

EPIDEMIOLOGICAL CHARACTERISTICS OF STREPTOCOCCUS SUIIS INFECTION

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Streptococcal infections have a significant impact on pig farming worldwide, including in Ukraine, resulting in significant economic losses for livestock farmers. Streptococcus is considered one of the primary zoonotic diseases in pigs, often causing meningitis, septicemia, or arthritis, and can also be dangerous for humans. For many years, experts have managed to control the spread of this infection quite effectively. However, in recent years, due to antibiotic resistance, this infection is becoming more widespread. Given the importance and danger of this pathogen for pigs, this problem is still relevant in pig farming. The main etiological factor of streptococcal infections in pigs is Streptococcus suis (S. suis), which are gram-positive bacteria that are either aerobic or facultatively anaerobic. Most streptococci have capsules, do not form spores, and cannot move. The structure of the streptococcal antigen is complex and consists of three primary components, namely group-specific antigen, type-specific antigen, and nuclear protein antigen. β -type hemolytic streptococcus has highly pathogenic properties, and there are 35 serotypes based on different characteristics of the capsular antigen. S. suis serotype 2 is commonly isolated from clinically ill piglets and is considered the most virulent subtype of the organism. The pathogenicity of this bacterium for animals is related to its primary pathogenic factors, including the capsule, toxin, and enzymes.

S. suis is widespread, and newborn piglets, suckling piglets, and pregnant sows are most susceptible to infection, while adult pigs are more resistant. S. suis tends to peak during weaning and mating, and piglet susceptibility to S. suis decreases with age. S. suis occasionally infects dogs, cats, rodents, cattle, sheep, and horses. It can also infect humans and has become a serious endemic threat to public health. The primary modes of transmission are alimentary and respiratory infections. People typically become infected by contacting sick animals. Infected animals often show symptoms such as pneumonia, wet dermatitis, meningitis, peritonitis, osteomyelitis, arthritis, and pharyngitis, among others. Outbreaks of the disease caused by S. suis usually occur throughout the year without any significant seasonal patterns.

Key words: streptococcosis, S. suis infection, etiology, epidemiology.

DOI <https://doi.org/10.32782/bsnau.vet.2023.1.1>

Introduction. In the swine industry, streptococcosis is considered a major infectious disease that often co-occurs with meningitis, septicemia, or arthritis. These diseases result in significant financial losses for the pig business and are significant factors in the need to treat large herds of pigs with antibiotics as a preventative measure. It has been established that *Streptococcus suis* (S. suis), first reported in 1954, is the etiological agent for this type of persistent bacterial illness (Gottschalk, Segura, & Xu, 2007; Hughes et al., 2009). S. suis can

also infect humans and has become a serious endemic public health threat.

S. suis is a gram-positive bacterium, aerobic or facultative anaerobic. The bacteria are 1~2 μm in diameter, single or double, oval in shape, and present long chains in liquid medium. The longer the chain, the stronger the pathogenicity (Segura, Fittipaldi, Calzas, & Gottschalk, 2017). Most streptococci have visible capsules in young cultures and do not have flagellates, form spores, or move. The colonies are small, pale, transparent, and slightly sticky.

Streptococcus produces obvious β -type hemolysis on the blood plate. The colonies are round, moist, smooth, and translucent after being cultured at 37 on the blood Martin agar plate for 24h. *Streptococcus suis* produces α or β hemolysis on the blood plate, usually α hemolysis first and β hemolysis after delayed culture, or there is no hemolysis around the colony. Scraping the colony can reveal alpha or beta hemolysis. *Streptococcus suis* type 2 shows alpha hemolysis on sheep blood plate and beta hemolysis on horse blood plate.

