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 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

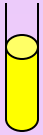
Factors of virulence:
gelatinase, feromon substance, *colonization factors, bacteriocins* - inhibition of other bacteria
VanA, B, C gens causes rezistence to vancomycin (*C* is gen of primary resistence, *VanA/B* of secondary resistance, transferable through plasmides)

Treatment: primary resistant to cefalosporines
Liht urinary tract infection: ampicillin, ampicillin with β–lactamase inhibitors, nitrofurantoin, possible glycopeptides.
Wound infections, sepsis and endocarditis: combination of aminoglykoside + penicillin/ampicillin or glycopeptides (vancomycin, teicoplanin)

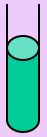
VRE (vancomycin resistant enterococci) – linezolid, quinupristin/dalfopristin

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

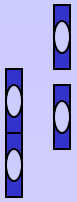
Corynebacterium difteriae

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

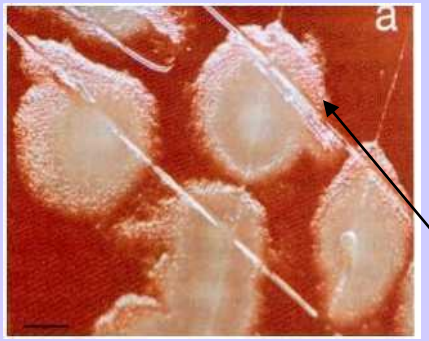
Other *Corynebacteria (C. jejkeium etc.)*

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*

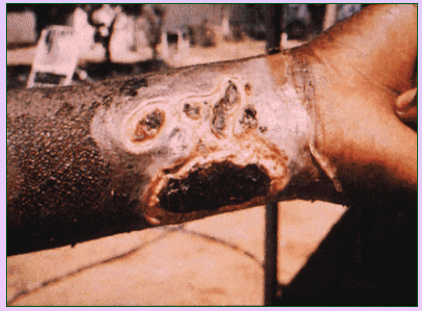


B. antracis



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.



B. cereus

Microscopy: G+rods, central terminated spores

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

Pathogenicity: 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

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