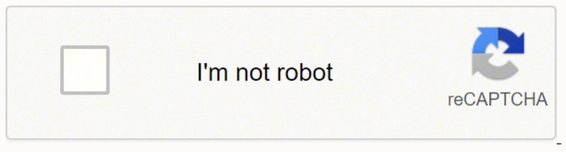

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Periodontosis—an analysis and clarification of its status as a disease entity. H. Phagocytosis and virulence of different stains of Porphyromonas gingivalis. [PubMed: 8910821]40.Genco R., Kornman D., Williams R., Offenbacher S., Zambon J., Ishikawa I., Listgarten M., Michalowicz B., Page R., Schenkein H., Slots J., Socransky S., Van Dyke T. Eur. 1996;1:926–932.41.Gibbons, R. S. [PubMed: 11345522]54.Haffajee A. [PubMed: 6713708]112.Sbordone L., Ramaglia L., Gulletta E., Iacono V. 1998;62:822–839. Risk assessment for periodontal diseases. gingivalis results in an interleukin-1β (IL-1β) mRNA response, decreased IL-8 accumulation (25), and inhibition of neutrophil migration through the epithelium (84), all factors that have an impact on the host defense system. Biol. [PMC free article: PMC313746] [PubMed: 2254014]116.117.Shenker B. Occurrence and nature of bacterial IgA proteases. nucleatum also adheres to and invades primary cultures of human gingival epithelial cells (55). K., Van Dyke T. Virulence factors of Porphyromonas gingivalis. C., Levine M. Microbiol. naeslundii. Several other less clearly defined factors should be included as part of a risk assessment for each patient. 1995;63:3878–3885. Dent 20451–456., 458–460, 462. [PubMed: 10522223]8.Asikainen S., Chen C., Slots J. The bacteria of periodontal disease. actinomycetemcomitans including cell invasion (90, 91, 93).P. E., Kieba I. [PMC free article: PMC313300] [PubMed: 2707855]70.Kornman K. C., Nieuw Amerongen A. As a part of the innate response, secretory IgA and numerous enzymes and antimicrobial factors present in the saliva neutralize microbial components. Biophys. [PubMed: 9495608]134.Tsai C. 1980;15:144–151. O., Tseng C. S., Crane A., Wang H. E., Nesbitt W. 1981;8:281–294. W., Demuth D. The role of Actinobacillus actinomycetemcomitans in the pathogenesis of periodontal disease. [PubMed: 9118282]98.O'Toole G., Kaplan H. This may be because both antibody titers and function (avidity) may vary with the patient's age and disease, making some individuals more or less susceptible to tissue breakdown (30). [PMC free article: PMC229599] [PubMed: 9003615]136.Van Winkelhoff A. Defensins. [PubMed: 10204480]75.Krisanaprakornkit S., Weinberg A., Perez C. Beyond the specific plaque hypothesis: are highly leukotoxic strains of Actinobacillus actinomycetemcomitans a paradigm for periodontal pathogenesis? 1993;8:225–229. S., Rosenbloom J., Rosenbloom J. Both systemic antimicrobials and locally delivered antimicrobial agents administered directly into periodontal pockets have been used in treatment of periodontal diseases.Systemic antibiotics have been effective and are specifically recommended in the treatment of aggressive forms of periodontal disease (72, 136). Through these processes, A. 1999;78(Special Issue):259.108.Sandros J., Madianos P. The oral microflora and human periodontal disease, p. IL-6 stimulates osteoclast formation and therefore may also play a role in bone resorption. 1996;64:2988–2997. Therefore, additional studies are necessary to better define the roles of proposed periodontal pathogens and their virulence factors in vivo in the destructive processes of periodontal disease.Despite the importance of infection and colonization by bacteria in periodontal infections, the immune status of the host and effectiveness of the host response are key determinants of disease susceptibility.In periodontal disease, bacterial colonization of the subgingival area results in both an innate host response and an acquired immune response. Considering that the gingival crevicular fluid is capable of being replaced 40 times in 1 h (45), the drug should ideally be substantive (retained on root surfaces) or be delivered in a slow- or controlled-release formulation. 1999;28:520–526. [PMC free article: PMC1684137] [PubMed: 3578282]13.Boughman J. For example, higher titers of an antibody reactive to the LPS of a virulent strain of A. B., Lantz M., Marucha P. [PubMed: 1067529]84.Madianos P. J., Genco R. J., Singer R. H., Boughman J. 1992;63:322–331. Infect. Commun. [PMC free article: PMC108102] [PubMed: 9529095]26.Darveau R. A., Wilson M. 1994;5:78–111. Although recent studies have suggested a role for environmental (39), behavioral (50, 51), and genetic (71) risk factors in periodontal disease progression, most, if not all, forms of periodontitis should be viewed as infectious diseases. 2000;54:413–437. nucleatum, B. Genetic analysis of juvenile periodontitis in families ascertained through an affected proband. Virulence factors of Actinobacillus actinomycetemcomitans. Epidemiology of periodontal diseases. L. For example, A. 2000;54:49–79. Nucleotide sequence of the leukotoxin gene from Actinobacillus actinomycetemcomitans: homology to the alpha-hemolysin/leukotoxin gene family. Ann. A unique genetic marker for chronic periodontitis recently linked this form of periodontal disease to an inherited allele responsible for IL-1β overproduction (71). Therapeutic strategies now being used in clinical practice include administration of low-dose doxycycline, which inhibits matrix metalloproteinases, i.e., collagenase (16, 44). M., Genco R. 1975;39:396–402. 1995;6:161–175. 1979;25:427–439. 1986;6:341–350. 2000;15:1–9. Taxonomy and virulence of oral spirochetes. F. J., Ho A. J., de Graaff J. The tooth-associated plaque primarily comprises gram-positive rods and cocci, whereas the plaque associated with the epithelial tissue lining the gingival crevice is predominated by gramnegative rods, filaments, and spirochetes. Studies have shown that in shallow to moderately deep pockets, good oral hygiene can change the subgingival flora to one more compatible with periodontal health (138).With the continued evolution of the specific plaque hypothesis, there is increased interest in chemical antimicrobials, which have the ability to suppress and/or eradicate the pathogenic players. V., Gunsolley J. However, the protective role of the adaptive immune response is not fully understood. The architecture of the biofilm and its matrix may inhibit penetration of antimicrobials, the biofilm may contain inactivating substances, and phenotypic changes in biofilm bacteria, such as slower growth, may increase their resistance to antimicrobial agents. gingivalis can invade human endothelial cells by a mechanism that involves fimbriae, cytoskeletal rearrangements, protein phosphorylation, energy metabolism, and P. The biological effects of the CDT, however, extend beyond immunosuppression and could play a role in other phenomena associated with A. M., Losh G., Graham S., Johnson G. [PubMed: 7886453]115.Shenker B. D. Risk indicators for alveolar bone loss. intermedia, and F. Scand. Inhibition of Actinobacillus actinomycetemcomitans leukotoxicity by bacteria from the subgingival flora. Recently, the organism was also shown to produce a cytolethal distending toxin (CDT) (88, 117, 130). E. The distribution and transmission of Actinobacillus actinomycetemcomitans in families with localized juvenile periodontitis. P., Brissette C. [PubMed: 9673165]31.Ebersole J. 63–104. Each of these cytokines is found in elevated levels in inflamed gingival tissues. [PubMed: 6445976]97.Offenbacher S. Influence of anti-Actinobacillus actinomycetemcomitans Y4 (serotype b) lipopolysaccharide on severity of generalized earlyonset periodontitis. P., DiRienzo J. In addition, β-defensin expression on other mucosal surfaces, such as buccal mucosa, tongue, and salivary derived defensins, may limit initial bacterial colonization by certain periodontal pathogens (75, 87, 114). Y., Ueno Y., Komatsuzawa H., Fujiwara T., Kurihara H., Suginaka H., Oswald E. [PubMed: 6947992]10.Baehni P., Tsai C. Alternatively, periodontal disease may be found in individuals with defects in either their innate or acquired immunity which limits their ability to mount an adequate response. Degradation of endogenous plasma membrane fibronectin concomitant with Treponema denticola 35405 adhesion to gingival fibroblasts. 1997;65:3983–3990. 