Actinomyces

Actinomyces, which generally have low virulence and are part of the normal flora of the mouth and gastrointestinal tract, gain access to tissues by a breech in the mucous membranes of the mouth, oropharynx, and gastrointestinal tract or by aspiration.

From: Principles and Practice of Pediatric Infectious Disease (Third Edition), 2008

Related terms:

Dental Caries, Actinomycosis, Nocardia, Serositis, Lesion, Protein, Streptococcus, Prevotella, Bacterium

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Nocardia and Actinomyces

Petar Pujic, ... Verónica Rodríguez-Nava, in Molecular Medical Microbiology (Second Edition), 2015

Mechanisms of *Actinomyces* Pathogenicity

Actinomyces belong to the normal indigenous microflora, so they are considered as facultative pathogens. Actinomycosis is usually associated with the breakdown of normal physical barriers, such as disruption of mucosal membranes [140]. Little more is known about the molecular mechanisms by which the Actinomyces cause actinomycosis. Most infections with *Actnomyces* spp. are polymicrobial and members of the Streptococcus genus are the most commonly associated organisms. They act synergistically by inhibiting host defence mechanisms and reducing oxygen tension in the affected tissue, which enhances growth of *Actinomyces* spp. [146].

More is understood about how species of *Actinomyces* cause plaque formation and periodontal disease. The hydrolysis of urea by the urease enzymes of oral bacteria may have a major impact on oral microbial ecology and be intimately involved in oral health and diseases. Neutral pH environments and excess carbohydrate availability could promote urease expression of *Actinomyces* in biofilms, but only neutral pH environments could up-regulate the *ureC* gene expression and the pH regulates *ureC* gene expression at a transcriptional level [147].

In addition, *Actinomyces* are able to produce extracellular or cell-associated polymers, such as dextran, levan, glycogen, and *N*-acetylglucosamine-rich slime polysaccharides, which enable the organism to attach to enamel by making the cells sticky [138].

Actinomyces spp. possess fimbriae, involved in the pathogenicity. Type 1 fimbriae are involved in the adherence of the organism to salivary proline-rich proteins that coat the tooth surface. Type 2 fimbriae mediate the receptor-dependent co-aggregation between *Actinomyces* and oral streptococci as well as host cells during the development of oral biofilms [138]. Detailed molecular and biochemical studies have been carried out using Actinomyces naeslundii, Actinomyces viscosus T14V, *Actinomyces oris* and *Actinomyces naeslundii* strains. The genes that encode the structural proteic subunits of type 1 and type 2 fimbriae have been identified. FimP and FimA require sortase-like genes to assemble type 1 and 2 fimbriae, respectively. Both contain a pilin motif and an E box, which are common features of Gram-positive bacterial major pilin subunits [117]. The *fimQ-fimP-srtC1-fimR* gene cluster encodes the type 1 fimbriae that contain a major subunit, FimP, and a minor subunit, FimQ. Similarly, the *fimB-fimA-srtC2* gene cluster encodes the type 2 fimbriae composed of a shaft protein, FimA, and a tip protein, FimB. The type 1 gene cluster of strain T14V contains seven open reading frames with the gene order *orf3-orf2-orf1-fimP-orf4-orf5-orf6*. Only the insertions in *orf1*, *orf2*, and *fimP* eliminated adhesion to proline-rich proteins. The type 2 gene cluster encodes three proteins and exhibits the gene order *orf977-fimA-orf365*. An insertion mutation in *orf365* prevents the assembly of the fimbriae but not the synthesis of the fimbrial antigen, whereas an insertion mutation in *fimA* eliminated the synthesis of this antigen [148]. The sequencing of the genes for FimA proteins from *Actinomyces naeslundii* genospecies 1 and 2 and *Actinomyces odontolyticus* indicated the presence of three subtypes of FimA with different carbohydrate specificities. E box and LPXTG motifs, which are required for the sortase activity, displayed 80–100% sequence identity between the three subtypes. *Actinomyces* display seven conserved proline-containing regions and involve a diversity of unique proteins FimA [149]. Sequence alignment of several major fimbrial proteins revealed that FimA and FimP also contain several other conserved motifs that have yet to be characterized [148].

