



**UNIWERSYTET MEDYCZNY
IM. PIASTÓW ŚLĄSKICH WE WROCŁAWIU**

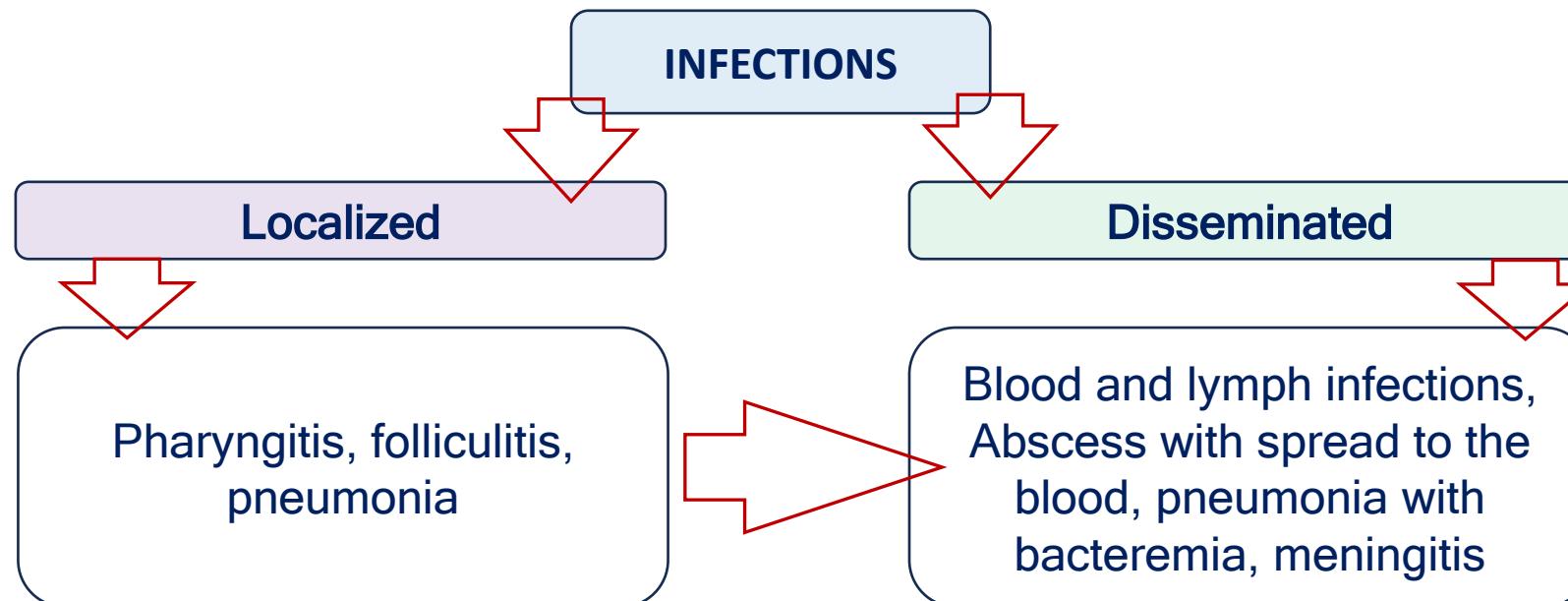
Przedmiot: Mikrobiologia (1)
Temat: Gram-positive cocci

Academic Year 2023/2024

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Wydział: Lekarski
Kierunek: Lekarski
Poziom studiów (jedn. mgr.)
Forma studiów (stacjonarne, niestacjonarne)
Rok studiów: II

Tytuł naukowy/zawodowy: prof. dr hab.
Imię, nazwisko osoby prowadzącej zajęcia: Beata Sobieszczańska
Stanowisko osoby prowadzącej zajęcia: profesor
Uniwersytet Medyczny im. Piastów Śląskich we Wrocławiu
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Endogenous infections:

Reservoir: human (body's microflora, part of the microbiota)

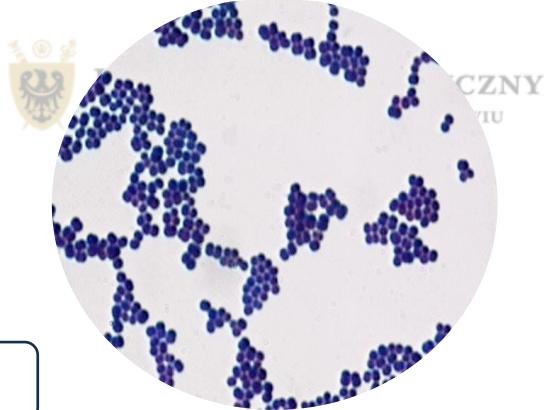
Source: human

They do not spread from person to person = **non-contagious**

Non-communicable vs. non-infectious diseases

Bacteria, viruses, fungi, parasites - infectious agents (infectious diseases): contagious or non-contagious

Non-communicable diseases are non-contagious - caused by non-infectious factors, e.g. diabetes, cardiovascular diseases