1982;128:148–154. M., Ryan M. 1991;62:207–210. Occurrence of invading bacteria in radicular dentin of periodontally diseased teeth: microbiological findings. E., Taichman N. Y. Invasion of epithelial cells by Actinobacillus actinomycetemcomitans: a dynamic, multistep process. C., Hansen E. [PubMed: 6368611]87.Mathews M., Jia H. 1999;181:199–204. The protective nature of host responses in periodontal diseases. In contrast, aggressive periodontitis involves rapid attachment loss and bone destruction, and the destruction seen is usually not commensurate with the amount of microbial deposits. Adv. [PubMed: 8156311]60.Iino Y., Hopps R. Porphyromonas gingivalis invasion of gingival epithelial cells. gingivalis, F. 1998;25:99–105. A., Leys E. gingivalis proteases in the invasion process. A., Halloran S. Although not completely supported by these criteria for causation, there is also evidence that E. [PubMed: 7669215]36.Frandsen E. [PubMed: 11018133]69.Kolodrubetz D., Dailey T., Ebersole J., Kraig E. nucleatum to evade host defenses and to invade tissues. Helicobacter pylori and Porphyromonas gingivalis lipopolysaccharides are poorly transferred to recombinant soluble CD14. 2000;71:1699–1707. The non-specific theory in microbial etiology of inflammatory periodontal diseases. P., Tanner A., Page R. Disadvantages of systemic antimicrobials include bacterial resistance, side effects (including superinfections and gastrointestinal irritation), compliance, and the fact that periodontal destruction is often localized to a few teeth.Local delivery alleviates many concerns associated with systemic antibiotics and offers the ability to reach bacteria at the base of a pocket and retain activity for periods sufficient to have bactericidal or bacteristatic effects on offending pathogens. 1999;67:1227–1237. Invasion is via a "zipping" mechanism and requires the involvement of actins, microtubules, signal transduction, protein synthesis, and energy metabolism of the epithelial cell and protein synthesis by F. E., Bleeg H. [PubMed: 2670940]77.Lally E. Annu. [PubMed: 7548622]114.Schonwetter B. In E. H., Luh K. Beachey (ed.), Bacterial Adherence. The immune response also changes. S., Trummel C. gingivalis colonization of the plaque biofilm. [PubMed: 11154406]64.Jorgenson R. A., Welham D. C., Sparks B. Oral Surg. 1990;61:579–584. L., Duff G. Immune suppression induced by Actinobacillus actinomycetemcomitans. Herpesviruses in human periodontal disease. 1994;33:370–377. nucleatum invasion of epithelial cells is accompanied by high levels of IL-8 secretion. [PubMed: 9391752]93.Meyer D. Internalization of P. [PubMed: 393729]120.Slots J. Flow cytometry to monitor phagocytosis and oxidative burst of anaerobic periodontopathogenic bacteria by human polymorphonuclear leukocytes. gingivalis, a coaggregation partner with F. [PubMed: 295292]127.Socransky S. A., Smith D. Sci. The importance of the biofilm in plaque colonization and bacterial interactions and the impact these interactions have on expression or inhibition of specific virulence factors is not fully understood. 1984;11:193–207. gingivalis is an active process that requires energy production by both the epithelial cell and the bacterium (58, 79). 1999;20:136–167. [PubMed: 8164120]51.Grossi S. Other tissue-destructive host-derived factors include tumor necrosis factor alpha and PGE2, both of which stimulate MMP production and induce bone resorption. Evidence for invasion of a human oral cell line by Actinobacillus actinomycetemcomitans. 2000;15:218– 225. They appear to contribute to the local generation of the proinflammatory molecules, bradykinin and thrombin, ultimately having an indirect effect on bone resorption (102).Human gingival fibroblasts incubated with T. [PubMed: 9226388]44.Golub L. Immunol. gingivalis, P. J., Levin L. Enzymatic and antigenic characterization of immunoglobulin A1 proteases from Bacteroides and Capnocytophaga spp. The focal infection theory resulted in unnecessary and unsupported tooth extractions. V. Subgingival plaque is either designated as tooth-associated or tissue-associated. 1997;35:455–461. W., Koch G., Dunford R., Zambon J. van Houte. Periodontal diseases: pathogenesis. 1987;40:443–452. 1993;28:219–226. Informational paper. [PubMed: 9495612]129.Sreenivasan P. R., Zambon J. gingivalis can adhere to many of the early plaque formers (80). Cloning of a Streptococcus sanguis adhesin which mediates binding to saliva-coated hydroxyapatite. T., Salinas C. Rationale of mechanical plaque control. It is believed that it must be a "mild" alteration in function, because there may be no apparent infections elsewhere in the body (102).Although bacteria are capable of directly causing destruction of the periodontal tissues, most of the destruction that occurs is the result of an indirect process whereby host cells are activated producing tissue-degradative substances. Oral Sci. Molecular pathogenesis of the cell surface proteins and lipids from Treponema denticola. 1990;58:920–929. G., Machtei E. On the host side, P. It is becoming clear that, while these assays do provide important information on specific pathogenic properties, they may not accurately mimic the in vivo environment. [PubMed: 8734698]83.Loesche W. [PubMed: 11018124]99.Page R. B., Davern L. Today, drugs are available that target groups of microorganisms. Thus, the tooth itself may serve as a reservoir for bacterial colonization.Although the primary cause for connective tissue destruction is the result of proteolytic activity of host cells, bacteria produce several enzymes that damage the extracellular matrix proteins, including collagenase.P. gingivalis produces numerous hydrolytic, proteolytic, and lipolytic enzymes (58). [PubMed: 1065840]90.Meyer D. 1985;48:534–539. A., Suzuki J. Re-interpretation of the evidence for X-linked dominant inheritance of juvenile periodontitis. Therefore, as stated above, mechanical debridement remains an important component of periodontal therapy with antimicrobial therapy serving as an adjunct to this therapy.Recently, new treatment strategies have been aimed at modulation of the host immunoinflammatory response. C., Gibson C. gingivalis does not stimulate E-selectin expression on endothelial cells and therefore hinders leukocyte extravasation (26). Socransky (126) proposed a modified series of criteria for microbial causation for periodontitis which included (i) association of the microorganism with periodontitis, (ii) demonstration that elimination of the bacteria reduced the disease, (iii) evidence of a host response to the pathogen, (iv) demonstration of ability of the pathogen to cause disease in an animal model, and (v) evidence that the pathogen produces virulence factors that contribute to the disease process (126). 1999. Relationship between conversion of localized juvenile periodontitissusceptible children from health to disease and Actinobacillus actinomycetemcomitans leukotoxin promoter structure. intermedia, P. [PMC free article: PMC96577] [PubMed: 10338476]88.Mayer M. Am. J. The α (classical)-defensins are found in the primary granules of neutrophils and are believed to produce their antimicrobial activity by forming channels in the bacterial or fungal membranes. actinomycetemcomitans. [PubMed: 8388449]110.Saxén L. [PubMed: 9002881]19.Clark W. B., Beem J. Biochemical composition of human saliva in relation to other mucosal fluids. Evidence of a substantial genetic basis for risk of adult periodontitis. intermedia was shown to invade a KB cell line. nucleatum, may provide the proteolytic activity needed for this conversion (68). G., Pirk F. Dis. D., Seachord C., Ratcliffe K., Bainbridge B., Aruffo A., Darveau R. Other agents, which block specific cytokines or inhibit PGE2, have also shown therapeutic potential and continue to be evaluated (70).Finally, because of the complex microbial picture in the pathogenesis of periodontal diseases, the development of a vaccine for treatment of periodontal disease will not occur readily. denticola outer sheath components, such as surface proteases, membrane lipids, and lipoproteins, may contribute to the induction of an inflammatory reaction in the periodontal tissues that can contribute to tissue damage (61).A. Microbial etiological agents of destructive periodontal diseases. Likelihood of transmitting Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis in families with periodontitis. F., McCray, Jr P. [PubMed: 1593411]57.Haubek D., Poulsen K., Westergaard J., Dahlen G., Kilian M. Host responses induced by co-infection with Porphyromonas gingivalis and Actinobacillus actinomycetemcomitans in a murine model. Recolonization of a subgingival microbiota following scaling in deep pockets. P., Baehni P. This pathogen also can agglutinate and lyse red blood cells (47) and induce membrane blebbing of epithelial cells (17). gingivalis produces a capsule that inhibits phagocytosis and decreases interactions with bacterial serum proteins (131); this resistance to phagocytosis varies between strains (20). S., Gibson C. Protein degradation by Prevotella intermedia and Actinomyces meyeri supports the growth of non-protein-cleaving oral bacteria in serum. 