The structure of streptococcus antigen is complex, with three main components: group-specific antigen, type-specific antigen, and nuclear protein antigen (Collin & Ehlers, 2013). (1) Group-specific antigen (C antigen), a polysaccharide component in the cell wall of the hammer bulb, has group specificity and is haptens, with an antigenic determinant of amino carbohydrate. Based on this, the Lancefield method divides streptococcus into 20 serogroups according to different antigens. (2) Type-specific antigen, a protein component of streptococcus cell wall, is located on the surface of C antigen, so it is also called surface antigen (Feng et al., 2014). The antigen can be divided into four components: M, T, R, and S, among which the M component is related to bacterial virulence, has anti-phagocytic activity, and is related to immunity. Based on the different M proteins, the bacteria can be classified into their respective groups. For example, group C can be divided into more than 20 types, group D into 10 types, and group E into 6 types. (3) The nucleoprotein antigen, also known as P antigen, is a non-specific antigen. The P antigen of various streptococci has the same property and is the main component of bacteria, without group or type specificity.

According to the hemolysis phenomenon, streptococci can be divided into α , β , and γ streptococci. α -type hemolytic streptococci have weak virulence and are more conditional pathogenic bacteria. β -type *Streptococcus haemolyticus* is highly pathogenic, while γ -type *Streptococcus haemolyticus* is generally not pathogenic.

Based on the distinctive characteristics of the capsule antigen, bacteria can be classified into 35 serotypes (types 1-34 and 1/2) (Okwumabua, Williamson, Pearson, & Sahl, 2020). Serotype 2 of *S. suis* is commonly isolated from sick piglets and is believed to be the most virulent subtype of the organism. *Streptococcus* has a relatively active biochemical reaction and can ferment lactose, sucrose, trehalose, and heptoxin, but does not ferment mannose and arabinose. The pathogenicity of this bacterium to animals is associated with its main pathogenic factors, including the capsule, toxin, and enzymes (T. Li & Yu, 2019).

The classical virulence factor of *S. suis* 2 (SS2) is the capsular polysaccharide (CPS). CPS is a typing marker of SS2 and an important protein and virulence factor for SS2 to resist phagocytosis by macrophages. The capsule mainly comprises N-acetylneuraminine, galactose, glucose, rhamnose, and N-acetylglucosamine (T. Li & Yu, 2019; M. Liu, Xia, Liu, & Kasianenko, 2021). Compared to the wild strain, the CPS deletion strain's resistance to phagocytosis and macrophage killing was significantly reduced. CPS not only contributed to SS2 strains' resistance to phagocytosis-

mediated killing but also played a significant role in evading trapping and further killing by neutrophil extracellular traps (NETs) (J. Zhao et al., 2015).

Sulysin (Sly) is a cholesterol-dependent pore-forming cytotoxin found in most virulent strains of SS2. It is expressed in the medium and secreted in the supernatant. The mature Sly protein has a molecular weight of approximately 54kDa. However, Sly loses its hemolytic activity after oxidation, and its hemolytic activity can be inhibited by cholesterol (Lv et al., 2014). Sly plays a crucial role in damaging various cells and the process of SS2 invading the central system and destroying the blood-brain barrier (BBB). Furthermore, Sly can induce changes in host cytoskeleton and the release of pro-inflammatory and immunomodulatory cytokines and chemokines such as interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF- α), and interleukin-10 (IL-10) (Vötsch et al., 2019).

Muramidase-released protein (MRP) and Extracellular factor (EF) are two important virulence factors of *Streptococcus suis* 2 (SS2). MRP is a 136 kDa cell wall protein that is released after the action of bacterial enzymes on virulent SS2. Research has found that MRP may play a significant role in crossing the blood-brain barrier (BBB) and binding to the host cell, and the variable regions of MRP are involved in virulence (Q. Li, Fu, et al., 2017). On the other hand, EF is an extracellular protein with a molecular weight of approximately 110kDa that is encoded by the epf gene. EF is a secreted protein and can only be detected in the supernatant of the culture medium. Both MRP and EF are related to the virulence of SS2, but natural strains lacking MRP and EF can still be pathogenic. It may be that the strains expressing MRP and EF are more virulent than the strains without MRP and EF (Guo et al., 2021). However, both MRP and EF can be expressed in SS2 strains isolated from diseased pigs in China, while the non-pathogenic strains did not express MRP and EF (J. Wang et al., 2017).