Family: Micrococcaceae

Genus: *Micrococcus*

Genus: *Staphylococcus*

Względnie beztlenowe

Species: *S. aureus*

Species: *S. epidermidis*

Species: *S. saprophyticus*

Species/strains

Undemanding

Family: Streptococcaceae

Genus: *Streptococcus*

Species: *S. pyogenes*

Species: *S. agalactiae*

Species: *S. pneumoniae*

Species: *S. orale/viridans*

Species/strains

Fastidious

Genus: *Enterococcus*

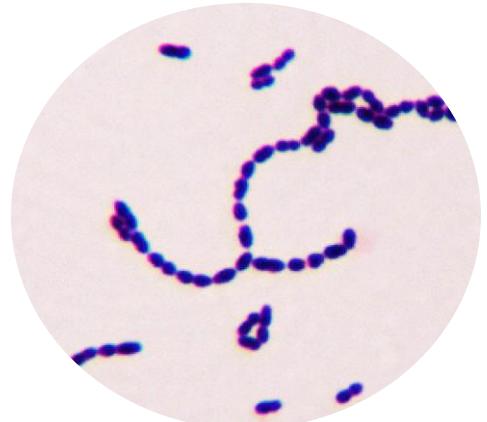
Species: *E. faecalis*

Species: *S. faecium*

Species/strains

Family: Peptostreptococcaceae

Genus: *Peptostreptococcus*



Genus: Staphylococcus

Coagulase-negative

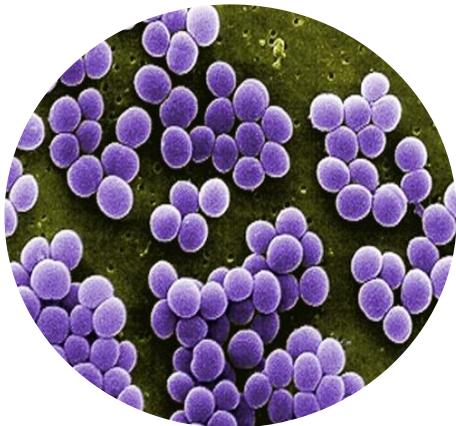
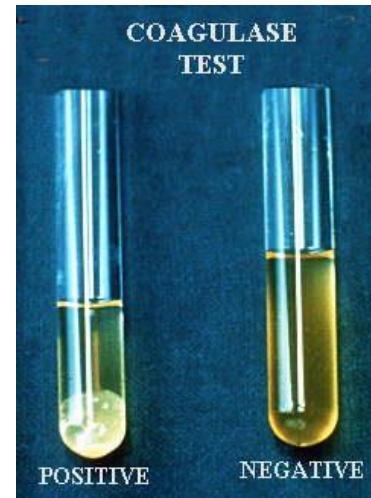
S. epidermidis

S. saprophyticus

Endogenous flora

Coagulase-positive

S. aureus

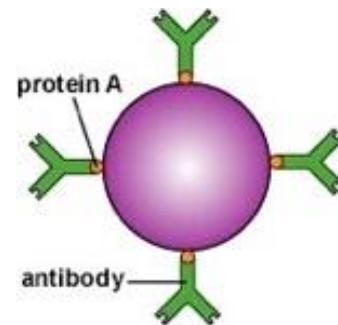


Virulence factors

S. aureus

Cell-bound

Peptidoglycan (LTA)
Coagulase
A protein
Glycocalyx



Extracellular

Catalase
Coagulase
Fibrinolysin
Hyaluronidase
Hemolysins

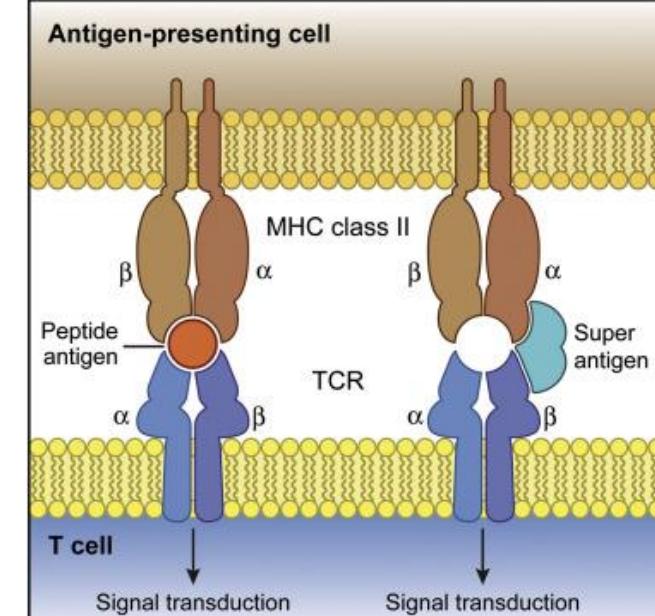
Toxins:

Panton-Valentine Leukocidin (PVL)
Epidermolytic toxins (exfoliating toxins)
Enterotoxins
TSST-1 toxin

Weakened immune response

SUPERANTIGENS

Excessive activation of lymphocytes, secretion of cytokines "cytokine storm" (shock)