1996;1:821–878. [PubMed: 2027073]3.American Academy of Periodontology. Adherence of Peptostreptococcus micros morphotypes to epithelial cells in vitro. 1994;65:260–267. In addition, they are effective in conjunction with scaling and root planing in deep periodontal pockets that are nonresponsive (46, 82, 137). [PMC free article: PMC108622] [PubMed: 9746611]131.Sundqvist G., Figdor D., Hänström L., Sörlin S., Sandström G. Advantages of using systemic antibiotics in treatment of periodontal diseases include treatment of multiple sites and potential microbial reservoirs (tongue, tonsils, and buccal mucosa); treatment of organisms at the base of the pocket and in the tissue because of systemic absorption and delivery into oral tissues, gingival crevicular fluid, and saliva (137); availability of a variety of drugs and specific combinations from which to choose; and lower cost than locally delivered antimicrobials. J., Rosling B. N., Dale B. J., Tijhof C. [PubMed: 333085]126.Socransky S. Diehl, Gunsolley J. 1992;63:52–57. Cytokines such as IL-1, IL-6, and IL-8 are likely to be important in the destructive process (5). J., DiRienzo J. Assessment of risk for periodontal disease. Evidence is accumulating supporting the host response as a major determinant of disease susceptibility.Innate immunity plays a significant role in the response to microbial colonization seen in periodontal diseases. A. [PubMed: 5284178]12.Beaty T. Pediatr. 1999;34:136–144. Lectin-like receptors appear to be involved in coaggregation among streptococci, and lipoproteins and pili play a role in cell-cell interactions among other early colonizers. gingivalis LPS also is a poor activator of the release of tumor necrosis factor by mononuclear cells (22). nigrescens, Prevotella melaninogenica, and Prevotella loeschii) (63).In summary, many proposed virulence factors have been identified for specific periodontal pathogens. L., Holt S. 1989;159:256–262. intermedia, F. Recolonization of the subgingival microflora after scaling and root planing in human periodontitis. micros, and T. [PMC free article: PMC173817] [PubMed: 8550223]86.Magnusson I., Lindhe J., Yoneyama T., Liljenberg B. [PubMed: 2052893]132.Theilade E. These studies clearly demonstrate the multifactorial nature of periodontal diseases and the need for further research to better identify individuals at risk and for prescription of earlier and more definitive treatment.The treatment of periodontal disease has primarily relied on mechanical therapy: root debridement performed either with or without surgical access to reduce the overall plaque mass. [PMC free article: PMC260762] [PubMed: 2570751]20.Conrads G., Herrler A., Moonen I., Lampert F., Schnitzler N. E., Norderyd O. 1997;8:389–409. L., Schenkein H. [PubMed: 2213468]113.Schenkels L. For instance, H_2O_2 production by Streptococcus sanguis is lethal to A. forsythus (40). Host modulation as a therapeutic strategy in the treatment of periodontal disease. 1979;24:233–243. [PMC free article: PMC174269] [PubMed: 8751905]23.Darenfed J., Grenier D., Mayrand D. P., Guthmiller J. The cell cycle-specific growth-inhibitory factor produced by Actinobacillus actinomycetemcomitans is a cytolethal distending toxin. A., Slots J. [PubMed: 7667395]39.Genco R. J., Vitale L. [PubMed: 7054277]119.Slots J. gingivalis proteases (28).Invasive capabilities have been evaluated for other periodontal pathogens. A., Scheinkein H. [PubMed: 9211642]91.Meyer D. Science. Intrafamilial transmission of Actinobacillus actinomycetemcomitans. actinomycetemcomitans and the capsule of P. G., Genco R. C., Nakashima K., Schenkein H. B., Katz J., Eldridge J. While clinical and laboratory strains have the ability to invade epithelial cells, considerable variation occurs in the invasive potential among the various strains (109). Pharmacokinetic principles controlling efficacy of oral therapy. H., Michalek S. 1996;104:363– 371. [PubMed: 10522226]35.Fives-Taylor P., Meyer D., Mintz K. nucleatum, Capnocytophaga gingivalis, Capnocytophaga ochracea, Capnocytophaga sputigena, E. 1998;12:12–26. Although antimicrobials reduce the subgingival flora, their effectiveness against plaque as a part of a biofilm is not known. gingivalis render polymorphonuclear leukocytes inactive (102).One of the hallmarks of pathogenesis is the ability of the pathogenic microorganism to invade surrounding tissues, yet another mechanism to evade the host defense. [PubMed: 8707987]139.Wilson M., Henderson B. Chest. 1998;69:998–1007. Here, the bacteria may survive, replicate, and eventually be released back into the extracellular environment. 1976;9:65. 1999;20:168–238. 1997;24:346–353. Int. nucleatum may have a synergistic interaction with P. Relationship between some subgingival bacteria and periodontal pocket depth and gain or loss of periodontal attachment after treatment of adult periodontitis. [PubMed: 11128917]95.Moore W. [PubMed: 3540019]133.Tinoco E., Sivakumar M., Preus H. 1988;56:1150–1157. S., Hutcherson S. Periodontal disease is one of the most common diseases of the oral cavity and is the major cause of tooth loss in adults. 1995;66:23–29. C., Marazita M. C., McArthur W. 1998;66:4222–4228. E., Listgarten M. A summary of recent work. denticola are etiologic factors in periodontitis (40).Pathogenic bacteria must be able to (i) colonize the host, (ii) evade host defense mechanisms, and (iii) damage host tissues. denticola demonstrate a variety of pathologic responses, including cell detachment, reduced cell proliferation, and cell death (33). Actinobacillus actinomycetemcomtians with chest wall involvement and rib destruction. T.-J., Kinder Haake S., Park N.-H., Kuramitsu H., Genco R. In individuals in whom either the innate or acquired response is altered, the result is likely to be aggressive/progressive destruction. VII. 1977;48:497–504. FEMS Microbiol. W., Kramer A. [PubMed: 1573546]128.Socransky S. [PMC free article: PMC53231] [PubMed: 1688654]66.67.Kilian M., Thomsen B., Petersen T. C., Hammond B. P., Fives-Taylor P. Risk indicators for attachment loss. W., Shi W., Huang G. [PubMed: 3860517]124.Slots J., Listgarten M. nucleatum and potential contribution to tissue destruction during periodontitis. E., Wooten R., Cohen M. All these factors attempt to minimize the effect of the offending biofilm complex and may function synergistically.As plaque extends subgingivally, the flora becomes more complex and exists in a more protected environment. J., Papaioannou W., Quirynen M., van Steenbergen T. M. 1997;5:224–228. J., Chan A., Belton C. Judicious use of all antibiotics must be adhered to in light of this and increasing concerns of bacterial resistance. Effect of subgingival scaling on systemic antibody responses to oral microorganisms. [PubMed: 3466908]121.Slots J. 1983;54:707–711. Proc. Risk assessments in the diagnosis and treatment of periodontal diseases. [PubMed: 10791704]22.Cunningham M. [PMC free article: PMC108509] [PubMed: 9712771]76.Lally E. C., Moore L. [PubMed: 11154429]81.Listgarten M. forythus, P. Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis in periodontal disease: introduction. Y., di Giovine F. 1984;25:332–335. [PMC free article: PMC173152] [PubMed: 7534275]27.Dennison D. E., Tew J. However, studies from the 1930s to 1970s focused on the nonspecific plaque hypothesis which implicated the overall mass of the microbiota as the key factor in the initiation of tissue destruction rather than stressing the significance of specific bacterial species (132). 