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Nocardia and actinomyces

Grant C. Paulsen MD, Paul K. Sue MD, in Pediatric Transplant and Oncology Infectious Diseases, 2021

Epidemiology

Actinomyces species are slow-growing, microaerophilic to facultative anaerobic, gram-positive, filamentous branching bacilli that are known to cause infection in three distinct anatomic sites: cervicofacial, thoracic, and abdominal.[26] They belong to the order Actinomycetales, along with *Mycobacteria* and *Nocardia*. There are more than 40 known species of *Actinomyces*, and infections have commonly been reported owing to *A. israelii, A. odontolyticus, A. mayeri, A. naeslundii, A. neuii, A. turensis,* and *A. graerenitzii*.[36]

In general, pediatric actinomycosis is uncommon, representing less than 3% of reported *Actinomyces* cases.[37] *Actinomyces* species are generally opportunistic pathogens, with disease reported after penetrating or nonpenetrating trauma as well as any breach of the mucosal barrier. The pathogens are also notorious for their ability to directly cross tissue planes and extend into bone to cause soft tissue abscesses and chronic suppurative granulomatous infections.

Actinomycosis in pediatric SOT and HSCT recipients remains a rare infection, with reported cases in the literature occurring only in adults. An adult center reporting on 16 years of actinomycosis found 2 of 36 (6%) proven infections occurred in SOT recipients and 6 (17%) occurred in patients with concurrent malignancy. Alcohol abuse and foreign bodies/devices are commonly reported factors associated with actinomycosis in adults, whereas breaks in the mucosal barrier, including trauma, perforation, or surgery, are often risks in pediatrics.[38] In another analysis of 366 surveillance and clinically triggered bronchoscopy specimens after adult lung transplantation, only a single culture (0.3%) was positive for *Actinomyces*.[39] Although there are reports of actinomycosis in patients with human immunodeficiency virus as well as patients with other immune-compromising conditions, such as autoimmune disorders and certain primary immunodeficiencies, the degree of association between SOT and/or HSCT and the risk of *Actinomyces* infection remains elusive. A national study of actinomycosis after renal transplantation found an estimated prevalence of 0.02% (7 cases of 34,268 renal transplant recipients).[36] All cases were in adult SOT recipients, and the median time between transplant and diagnosis was 104 months. Based on population data in the same region, the authors estimated that actinomycosis prevalence may be increased up to 10-fold in adult kidney transplant recipients. As there are no similar data for pediatric SOT, HSCT, or oncology patients, the associated risk in these populations remains unknown. Although overall treatment outcomes are generally good, as with many other opportunistic infections, an infection with invasive actinomycosis is potentially a marker of poor overall prognosis.[40]

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Anaerobic Gram-Positive Nonsporulating Bacilli (Including Actinomyces)

Anat R. Feingold, Debrah Meislich, in Principles and Practice of Pediatric Infectious Diseases (Fifth Edition), 2018

Actinomyces Species

Actinomyces spp. are filamentous, branching, gram-positive, pleomorphic, non–spore-forming, catalase-negative, anaerobic or microaerophilic/capnophilic bacilli. Although many species have been associated with disease in humans, actinomycetes usually are soil dwelling organisms.[16] Actinomyces cannot be distinguished from Nocardia on Gram stain.[17] However, *Nocardia* spp. grow aerobically and stain with acid-fast technique, and *Actinomyces* spp. do neither. With the use of 16S (small subunit) ribosomal RNA (16S rRNA) sequencing, at least 21 species of *Actinomyces* have been identified in humans. *A. israelii* is the predominant disease-causing species (median, 73% of cases), although other *Actinomyces* spp., including *A. naeslundii* (the second most frequent *Actinomyces* sp.)[16] *A. meyeri, A. odontolyticus, A. gerencseriae,* and *A. viscosus,* as well as Propionibacterium *propionicum,* also have been implicated.[18,19] Many different *Actinomyces* spp. are associated increasingly with infections at many body sites, and currently, 25 *Actinomyces* spp. have been described from human material.[20] Most actinomycotic infections are polymicrobial, involving other aerobic and anaerobic bacteria. Coisolates depend on the source or site of infection and include Aggregatibacter (formerly *Actinobacillus*) *actinomycetemcomitans,* Eikenella corrodens, Bacteroides, Fusobacterium, Capnocytophaga, aerobic and anaerobic streptococci, Staphylococcus, and Enterobacteriaceae.

