Enterococcus (Enterococcus faecalis, Enterococcus faecium etc.)

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

<u>Cultivation:</u> 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

<u>Pathogenicity:</u> part of normal digestive tract flora, more frequent in long therm 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, catether 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 resistence, 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.

<u>Wound infections, sepsis and endocarditis</u>: 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





<u>Cultivation:</u> 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)

<u>Virulence factors</u>: lysteriolysin, internalins (intracellular alive)

Treatment: fluoroquinolons

Laboratory dg.:

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

Corynebacterium difteriae



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

<u>Cultivation:</u> does not grow on MH, but on BA, on telur media (Clauberg)

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

Factors of virulence: diphteric toxin

Therapy: vaccination, antidiphteric globulin (deserters!), PNC, tracheostomy, cortikoids

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. jejkeium etc.)

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

Cultivation: any growth on MH, but BA

Pathogenicity: wound infection, sepsis, urinary tract infections

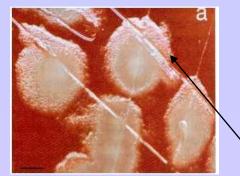
Factors of virulence: haemolysins

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

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

Rod Bacillus

B. antracis



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.









Microscopy: G+rods, central terminated spores

<u>Cultivation:</u> on BA flat colonies with β haemolysis, PEMBA-blue colonies

<u>Pathogenicity</u>: component of gastrointesinal flora, contamination of food, causing diarrhea, vomitting. Diarrhea is caused by thermolabil enterotoxin (source: sauce), vomitting is caused by thermostabil toxin (source: rice). Also causes eye + wound infection

Factors of virulence: enterotoxins

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

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