2000;15:341–349. [PMC free article: PMC97053] [PubMed: 10569761]24.Darveau R. [PubMed: 3793857]14.Bueno L. Pellicle receptors for Actinomyces viscosus type I fimbriae in vitro. N., Sandros J. B., Weitkamp L. J., Hausmann E. 1995;17:365–379. Models of invasion of enteric and periodontal pathogens into epithelial cells: a comparative analysis. Br. J. gingivalis involves a receptor-mediated endocytosis pathway (108). E., Slots J. Development of classification system for periodontal diseases and conditions. actinomycetemcomitans (serotype B) was shown to be protective for young individuals with generalized aggressive disease (15).For most people suffering from periodontal disease, it is hypothesized that the innate and acquired immune responses are protective resulting either in minimal periodontal destruction or an arrested disease state. Res. Chem. [PMC free article: PMC258118] [PubMed: 1879920]43.Giuliana G., Ammatuna P., Pizzo G., Capone F., D'Angelo M. Consequently, the host's protective nature may by countered by its destructive potential. micros adherence to epithelial cells has been shown to vary with morphologic characteristics of particular strains (74). 1994;65:848–854. Acquisition of plasmin activity by Fusobacterium nucleatum subsp. An autosomal-dominant form of juvenile periodontitis: its localization to chromosome 4 and linkage to dentinogenesis imperfecta and Gc. J. [PubMed: 10863370]7.Asikainen S., Chen C. P., Tsai C. [PubMed: 379300]107.Saarela M., Lippmann J. 1994;62:3033–3037. D., Zasloff M. nucleatum.The invasive ability of A. Porphyromonas gingivalis invades oral epithelial cells in vitro. micros, and S. [PubMed: 3629258]50.Grossi S. In this same study, B. 1996;23:263–267. 1996;64:3601–3608. Attempts to apply Koch's postulates to specific bacteria have been hampered because these pathogens often cannot be grown in pure culture (e.g., large spirochetes), they may have long incubation times, they can occur in an asymptomatic carrier state, and they may exhibit a limited host range (121). 2001;25:37–58. Finally, antimicrobials should not be used as a replacement for good mechanical therapy, but rather should be used in conjunction with mechanical therapy to optimize the therapeutic effectiveness of each. Hum. R., Debski B. Even in the presence of such immune responses, the bacterial challenge may be too severe to overcome. F., Taichman N. [PMC free article: PMC175572] [PubMed: 9316996]85.Madianos P. [PubMed: 10522220]122.Slots, J., and C. Increased prevalence of several genera of proposed importance in the development of periodontitis may be seen as the subgingival plaque matures. nucleatum, T. Third, antibody formation is specific for specific periodontal organisms, i.e., A. T. 1995;63:1311–1317. However, certain bacteria, such as Fusobacterium nucleatum, serve as important bridges between these noncoaggregating, early colonizing bacteria and the late colonizers (68).As the biofilm begins to mature, there is a progressive shift from a gram-positive, facultative flora to one predominated by gram-negative, anaerobic species. H., Lippmann J. G., Higginbottom F. 1992;63:169–173. In vitro, β-defensins exhibit broad-spectrum antimicrobial activity against gram-positive and gram-negative bacteria and fungi (38). 1996;11:274–281. D., Bailey T., Seachord C., Ratcliffe K., Bainbridge B., Dietsch M., Page R. These include genetic factors, age, gender, socioeconomic status, stress, human immunodeficiency virus infection/acquired immunodeficiency syndrome, osteoporosis, infrequent dental visits, previous history of periodontal disease, and bleeding on probing.Until recently, aggressive periodontal diseases were unique in their risk associated with genetic predisposition. corrodens were shown to be noninvasive.In addition to invasion of the surrounding soft tissues, a recent study demonstrated the invasion of P. S., McLaughlin R. 1995;66:191–205. Expression of the peptide antibiotic human β-defensin 1 in cultured gingival epithelial cells and gingival tissue. 1996;11:387–394. When performed as a part of routine periodontal maintenance, periodontal pathogens are suppressed to a level where an equilibrium is established between the host and the pathogen, which does not result in progressive loss of attachment in most patients (86, 112). (Vertical and/or horizontal transmission has been shown both for A. Delineation of a segment of adsorbed salivary acidic proline-rich proteins which promotes adhesion of Streptococcus gordonii to apatitic surfaces. And Eikenella corrodens demonstrates an aggregating factor, which is believed to play an important role in the accumulation of plaque (32).Periodontal pathogens use a variety of means to interfere with host defense mechanisms, thereby prolonging their presence in the periodontal pocket. [PMC free article: PMC414468] [PubMed: 113347]135.Tuite-McDonnell M., Griffen A. Microbial complexes in subgingival plaque. [PubMed: 10328661]6.Armitage G. Criteria for the infectious agents in dental caries and periodontal disease. nucleatum and P. E., Ho A. Tannock (ed.), Medical Importance of the Normal Microflora. A., Lamont R. Antimicrobial defensin peptides form voltage-dependent ion-permeable channels in planar lipid bilayer membranes. [PMC free article: PMC302918] [PubMed: 8005694]34.Fives-Taylor P. gingivalis and a high molecular weight carbohydrate on A. Lett. Efficacy of clindamycin hydrochloride in refractory periodontitis. Actinobacillus actinomycetemcomitans apaH is implicated in invasion of epithelial cells. The designated "red" complex (Treponema denticola, Porphyromonas gingivalis, and Bacteroides forsythus) and the "orange" complex (Prevotella intermedia, Prevotella nigrescens, Peptostreptococcus micros, F. Additional evidence has shown that P. V., Burmeister J. Mechanisms for each of these required steps for pathogenesis have been identified for many of the periodontal pathogens. These interactions are mediated by P. Protease inhibitors can inhibit invasion, suggesting a role for P. [PMC free article: PMC261367] [PubMed: 3886548]32.Ebisu S., Nakae H., Fukuhara H., Okada H. Consensus report: periodontal diseases; pathogenesis and microbial factors. 1996;64:3908–3910. nucleatum. G., Zambon J. [PubMed: 10202019]118.Shenker B. However, the crevicular fluid bathing the gingival sulcus or pocket contains many factors capable of resisting bacterial progression such as lysozyme, bradykinin, thrombin, fibrinogen, complement, antibodies, and neutrophil-derived components (24).Recent investigations have looked toward defensins (innate immune peptides with antimicrobial properties) and their role in periodontal and other oral infections. J., McKay T., Datar S., Miller M., Chowhan R., Demuth D. 1985;12:540–552. Local chemokine paralysis, a novel pathogenic mechanism for Porphyromonas gingivalis. Position paper. M., Izutsu K. [PubMed: 1812467]48.Grenier D., Mayrand D., McBride B. intermedius in radicular dentin (43). 1989;4:12–18. For example, it has been suggested that F. Bacteroides gingivalis, Bacteroides intermedius and Actinobacillus actinomycetemcomitans in human periodontal diseases. USA. Concordance of Porphyromonas gingivalis colonization in families. Although the specific antimicrobial mechanism for the β-defensins is not known, they are believed to act similarly to the α-defensins.The neutrophil response is the host's first innate cellular response and probably the most significant in limiting the subgingival biofilm accumulation (24). Prevalence in patient groups and distribution of biotypes and serotypes within families. Structural features of salivary function. 