Corynebacterium Xerosis

Corynebacterium xerosis bacteria are found in great numbers in the lesion.

From: Ocular Pathology (Seventh Edition), 2015

Related terms:

Alkaloids, Bacterium, Microorganism, Sjoegren Syndrome, Corynebacterium, Xerosis, Micrococcus luteus, Escherichia coli, Candida Albicans

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Coryneform Gram-Positive Bacilli

Laura Lucía Rojas-García, in Reference Module in Biomedical Sciences, 2021

Mechanisms of resistance to antibiotics

In the evolution of antibiotic resistance of the genus *Corynebacterium* most studies conclude that extrachromosomal genetic elements are involved in resistance gene transfer. Horizontal gene transmission plays an important role in their pathogenicity. Antibiotic resistance genes are localized in large plasmids, for example, the pTP10 plasmid is responsible for the resistance to chloramphenicol, tetracycline, streptomycin, and erythromycin described in Corynebacterium xerosis (Oliveira et al., 2017).

Many species such as *C. striatum, C. pseudodiphtheriticum, C. jeikeium* and *C. coyleae* (Fernández-Natal et al., 2008) are resistant to macrolides because they possess ermX genes. For this reason, erythromycin, which had traditionally been the treatment of choice, has ceased to be used unless the sensitivity report of the laboratory confirms its sensitivity. It has also been linked to resistance to clindamycin, and trimethoprim-sulfamethoxazole.

Another example is the existence of multidrug-resistant strains, such as *C. amycolatum*, one of the most common strains of corynebacteria. This property is defined by the existence of resistance to three or more groups of antibiotics commonly used in the treatment of infection such as beta-lactam, aminoglycosides, macrolides, MLSB

group and quinolones. The presence of mutations in the gyrA gene is related to resistance to treatment with quinolones (Ortiz-Perez et al., 2010).

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Corynebacteria (including diphtheria)

K.F. Smith, D.M. Oram, in Encyclopedia of Microbiology (Third Edition), 2009

Infections caused by Corynebacterium species found on the skin

Coryneform bacteria of multiple species are part of the microflora of healthy human skin but some of these bacterial species can cause disease under specific conditions. The four species discussed in this paragraph are occasionally associated with human disease. In clinical settings Corynebacterium *striatum* and *Corynebacterium amycolatum* are often misidentified as Corynebacterium xerosis, but true human infections caused by *C. xerosis* are exceedingly rare. On the other hand, both *C. striatum* and *C. amycolatum* do cause invasive infections in immunocompromised patients. Like *C. xerosis, C. striatum* is often found on healthy skin but it is increasingly associated with serious infections such as bacteremia, endocarditis, osteomyelitis, breast abscess, and pulmonary infections. Most *C. striatum* isolates are susceptible to antibiotics but the development of resistant strains is always a concern. On the other hand, multiresistance is very common in *C. amycolatum* strains. *C. amycolatum* was established as a species in 1988 and it is unusual in that it lacks detectable mycolic acids in its cell wall. Nonetheless, analysis of the 16S RNA sequences indicates that it is a member of the genus *Corynebacterium*. Infections caused by *C. amycolatum* include peritonitis, bacteremia, and wound infections. *C. amycolatum* infections are likely underreported because *C. amycolatum* is often identified in clinical laboratories as a member of the normal flora and not the etiological agent of disease. Corynebacterium minutissimum is yet another member of the normal flora on human skin but it can be associated with the polymicrobial disease erythrasma. Erythrasma is a skin infection characterized by scaly, reddish patches in areas where skin surfaces touch, such as the groin. The role of *C. minutissimum* in these infections is not clear since the condition is clearly caused by multiple contributing factors including several bacterial species.