Fibronectin- and fibrinogen-binding protein (FBPS) is an unanchored adhesion protein that enhances the colonization ability of *Streptococcus suis* (S. Zhang et al., 2016). FBPS consists of two domains with unique folds, where the C-terminus binds to host cells through fibronectin, while the N-terminus attaches to the bacterial surface, promoting the adhesion of SS2 to host cells (Xia et al., 2019). FBPS deletion significantly reduces the virulence of the strain compared to the wild-type strain (M. Liu et al., 2021). FBPS can also activate the signaling pathway through the β 1 integrin receptor to induce chemokine production (Musyoki et al., 2016).

Another novel virulence factor is the **surface secretory component, H-factor binding protein (FhbP)**. Factor H is a vital regulator of the complement pathway, and many pathogens recruit factor H to their surface to reduce complement-mediated killing and enhance adhesion and invasion of host cells. The first H-binding protein discovered was named Fhb. Fhb defective mutants were shown to be completely non-toxic and to have reduced survival in whole human blood or human neutrophils in pig infection models (Pian et al., 2012). Later, a second factor H-binding protein, named Fhbp, was reported. Pre-incubation of

eight SS2FhbP proteins with FhbP polyclonal antibodies significantly reduced the ability of SS2 to bind H factor (Q. Li, Ma, et al., 2017). Studies have shown that the binding of H factor to cells enhances the adhesion and invasion of human laryngeal carcinoma epithelial cells (HEp-2) by SS2.

Capsular polysaccharide sialic acid. Sialic acid is a component of the capsular polysaccharide of *Streptococcus suis*. In comparison to wild-type strains, it has been found that these strains can damage the blood-brain barrier (BBB) and cause meningitis (Shi et al., 2012). In vitro studies with whole blood cells from mice have shown that the secretion levels of monocyte chemotactic protein-1 (MCP-1) and interleukin-6 (IL-6) in the sialic-deficient group were higher compared to those in the wild-type group, and the capsule sialic acid may inhibit the host's recognition and immune response to SS2. The substitution of α -2,6 sialtransferase in SS2 and SS14 by α 2,3-6 sialtransferase affects the synthesis of bacterial CPS (Roy et al., 2018), suggesting that sialic acid and its linking form (α -2,6 bonds) affect *Streptococcus suis* virulence by affecting the synthesis of CPS.

S. suis serine-rich repeat protein 1. Serine-rich repeat protein (SRRP) plays a crucial role in the pathogenicity of *Streptococcus suis*. *S. suis* serotype Chz has a secreted protein encoded by a 50K gene island, named *S. suis* serine-rich repeat protein 1 (SssP1) (Y. Zhang et al., 2018). SssP1 contains 4,647 amino acid residues and is composed of 3 non-repeating regions and 3 repeating regions, making it the longest SRRP discovered in recent years. Animal infection tests using mouse models have shown that the median lethal dose of the sssP1 gene deletion strain (Δ sssP1) was significantly increased compared to the wild-type strain. Moreover, Δ sssP1 exhibited reduced adhesion to HEp-2 cells and human microvascular endothelial cells (HBMECs). This novel pathogenic mechanism mediated by SssP1 protein indicates its importance in *Streptococcus suis* virulence.

Heme-binding protein SntA: The outer membrane protein SntA was identified as a cell wall-anchored heme-binding protein. Incubation of bacteria with human serum showed that the surface C3 deposition and membrane attack complex (MAC) of the SS2 SntA gene deletion mutant (Δ sntA) was significantly higher than that of the parent and complementary strains. The Δ sntA strain showed reduced anti-phagocytosis, blood survival, and in vivo colonization in mice infection (Deng et al., 2018). SntA can interact with C1q, one of the components of complement C1, and inhibit complement activation through classical pathways. SntA can also lead to complement depletion through classical and lectin pathways, thereby mediating complement escape. The SntA protein is involved in the acquisition of heme by SS2. In vivo infection tests in pigs have shown that the parent strain causes more severe symptoms than the Δ sntA strain. Δ sntA is a newly discovered protein responsible for the virulence of *Streptococcus suis*.

