

# Streptococcus pyogenes

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***Streptococcus pyogenes*** is G+, facultatively anaerobic,  $\beta$ -hemolytic coccus, which grows in pairs or forms chains. He belongs to *Group A* (according to Lancefield). It is primarily pathogenic to humans and is the only natural source of infection. It is the cause of *infections of the respiratory tract, skin, systemic infections* and the cause of post-streptococcal sterile sequelae.



## Properties

### Virulence factors

#### Surface virulence factors

##### Group A polysaccharide antigen

Wall polysaccharide based on N-acetylglucosamine and rhamnose, which determines belonging to group A (serological classification according to Lancefield). Antibodies that form against it can attack the surface of the heart valves. It can be detected in a throat swab using a rapid test (so-called Streptest) already in the office and thus help the doctor identify the cause of the infection and initiate appropriate treatment.

##### M-protein

It is the main antigen determining the virulence of the strain. It is a fibrous structure protruding from the bacterial wall, there are different serotypes. It allows **adhesion** to the mucosa and **protects** bacteria **from phagocytosis**. It is associated with the development of rheumatic fever or glomerulonephritis (probably due to antigenic similarity to the membrane of the renal glomeruli). It has anti-phagocytic properties - it contains sites for the binding of the regulatory factor complement in H and fibrinogen. Inhibits the complement cascade.

##### F-protein and other adhesive molecules

They mediate attachment to different cells. F-protein interacts with fibronectin (binding to airway epithelium).

##### sheath

Hyaluronic acid (identical to tissue), anti-phagocytic properties, "mimicry", mucoid colonies.

#### Extracellular virulence factors

##### Erythrogenic (pyrogenic) toxins

They cause *Scarlet fever* and function as superantigen. They over-stimulate the cells of the immune system, there is a rise in temperature, overproduction of various mediators of inflammation, suppression of the antibody response, etc., toxic shock may occur. They increase sensitivity to endotoxin.

##### Streptolysin O

- It destroys cell membranes, killing them.
- In plasma, the activity is blocked, antigenic.
- It has cardiotoxic properties.
- Causes **lysis of white bloodline** (leukocytes, monocytes).
- It contributes to the emergence post-streptococcal rheumatic fever.
- **ASLO** - antistreptolysin O, antibodies, to a retrospective diagnosis of a recent streptococcal infection and to a possible post-streptococcal consequence.

##### Streptolysin S

It is responsible for  $\beta$ -hemolysis on blood agar. It kills leukocytes that phagocytose streptococcus, non-immunogenic.

##### Streptokinase

There are enzymes that activate fibrinolysis and allow penetration into tissues. They used to be used for treatment of thrombosis.

##### Hyaluronidase

It is another enzyme that facilitates spread in tissues.

## Deoxyribonuclease

It cleaves DNA from broken cells. Antibodies are formed against it, which can be detected and are therefore used for diagnosis.

## C5a-peptidase

It is an enzyme that prevents complement from functioning properly.

## Diseases

Children with young adults (about 10% of the population) often have asymptomatic carriers. However, the presence of streptococcus is limited by the natural microflora and antibodies against protein M in the bacterial wall.

### Respiratory diseases

- **Sleep angina** (lat. *tonsillitis acuta*) – makes up about 10-30% of pharyngitis (the rest is caused mainly by viruses)<sup>[1]</sup>, the disease is accompanied by general symptoms, inflammation of the tonsils with coxs, sore throat and runny nose, cough is not present.
- **Scarlet fever** (lat. *scarlatina*) – disease occurring on first contact with *S. pyogenes* (mostly in children) associated with a rash.
- **Inflammation of the middle ear** (lat. *otitis media*).

### Skin diseases (pyoderma)

- **Impetigo** – skin inflammation with blisters and scabs often affecting children.
- **Růže** (lat. *erysipelas*) – inflammation of the skin characterized by red spots of the characterphlegmony (unbounded inflammation) with general symptoms.

### Deep tissue diseases

- **Celulitis** (inflammation of the subcutaneous tissue), which may progress to **necrotizing fasciitis** (life-threatening muscle ligament infection requiring surgery).

### Invasive and toxic diseases

- **TSLS** (*toxic shock-like syndrom*) – toxic shock syndrome;
- necrotizing fasciitis;
- sepsis, meningitis.

### Sterile consequences

The so-called sterile sequelae are diseases that occur after streptococcus is no longer present. The cause of their formation is complex, they probably involve the effect of some bacterial products (eg streptolysin O and S) and affect the patient's immune system. The disease is caused by the similarity of bacterial antigens to host antigens and due to the deposition of immunocomplexes, ie antigen-antibody complexes, in tissues. The body then begins to damage its own tissues. These include:

- **Rheumatic fever** (lat. *febris rheumatica*) – joint injuries, heart at risk of valve defects and brain (*chorea minor* - motor disorder, so-called St. Vitus dance). It occurs about 3 weeks after untreated streptococcal angina.
- **Glomerulonephritis** - kidney damage caused by deposition of immunocomplexes in the basement membrane of glomeruli and activation of complement. There is pain in the hips, blood and proteins in the urine and blood pressure disorders.

The development of these consequences can be prevented by timely and properly managed antibiotic treatment.

### Carrier Streptococcus pyogenes

The prevalence of "Streptococcus pyogenes" in the pediatric population is 15 to 20%, depending on the region, and lower in the adult population. Asymptomatic carriers are not at risk of developing suppurative or non-suppurative complications and are not considered an essential reservoir for the spread of streptococcal infection. Therefore, there is no need to identify or treat these asymptomatic carriers. It is not recommended to perform control cultures after therapy (*cost-benefit*).<sup>[2][3]</sup>

## Therapy



Impetigo

*S. pyogenes* is well sensitive to antibiotics, the drug of first choice is penicillin. In the case of penicillin allergy, treatment with macrolide antibiotics (eg clarithromycin) or lincosamides is possible. If the infection is severe, accompanied by tissue necrosis, surgery is appropriate.

## Prevention

Vaccine against infections caused by *S. pyogenes* is not currently available.

## Diagnostics

Diagnosis is performed by culture, microscopy and serology. Can be supplemented with biochemical and bacitracin tests.

- Microscopy - G + cocci with chains, do not sporulate.
- Cultivation - on blood agar grows in small colonies with a zone of complete  $\beta$ -hemolysis (clearing).
- Serologically - detection of C, M and T antigens (see above).
- Bacitracin test - *Str. pyogenes* does not grow in the presence of 0.04  $\mu$ l bacitracin. Thus, group A streptococcus can be distinguished from other groups.
- Catalase test - negative.

## Photo gallery



Streptococcus  
pyogenes-blood  
agar-detail  
hemolyzy.jpg

Streptococcus  
pyogenes on blood  
agar

Streptococcus  
pyogenes, detail of  
 $\beta$ -hemolysis

## Links

### related articles

- **Streptokokové infekce:** Group A streptococcal infection • Scarlet fever • Burn sore throat • Erysipelas • Impetigo • Infections caused by virulent streptococci • Complications and treatment of streptococcal infections • Rheumatic fever

### External links

- Streptococcus pyogenes (česká wikipedie)
- Streptococcus pyogenes (anglická wikipedie)

### Reference

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### Source

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