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Endocarditis and Intravascular Infections

Vance G. FowlerJr., ... Arnold S. Bayer, in Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (Eighth Edition), 2015

Gram-Positive Bacilli

IE due to various species of corynebacteria (diphtheroids) has recently been reviewed.[440] It is uncommon and usually occurs on damaged or prosthetic valves,[441] although native valve infections (e.g., Arcanobacterium *[Corynebacterium] haemolyticum* in a drug addict) are reported rarely. About 19 cases of IE due to Corynebacterium pseudodiphtheriticum (previously *Corynebacterium hofmannii*) have been reported; native valves were involved in approximately half of these cases.[442] Corynebacterium xerosis is a very rare cause of native valve IE. Nontoxigenic Corynebacterium diphtheriae IE has been reported in more than 40 patients. A cluster of seven cases in 1 year from New South Wales, Australia,[443] emphasized the aggressive nature of the infection, including major vascular complications, the frequent occurrence of septic arthritis (in four of seven patients), and involvement of native valves. Injection drug use also is a predisposing factor.

The isolation of Listeria *monocytogenes* has been reported in 44 cases of IE.[444,445] Most cases of IE due to *Listeria* spp. have occurred in patients without any underlying defect in host defenses, although preexisting heart disease is present in approximately 50%. The mean age in the reported series was 51 years, and the overall mortality rate was 48%.[445]

Lactobacilli also have been reported to cause a subacute form of IE, but these cases are rare (only 41 reported).[446,447] Despite an initial response to therapy, relapse of this infection is not unusual (approximately 60% of cases). Most cases occur on structurally abnormal native valves after dental manipulation.[447] Therapy with single agents is often unsatisfactory because lactobacilli, similar to enterococci, are tolerant to penicillins. Medical cure has been difficult to achieve in the past. These organisms also may require several weeks for isolation on blood culture.

More than 90% of 49 serious infections caused by Erysipelothrix rhusiopathiae were characterized as IE.[448] Occupational or vocational animal or fish exposure is a major risk factor, and approximately one third of patients are alcoholic. Most patients are men. A characteristic erysipeloid skin lesion is present in approximately 40% of cases, and the organism exhibits significant aortic valve tropism (involved in 70% of patients).[448] The overall mortality rate is 38%.

Most cases of Bacillus IE involve the tricuspid valve in narcotic addicts, but nonaddicts and prosthetic valve recipients also have been affected.[449] Rothia dentocariosa is a rare cause of IE (six cases reported) but has led to significant central nervous system complications.[450,451]

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Clinical Syndromes and Cardinal Features of Infectious Diseases: Approach to Diagnosis and Initial Management

Susan E. Coffin, Theoklis E. Zaoutis, in Principles and Practice of Pediatric Infectious Diseases (Fourth Edition), 2012

Postoperative Mediastinitis and Sternal Osteomyelitis

Mediastinitis is inflammation or infection of the mediastinal area (defined as the extrapulmonary area of the thoracic cavity between the lungs). This area contains the thymus, trachea and bronchi, esophagus, aorta and aortic arch, heart, pericardium, lymph nodes, and nerve tissue. Historically, infections of the mediastinum have been divided into acute (abrupt onset, with or without toxic appearance) and chronic (indolent) processes.[197–199] Although acute mediastinitis is uncommon, it can be serious and life-threatening.

Epidemiology and Pathogenesis

Mediastinitis after cardiac surgery is an infrequent yet serious complication of median sternal incision, with an estimated incidence ranging from 0.15% to >5% of all cardiothoracic operations.[200] In the few studies restricted to children, mediastinitis rates after cardiac surgery varied between 0.1% and 5%, with the highest rates found in neonates.[201–203] Because most pediatric cardiac operations involve correction of congenital anomalies, infection rates and risk factors for infection may differ from those identified from adult studies. Using a large multicenter database, factors that were independently associated with the risk of mediastinitis included: young age, prolonged preoperative hospitalization, preoperative ventilator support, and genetic abnormality.[204] No data are available on whether rates of mediastinitis in children vary according to type of reconstructive surgery or congenital anomaly.

