Al-Kitab Journal for Pure Sciences (2026); 10(1):35-53.
https://doi.org/10.32441/kjps.10.01.p4

Al-Kitab Journal for Pure Sciences
ISSN: 2617-1260 (print), 2617-8141(online)
https://isnra.net/index.php/kjps

Neisseria meningitides and Study of Epidemiology, Molecular Characteristics and Prevention of Meningococcal Infection

Hanan Sami Nouri[1*], Najlaa Abdulla Fathi[1], Ibrahim Talal Dawood[2]

[1]Department of Biology, College of Science, University of Mosul, Mosul, Iraq.

[2]Department of Basic, College of Nursing, University of Mosul, Mosul, Iraq.

[*]Corresponding Author: hanan.sami@uomosul.edu.iq

Citation: Nouri HS, Fathi NA, Dawood IT. Neisseria meningitides and Study of Epidemiology, Molecular Characteristics and Prevention of Meningococcal Infection. Al-Kitab J. Pure Sci. [Internet]. 2026 Feb. 03;10(1):35-53. DOI: https://doi.org/10.32441/kjps.10.01.p4

Keywords: *Neisseria Meningitidis, Meinigococcal Infection, Coagulopathies, Molecular Characteristics, Prevention, Epidemiology.*

Article History

Received	11 Feb.	2025
Accepted	17 May.	2025
Available online	03 Feb.	2026

Abstract:

Neisseria meningitidis is an encapsulated, aerobic, Gram-negative diplococcal bacterium that is oxidase-positive. It grows on chocolate agar, soybean agar, and Mueller-Hinton agar. This organism causes meningitis, a serious bacterial disease characterized by fever, headache, nausea, neck and muscle pain. It is transmitted via airborne droplets originating from respiratory and throat secretions. *Neisseria meningitidis* colonizes the upper layers of the mucosal tissues in infected individuals, invades the bloodstream, and can lead to severe septicemia and meningitis. The bacterium is responsible for high mortality rates in children as well as adults worldwide. Despite the use of antibiotics, the current mortality rate remains around 10%. The phenotypic classification of *Neisseria meningitidis* includes twelve serogroups based on variations in surface structures such as the polysaccharide capsule, lipooligosaccharide, and outer membrane proteins. Among these twelve serogroups, five (A, B, C, W, Y) are the primary causes of disease and have contributed significantly to the global spread of meningococcal infections. The bacterial capsule plays a crucial role in inducing bactericidal antibodies,

particularly those targeting the polysaccharide capsule, which is essential for vaccine development. An exception is the B-polysaccharide, which has low immunogenicity. The capsule has several functions: it protects against the host immune system by preventing phagocytosis, facilitates colonization of the nasopharynx, and protects the bacteria during bloodstream dissemination, enabling it to reach the meninges and cause meningitis. Furthermore, the capsule contributes to antibiotic resistance and is a key component in vaccines, particularly those containing polysaccharide or protein-conjugated polysaccharide formulations. Vaccination is considered one of the most effective preventive measures against meningococcal disease. *Neisseria meningitidis* also possesses two types of (cilia), which aid in adhesion to epithelial cells. Recent studies have identified genetic mutations that introduce new mechanisms for developing antibiotic resistance, such as mutations in the *rpsJ* and *TetM* genes. To enhance prevention strategies against meningococcal disease, a novel antigen named 4CMenB has been identified through serogroup analysis. A protein-polysaccharide conjugation approach was applied, leading to improved T-cell immunity and memory cell immune responses. Third-generation cephalosporins are commonly used for the treatment of drug-resistant strains. Objective: To summarize the molecular characteristics of *Neisseria meningitidis*, the causes of infection, preventive strategies, and its epidemiology.

Keywords: *Neisseria Meningitidis*, Meinigococcal Infection, Coagulopathies, Molecular Characteristics, Prevention, Epidemiology.

النيسريا السحائية ودراسة علم الأوبئة والخصائص الجزيئية والوقاية من عدوى المكورات السحائية

حنان سامي نوري [1], نجلاء عبد الله فتحي [1], إبراهيم طلال داؤود [2]

[1]قسم علوم الحياة ، كلية العلوم، جامعة الموصل، الموصل، العراق.

