

Slots J. Predominant cultivable organisms in juvenile periodontitis.

Scand. J. Dent. Res. 84:1-10, 1976.

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Localized juvenile periodontitis affects the permanent teeth of young individuals and can cause pronounced damage to a dentition. This study represented the first comprehensive description of the pathogenic organisms in the disease. Deep periodontal lesions harbored a characteristic Gram-negative microflora. The key pathogen was isolated, characterized, and later classified as *Actinobacillus actinomycetemcomitans*. Knowledge about the organism's epidemiology, transmissibility, and susceptibility to treatment has dramatically changed the prevention, treatment, and prognosis of the disease. [The SCI® indicates that this paper has been cited in more than 165 publications.]

Long in the Tooth

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Localized juvenile periodontitis afflicts 0.1-1.0 percent of teenagers in the US and European populations and may occur with higher prevalence in some Third-World countries. The disease has a familial predisposition and is particularly prevalent among African-Americans. Localized juvenile periodontitis may lead to loss of the upper and lower incisor and first molar teeth within a period of a few years.

Until the mid-1970s, nothing was known about the pathogenic microorganisms in localized juvenile periodontitis. Some researchers even held the view that the disease was due to degenerative rather than infectious causes. It often failed to respond to conventional periodontal therapy.

This article identified a certain Gram-negative bacterium, later classified by this author as *Actinobacillus actinomycetemcomitans*, to be the most likely putative pathogen of localized juvenile periodontitis. Many laboratories in the US, Europe, and Asia later confirmed this finding. Today, localized juvenile periodontitis is the periodontal disease entity that most convincingly has been related to a given bacterium.

The localized nature and other clinical features of the disease are the result of interactions between *A. actinomycetemcomitans* virulence factors and protective host

mechanisms. *A. actinomycetemcomitans* colonizes the oral cavity in childhood and is poised to initiate periodontal destruction on the first teeth to erupt, viz. the incisors and first molars; the teeth that are also affected in localized juvenile periodontitis. The periodontal *A. actinomycetemcomitans* infection gives rise to a strong antibody response. The antibodies have the potential to control the small number of *A. actinomycetemcomitans* cells seeding to surfaces of later-erupting teeth, thereby preventing breakdown of those teeth. High antibody levels eventually may also neutralize the microbial insult in the original lesions, if the teeth are still remaining. The familial distribution of the disease stems from transmission of *A. actinomycetemcomitans* among susceptible family members. The transmission route has been delineated by microbial fingerprinting, including biotyping, serotyping, and, most recently, restriction fragment length polymorphism patterns.

Treatment and prognosis of the localized juvenile periodontitis has dramatically improved with the identification of *A. actinomycetemcomitans* as the key pathogen. In addition to residing in deep periodontal pockets, *A. actinomycetemcomitans* is capable of invading gingival tissue. This is probably the reason that mechanical tooth cleaning fails to eradicate the organism and arrest the disease. On the other hand, selected systemic antimicrobial therapies are able to eliminate the organisms from oral sites and stop disease progression. Systemic antimicrobial therapy is now used routinely as an adjunct to conventional periodontal therapy and has significantly enhanced the prognosis of the patient. Localized juvenile periodontitis represents a prime example of how a systematic research approach has led to a radical change in the understanding, treatment, and prognosis of a previously serious oral disease.

In research, localized juvenile periodontitis is widely used as a model to delineate the molecular events in destructive periodontal disease.¹ Data from studies of localized juvenile periodontitis have been instrumental in generating new hypotheses on the molecular biology of certain forms of adult periodontitis.

1. Slots J & Schonfeld S E. *Actinobacillus actinomycetemcomitans* in localized juvenile periodontitis. (Hamada S, Holt S C & McGhee J R, eds.) *Periodontal disease: pathogens & host immune responses*. Tokyo, Japan: Quintessence, 1991. p. 53-64.

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