The pathogenesis of infection is multifactorial but generally requires intraoperative introduction of pathogenic bacteria into the operative site; thus, perioperative aseptic technique and surgical technique are critical to prevent mediastinitis.[205,206] Risk

factors for mediastinitis in adults include diabetes mellitus, chronic obstructive pulmonary disease, prolonged duration of perfusion (cardiopulmonary bypass) or time of aortic cross clamping, higher body mass index or obesity, preceding infections (e.g., pneumonia, tracheal infections), and receipt of corticosteroids.[197,199,200,205–209] Outbreaks of mediastinitis have been associated with preoperative colonization, operating room personnel (e.g., anesthesiologist, intraoperative nurses, or surgeons), other intraoperative factors (e.g., contaminated cardioplegia solution or inadequate sterile surgical technique), and postoperative exposures.[170,171,210,211]

Staphylococci are the predominant pathogens causing postoperative mediastinitis.[208,212,213] In children, *S. aureus* accounts for 38% to 96%, and coagulase-negative staphylococci for 10% to 52%, of episodes of mediastinitis in some series.[201,214] Gram-negative organisms and *Candida* spp. are less common pathogens in children.[215,216] A wide variety of pathogens have been reported to cause mediastinitis after cardiac surgery in adults, including staphylococci, Enterobacter cloacae, *Escherichia coli,* Klebsiella spp., Pseudomonas spp., Proteus spp., enterococci, Bacteroides fragilis, Corynebacterium xerosis, Mycoplasma spp., nontuberculous mycobacteria, Aspergillus spp., Haemophilus spp., Nocardia spp., and Rhodococcus *bronchialis.*[217,218]

Clinical Manifestations and Laboratory Diagnosis

Common signs and symptoms of postoperative mediastinitis include local tenderness, wound dehiscence, increased erythema of the wound with or without purulence, and an unstable sternum (movable appositional edges). Infants merely can be fussy and display expiratory grunting. Fever occurs on average 5 days after surgery, and local signs occur a mean of 9 days after surgery.[201,216] In older children, signs and symptoms can appear later; in one study, infections were diagnosed at a mean of 15 days postoperatively.[201] Infections due to *Nocardia, Rhodococcus,* or *Candida* species, or nontuberculous mycobacteria, can have an indolent onset, sometimes with incubation periods >30 days. Infections associated with these organisms can have minimal local signs (only serosanguineous drainage), and little or no fever.

Laboratory tests usually reveal a moderate leukocytosis with an increased frequency of neutrophils or an elevation in the erythrocyte sedimentation rate or C-reactive protein value. The diagnosis of acute suppurative mediastinitis is suggested by radiographic evidence of widened mediastinum, mediastinal emphysema, and pleural effusions. The presence of gas in the soft tissues is highly suggestive of esophageal perforation. Computed tomography (CT) has been used to differentiate mediastinitis from mediastinal abscess;[219,220] however, in the absence of mediastinal gas, CT may not differentiate mediastinitis from benign postoperative changes. Among heart transplant recipients, gallium scintigraphy has been used to confirm mediastinitis when CT scan is not diagnostic.[221] Gadolinium-enhanced magnetic resonance

imaging (MRI) also can help delineate the extent of infection and differentiate among mediastinal tissues, masses, and inflammatory tissue.[222,223] A definitive diagnosis can be made by Gram and acid-fast stains and culture for bacteria, mycobacteria, and fungi performed on specimens obtained via CT-guided aspiration. If esophageal perforation is suspected, fluoroscopy with water-soluble contrast media may aid in the diagnosis of mediastinitis and allow localization of the level of perforation.