[2]قسم الأساسية، كلية التمريض، جامعة الموصل، الموصل، العراق.

hanan.sami@uomosul.edu.iq [1], najasbio75@uomosul.edu.iq[2], ibrahim.alsawaf@uomosul.edu.iq[3]

الخلاصة:

جرثومة النيسيرية السحائية (*Neisseria meningitis*) " هي عبارة عن بكتريا عصوية ثنائية المكورات هوائية ومغلفة سالبة لصبغة كرام موجبة للاوكسديز "وتنمو على أوساط (اكار الشوكولاتة ،اكار فول الصويا، اكار مولر هنتون) وتسبب مرض التهاب السحايا *meningitis* وهو مرض بكتيري خطير يتميز بالحمى ، صداع ،غثيان ،الام الرقبة والعضلات وينتقل عن طريق الرذاذ المحمول جوا من افرازات الجهاز التنفسي والحلق تسببه *Neisseria meningitis* تستعمر الجزء العلوي لطبقات الانسجة المخاطية لدى المصابين وتخترق مجرى الدم وتسبب تعفن الدم الشديد والتهاب السحايا .تسبب

ال*Neisseria* نسب وفيات عالية لدى الأطفال وكذلك البالغين في جميع أنحاء العالم وعلى الرغم من استخدام المضادات الحيوية إلا انه وجد أن معدل الوفيات الحالي يبلغ حوالي ١٠٪.يمكن تقسيم التصنيف المظهري للمكورات السحائية الى (١٢) مجموعة مصلية تعتمد على الاختلافات المظهرية من وجود كبسولة متعدد السكرايد ومتعدد السكرايد الدهني وبروتين الغشاء الخارجي أو عدمها ،إذ أن هناك خمس أصناف من بين المجموعات المصلية أل (١٢) هم السبب الرئيسي للإصابة بالمرض ألا وهي (*A,B,C,W,Y*) والتي ساهمت في تطور المرض عالميا ، إن الأجسام المضادة البكتيرية القاتلة المتخصصة تحفز من قبل غلاف الكبسولة*Polysaccharide* وهذا مهم في تصنيع اللقاحات باستثناء *B*– *Polysaccharide* الذي يكون ضعيف مناعيا ، وللكبسولة فوائد كثيرة منها الحماية من الجهاز المناعي اذ تمنع البلعمة وتسهل الكبسولة استعمار البلعوم الأنفي، ويحمي البكتيريا أثناء انتقالها في مجرى الدم مما يسمح لها بالوصول إلى السحايا والتسبب في التهاب السحايا، أيضا المساهمة في مقاومة المضادات الحيوية، وللكبسولة دور في اللقاحات نظرًا لدورها الأساسي في الضراوة مثل اللقاحات الحاوية على متعدد السكاريد أو المترافقة بالبروتين ويعد التطعيم وسيلة من وسائل الوقاية ضد الأمراض وللبكتريا فئتين من الأهداب *cilia* والتي تساهم في الالتصاق بالخلايا الظهارية واكتشفت الأبحاث مؤخرا أن هناك طفرات في الجينات لها آليات جديدة في تطوير مقاومة المضادات الحيوية مثل جين *TetM* ، *rpsj*.للوقاية من الأمراض السحائية وتم اكتشاف المستضد الجديد المسمى (*CMenB*)٤ من خلال المجموعات المصلية وتم تطبيق اقتران متعدد السكارايد بحامل بروتيني *Proteinpolysaccharide* مما أدى الى تعزيز مناعة الخلايا التائية والاستجابة المناعية لخلايا الذاكرة ، وغالبا ما يستخدم الجيل الثالث من السيفالوسبورين لمعالجة السلالات المقاومة للأدوية. الهدف : تلخيص الخصائص الجزيئية للنيسيريا السحائية ،وأسباب الإصابة بالمرض ،الوقاية من المرض. الوبائية.

الكلمات المفتاحية: النيسرية السحائية، عدوى المكورات السحائية، اعتلالات التخثر، الخصائص الجزيئية، الوقاية، الوبائية.

1. Introduction:

Neisseria meningitidis is a non-spore-forming gram-negative β proteo-bacterium, a fastidious, capsulated, aerobic diplococcus bacterium, oxidase-positive, the organism grows at 37 °C and able to be cultured on various type of media such as chocolate agar, trypticase soy agar, and Mueller-Hinton agar where the grow of colonies in size about 1–2 µm. The bacteria appear under microscope as gram-negative cocci as in pairs and sometimes in clusters. The bacteria consist of three layers first called cytoplasmic membrane, then a thin peptidoglycan layer and finally an external membrane which having outer-membrane proteins, phospholipids, and also lipooligosaccharide [1, 2].