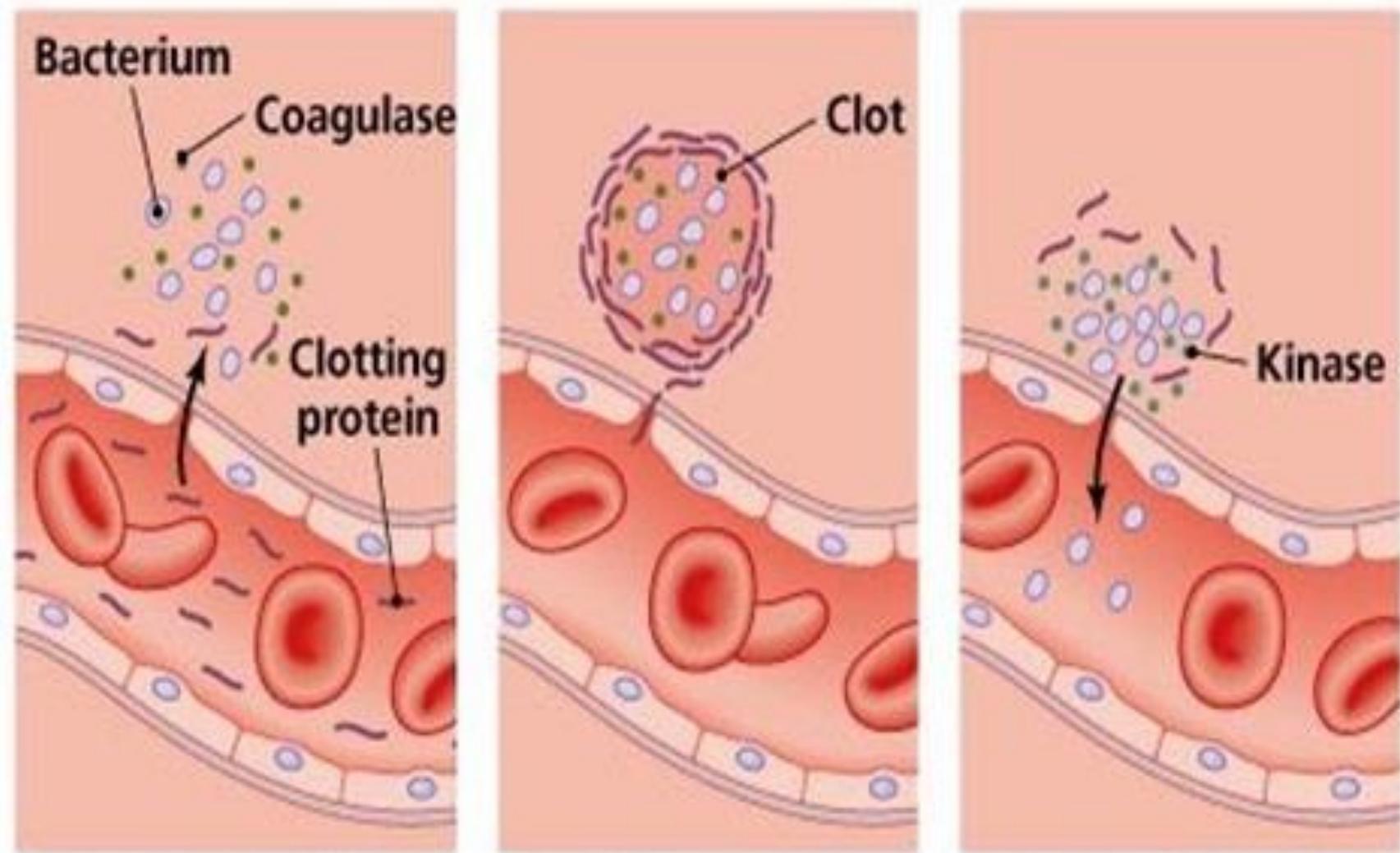
Coagulase - role in the formation of abscesses - a characteristic feature of *Staphylococcus aureus*

