Taani, D.Q., 2009. Smoking and caries experience in subjects with various form of periodontal diseases from a teaching hospital clinic. *Int. J. Dent. Hyg.* 7 (1), 55–61.
- Al Omari, M.A., Al Habashneh, R., Taanni, D.Q., 2008. Dental caries experience in patients with aggressive periodontitis compared to those with chronic periodontitis. *Quintessence Int.* 39 (10), 847–851.
- Armitage, G.C., Cullinan, M.P., 2010. Comparison of the clinical features of chronic and aggressive periodontitis. *Periodontology* 2000 53 (1), 12–27.
- Barnett, M.L., Press, K.P., Friedman, D., et al, 1986. The prevalence of periodontitis and dental caries in Down's syndrome. *J. Periodontol.* 57, 288–293.
- Bial, J.J., Mellonig, J.T., 1987. Radiographic evaluation of juvenile periodontitis (periodontosis). *J. Periodontol.* 58 (5), 321–326.
- Broadbent, J.M., Page, L.A., Thomson, W.M., Poulton, R., 2013. Permanent dentition caries through the first half of life. *Br. Dent. J.* 215 (7), E12.
- Cogulu, D., Sabah, E., Uzel, A., et al, 2006. Genotyping of *Streptococcus mutans* by using arbitrarily primed polymerase chain reaction in children with Down syndrome. *Arch. Oral Biol.* 51 (3), 177–182.
- Darby, I.B., Angkasa, F., Duong, C., et al, 2005. Factors influencing the diagnosis and treatment of periodontal disease by dental practitioners in Victoria. *Aust. Dent. J.* 50 (1), 37–41.
- Fine, D.H., Furgang, D., Goldman, D., 2007. Saliva from subjects harboring *Actinobacillus actinomycetemcomitans* kills *Streptococcus mutans* *in vitro*. *J. Periodontol.* 78 (3), 518–526.
- Fine, D.H., Goldberg, D., Karol, R., 1984. Caries levels in patients with juvenile periodontitis. *J. Periodontol.* 55 (4), 242–246.
- Masamatti, S.S., Kumar, A., Virdi, M.S., 2012. Periodontal diseases in children and adolescents: a clinician's perspective part. *Dent. Update* 39 (8), 541–544 (547-8, 551-2).
- Rudney, J.D., Staikov, R.K., 2002. Simultaneous measurement of the viability, aggregation, and live and dead adherence of *Streptococcus crista*, *Streptococcus mutans* and *Actinobacillus actinomycetemcomitans* in human saliva in relation to indices of caries, dental plaque and periodontal disease. *Arch. Oral Biol.* 47 (5), 347–359.
- Sioson, P.B., Furgang, D., Steinberg, L.M., et al, 2000. Proximal caries in juvenile periodontitis patients. *J. Periodontol.* 71 (5), 710–716.
- Vellyyagounder, K., Kaplan, J.B., Furgang, D., et al, 2003. One of two human lactoferrin variants exhibits increased antibacterial and transcriptional activation activities and is associated with localized juvenile periodontitis. *Infect. Immun.* 71 (11), 6141–6147.