Management and Outcome

Aggressive surgical drainage and debridement generally are required to cure mediastinitis after cardiac surgery. For superficial mediastinal infections, incision, drainage, packing of the wound, and antimicrobial therapy may be effective. For deep infections, debridement with removal of infected and devitalized tissue, mediastinal irrigation, and antimicrobial therapy may be necessary. Surgical debridement (with closed-tube irrigation) and systemic antimicrobial therapy usually are sufficient. In severe infections, it may be necessary to leave the wound open, with subsequent secondary closure.[194,224]

For postoperative mediastinitis, selection of empiric agents should be based on the prevalent pathogens associated with cardiac SSIs in the institution and the patient's endogenous flora if known; therapy effective for *S. aureus* and coagulase-negative staphylococci must be provided. The combination of vancomycin and a third- or fourth-generation cephalosporin is a common empiric regimen. No studies have been conducted to evaluate the optimal regimen or duration of antimicrobial therapy for mediastinitis; however, 3 to 8 weeks is generally recommended, depending on the severity of the infection and the extent of bone involvement.[225] There is no role for directly instilled (topical) antibiotic therapy.

Sternal osteomyelitis can accompany severe, deep mediastinal infections. These infections most commonly follow surgery involving a median sternotomy. The pathogens responsible for sternal osteomyelitis are similar to those causing mediastinitis, and *S. aureus* and coagulase-negative staphylococci are the predominant organisms. Treatment of sternal osteomyelitis requires debridement of infected bone and a minimum of 4 to 6 weeks of antimicrobial therapy.[202,226,227]

Prevention

Antimicrobial prophylaxis has not been shown in placebo-controlled trials to decrease the risk of mediastinitis; however, because this infection can be catastrophic, perioperative prophylaxis frequently is used. Centers with lower rates of mediastinitis employ: (1) strict perioperative adherence to careful aseptic technique; (2) attention to surgical measures, including hemostasis and precise sternal closure; and (3) interventions targeted to identified risk factors.[196,205,228] Some authorities

have proposed the use of intraoperative ultraviolet irradiation, preoperative decolonization, or preoperative chlorhexidine washes of patients who are carriers of *S. aureus* to help prevent infections.[151,172,228]

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Biological Activities of Extracellular Yeast Glycolipids

Ekaterina Kulakovskaya, Tatiana Kulakovskaya, in Extracellular Glycolipids of Yeasts, 2014

4.3 Biological Activities of MELs and Sophorolipids

There is a widely-held view that extracellular glycolipids perform primarily the function of biosurfactants, that is, compounds facilitating solubilization and absorption by the cells of various organic hydrophobic compounds that are present in the medium and can be utilized by microorganisms as growth substrates. A comprehensive review of this role of extracellular glycolipids is presented in Spencer et al. (1979), Lang and Wagner (1987), Rosenberg and Ron (1999), Cameotra and Makkar (2004), Kitamoto et al. (2002), Rodrigues et al. (2006), Langer et al. (2006), Van Bogaert et al. (2007a), Arutchelvi et al. (2008), Bolker et al. (2008), Arutchelvi and Doble (2011), and Van Bogaert and Soetaert (2011).

The antibiotic activity of sophorolipids and MEL has been reported. The growth of Gram-positive bacteria (Bacillus subtilis and Micrococcus luteus) was suppressed by 3–10 mg/l of MEL or 0.12–0.48 mg/l of sophorolipid (Kitamoto et al., 2002), but higher concentrations (up to 400 mg/l) were needed to suppress the growth of Gram-negative bacteria. Sophorolipids inhibit the growth of Gram-positive bacteria such as Staphylococcus epidermidis, Staphylococcus aureus, Streptococcus faecium, Propionibacterium acnes, and Corynebacterium xerosis (Hommel et al., 1987; Lang et al., 1989).

Synthesized analogs of sophorolipids were obtained and their antibiotic activities were studied (Azim et al., 2006). These compounds consist of amino acids linked by amide bonds to the carboxyl group of fatty acid residues of sophorolipids. All tested analogs showed antibacterial activity against both Gram-positive and Gram-negative bacteria. Leucine conjugate was the most efficient: the MICs for Moraxella sp. and *Escherichia coli* were 0.83 mg/ml and 1.67 mg/ml, respectively. All compounds displayed virus-inactivating activity with 50% effective concentrations below 0.2 mg/ml (Azim et al., 2006).