1984;29:59–63. H., Mintz K. As a result of the polymicrobial infection, the bacteria act in concert supporting nutrition and aggregation factors necessary for the biofilm development. The conversion of the cell-bound plasminogen to plasmin may allow F. P., Cunningham M. 1995;267:1645– 1648. [PubMed: 2921370]82.Loesche W. E., Fives-Taylor P. W. 1989;57:1465–1469. S., Newman M. 1990;87:210–214. Periodontosis: a phenotypic and genetic analysis. Chen. [PubMed: 10585538]62.Jansen H. [PubMed: 9847886]101.Pihlstrom B. 1986;13:905–911. Rheumatol. Future treatment considerations should include reservoirs for the infection (i.e., buccal mucosa and tongue) and acquisition of the pathogens from family members (52, 135, 141). H., Sreenivasan P. 1993;61:1239–1245. While the space limitations of the subgingival environment (pocket) attempt to limit the expansion of the subgingival microbial complex, the apical migration of the epithelium and destruction of the attachment apparatus adjacent to the tooth (deeper periodontal pockets) allows for expansion of the subgingival biomass. The purpose of this section is not to provide an exhaustive evaluation of each of these mechanisms. 1996;10:45–78. Delineation of unique features and comparison to homologous toxins. Genet. L., Taubman M. A synergistic effect is also seen with A. Subgingival microflora and periodontal disease. Nonsurgical treatment of patients with periodontal disease. Chronic periodontitis is the result of a polymicrobial infection with variable microbial patterns. For example, a single clinical isolate of P. The interleukin-1 genotype as a severity factor in adult periodontal disease. C., Beck J. Meanwhile, twin studies demonstrate that chronic periodontitis has a 50% heritable component (94). Unlike other infections whereby treatment correlates with elimination of the offending pathogen, the successful treatment of this polymicrobial infection occurs not by elimination of pathogens but by an alteration where a level of symbioses or homeostasis can occur between the offending bacteria and the defensive host.Periodontal diseases result from a polymicrobial infection of the subgingival crevice. Of equal importance is the necessity for good plaque control performed by the patient. 1997;14:12–32. 1989;264:15451–15456. M., Lee H. Oral Microbiol. 1992;27:615–622. 1976;42:32–41. Ribotyping shows intrafamilial similarity in Actinobacillus actinomycetemcomitans isolates. 1996;23:346–354. These include inhibition of polymorphonuclear chemotaxis, production of immunosuppressive factors, secretion of proteases, which cleave immunoglobulin G (IgG), and production of Fc binding proteins (139). 1997;24:478–485. The periodontal diseases are a diverse group of clinical entities in which induction of an inflammatory process results in destruction of the attachment apparatus, loss of supporting alveolar bone, and, if untreated, tooth loss. 1971;42:516–520. 1999;67:6439–6444. This sequential deposition begins with the adherence of early colonizers, streptococci and actinomycetes spp., to host-derived glycoproteins, mucins, and other proteins coating the tooth surface (41). 1996;67:935–945. T., Kieba I. N., Koertge T. J., Burmeister J. J., McArthur W. Immun. The bone-resorbing activities in tissue culture of lipopolysaccharides from the bacteria Actinobacillus actinomycetemcomitans, Bacteroides gingivalis, and Capnocytophaga ochracea, isolated from human mouths. Role of bacterial proteinases in matrix destruction and modulation of host responses. [PubMed: 7891246]52.Gunsolley J. actinomycetemcomitans, P. [PubMed: 9972117]45.Goodson J. Trends Microbiol. 1979;6:351–382. Clin. G., Reinholdt J., Kilian M. 1998;66:1660–1665. 1997;24:72–77. Other proteinases produced by P. In contrast, the mixed microbial milieu may be beneficial in suppressing growth of various species, inhibiting expression of various virulence factors or neutralizing virulence factors. [PubMed: 6936406]111.Saxén L., Nevanlinna H. gingivalis and Actinomyces naeslundii is mediated by fimbrillin and a 40-kDa membrane protein of P. gingivalis also can replicate and persist within KB cells (85). 1989;57:3003–3008. Effects on human peripheral blood lymphocyte responses to mitogens and antigens. [PubMed: 9448791]100.Papapanou P. V., Payne J., Sorsa T. In addition, in the process of mechanical root debridement, specific subgingival pathogenic species are inadvertently removed or reduced to levels which result in improved clinical health and/or stabilization in periodontal maintenance patients (81, 103, 123).Scaling and root planing (debridement) have routinely been shown to be effective in treatment of chronic periodontitis without the concomitant use of systemic or local antimicrobials (96). K., Meyer D. Oral Pathol. Oral Biol. M., Reife R. T., Golub E. [PMC free article: PMC174179] [PubMed: 8757825]92.Meyer D. The type C fimbriae and a cytoskeletal rearrangement were required for this invasion. Natl. Several primary players in the disease process have been identified and their virulence factors wellcharacterized. Biomaterial-centered infection: microbial adhesion versus tissue integration. 2000;24:153–192. Primary and secondary receptors for uptake may be the transferrin receptor and integrins, respectively (92, 107). [PubMed: 9673164]55.Han T. 1999;14:49–55. Most patients suffer from chronic periodontitis, an insidious disease in which the destruction is consistent with the presence of bacterial plaque and mineralized plaque or calculus (Color Plate 1 [see color insert]). 1999;162:4773–4780. Kluwer Academic Publishers, London, United Kingdom.123.Slots J., Emrich L. [PMC free article: PMC258078] [PubMed: 1855989]94.Michalowicz B. T., Korostoff J. L., Selsted M. nodatum, Campylobacter rectus, P. 1993;4:251–259. Both complexes could be associated with clinical parameters of disease supporting the polymicrobial nature of periodontitis.Although it is clear that periodontal disease is a polymicrobial infection, there has historically been an interest in identifying specific microorganisms that contribute to the disease process. As the biofilm develops, there are areas of high and low bacterial biomass interlaced with aqueous channels which will provide for movement of essential nutrients (derived primarily from the gingival crevicular fluid) for the growth of the organisms and removal of metabolic waste products (24). Recently, there has been increasing interest in the relationship of periodontal disease to important systemic diseases, such as cardiovascular disease and complications in pregnancy (97).Historically, the etiology of periodontal diseases has focused on bacterial plaque, microbial by-products, and the host immune response. E., Califano J. The individual or combined actions of these inflammatory molecules can result in significant tissue destruction.Bacterial plaque is the primary etiologic factor associated with periodontitis, yet there are several other variables that may place an individual at risk for developing disease (3, 99, 100, 101). 1999Evaluation of Periostat for patient management Compend. E., Fuerst P. [PubMed: 10384401]21.Contreras A., Slots J. gingivalis fimbriae and may be facilitated by proteolytic enzymes (80).T. E., Cisar J. These channels increase membrane permeability in a charge- or voltage-dependent manner, ultimately resulting in cell lysis or cell death (65).The cationic β-defensins produced by epithelial cells represent a local defense mechanism in contrast to the more systemic response seen with the neutrophil-derived α-defensins. L., Roulston D., Schwartz S., Suzuki J. [PMC free article: PMC97547] [PubMed: 10816455]56.Hart T. A direct relationship exists between periodontal disease and the prevalence of smoking, and the prevalence and severity of periodontitis is significantly higher in patients with type I and type II diabetes. actinomycetemcomitans LPS and acid and alkaline phosphatases induce bone resorption (60), and collagenolytic activity has been observed in both media and cell sonicates of the organism (105). actinomycetemcomitans (previously described as the immunosuppressive factor) (115, 117, 118) induces cell cycle arrest in lymphocytes. gingivalis, A. Requirements for invasion of epithelial cells by Actinobacillus actinomycetemcomitans. For example, recent data imply that strains of A. The case for periodontosis as a clinical entity. In certain populations, the protective role of antibodies has been demonstrated. 