Neisseria meningitidis may penetrate and colonize the human nasopharynx then be able to enter the bloodstream and cross through the blood-meningeal barrier and cause severe

septicemia or meningitis. *Neisseria meningitidis* causes notable disease and mortality in children, also adults worldwide in spite of the use of proper antibiotics; meningococcus is relevant public health issues worldwide, some factors may play a relevant role in disease like genetic and capsular structure of pathogenic strains [3]. **Figure 1.**

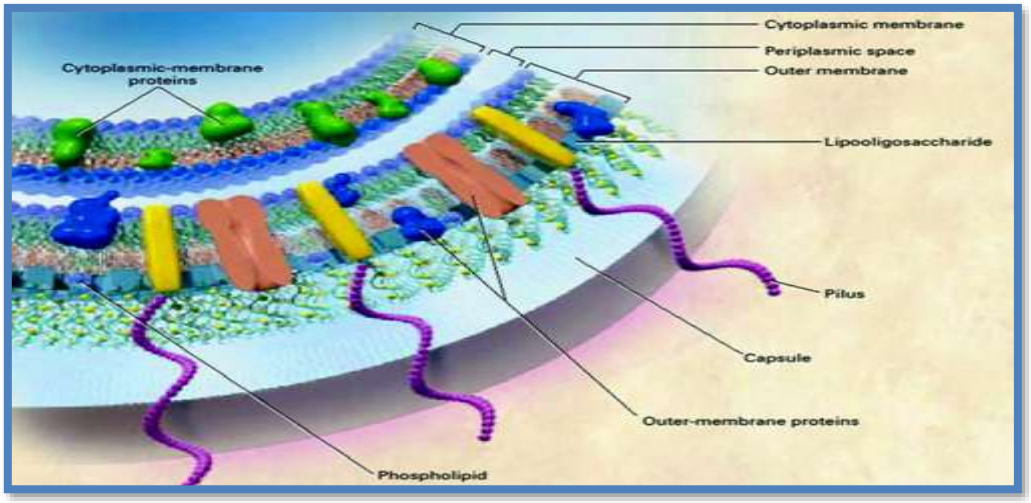

Figure 1: Cross-sectional view of the meningococcal cell membrane [1].

Neisseria meningitidis commonly exist within the higher tract of humans and in most cases colonize in tissue layer mucosal surfaces It found in the throat's epithelial lining without causing any disease symptoms [4]. Infection of human beings with disease through respiratory droplets or sometimes from asymptomatic carriers. Immune response generally is sufficient to stop spreading the disease [2]. N. meningitidis were classified using common bacteriologic methods; recommendations of the World Health Organization must be followed as applicable [5]. **Figure 2**.

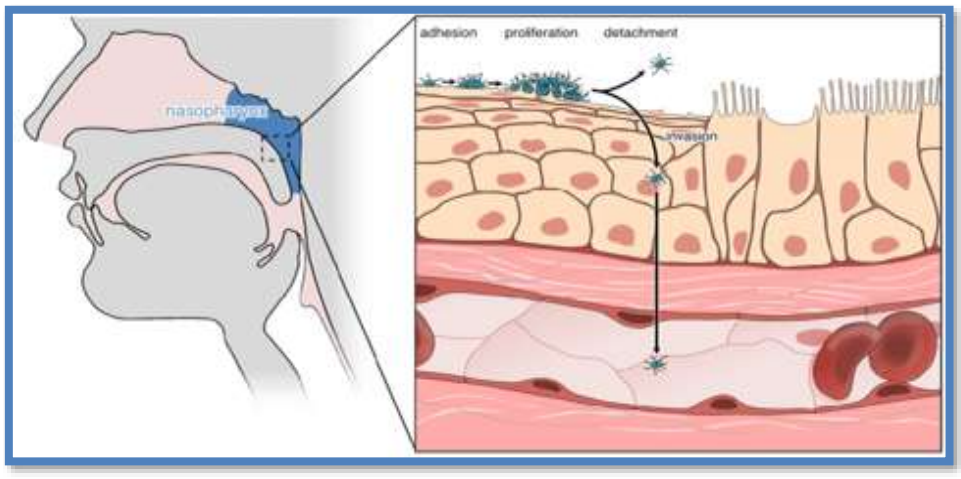

Figure 2: *N. Meningitides* Life cycle. Human nasopharynx represent the natural reservoir of *N. Meningitidi* [6].

The phenotypic classification of meningococci can be split into twelve serogroups depend on structural differences due to presence capsular lipoligosaccharide structure and in serotypes and sub-serotypes according to presence the outer- membrane protein) [7]. Only five between the twelve serogroups, as (A, B, C, W and Y), that led extremely to an evolution of disease infections global). 12 serotypes, which are (A, B, C, W, Y) (X, Z, Z, E, H, I, L) the five types only cause diseases and epidemics remaining seven serotypes are rare and do not cause epidemic disease. Therefore, the focus is on the five pathogenic serotypes. The role played by these five serogroups quite changes according to changing in time period and also geographical area [8, 9]. Figure 3, 4.