Some authors disprove the antibacterial activity of sophorolipids and believe that sophorolipids can be used as immunomodulators without any effect on the host microflora (Sleiman et al., 2009).

The data on the fungicidal activities of sophorolipids and MEL should be considered with care, because their effective concentrations are rather high. The MICs against C. albicans were above 400 mg/l for MEL-A and MEL-B and above 200 mg/l for the sophorolipid of *C. apicola* (Kitamoto et al., 2002). These concentrations are not much more effective than MIC of the known detergent SPAN-20 (sucrose nonadecanoate).

The antifungal activities of *St. bombicola* sophorolipids and some of their derivatives were tested by the standard MIC assay during the cultivation of test cultures in plates on the medium containing yeast extract and wort at a concentration of 5 mg/ml. The natural sophorolipid mixture suppressed the growth of *C. albicans* and *C. tropicalis* by 30% and 25%, respectively, while some derivatives esterified to the fatty acid residue (ethyl-17-l-[(2□-*O*-□-d-glucopyranosyl-□-d-glucopyranosyl)-oxy-]-cys-9-octadecanoate-6□6□□-diacetate and methyl-17-l-[(2□-O--□-d-glucopyranosyl-□-d-glucopyranosyl)-oxy-]-cys-9-octadecanoate) were capable of 100% growth suppression of these two species (Gross and Shah, 2005).

Diverse biological activities of sophorolipids and MEL are associated primarily with their surfactant and amphiphilic properties.

For instance, it has been shown that MEL-A substantially increases the efficiency of transfection by cationic liposomes. It is supposed that MEL-A induces the membrane fusion of cationic liposomes and target cells and thereby accelerates the transfer of genetic material (Inoh et al., 2001, 2004Inoh et al., 2001Inoh et al., 2004).

The biological activities of MEL and sophorolipids were actively studied in the context of their influence on various regulatory processes in mammalian cells. These data were summarized in the reviews (Kitamoto et al., 2002; Cameotra and Makkar, 2004; Arutchelvi and Doble, 2011; Van Bogaert and Soetaert, 2011).

The experiments with cell cultures have shown the anticancer activity of MEL and sophorolipids. MEL can suppress the proliferation of lymphocytes affected by leukemia in cell culture and induce their differentiation (Isoda et al., 1997), inhibit the proliferation of melanoma cells and, at sufficiently high concentrations, induce their apoptosis (Zhao et al., 1999, 2001Zhao et al., 1999Zhao et al., 2001).

Sophorolipids also suppressed the reproduction of liver carcinoma cells (Chen et al., 2006a,bChen et al., 2006aChen et al., 2006b). The 10 sophorolipid molecules differing in acetylation degree of sophorose, unsaturation degree of hydroxyl fatty acid, and lactonization or ring opening were tested as inhibitors of esophageal cancer cells (Shao et al., 2012). The inhibition of diacetylated lactonic sophorolipid (total inhibition at 30 μg/ml) was stronger than that of monoacetylated lactonic

sophorolipid (totally inhibition at 60 μg/ml). The sophorolipid with one double bond in fatty acid part had the strongest cytotoxic effect on two esophageal cancer cells. Acidic sophorolipid showed higher anticancer activity (Shao et al., 2012).

Cytotoxicity of the natural sophorolipid mixture and individual chemical derivatives of these compounds against pancreas carcinoma cells was investigated (Fu et al., 2008). It was found that sophorolipid methyl esters proved to be more toxic for these carcinoma cells compared to the natural sophorolipids. Sophorolipids in the acidic form and the sophorolipid containing two acetate groups and existing in the lactone form demonstrated similar toxicities (the death of 40–50% cells after 24-h treatment at a concentration of 0.5 mg/ml). Under these conditions, the mononuclear cells of peripheral blood were insensitive to all of the used sophorolipids and their derivatives (Fu et al., 2008).