LNY
U

Reservoir: humans

Source: diseased humans,
carriers

Transmission: direct and
indirect contact, food-borne,
air-borne



Bacteria produce
coagulase

Clot forms

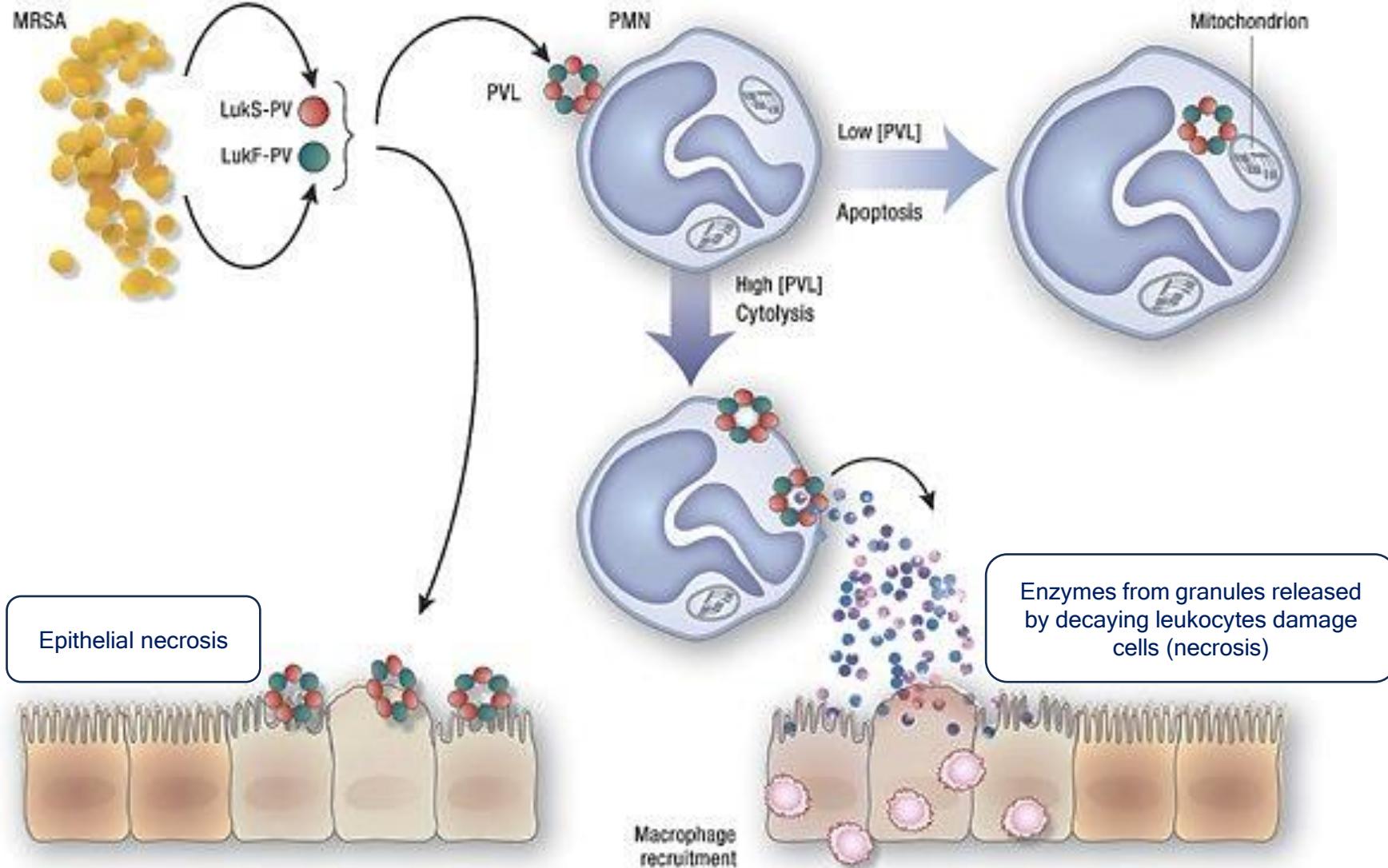
Bacteria later produce
kinase, dissolving clot
and releasing bacteria

Leukocidin (leukotoxin) Panton-Valentin (PVL) - role in tissue necrosis



Secreted PVL (in the form of subunits) forms a heptamer that is incorporated into leukocyte membranes

Produced by *S. aureus* strains causing community-acquired infections
Often due to CA-MRSA (community-acquired MRSA), i.e., *S. aureus* strains resistant to β -lactam antibiotics



Localized purulent infections

Folliculitis
Impetigo
Abscesses, furuncles, carbuncles, cellulitis
Wound infections; necrotizing fasciitis
Osteomyelitis
Pneumonia (also necrotizing pneumonia)
Lung abscesses
Otitis media, sinusitis
Urinary tract infections

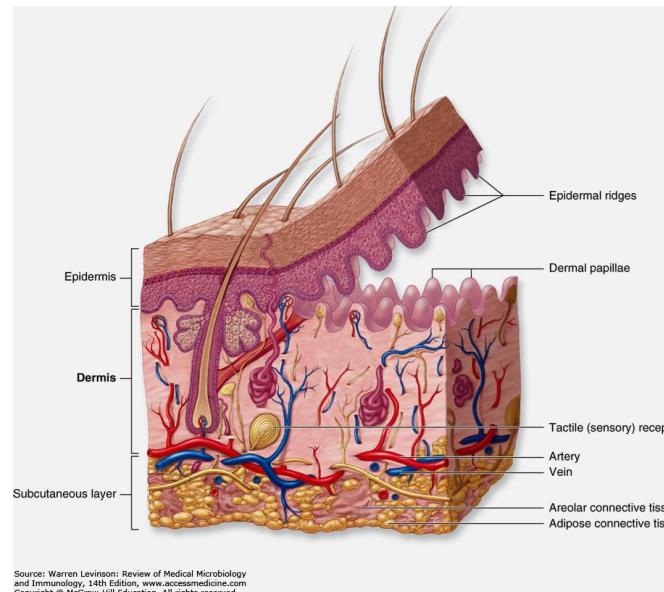
S. aureus

Superantigens-associated diseases

Food poisoning - enterotoxins
Scaled skin syndrome,
 (Reiter-Lyell disease), **bullous impetigo** - exfoliating toxins
Staphylococcal toxic shock syndrome - TSST-1