1980;7:276–288. forsythus. [PMC free article: PMC173546] [PubMed: 7558295]80.Lamont R. 1979;6(Extra Issue):16–19. [PubMed: 1582318]141.Zambon J. gingivalis where a coinfection with these organisms showed enhanced induction of the humoral and cell-mediated response (18). gingivalis produces proteinases that cleave IgA1, IgA2, and IgG (36, 66, 67), including hydrolysis of immunoglobulin already bound to the bacterial surface (48). [PubMed: 8845187]140.Yuan A. Invasion of epithelial cells by P. rectus, oral spirochetes, and is specific for certain virulence factors such as the leukotoxin and other components of A. Actinobacillus actinomycetemcomitans immunosuppressive protein is a member of the family of cytolethal distending toxins capable of causing a G2 arrest in human T cells. nucleatum subspecies, Eubacterium nodatum, Streptococcus constellatus, and three Campylobacter species) were generally found together, and evidence showed that colonization by the red complex was preceded by colonization by orange complex species. intermedia/nigrescens, P. actinomycetemcomitans and P. Rather, they are present as a biofilm—a "community of microorganisms attached to a surface" (98). 1994;5:66–77. Actinobacillus actinomycetemcomitans in human periodontal disease. [PubMed: 9706864]5.American Academy of Periodontology. K., Tack B. [PMC free article: PMC414288] [PubMed: 222679]11.Baer P. P. F., Walsh L., Schork M. R. [PubMed: 10522227]59.Hughes R. [PubMed: 11155181]102.Potempa J., Banbula A., Travis J. 1996;34:1576–1578. 1987;55:631–638. [PubMed: 3908641]47.Grenier D. 1996;67:282–290. denticola, Treponema medium, and B. In addition, it has been proposed that T. Three β-defensins, HBD-1, HBD-2, and HBD-3, are expressed in the gingival epithelial tissue, which makes them excellent candidate peptides contributing to the defenses at mucosal surfaces, including the periodontium (75, 87). Ther. actinomycetemcomitans, and B. 1999;67:2740–2745. G., Moore D. I., Hariharan G., Lally E. 2000. [PubMed: 9049801]72.Kornman K. The supragingival (above the gingival surface) and subgingival (beneath the gingival surface) habitats differ in terms of pH, redox potential, and nutrient availability. Further studies on the degradation of immunoglobulins by black-pigmented Bacteriodes. M., Neyer D. [PubMed: 8930584]109.Sandros J., Papapanou P., Dahlen G. B., Lamster I., West T., Socransky S., Seiger M., Fasciano R. II. The microbial challenge in periodontitis. C., Mayer M. T., Kornman K. 1993;9:55–62. Identification of a cytolethal distending toxin gene locus and features of a virulence-associated region in Actinobacillus actinomycetemcomitans. IL-1 promotes bone resorption, stimulates release of the eicosanoid prostaglandin E2 (PGE2) by monocytes and fibroblasts, and stimulates the release of matrix metalloproteinases (MMPs) important in degradation of the extracellular matrix. actinomycetemcomitans adheres to the tooth or epithelium via surface proteins, microvesicles, and fimbrae (139). Analysis of the Actinobacillus actinomycetemcomitans leukotoxin gene. J., van der Hoeven J. Chemotherapy of dental plaque infections. Biofilm formation as microbial development. forsythus, Campylobacter curvus, and E. The localized form of aggressive periodontitis is an unusually unique disease relative to other forms of periodontitis in that it usually occurs during adolescence (a group traditionally exhibiting a low incidence of periodontal disease); the subgingival microbiota demonstrate an unusually high association (96.5%) with a single bacterium, Actinobacillus actinomycetemcomitans (141); bone resorption progresses at a rate three to four times faster than that observed for chronic periodontitis (106), may spontaneously arrest (11), and is localized to very specific teeth (first molars and incisors); finally, the disease tends to cluster in families suggesting that predisposition to the disease may be genetically regulated (12, 13, 56, 64, 89, 110, 111).More than 500 different bacterial species have been estimated to reside in the subgingival plaque (95). [PubMed: 7990021]73.Kraig E., Dailey T., Kolodrubetz D. The result is the initiation and progression of periodontitis. The pathogenesis of periodontal diseases. Utility of 5 major putative periodontal pathogens and selected clinical parameters to predict periodontal breakdown in patients on maintenance care. A., Sullivan P., George C., Nitkin L., Rosenberg E. I. These genera include, but are not limited to, Treponema, Bacteroides, Porphyromonas, Prevotella, Capnocytophaga, Peptostreptococcus, Fusobacterium, Actinobacillus, and Eikenella.Certain periodontal bacteria are often found together in subgingival plaque samples. Oral microbial communities: biofilms, interactions and genetic systems. gingivalis can also interact with later colonizers such as F. 1994;5:112–141. While the data are not entirely clear that periodontal diseases play a strong role in the development of other diseases, the systemic communications of this polymicrobial infection need to be further explored.1.Alaluusua S., Saarela M., Jousimies-Somer H., Asikainen S. 1999;20:7–13. For example, bacterial derived proteinases destroy tissue providing polypeptides utilized for growth by other organisms (62).Although primarily a subgingival microorganism, P. [PMC free article: PMC3523333] [PubMed: 9776028]15.Califano J. Arch. Periodontal risk assessment, diagnosis and treatment planning. 1990;58:3856–3862. J., Giordano J., Soehren S., Hutchinson R., Rau C. Periodontal Res. Following uptake, A. 1989;68:1625–1632.46.Gordon J. In addition, salivary and masticatory influences that have an impact on the supragingival microflora do not have the same influence on subgingival bacteria (7). Characteristics of hemolytic and hemagglutinating activities of Treponema denticola. Cloning and expression of the leukotoxin gene from Actinobacillus actinomycetemcomitans. S., Stolzenberg E. A., Yang P., Astemborski J. A., Phillips R. For the first time, evidence further delineates the role of virulent bacteria to include certain pathogenic strains or clones of bacterial species in the initiation and progression of particular periodontal diseases (53).As previously stated, more than 500 different bacterial species can be found in the oral cavity (95). Porphyromonas gingivalis infection of oral epithelium inhibits neutrophil transepithelial migration. P., Belton C. 2001;12:116–124. [PMC free article: PMC281353] [PubMed: 8454326]130.Sugai M., Kawamoto T., Peres S. Dent. 1996;67(Suppl.):1041–1049. The microorganisms colonizing these surfaces are not present in a free-floating planktonic state. M., Walker C. D., Bixler D. Two of these variables are clearly defined risk factors: tobacco smoking and diabetes. actinomycetemcomitans subsequently escapes from the vacuole, replicates rapidly in the cytoplasm, and is transmitted to adjacent cells through bacteria-induced protrusions of the host cell membrane. [PMC free article: PMC96451] [PubMed: 10024565]89.Melnick M., Shields E. 1999;20:65–81. Systemic antibiotic therapy in periodontics. The CDT of A. [PubMed: 9567966]28.29.30.Ebersole J. In recent years, the connection of periodontal diseases with systemic diseases has been revisited and studies suggest that periodontal diseases may be a risk factor in preterm low birth weight deliveries, cardiovascular disease, diabetes, respiratory diseases, and other diseases (4). Among the proteolytic enzymes are two cysteine proteases (gingipains). C., Kesavalu L., Walker S., Genco C. [PMC free article: PMC258561] [PubMed: 2318535]74.Kremer B. J. This cytotoxic protein specifically kills a subset of leukocytes in vitro that includes polymorphonuclear leukocytes and peripheral blood monocytes (10, 134). [PubMed: 11155157]18.Chen P. Meanwhile, our understanding of the pathogenic process has been hindered by the fact that it is usually the result of a polymicrobial infection including indigenous organisms with little pathogenic potential.There are two main categories of periodontal disease in which loss of supporting structures around the tooth occurs: chronic periodontitis and aggressive periodontitis (6). [PubMed: 1313103]137.Van Winkelhoff A. A., Herscheid A. Biochem. Rather, examples of pathogenic processes used by selected periodontal pathogens are presented with appropriate review articles listed for further reference.As discussed in the section on development of the biofilm, adhesion is a necessary element in the colonization of subgingival bacteria, either directly to the periodontal tissues or through the association with other organisms by coaggregation and coadhesion. These factors include lysozyme, lactoferrin, peroxidases, antimicrobial peptides, histatins, defensins, and cathelicidins (78, 113). 