The effects of MEL and sophorolipid on the initiation of nerve ending growth were investigated. The addition of these glycolipids was shown to cause a considerable growth of nerve endings. MEL-A enhances the acetylcholine esterase activity to a level similar to that caused by the nerve tissue growth factor (Isoda et al., 1999). Other authors have shown that MEL increases the level of galactoceramide and the growth of nerve endings in pheochromocytoma cells (Shibahara et al., 2000). In addition, the treatment of the cells of this line with MEL leads to cell cycle interruption in the G1-phase and suppression of cell differentiation (Wakamatsu et al., 2001). The mechanism of MEL action is still unknown.

MELs have a high affinity to the human immunoglobulin G. The maximum binding capacity was shown for MEL-A (Im et al., 2001, 2003Im et al., 2001Im et al., 2003). It is evident in favor of potential involvement of this glycolipid in binding of other important regulatory proteins and possible regulation of their activities.

Sophorolipids can modulate inflammatory response and reduce mortality under experimentally induced sepsis in rats (Bluth et al., 2006; Hardin et al., 2007). Sophorolipids reduced the production of nitrogen oxide and cytokins by macrophages *in vitro* (Bluth et al., 2006). It was shown that the survival of rats could be enhanced by 40% at a dose of 5 mg/kg of animal weight, while the toxic effect occurred at 75- to 150-fold higher doses (Hardin et al., 2007).

MELs, similar to sophorolipids, influence the inflammatory process (Morita et al., 2011d). The experiments with mast cells have shown the anti-inflammatory effect of MEL due to exocytosis suppression and the inhibition of antigen-induced secretion of leukotriene C(4) and cytokine TNF-□. This effect is determined by suppression of the activity of several signaling systems, including those related to the increase in Ca^{2+} and MAP kinase concentrations and the activities of other signaling systems. MEL also suppressed the phosphorylation of the receptor protein SNARE, which

plays a key role not only in exocytosis but also in the intracellular movement of vesicles.

It was shown that 5% and 10% MEL-A solutions yielded 73% and 91% survival of skin cells treated with the damaging concentrations of SDS. MEL-B possessed similar protective properties (Morita et al., 2009a–c, 2011b–dMorita et al., 2009a-Morita et al., 2009bMorita et al., 2009cMorita et al., 2011bMorita et al., 2011cMorita et al., 2011d; Yamamoto et al., 2012). It was close to the protectant properties of natural ceramides used in cosmetics for regeneration of damaged skin. The antioxidant properties of MEL were shown using the model of the peroxide-induced oxidative stress of human fibroblast culture; MEL-C proved to be the best antioxidant (Takahashi et al., 2012). It was also shown that MEL-A at a concentration of 0.001 μg/l stimulated the activity of hair bulb cells (Morita et al., 2010a); hence, it was proposed to use mannosylerythritols as agents for regeneration of damaged hair and stimulation of hair growth (Morita et al., 2010b).

The diacetylated MEL (MEL-A) produced from soybean oil significantly increased the viability of the fibroblast cells over 150% compared with that of control cells (Morita et al., 2010c). The monoacetylated MEL (MEL-B) hardly increased the cell viability. The viability of the fibroblast cells decreased with the addition of more than 1 μg/l of MELs, whereas the cultured human skin cells showed high viability with 5 μg/l of MELs. The papilla cells were dramatically activated with 0.001 μg/l of MEL-A produced from soybean oil: the cell viability reached at 150% compared with that of control cells (Morita et al., 2010c).

Sophorolipids have a spermicide activity and can inactivate the human immunodeficiency virus (Shah et al., 2005).

Sophorolipids and MEL are characterized by structural diversity, including different degrees of acetylation and lengths of fatty acid chains. The question about the interrelationship between the structures and effects of these compounds on animal and humans cells is still far from being solved.