Disseminated infections

Blood infections (bacteremia, sepsis, septic shock)
Endocarditis
Meningitis
Brain abscesses



Genus: *Staphylococcus*

Coagulase-negative

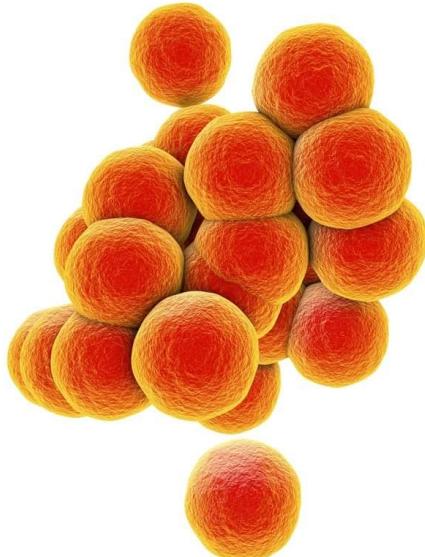
S. epidermidis

S. saprophyticus



Endogenous flora

Potential pathogens



S. saprophyticus

Urinary tract infections (UTI)

in young women (often recurrent)

Treatment: nitrofurantoin, TMP-SMX
amoxicillin/clavulanate, cefaclor,
fosfomycin, fluoroquinolones (ofloxacin,
ciprofloxacin, levofloxacin)

S. epidermidis

Catheter-related bacteremia

(30% of nosocomial bacteremia)

(can induce shock, meningitis, endocarditis,
pneumonia, osteomyelitis, phlebitis)

Postoperative endocarditis

Osteitis after implantation of prostheses

Bacteremia after infection of burn wounds

Treatment as for *S. aureus* infections

Gronkowce

Treatment

Groups of antibiotics:

Strains susceptible to β -lactams (MSSA): cloxacillin (drug of choice), amoxicillin/clavulanic acid, cephalosporins (I - IV), macrolides, tetracyclines, clindamycin, TMP-SMX

Strains resistant to β -lactams (MRSA): glycopeptides, lincosamides, daptomycin, ceftaroline, rifampicin (always in combination), tigecycline, tetracyclines, clindamycin, TMP-SMX, fluoroquinolones, aminoglycosides

Localized infections

Infections of the skin and soft tissues, bones, joints, and lungs (depending on the severity of the infection - outpatient vs. hospitalized, depending on whether the strain is sensitive or resistant to β -lactams)
risk of dissemination into the blood

Disinfection, ointments with mupirocin, fusidic acid, retapamulin), TMP-SMX, clindamycin - **local or oral treatment**

Oral or parenteral antibiotics: isoxazolyl penicillins (nafcillin, cloxacillin), amoxicillin/clavulanic acid, cephalosporins, clindamycin, TMP-SMX, tetracyclines, macrolides, fluoroquinolones

Localized infections disseminated to blood

Parenteral: isoxazolyl penicillins (cloxacillin), cephalosporins, glycopeptides, daptomycin, Linezolid, ceftaroline, rifampicin, tigecycline, clindamycin, TMP-SMX, aminoglycosides, fluoroquinolones

Streptococci

Serologic groups: A, B, C, D, E, F, G ...

Hemolysis alpha

S. orale (viridans)
S. pneumoniae

Hemolysis beta

S. pyogenes A (GAS)
S. agalactiae B (GBS)

Hemolysis gamma

Enterococcus

***S. viridans* group** (many species colonizing the oral cavity: *S. mitis*, *S. mutans*, *S. salivarius*, *S. sanguis*, etc.)

Physiological microflora of the oral cavity

Endogenous infections: caries, mixed infections in the oral cavity (abscesses), subacute endocarditis

Enterococcus (*E. faecalis*, *E. faecium*)

Physiological intestinal microflora
Endogenous infections: urinary tract infection, subacute endocarditis, catheter-related bacteremia; less frequently: intra-abdominal infections, post-operative wound infections