1991;59:2948– 2954. 1999;70:457–470. Collagenolytic activity associated with Bacteriodes species and Actinobacillus actinomycetemcomitans. The influence of supragingival plaque control on clinical and microbial outcomes following the use of antibiotics for the treatment of periodontitis. J., Christersson L. Oral Med. However, a vast number of pathogens potentially exist that have not been identified or characterized. 1987;237:1588–1595. A., Diehl S. The biofilm continues to develop as late colonizers, such as veillonellae, prevotellae, propionibacteria, and certain streptococci, begin to colonize the tooth surface (98). [PubMed: 9567964]25.Darveau R. 1991;99:117–129. Educ. S., Chilton N. Actinobacillus actinomycetemcomitans in families afflicted with periodontitis. [PubMed: 7504235]2.Alaluusua S., Asikainen S., Lai C. 101–127. Interaction of inflammatory cells and oral microorganisms. [PubMed: 3279073]125.Socransky S. B., Kuo S. Bacteria are the primary etiologic factor of periodontal diseases, however, recent evidence also lists yeast and herpesviruses as putative pathogens (21, 122). Local delivery results in a substantially higher drug concentration in the pocket than the equivalent systemic agent.Local delivery of any antimicrobial is not, however, a substitute for systemic antibiotics when indicated in specific periodontal diseases. [PubMed: 10650360]17.Chan E. R., Wenk R. actinomycetemcomitans was recently shown to be inhibited by other subgingival inhabitants (P. W., Wilson, Jr T. As reviewed by Dennison and Van Dyke (27), the pivotal role of these cells in protection from periodontal diseases has been proven, because individuals with a decreased number or function demonstrate a marked increase in susceptibility to rapid and severe periodontal destruction.The importance and correlation of adaptive/acquired immunity in periodontal diseases is proven by several factors. 2000;35:3–16. Antibiotic selection when choosing local delivery is empirical; therefore, in cases where one needs to know the specific bacteria and antimicrobial susceptibility (i.e., aggressive or nonresponsive disease), microbiologic testing and antibiotic susceptibility are recommended.Periodontitis is a mixed infection. Rev. [PubMed: 2647082]78.Lamkin M. What is realized, however, is that given the right combination of bacteria, indigenous colonizers may become opportunistic pathogens.Currently, our therapy remains primarily directed toward controlling the bacterial etiology at the site of infection. Although the nature of these interactions in biofilm development is currently being investigated in in vivo model systems, many of the previous studies were based on in vitro assays on either planktonic cells or in vitro-generated biofilms. The acute inflammatory response and the role of phagocytic cells in periodontal health an disease. Yang, Lee L., Chang D. Porphyromonas gingivalis FDC381 multiplies and persists within human oral epithelial cells in vitro. Pharmacol. [PubMed: 2628862]49.Gristina A. [PubMed: 8739166]104.Ranney R. Most strains adhere well to extracellular and basement membrane proteins, such as fibronectin and laminin (17). 1985;56(Suppl):75–80. gingivalis (30). A., Smith C., Kent, Jr R. J., and J. Cellular events concurrent with Porphyromonas gingivalis invasion of oral epithelium in vitro. Microbiological and clinical results of metronidazole plus amoxicillin therapy in Actinobacillus actinomycetemcomitans-associated periodontitis. [PubMed: 2231231]53.Guthmiller J. Acad. This shift is associated with the development of the biofilm beneath the gingival surface. [PubMed: 9567937]138.Westfelt E. To support this, periodontal bacteria have been cultured from other target organs and prosthetic joints (49, 140). One of the best-studied virulence factors is the leukotoxin produced by A. 1986;13:912–917. In G. The bacterial etiology of destructive periodontal disease: current concepts. Bacterial adherence and the formation of dental plaques, p. [PubMed: 29539847] actinomycetemcomitans associated with localized aggressive periodontitis differ from strains of the bacterium found in other forms of periodontal disease or in health (14, 57, 142). The mechanisms of Eikenella corrodens aggregation by salivary glycoprotein and the effect of the glycoprotein on oral bacterial aggregation. The microbiology of early-onset periodontitis: association of highly toxic Actinobacillus actinomycetemcomitans strains with localized juvenile periodontitis. Chapman & Hall, London, United Kingdom.42.Gibbons R. W., Koch G., Dunford R. Med. [PubMed: 1460548]33.Ellen R., Song M., McCullock C. J., Jenkinson H. M., Lally E. Focal infection revisited. D., Socransky S. 2000;68:3140–3146. J., Haraszthy V. L., Moeschberger M. S., Brooks C. E., Meyer D. 1998;69:841–850. L., Dalton R. [PubMed: 1054138]65.Kagan B. Heredity of juvenile periodontitis. [PubMed: 9673163]96.Mousques T., Listgarten M. 1996;64:660–664. J., Haffajee A. gingivalis, that invade epithelium may not be as susceptible to standard mechanical debridement and require supplemental antibiotics and/or surgery for eradication (109, 129). 1981;3:89–100.105.Robertson P. K., Fives-Taylor P. A., Bueno L. 1979;50:311–315. The sites colonized by these different microorganisms are diverse, ranging from the nonshedding tooth surface to the continually shedding epithelium covering the mucosal surfaces. 1999;4:1–6. C., Ranney R. R., Demuth D. [PubMed: 8884652]4.American Academy of Periodontology. Comparative longitudinal study of 2 methods of scheduling maintenance visits: 4-year data. Periodontal disease as a potential risk factor for systemic diseases. S., Haffajee A. Interactions between periodontal bacteria and human oral epithelial cells: Fusobacterium nucleatum adheres to and invades epithelial cells. Detection of leukotoxic activity of a plaque-derived gram-negative microorganisms. R., Taichman N. D., Cugini M. Dev. Benefits of mechanical debridement include: removal of calculus and endotoxin, disruption of the plaque biofilm complex, induction of potentially protective antibody responses to certain pathogens (31), and increased numbers of beneficial bacteria, such as streptococci (9). In addition, periodontal pathogens, such as A. R., Rosenbloom J., Golub E. J., Machtei E. N., Nannmark U., Dahlén G., Sandros J. [PubMed: 9467371]9.Axelsson P., Lindhe J. Periodontol. 1989;16:105–115. Effect of scaling and root planing on the composition of human subgingival microflora. 1990;61:643–648. N., Papapanou P. Ability of bacteria associated with chronic inflammatory disease to stimulate E-selectin expression and promote neutrophil adhesion. Epithelial antibiotics induced at sites of inflammation. gingivalis, which may be an important factor in colonization and development of the plaque biofilm (61).Other important periodontal pathogens are also known for their ability to aggregate and/or adhere. 1996;81:533–543. H., Fives-Taylor P. Microbiology of periodontal disease: present status and future considerations. G. actinomycetemcomitans is taken up in a host-derived membrane-bound vacuole by an active process involving signaling between the bacterium and KB cell microvilli (129). The leukotoxin gene was cloned and sequenced (69, 73, 76, 77) and, on the basis of sequence homology, was found to be a member of the RTX (repeats in toxin) family of pore-forming bacterial toxins including toxins from such other species as Pasteurella haemolytica and Actinobacillus pleuropneumoniae, producing devastating infections in cattle and swine, respectively. The lipopolysaccharide (LPS) produced by P. There are indications that these proteases are involved in several functions including adherence to host cells, inhibition of host defenses, and damage to host cells. Cont. actinomycetemcomitans also produces a factor that inhibits fibroblast proliferation in cell cultures and their production of substances of the extracellular matrix, thus modulating tissue turnover (116).In addition to the independent virulence mechanisms by individual species and strains, complex, interdependent interactions also exist that occur among the different genera in the plaque biofilm, which can affect their pathogenic potential. Bacterial life in a biofilm environment can be very different from the planktonic state. C., Aruffo A. A., Scheinken H. The members of the RTX family all share a common gene organization, and the toxins contain tandemly repeated nonapeptides that have the consensus sequence GGXGXDX(L/I/V/W/Y/F)X.A. actinomycetemcomitans elaborates many other factors that may allow the organism to evade detection/destruction by the host's immune system. However, neither a different clinical isolate nor the type strain was able to invade the cell line (29). 