Pilin: The srtBCD gene cluster in *S. suis* is considered a pili-associated cluster, which contains three sorting enzyme genes (srtB, srtC, and srtD) and several pili protein genes (sbp1, sbp2, sbp3, sbp4). SBP1 is expressed in highly virulent SS2 chrysanthemum strains and is related to the

adhesion of SS2 to HEp-2 cells (Qian et al., 2018). SBP1 does not affect SS2 invasion of HEp-2 cells, macrophage-mediated phagocytosis, and bacterial survival in RAW264.7 cells (mouse macrophages). Deletion of the sbp1 gene does not reduce the lethality of SS2 to zebrafish and mice; sbp2 gene was only widely found in the highly pathogenic strains of SS2 (Yu et al., 2016). The recombinant protein SBP2 can also adhere to HEp-2 cells, and SBP2 can bind laminin (LN) and fibronectin (FN), suggesting that SBP2 may promote the adhesion process.

Oligopeptide-binding protein: The oligopeptide-binding protein (OppA) is an immunogenic protein expressed during the infection of piglets by the SS2 strain (F. Zheng et al., 2018). The OppA deletion mutant (Δ oppA) of SS2 exhibited slower growth than the wild-type strain. Adhesion of Δ oppA to human epithelial cells was significantly reduced compared to the wild-type strain. Mouse infection studies showed that OppA deletion significantly weakened the pathogenicity of highly virulent SS2 strains, suggesting that OppA plays an important role in SS2 infection. The OppA protein of SS2 can bind to laminin (LN) and fibronectin (FN), and the recombinant OppA protein can adhere to human HEp-2 cells, indicating that OppA may be involved in the adhesion process of SS2.

Enzymes: Dipeptidyl peptidase-4 (DPP-4) is an essential virulence factor in SS2. SDS-PAGE analysis has revealed that *Streptococcus suis* DPP-4 exhibits hydrolytic activity against porcine antimicrobial peptide PR-39 protein, indicating its role in bacterial resistance to host immune defenses (LeBel et al., 2018). Addition of recombinant DPP4 to growth medium resulted in decreased sensitivity of porcine frontalong to PR-39. Stimulation of HBmecs with PR-39 led to increased secretion of the chemokine interleukin-8 (IL-8). However, pretreatment of PR-39 with DPP-4 prevented the increase in IL-8 secretion, suggesting that DPPIV produced by *S. suis* can degrade PR-39 and reduce its bactericidal and immunomodulatory effects.

5'-Nucleotidase enzyme: The 5'-nucleotidase enzyme of *Streptococcus suis* has been found to convert 2'-deoxyadenosine monophosphate to 2'-deoxyadenosine and synthesize adenosine in the blood of mice (Dai et al., 2018). 2'-deoxyadenosine can induce caspase-3 dependent death in mouse macrophages, and in vivo infection experiments have shown that the synthesis of 2'-deoxyadenosine by the 5'-nucleotidase enzyme causes mononucleosis in the blood of mice. Transcriptome analysis of blood components in mice infected with virulent and defective strains revealed that 5'-nucleotides may suppress neutrophil function and immune response through adenosine-mediated suppression. These findings suggest that *S. suis* inhibits the host immune response through 5'-nucleotidase-mediated syntheses of 2'-deoxyadenosine and adenosine, which is a newly discovered pathogenic mechanism in *S. suis*.