An important "nosocomial pathogen,"
Resistant to many antibiotics

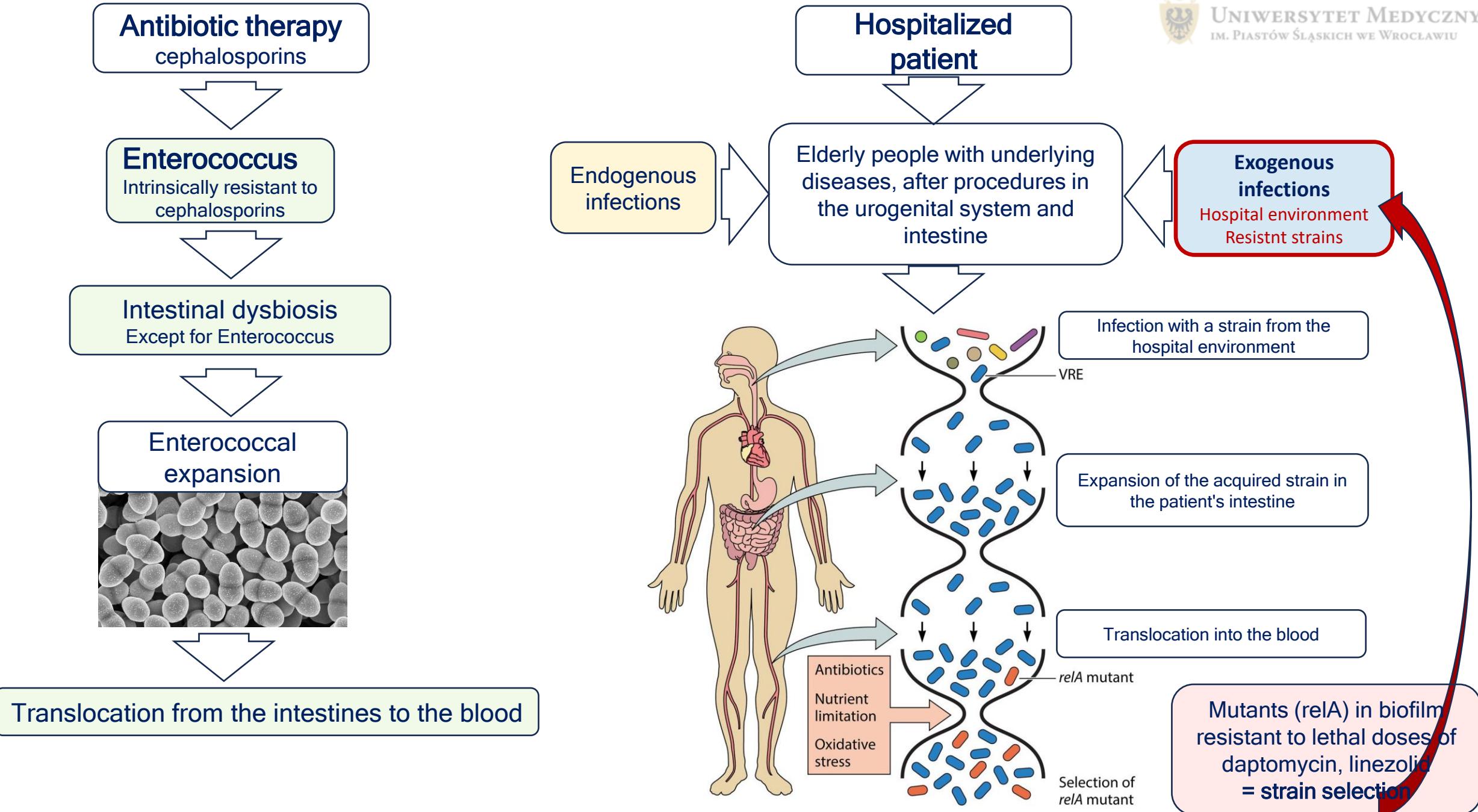
Treatment of *E. faecalis*: penicillins (ampicillin, piperacillin), glycopeptides, Linezolid, tigecycline, fosfomycin, chloramphenicol.
E. faecium: glycopeptides, daptomycin, tigecycline, Linezolid, rifampicin, fosfomycin

Streptococcus bovis (grupa D)

Physiological microflora of the large intestine

Endogenous infections: colon cancer, bacteremia, biliary tract disease, fulminant sepsis, and meningitis in newborns





Streptococcus pyogenes (GAS)

Reservoir: humans

Source: diseased humans, carriers

Transmission: direct contact, air-borne

route, rarely: food-borne (milk)

Carriage: pharynx (rectum)

