Localized juvenile periodontitis (LJP) is a distinct clinical form of periodontal disease. In its classic form, it is a disease of adolescents in which individuals between 12 and 20 years of age demonstrate a rapid loss of supporting alveolar bone around the first permanent molars and incisors (1, 8). The classic condition exhibits apparent minimal clinical inflammation, bacterial plaque, calculus, or pain. It is a particularly intriguing condition in that a hereditary component has been hypothesized (4, 38). In a study of LJP patients, Newman et al. (35) described two numerically dominant groups of microorganisms, groups III and IV, which were later shown to include strains of Actinobacillus actinomycetemcomitans (46).

A cross-sectional study of 42 patients by Slots et al. (39) revealed a carrier rate of A. actinomycetemcomitans in 20% of normal juveniles, 36% of normal adults, and 50 and 90% of adult periodontitis and LJP patients, respectively. Zambron et al. (53) examined 403 patients for the incidence of A. actinomycetemcomitans and found 95% of juvenile periodontitis patients harbored the organism, whereas only 15% of his other population groups did. In a recent study, Mandell and Socransky (31) examined the subgingival flora of deep pockets displaying radiographic bone loss. They found a significant association between A. actinomycetemcomitans and LJP but not with gingivitis or adult periodontitis.

The purpose of this investigation was to longitudinally follow sites in an LJP population and their siblings. Utilizing the running median technique (11), periodontal pockets undergoing active destruction were identified and cultured for a group of organisms, including Bacteroides intermedii, Actinomyces spp., Fusobacterium nucleatum, Capnocytophaga spp., Streptococcus spp., A. actinomycetemcomitans, and Eikenella corrodens. These groups were sought because prior reports (34, 40, 42) related each of these organisms with health or disease.

Eight patients aged 10 to 18 years were selected for study. Longitudinal attachment measurements were performed (11) on days 1, 7, 30, and 37 after entry into the study by the patients. Active disease was defined as a loss of connective tissue attachment of ≥2 mm. A total of 168 sites per patient (6 sites per tooth) were followed, so that over 1,200 total sites were studied. Of these sites or pockets, eight displayed active disease. An additional 16 sites were selected as controls. Pocket depths averaged 8.4 mm (range 5 to 10 mm) in the diseased sites versus 4.8 mm (range 3 to 7 mm) in the control sites. The sampled sites were either molars or incisors, because these areas are most frequently affected in LJP (20). Subgingival plaque samples were taken from the base of the periodontal pockets with a sterile Morse (00) scaler inserted into the depth of the pocket. The tip was aseptically transferred to prereloded one-quarter strength Ringer solution, sonicated for 5 s (Heat System Ultrasonic, Plainview, N.Y.) at settings previously determined to give the highest recoverable bacterial counts (unpublished data) to disperse the plaque (32), and then 10-fold dilutions were made under an O2-free atmosphere. One-tenth-ml samples were plated on triplicate plates of selective and elective media for the following organisms: (i) A. actinomycetemcomitans (31) colonies were subcultured and antigen type was determined (6); (ii) E. corrodens (C. Walker, A. C. R. Tanner, C. Smith, and S. S. Socransky, J. Dent. Res. 58(Special Issue A):315, abstr. no. 108, 1978); (iii) Streptococcus mutans and Streptococcus sanguis (9); (iv) F. nucleatum (51); (v) Actinomyces viscosus and Actinomyces naeslundii (54); and (vi) B. intermedius (43) colonies were selected and identified by the method of Johnson and Holdeman (20). Total counts were determined on 5% Trypticase soy blood agar (BBL Microbiology Systems, Cockeysville, Md.), and the wet spreaders Capnocytophaga sputigena, Capnocytophaga ochracea, and Capnocytophaga gingivalis were isolated and identified (41). The plates were incubated in either air plus 10% CO2 or 80% N2-10% CO2-10% H2 for 7 days at 35°C in Brewer jars.

The results are summarized as log viable counts in Table 1. There was essentially no difference in the active versus inactive sites for the majority of organisms examined by the Mann-Whitney test. There was, however, a significant association of both A. actinomycetemcomitans (Y4 antigen type) and E. corrodens with disease-active versus nonactive sites. A. actinomycetemcomitans was found to occur in active sites at levels 100-fold greater than those of the nonactive sites. E. corrodens levels were 50-fold higher in the active versus the inactive sites.

A. actinomycetemcomitans appears to make up a small but significant component of the human bacterial plaque (23, 26, 52). The organism has been isolated from a variety of infections, including actinomycosis (10, 12, 14-16, 24, 25, 47, 49), bacterial endocarditis (29, 33, 36, 50), brain and thyroid abscess (3), and cystitis (48). A. actinomycetemcomitans demonstrates a number of biological characteristics of interest. Y4, an oral isolate of A. actinomycetemcomitans
has displayed the following biological activities: (i) a leukotoxin (44) which is cytotoxic for human peripheral blood polymophonuclear leukocytes and monocytes (45); (ii) a lipopolysaccharide which stimulates bone resorption, is toxic to macrophages, inhibits platelet aggregation, and demonstrates lethality in a mouse model system (22); and (iii) rapid alveolar destruction in monoinfected gnotobiotic rats (19).

* E. corrodens, a microaerophilic gram-negative bacillus, is part of the flora of the mouth, upper respiratory tract, and female genital tract (30). *E. corrodens* has been cultured from brain abscesses, subdural empyemas, meningitis, sinus infections, and osteomyelitis (5, 7, 21, 37). When monoinfected in gnotobiotic rats, it can cause significant tissue and alveolar bone loss (28). Tanner et al. isolated the organism in large proportions from an adult subject with advanced periodontal destruction (46).

Both *A. actinomycetemcomitans* and *E. corrodens* have been reported in the medical literature as interacting with other organisms to exaggerate an infectious process (2, 14, 15, 18, 20). It is possible that in LJP active disease sites these two organisms, either in combination with each other or with other as yet unidentified species, may lead to destruction. Ingram et al. (18), in his report of two brain abscess cases secondary to dental sepsis, demonstrated that both *E. corrodens* and *A. actinomycetemcomitans* were present, in addition to other bacteria.

Conversely, a number of organisms previously thought to be important in periodontal disease (*B. intermedius, F. nucleatum,* and *C. ochracea*) were not significantly elevated in active sites in the LJP patients. This may be due to the manner of site selection. In previous studies (31, 39, 46), healthy sites were compared with diseased sites based upon probing depths and evidence of radiographic bone loss. In this study, active sites were identified by longitudinal clinical monitoring of the attachment level changes (11). The control sites, which were not breaking down, often had deep pockets and radiographic bone loss. Whereas there was a significant difference in pocket depth (disease sites averaged 8.4 mm versus 4.8 mm for controls), there was no significant difference in total bacterial counts (Table 1). Traditionally, a pocket which averaged 4.8 mm (range, 3 to 7 mm) would be considered diseased. The running median technique (11) allows discrimination of these disease-active versus inactive pockets. One is able to see bacterial differences, based not on counts alone but also on qualitative and quantitative differences. Thus *B. intermedius, F. nucleatum,* and *C. ochracea* may represent opportunistic organisms.

Active periodontal disease in LJP patients may represent a blooming of existing organisms due to a lack of inhibiting organisms (13, 42), an abnormality in host resistance (27, 42, 44, 45), or specific properties of the infecting microorganism (17, 42, 44, 45). The results of this study suggest the possibility that both *E. corrodens* and *A. actinomycetemcomitans* are involved in the active phase of tissue destruction in LJP.

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### LITERATURE CITED

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