Serine/threonine kinase (STK) is involved in regulating bacterial stress response, biofilm formation, cell wall biosynthesis, development, metabolism, and virulence. Studies have shown that the ability of a SS2 strain with a defective stk gene (Δ stk) to adhere to and invade

HBMECs and mouse brain microvascular endothelial cells (bEnd.3 cells) and cross the blood-brain barrier (BBB) was significantly reduced compared to the wild-type strain (C. Zhang et al., 2017). The wild-type strain was found to infect claudin-5, which is a component of tight junctions in BBB, more efficiently than the Δ stk strain. However, no significant difference was observed in the Claudin-5 mRNA level in bEnd.3 cells infected by the wild-type strain and the Δ stk strain. In bEnd.3 cells infected with the Δ stk strain, the expression of ubiquitin E3 ligase was reduced by 1.5 times, indicating that STK may affect the expression of steroid D1 and promote the degradation of claudin-5 through ubiquitination regulation. This allows SS2 to pass through the BBB and cause infection.

Transcription factors and regulatory systems. VraSR Regulatory system. *Staphylococcus aureus* is capable of reacting to B-lactam and vancomycin (Yin, Daum, & Boyle-Vavra, 2006) via a phospho-transfer-mediated signaling pathway composed of histidine protein kinases (VraS) and response regulatory proteins (VraR). In *Streptococcus suis*, VraSR is a newly discovered binary regulatory system. A *urasR* gene deletion mutant (Δ vraSR) was constructed, which showed increased susceptibility to phagocytosis by human polymorphonuclear leukocytes (PMN) and sensitivity to oxidant and lysozyme (Chang et al., 2018). The virulence of the Δ vraSR mutant was significantly reduced in vitro and in vivo mouse infections. Therefore, VraSR is a key regulatory system that contributes to the survival of *Streptococcus suis* in the bloodstream and enhances resistance to host innate immunity.

The Spx protein is a widespread regulator in many bacteria, and two Spx proteins, SpxA1 and SpxA2, have been identified in SS2 (C. Zheng et al., 2017). Mutant strains, Δ sprA1 and Δ sprA2, were constructed, and it was found that SpxA1 helps SS2 resist oxidative stress, while SpxA2 helps SS2 resist sodium chloride and SDS stress. Both Spx regulatory proteins are also involved in the nutritional metabolism of SS2. In a mouse infection model, the two Spx regulatory proteins were shown to promote tissue colonization of SS2 and induce the host's synthesis of inflammatory factors. Comparison of the transcription profiles of wild and mutant strains showed that 165 and 404 genes were differentially expressed in Δ sprA1 and Δ sprA2, respectively, indicating that the Spx protein is a global regulatory factor.

Other virulence factors.

MurB-potABCD Operon: Role in Polyamine Transport and PG Synthesis

Polyamines play an important role in bacterial virulence, antibiotic resistance, biofilm formation, and innate immunity against the host. In *Streptococcus suis*, two polyamine transport systems, PotAB-CD and a bacterial adenosine triphosphate system, are involved in polyamine transport. PotA catalyzes hydrolysis of ATP to transport polyamines, while PotB and PotC form polyamine transport channels and PotD binds polyamines.

The acetylglucosaminolpyruvate reductase gene (*murB*) is also important in *S. suis*, as it is involved in peptidoglycan (PG) synthesis. Tests using SS2 parental strains and *potA*

gene deletion strains (Δ potA) revealed that PotABCD is responsible for polyamine uptake, with PotA being critical for energy production. Notably, the PG chain of Δ potA mutants was prolonged, and abnormal cell division morphology was observed, suggesting that *murB* co-transcription with the *potABCD* gene is biologically significant.

Furthermore, polyamines act as feedback regulators of PotA activity and as regulators of PG synthesis and hydrolysis. Thus, the *murB-potABCD* operon plays an essential role in the normal life of *S. suis*, with PotAB-CD facilitating polyamine transport and regulation of polyamines in PG synthesis.

