

Enterococcus (Enterococcus faecalis, Enterococcus faecium etc.)

Microscopy: G+ cocci in pairs or short chains, catalase negative

Cultivation: small greyish white colonies on blood agar with viridation

Some of them have yellow pigment, some are mobil

selective diagnostic Slanetz-Bartley (sodium azide) agar - pink to red colonies

Bile-aesculin agar: black colonies

Biochemistry: pyrrolidonylarylamidase (PYR-positive) and leucinaminopeptidase (LAP-positive)
high resistance, growing in 6,5% NaCl agar, large temperature interval

Pathogenicity: part of normal digestive tract flora, more frequent in long term hospitalised patients with medical devices or patients treated with broad spectrum antibiotics

Urogenital infections, wound infections, intraabdominal infections, endocarditis – more often in drug users or seniors, catheter sepsis, biliary tract infection



Factors of virulence:

gelatinase, *feromon* substance, *colonization factors*, *bacteriocins* - inhibition of other bacteria

VanA, *B*, *C* gens causes rezistence to vancomycin (*C* is gen of primary resistance, *VanA/B* of secondary resistance, transferable through plasmides)

Treatment: primary resistant to cefalosporines

Liht urinary tract infection: ampicillin, ampicillin with β -lactamase inhibitors, nitrofurantoin, possible glycopeptides.

Wound infections, sepsis and endocarditis: combination of aminoglykoside + penicillin/ampicillin or glycopeptides (vancomycin, teicoplanin)

VRE (vancomycin resistant enterococci) – linezolid, quinupristin/dalfopristin

Laboratory dg.:

microscopy, cultivation on BA, on Slanetz-Bartley medium

Latex agglutination – differentiation from streptococci, from other bacteria through PYR test and LAP

Phenotypic test (production of yellow pigment, moovement)

Biochemistry: fermentation of arabinosis and pyruvate:

E. faecium

arabinosis fermentation – change of the indicators colour

pyruvate negative

resistent to ampicilin

EN-coccus test



E. faecalis

without fermentation

pyruvate fermentation

susceptible to ampicillin



G+ rods

Listeria monocytogenes



Morphology: microscopy: G+ rods, catalase positive



Cultivation: chromogennous media, growth in cold, on BA form grey colonies with haemolysis – looks like enterococci, streptococci or difteroids

Pathogenicity: wound infection, new-born babies infection (meningitis or sepsis)

Virulence factors: lysteriolysin, internalins (intracellular alive)

Treatment: fluoroquinolons

Laboratory dg.:

microscopy, cultivation on chr. medium/ BA and bile-aesculin medium, catalase detection, BBL test

Corynebacterium diphtheriae



Microscopy: G+ rods with metachromatic granules, club-shaped looking like chinese signs, catalase positive

Cultivation: does not grow on MH, but on BA, on telur media (Clauberg)

Pathogenicity: strains producing toxin (microb attacked by fag) causes diphtheria with pharyngitis (couldn't take off without bleeding), can suffocate, arise of myocarditis etc. Non-toxic strains causes skin inflammations.

Factors of virulence: diphtheric toxin

Therapy: vaccination, antidiphtheric globulin (deserters!), PNC, tracheostomy, corticoids

Laboratory dg.: microscopy, staining of specific parts - granules (Lebranc), Clauberg medium - metal shiny colonies with blue zone around colonies, Lofler medium, detection of toxins through Elek test, PCR, demonstration on guinea-pig.

Other *Corynebacteria* (*C. jeikeium* etc.)

Microscopy: G+ rods with metachromat. granules, club-shaped form looks like chinese signs, arranged in palisades, catalase positive

Cultivation: any growth on MH, but BA

Pathogenicity: wound infection, sepsis, urinary tract infections

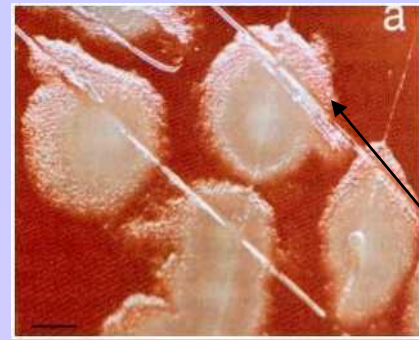
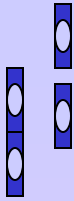
Factors of virulence: haemolysins

Treatment: vancomycin, teicoplanin, rifampicin, if possible - PNC

Laboratory dg.: microscopy, cultivation on BA, biochemistry...

Rod *Bacillus*

B. anthracis



Microscopy: G+rods looks like bamboo stick, spors (central terminated) – only in air

Cultivation: on BA – large, flat, spreading through the agar surface - caput medusae, ahaemolytical

Pathogenicity and pathogenesis: contact with ill person, dead animals or their productes (skin), spors invade into organism, germinate and produce toxin. Via entrance is disease devided into 3 forms.

1. skin - pustula maligna

2. pulminal – after inhalation arises hemoragic necrosis of nodes with mediastinitis ends as septic shock

3. intestinal – via contaminated food – causes bloody diarrhea, high temperature etc.

!! spors are easy to diffuse, that's why it is discussed as a biological warfare!!

Virulence: toxin (3components)

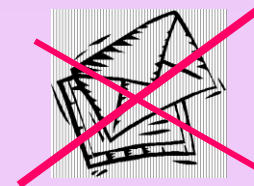
Therapy: PNC, ciprofloxacin, doxycyklin, chloramphenicol

Prevention: veterinary control of animal, vaccination of animal or people

Laboratory diagnosis: microscopy, cultivation on BA

Antigen detection - Ascoli termoprecipitation reaction, animal demonstration

!! Can do only laboratory with biosafety level III.



B. cereus



Microscopy: G+rods, central terminated spores

Cultivation: on BA flat colonies with β haemolysis, PEMBA-**blue** colonies

Pathogenicity: component of gastrointestinal flora, contamination of food, causing diarrhea, vomiting. Diarrhea is caused by thermolabile enterotoxin (source: sauce), vomiting is caused by thermostable toxin (source: rice). Also causes eye + wound infection

Factors of virulence: enterotoxins

Treatment: rehydration + linkosamids. Prevention: good food preparation
Eye infection: lincosamids + aminoglycosides

Laboratory dg.: microscopy, cultivation on BA/PEMBA, detection of granules
toxin detection via ELISA method or latex agglutination