Czynniki wirulencji



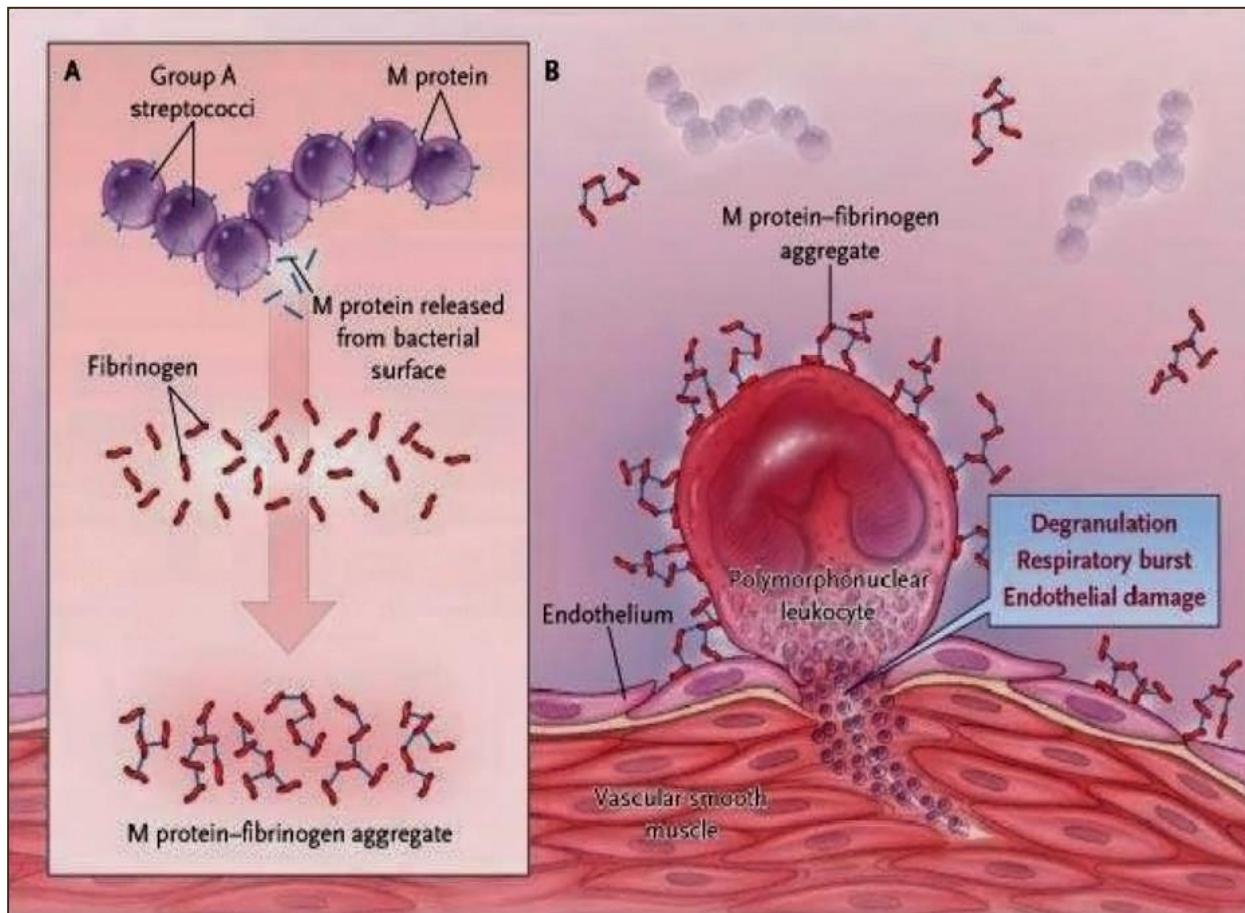
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Cell-bound

Peptidoglycan (LTA)
M protein
Capsule (hyaluronic)

Extracellular

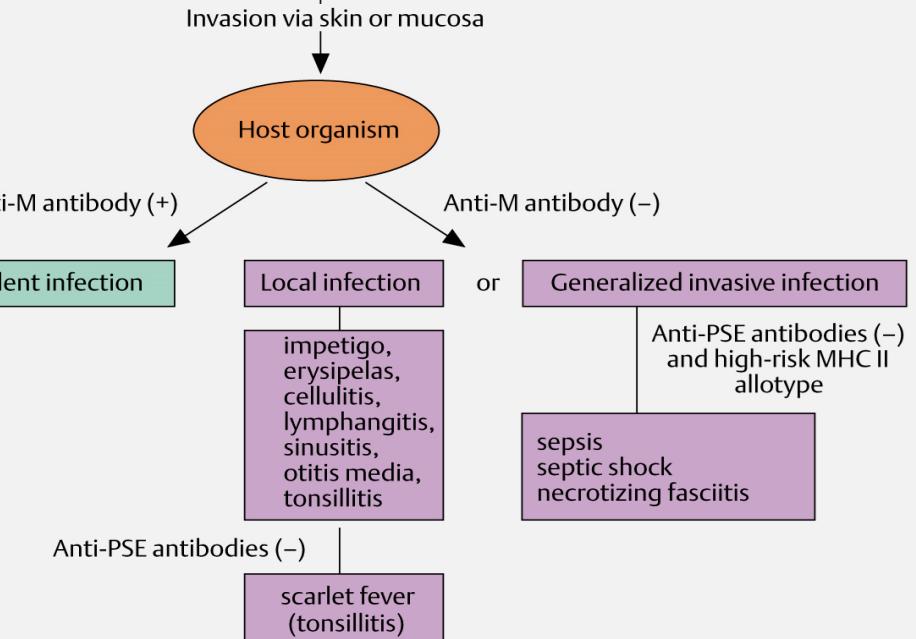
Hyaluronidase
Streptolysin O
Streptokinase
Pyrogenic exotoxin (SPE) Superantigen



**Streptococcal toxic shock syndrome
Scarlet fever (scarlet fever)
Necrotizing fasciitis**

Streptococcus pyogenes Infections

S. pyogenes (M protein, PSE, other pathogenicity factors)





Primary streptococcal infections

Tonsillitis (strep pharyngitis)