Numerous studies have demonstrated that sophorolipids and MEL are not toxic for noncancer human cells (Ikeda et al., 1986 a,bIkeda et al., 1986aIkeda et al., 1986b; Kitamoto et al., 2002). Nontoxicity of cellobiose lipids has also been shown for some cell lines (Mimee et al., 2005). Hence, they may be considered promising for application in cosmetology and medicine as natural detergents, immunomodulators, liposomal carriers for drug delivery, while cellobiose lipids may be considered as prospective fungicidal compounds.

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SI:Human microbiome and health

Reet Mändar, in Pharmacological Research, 2013

2.6 Coryneform bacteria in male genital tract

In many studies coryneform bacteria have been revealed from male urogenital tract. Mostly these bacteria tend to be often overlooked as commensals but some authors have associated these microorganisms with prostatitis [51–54]. Coryneform bacteria are aerobic, asporogenous, irregular Gram-positive rods. They belong to the phylum Actinobacteria. Their classification has undergone dramatic changes – genus Corynebacterium has been defined more narrowly and many species now belong to other genera like Arthrobacter, Cellulomonas and Rhodococcus, instead. With a notorious exception of Corynebacterium diphtheriae, the coryneform bacteria have been considered as part of the normal human flora or environmental contaminants, but were recognized increasingly as a cause of life-threatening diseases later [55,56].

The list of coryneforms found in male urogenital tract includes *Corynebacterium singulare*, *Corynebacterium freneyi*, *Corynebacterium afermentans*, Corynebacterium xerosis, *Corynebacterium* group ANF, *Corynebacterium striatum*, *Corynebacterium amycolatum*, *Corynebacterium macginley*, Corynebacterium jeikeium, *Dermabacter hominis*, Corynebacterium minutissimum and even *C. diphtheriae* [52,54,57–59]. A new coryneform species *C. seminale* (also known as *C. glucuronolyticum*) was discovered first from prostatitis patients [60]. Later it has been associated also with urethritis [61].

In addition to cuturable ones, also the unculturable or fastidious coryneforms may appear in male genital tract that remain undetected during routine cultures [53,54].

Nucleotide-based studies [54,62] showed that *Corynebacterium* sp. were the most common bacteria in the EPS or urine, respectively, among prostatitis patients. Tanner et al. [54] found with 16S rRNA gene probe an unexpectedly diverse list of *Corynebacterium* species, up to nine species from one patient were found, and some unidentified species were characteristic to men with prostatitis only. Interestingly, 7 of 11 men who had bacteria in EPS were susceptible to treatment with antibiotics. It has been also speculated (although not proved) that coryneforms could grow in the prostate as a biofilm that would enhance antibiotic resistance [54].

Our study group has investigated coryneform bacteria in inflammatory prostatitis patients and healthy controls [63,64]. These bacteria were present in 76% of inflammatory prostatitis patients (NIH IIIA and NIH IV categories) and 83% controls. Half of men harbored corynebacteria in both semen and urine, 22% of men harbored them in semen only and 3% in urine only. Their total concentration was greater in semen than in urine (median 5000 vs 100 CFU/ml). The subjects had up to 6 (mean

1.3) different coryneforms present. The most frequent species was *Corynebacterium seminale*. Two coryneform bacteria were significantly more frequently found from prostatitis patients – *Corynebacterium* group G and *Arthrobacter* sp. We subsequently set a threshold limit of ≥10^4 CFU/ml to bacterial concentration in order to reveal possible differences between patients and controls at quantitative level. In controls, only four bacterial groups managed to outnumber this threshold: *C. seminale*, *C. jeikeium*, *Corynebacterium* sp. and catalase-negative coryneforms. In prostatitis patients also *Arthrobacter* sp., Brevibacterium sp., *Cellulomonas*/Microbacterium, *Corynebacterium* group F1 and *Corynebacterium* group G exceeded that threshold. These data indicate that coryneform bacteria may appear a major component of male genital tract microbiota.

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