VirD4-VirB4 Secretion System. The virulence factors VirD4-VirB4, located on the 89K virulence island, belong to the first and N-type secretion systems (T4SS). In mice infected with SS2 and suffering from toxic shock syndrome (STSLs), this system stimulates an enhanced immune response from the host (Y. Zhao et al., 2011). VirD4 is one of the components of the N-type secretion system, and the *virD4* gene of SS2 was knocked out. Compared to the wild-type strain, the expression of pro-inflammatory cytokines was downregulated in mice or cell lines infected by the VirD4 gene-deletion strain, and the strain's ability to resist phagocytosis and virulence were reduced (Jiang et al., 2016). *Streptococcus suis* upregulates *virD4* expression under oxidative stress.

Epidemiology: Susceptible Hosts and Symptoms

S. suis is a widespread pathogen, with pigs being the most susceptible host. Epidemiological studies have shown that under natural conditions, all strains of pigs can be infected with *S. suis*. Newborn piglets, lactating piglets, and pregnant sows are the most susceptible, followed by feeder pigs, while adult pigs are the most resistant. *S. suis* tends to peak during weaning and mixing, and the susceptibility of piglets to *S. suis* decreases as they age. In addition to pigs, *S. suis* can also occasionally infect dogs, cats, rodents, cattle, sheep, and horses. Infected animals often develop symptoms such as pneumonia, wet dermatitis, meningitis, peritonitis, osteomyelitis, arthritis, pharyngitis, suppurative pneumonia, and even sudden death. Among experimental animals, domestic rabbits are the most sensitive to *S. suis*. Infections with highly virulent SS2 strains can cause rabbits to die within 24 hours. Subcutaneous, muscular, and intravenous inoculation can lead to an increase in body temperature and even death in rabbits. BALB/C mice are often used as animal models of *S. suis* infection. Although rare, *S. suis* can also infect humans, with symptoms including fever, chills, headache, fatigue, abdominal pain, diarrhea, and general discomfort. In some cases, it can lead to human meningitis, permanent deafness, sepsis, endocarditis, and even death. However, most of the infected people were individuals who had close contact with pigs.

Route of transmission: The main mode of transmission of *S. suis* from pig to pig is through respiratory infection, but it has not been established whether *S. suis* can infect humans through respiratory infection. Close contact with pigs is the most common source of human infection, primarily through the digestive tract and wounds. After an incubation period, the infection will manifest, causing inflammatory reactions

and septicemia. Diseased and infected pigs are the primary source of *S. suis* disease, often entering the body through the mouth or nasal cavity and settling in the tonsils. The pathogen is present in the urine, blood, viscera, nasal fluid, tonsils, saliva, and swollen joints of pigs. *S. suis* disease is typically transmitted horizontally by aerosol or direct contact, with pathogens being transmitted through the digestive, respiratory, and reproductive tracts. Pathogens can also be transmitted through contaminants. *Musca domestica* carrying *S. suis* can survive for 2 to 5 days and can mechanically carry pathogens within or between pig farms. Natural transmission can also occur between diseased mice and pigs. Additionally, birds have been reported as potential vectors, but this is yet to be confirmed. Mother-to-child transmission is also an important channel for the transmission of *S. suis*. If *S. suis* is present in the reproductive tract of the sow, piglets are at risk of infection during delivery, although the mechanism is not well understood. Surgical trauma is also a possible route of infection; as poor disinfection can lead to the spread of *S. suis* disease.

Susceptible population: *S. suis* can also infect humans, causing various diseases such as endocarditis, permanent deafness, meningitis, sepsis, DIC, suppurative arthritis, and endophthalmitis (Hlebowicz, et al., 2019). Butchers, slaughterhouse workers, housewives, and other professionals involved in raw pork processing and sales are at a higher risk of infection (Vadeboncoeur, et al., 2003). Farmers are also at a high risk of infection, especially those who kill, eat, and skin sick pigs. The first human infection of *S. suis* in Tianjin was reported from handling dead pigs with skin disease. People with weakened immune systems may experience more severe

symptoms upon infection (Arends, J., et al., 1988). *S. suis* infection in pigs can manifest as acute hemorrhagic septicemia, endocarditis, meningitis, arthritis, dysentery in lactating pigs, and abortion in pregnant pigs (Hughes, et al., 2009).