The head: otitis media, sinusitis, peritonsillar abscesses

Skin and soft tissue infections: cellulitis, purulent skin infections, impetigo, erysipelas

Respiratory system: pneumonia

Bones, joints: purulent infections

Systemic: blood infections (bacteremia), puerperal fever, post-traumatic meningitis, endocarditis

Pharyngitis

Infections involving erythrogenic toxin:

1. Necrotizing fasciitis
2. Scarlet fever
3. Streptococcal toxic shock syndrome

Scarlet fever

Skin infection

Necrotizing fasciitis

Complications after streptococcal infections immunological

After streptococcal pharyngitis:

- 1) Rheumatic fever
- 2) Acute rheumatic heart disease

Pathomechanism: autoantibodies against M protein

After skin infections:

- 3) Glomerulonephritis

Pathomechanism: immune complexes

Erysipelas

Rheumatic fever

Treatment:

Penicillin (benzyl or phenoxyethyl)
- drug of choice!

Clindamycin

Macrolides (except for azithromycin!)

Invasive infections - combined therapy:
penicillin + clindamycin
penicillin + linezolid



Clindamycin: inhibits the production of erythrogenic toxin, the synthesis of M protein

Reservoir: humans

Source: diseased humans, carriers

Transmission: direct and indirect contact, air-borne route

Streptococcus pneumoniae

Localized purulent infections:

Sinusitis
Otitis media
Pneumonia

Invasive infections (invasive

serotypes - mortality 15-25%):
meningitis, brain abscesses,
bacteremia, sepsis,
endocarditis

Treatment:

Penicillins + BLI, cephalosporins (II-IV gen.), carbapenems,
glycopeptides, daptomycin, tigecycline, linezolid, fluoroquinolones,
chloramphenicol, rifampicin, clindamycin, macrolides, tetracyclines,
TMP-SMX

Virulence factors



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LTA

Capsule (serotypes)

Hyaluronidase

IgA protease

Pneumolysin

Specific prevention - protection against invasive infections

Conjugate vaccine for children (> 6 years of age) and adults (> 50 years of age) - capsular antigen with protein (13-valent)

Polysaccharide vaccine (23-valent) for children (over 2 years of age) and adults (>50 years of age)

Streptococcus agalactiae (GBS)

Vaginal colonization in 5-30% of healthy women

Neonatal infections (1%):

sepsis (early onset),

meningitis (late onset)

Neurological complications

Pregnant women: puerperal fever, endometritis, bacteremia, endocarditis, cellulitis, abscesses, toxic shock-like syndrome

Infections in the elderly: bacteremia, pneumonia, endocarditis, osteoarthritis, pressure wound infections, diabetic foot, prostate disease, UTI

Treatment:

Penicillins (penicillin G, ampicillin, amoxicillin)

Macrolides (erythromycin)

Clindamycin

Glycopeptides

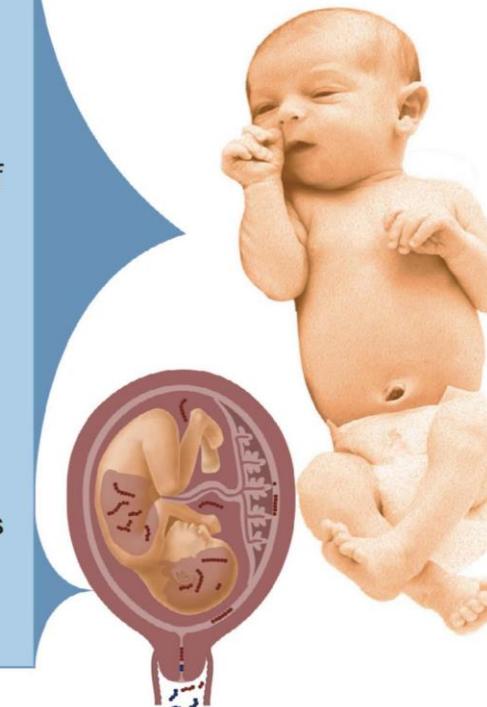
Daptomycin

Virulence factors

Capsule
Hemolysin
Pyrogenic toxin
(uncharacterized)

GBS Early-Onset Disease (EOD)

- Onset in first week of life
- Leading cause of sepsis in neonates
- Can also cause pneumonia
- meningitis
- Acquired by fetus in utero or during birth



Perinatal prevention

Vaginal swab in pregnant women 36/37 - 38th week of pregnancy

Positive results - parenteral penicillin or ampicillin 4 h before labor or if there is no data on the test, no test, GBS infection in a previous pregnancy, bladder infections, or fever during pregnancy

GBS Late-Onset Disease (LOD)

- Usually occurs 1-4 weeks after birth, but may occur up to 3 months of life
- Wider clinical spectrum
- May cause
 - meningitis
 - sepsis
 - septic arthritis
 - osteomyelitis
 - pneumonia

Reservoir: humans (physiological flora of the intestines, urogenital system)
Source: humans (endogenous infections)



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beata.sobieszczanska@umw.edu.pl