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Bacteriology of the Head and Neck Regions

Thomas S. Murray, Todd Cassese, in Head, Neck, and Orofacial Infections, 2016

Actinomyces Species

Actinomyces species are facultative, anaerobic, non–spore-forming, filamentous, gram-positive rods that commonly inhabit the oropharynx. Although individual bacteria are filamentous, colonies form fungus-like branched networks of hyphae.

Actinomyces grow slowly in culture and are not acid-fast, distinguishing them from Nocardia species. *Actinomyces* species are pathogenic locally in the oral cavity or through direct extension into fascial planes as abscesses. Of the myriad species of *Actinomyces* identified, the most common pathogens include *A. israelii, A. viscosus, A. naeslundii, A. turicensis,* and *A. radingae.* Systemic disease can also result through exposure of *Actinomyces* species to the bloodstream or through aspiration of organisms into pulmonary tissue. Local infection with *Actinomyces* species occurs at the level of the teeth and gingiva. *Actinomyces* species are isolated from supragingival plaques and root surface caries, and they are implicated in the development of gingivitis.[32]

Infection can spread when normal mucosal barriers are disrupted, leading to abscesses with connecting sinus tracts. These abscesses are most commonly found in the face and neck as cervicofacial actinomycosis, but they can also occur throughout the thorax, abdomen, pelvis, and central nervous system. Understanding of the virulence factors used by *Actinomyces* species is limited. One hypothesis is that cell wall lipoproteins induce an overzealous immune response through TLR2, leading to extension of disease beyond mucosal surfaces.[32] In addition, cell wall peptidoglycan has recently been shown to induce alveolar bone resorption and osteoclastogenesis, in addition to recruitment of inflammatory cytokines.[33]

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Clinical Bacteriology

Jeffrey K. Actor PhD, in Elsevier's Integrated Review Immunology and Microbiology (Second Edition), 2012

Actinomyces

Actinomyces spp. are gram-positive, obligate anaerobes known to reside in the mouth and intestinal tract. They are morphologically similar to fungus in that they form filamentous branches. Pathology due to proliferation of organisms usually occurs following injury or trauma to tissue, resulting in actinomycosis (abscess formation and swelling at the site of infection). Microscopic examination of pus reveals exudates with granular texture caused by sulfur granules, resulting from the bacterium and its waste. *A. israelii* is most commonly associated with actinomycosis; however, other Actinomyces bacteria are capable of causing disease. Actinomycosis can be treated with penicillin.

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Diseases Caused by Actinomyces Species

James Booth, in xPharm: The Comprehensive Pharmacology Reference, 2007

Epidemiology

Actinomyces species are found in humans as normal flora throughout the upper respiratory, gastrointestinal, and female genital tracts. They are particularly common in the oral cavity where nine species are found Sarkonen et al (2001), and they are prominent components of dental plaque and calculus Shuster (2002). Several species have been isolated from animals and can cause disease, such as lumpy jaw of cattle. While most infections are due to ones own normal flora, infections due to exogenous sources, such as human or animal bites, can occur. Actinomyces have not been isolated from the environment Smego and Foglia (1998).

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Cervical and Vaginal Cytology

Edmund S. Cibas, in Cytology (Third Edition), 2009

Actinomyces

Actinomyces species are gram-positive anaerobic bacteria that are normal inhabitants of the mouth and bowel. They are uncommon in the cervix and vagina, where they are almost always associated with a foreign body, most commonly an IUD. It is estimated that 7% of women with an IUD have Actinomyces spp. on their Pap,[117] and the frequency is related to the duration of continuous IUD use. When found incidentally on a Pap test, they are almost always harmless. In a small number of cases, however, women with an IUD develop pelvic actinomycosis, usually a tubo-ovarian abscess, presumably as a result of ascending infection. Case reporting has not been systematic, so it is impossible to judge the risk of this significant complication, but pelvic actinomycosis resulting from an IUD is considered exceedingly rare.[118]

CYTOMORPHOLOGY OF ACTINOMYCES:

•tangled clumps of bacteria (“cotton balls,” “dust bunnies”)•long, filamentous organisms•Figure 1.23

If *Actinomyces* are seen on a Pap, removal of the IUD is not necessary, and treatment of asymptomatic women is not recommended.[117]

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