Seasonality. Outbreaks of *S. suis* disease usually occur throughout the year without a clear seasonal pattern, although most occur between May and November and are endemic. The incubation period is typically around 7 days (Norton et al., 1999). Acute septicemia of *S. suis* disease can spread rapidly and cause heavy losses in a short period of time, making early prevention crucial. Chronic *S. suis* disease is often sporadic and endemic. Stress and changes in external factors are important contributors to *S. suis* infection (Wang et al., 2022). Changes in stress factors, such as overcrowding, sudden climate changes, poor ventilation, immunization, and mixing, can trigger outbreaks of *S. suis* disease. Co-infections with other pathogens such as blue ear disease virus, porcine pseudorabies virus, and *Bordetella bronchiseptica* are also common. In general, intensive pig farming practices are associated with a higher risk of infection (Timoney, 2022).

Conclusion. This study investigates the etiological factors of streptococcal infection in pigs and provides detailed information on the biological properties of the causative agent, *Streptococcus suis*. The study also highlights the key epidemiological aspects of the disease, including susceptible animals, routes of transmission, susceptible populations, and seasonality. Given the significant threat and widespread distribution of *Streptococcus suis*, future research will focus on investigating its antibiotic resistance to various antibacterial drugs.

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Епідеміологічна характеристика стрептококової інфекції свиней

На стрептококову інфекцію хворіє значна кількість свиней у більшості господарств України. Також дану інфекцію реєструють у інших країн світу, завдаючи тваринникам великих економічних збитків. Стрептококоз вважається однією із основних зоонозних хвороб свиней, яка часто супроводжується менінгітом, септицемією або артритом, а також є небезпечною для людини. Тривалий час фахівцям вдавалося контролювати цю інфекцію досить успішно, але останні кілька років через проблему антибіотикорезистентності бактерій до лікувальних препаратів ця інфекція стає все більш масштабнішою. Враховуючи важливість і безпеку збудника стрептококової інфекції для свиней, ця проблема є досить актуальною у свинарстві. Етіологічним чинником даної інфекції є *Streptococcus suis* (*S. suis*). Це грампозитивні, аеробні або факультативно-анаеробні бактерії. Більшість стрептококів мають капсули, не утворюють спор і не можуть рухатися. Структура стрептококового антигену складна, складається з трьох основних компонентів, а саме групспецифічного антигену, типоспецифічного антигену та антигену ядерного білка. Високопатогенні властивості має гемолітичний стрептокок β -типу. Відповідно до різних характеристик капсульного антигену, бактерії класифікують на 35 серотипів. Серотип 2 *S. suis* зазвичай виділяється від клінічно хворих поросят і вважається найбільш вірулентним підтипом даного мікроорганізму. Патогенність даного збудника для тварин пов'язана з її основними патогенними факторами, такими як капсула, токсин і ферменти.

Збудник *S. suis* широко поширений, найбільш сприйнятливими стрептококової інфекції є новонароджені поросята, поросята в період лактації та супоросні свиноматки, а найбільш стійкими є дорослі свині. Інфекція досягає піку в період відлучення, а сприйнятливість поросят до *S. suis* зменшується зі збільшенням віку. Крім свиней, *S. suis* зрідка інфікує собак, котів, гризунів, велику рогату худобу, овець, коней. *S. suis* також може інфікувати людей, і це стало серйозною ендемічною загрозою для здоров'я населення. Основними шляхами передачі інфекції є аліментарний і респіраторний. Люди заражуються під час контакту з хворими тваринами та при роботі з продукцією тваринництва. У заражених тварин часто розвиваються такі симптоми, як пневмонія, вологий дерматит, менінгіт, перитоніт, остеомієліт, артрит, фарингіт, гнійна пневмонія. Спалахи захворювання, що спричинені *S. suis*, зазвичай реєструються впродовж року, сезонність не встановлена.

Ключові слова: стрептококоз, *S. suis* інфекція, етіологія, епідеміологія.