1991;59:2719–2726. Subgingival colonization by Porphyromonas gingivalis. For example, adherence between P. Autosomal recessive inheritance of juvenile periodontitis: a test of a hypothesis. [PMC free article: PMC229068] [PubMed: 8735124]58.Holt S. Identification and expression of the Actinobacillus actinomycetemcomitans leukotoxin gene. F., Tew J. B. Pathogenesis of gingivitis and periodontal disease in children and young adults. corrodens, C. actinomycetemcomitans may not only evade host defenses but also gain access to underlying periodontal tissues.As seen with A. C., Veerman E. The efficiency of invasion varies among the clinical and laboratory isolates examined. 1992;101:1450–1452. Virulence factors of Actinobacillus actinomycetemcomitans relevant to the pathogenesis of inflammatory periodontal diseases. This suggests that different strains of the same species may demonstrate varying pathogenicity. Tetracyclines inhibit connective tissue breakdown by multiple non-antimicrobial mechanisms. [PubMed: 10194070]71.Kornman K. While specific microbial species are believed to play a role in the disease process, periodontal therapy today remains targeted toward removal of the plaque mass as opposed to elimination of specific pathogens. 1980. Salivary components no longer have access to the bacteria colonizing the subgingival environment. Oral ecology and person-to-person transmission of Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis. Development of dental plaque and subsequent tissue destruction, however, rely on complex interactions among these bacteria in the biofilm environment. I., Schlesinger D. Current view of risk factors for periodontal diseases. The diseases can be further characterized by the extent of bone loss (localized or generalized) and the severity of the disease (slight, moderate, or advanced). Crit. The significance of maintenance care in the treatment of periodontal disease. These salivary proteins are deposited within minutes on a clean tooth surface and are called the "acquired pellicle." Bacterial surface structures, such as pili and outer membrane proteins, as well as proteins and enzymes in the acquired pellicle, are important mediators of this initial attachment (19, 37, 42).Continued development of the plaque biofilm relies on physical interaction of bacteria of the same or different genera through coaggregation and coadhesion (68). 1998;25:134–144. 12 month results. More studies are currently underway to define the role of this gene in other forms of periodontal disease and understand the significance it may have in performing and prescribing periodontal treatment. C. These interactions promote P. gingivalis also can bind to epithelial cells, fibroblasts, and erythrocytes, and to components of the extracellular matrix. S., Oppenheim F. J., Hay D. This theory was subsequently challenged when different bacterial species were seen to play a pivotal role in the initiation and progression of periodontal disease, which supported the specific plaque hypothesis (83, 104, 119, 120, 124, 125).A persistent concern with the specific plaque hypothesis is the fact that all of the potentially pathogenic organisms can be isolated from healthy as well as diseased subjects (54). gingivalis [1, 2, 8, 133].) In addition, future research and treatment efforts will certainly continue in modulation of the host response and genetic heritability of periodontal diseases.In the 19th and early 20th centuries, periodontal disease was believed to be a focus of oral sepsis serving as a seed of infection for inflammatory systemic diseases (59). [PubMed: 6362631]61.Ishihara K., Okuda K. All of these factors may have an impact on the effectiveness of antimicrobial therapy. Cluster analysis and community ordination techniques were used to further define these relationships and to determine whether there were correlations between certain clusters and clinical parameters of disease (128). Many of these bacteria would not usually interact with each other in a way that results in aggregation. Craniofac. Highly toxic clone of Actinobacillus actinomycetemcomitans in geographically widespread cases of juvenile periodontitis in adolescents of African origin. actinomycetemcomitans has been demonstrated in vitro using a human epidermoid carcinoma cell line (KB) that is of oral origin (34, 35). E., Giannobile W. gingivalis. [PubMed: 6358452]142.Zambon J. denticola attaches to human gingival fibroblasts, possibly through a lectin-mediated mechanism. 1983;409:612–624. Periodontosis in sibs. [PMC free article: PMC260386] [PubMed: 3546133]37.Ganeshkumar N., Song M., McBride B. Currently, evidence of host cell invasion exists for A. J., Rams T. 1988;15:85– 93. B., Kolter R. Extraction and partial characterization of a leukotoxin from a plaque-derived gram-negative microorganism. E., Ganz T., Lehrer R. T., Vasel D., Weinberg A. [PubMed: 11276866]103.Rams T. 1982;17:275–283. Molecular biological techniques have enabled us to examine associations between specific strains of a bacterial species with distinct diseases. 1991;6:246–249. 1997;14:54–78. 1998;66:5008–5019. T., Demuth D. [PubMed: 9178115]63.Johansson A., Hanstrom L., Kalfas S. An example of this is aggressive periodontitis where numerous studies have demonstrated abnormalities in neutrophil function. While the communities found on soft tissues often comprise a single microbial species, the most prevalent oral biofilm, dental plaque, exists as a complex multispecies entity attached to the tooth surface.Within this multispecies biofilm are grampositive, gram-negative, aerobic, facultative, and anaerobic microorganisms that are deposited on the tooth surface in a sequential fashion. denticola coaggregates with F. Characteristics of Actinobacillus actinomycetemcomitans invasion of and adhesion to cultured epithelial cells. IL-8, a chemoattractant for neutrophils, selectively stimulates MMP activity from these cells. First, there is an elevation in the cell-mediated (both CD4+ and CD8+ cellular response) or humoral response (a composite of antibodies) with the presence of periodontitis and increasing severity of periodontitis. actinomycetemcomitans (127), and the leukotoxicity of A. nucleatum binds plasminogen that can be converted by protease activity to plasmin (23). [PMC free article: PMC259776] [PubMed: 3356463]38.Ganz T., Lehrer R. Second, both systemic and local antibody formation have been demonstrated following mechanical periodontal therapy most likely as a result of a treatment-induced bacteremia. [PMC free article: PMC174312] [PubMed: 8751948]16.Caton J. N. Production of β-defensin antimicrobial peptides by the oral mucosa and salivary glands. Immune suppression induced by Actinobacillus actinomycetemcomitans: effects on immunoglobulin production by human B cells. [PubMed: 6347000]68.Kolenbrander P. Bacterial specificity in adult periodontitis. actinomycetemcomitans, invasion of primary cultures of gingival epithelial cells by P. P., Hammond B. Results of these studies demonstrated that the bacteria could be sorted into five major groups that were given color designations. Following this approach, three bacteria have been recognized as causative agents of periodontitis: P. 1997;47:61–87. [PubMed: 6125580]106.Ruben M. However, broad-spectrum antibiotics are still widely used and have the disadvantage of killing beneficial bacteria as well. [PubMed: 8373982]79.Lamont R.

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