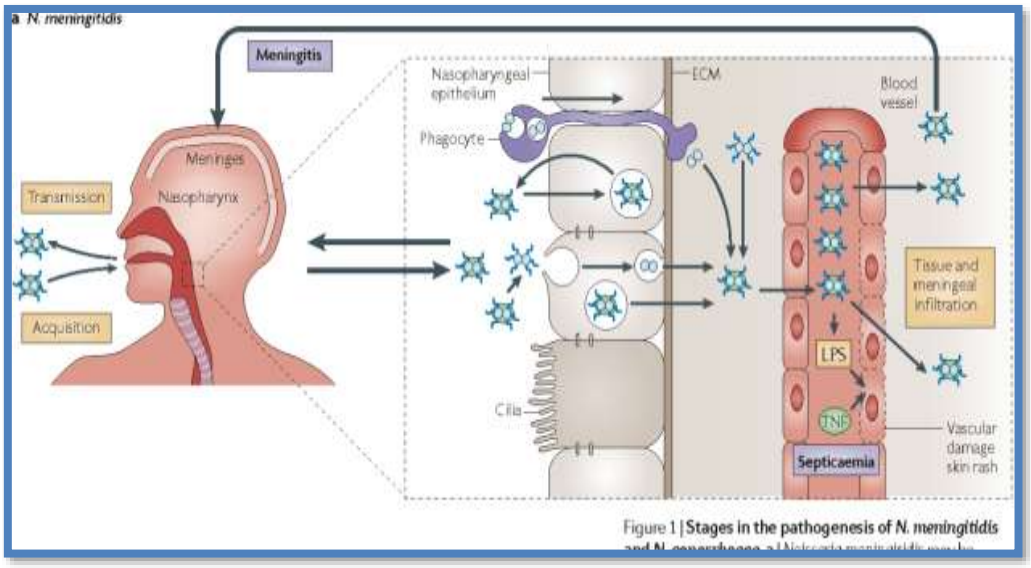

Figure 3: Stages in the pathogenesis of *N. Meningitides* [10].

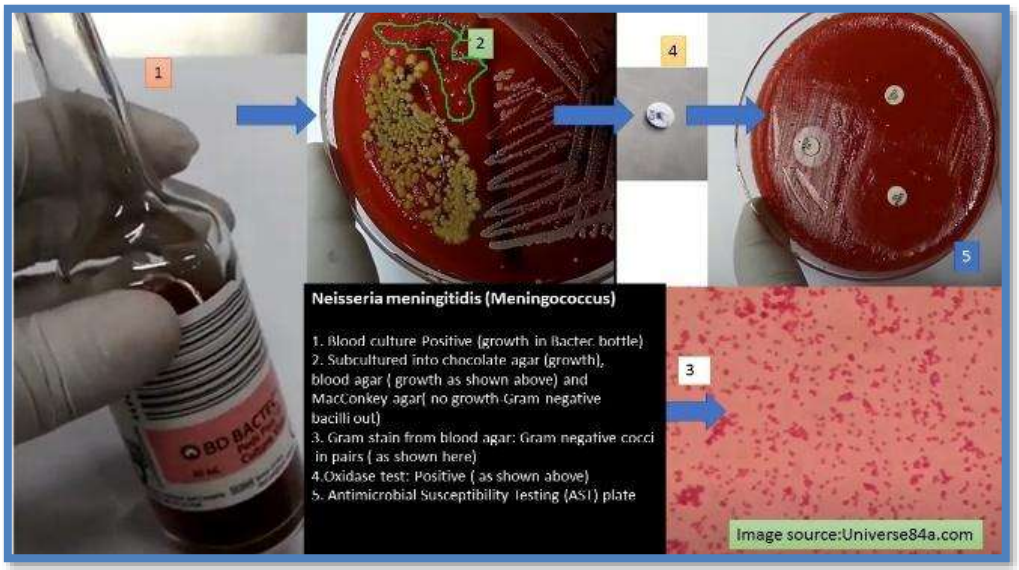

Figure 4: Meningococcus, Culture characteristics, Pathogenesis, Laboratory Diagnosis and Treatment [11].

Molecular typing methods are preferred as recent technique for clonal groups' identification; closely strains have the power to be a reason of outbreaks, overall, in vaccine predicting coverage, realization genome of N. meningitidis by detecting minimal changes in the genome [1, 12].

2. Epidemiological of *Meningococcal meningitis*

Meningococcal meningitis is a serious bacterial disease distinguished by unexpected fever, strong headache, nausea, and hard neck and may be accompanied by photophobia. The *Neisseria meningitidis* is present only in humans being and people become infected through airborne droplets from the secretions from respiratory tract and throat. Some disease infections with *Neisseria meningitidis* found with asymptomatic pharyngeal carriage and sometimes lead to acute disease. Epidemiology of meningococcal infections has notably different through the years in several places of the world. Meningococcal meningitis is an acute common health problem for the high fatality rate and, in some places, tended to be epidemics [13, 14].

Although presence of rapid and suitable treatment for disease, the fatality present is found to be around 10%, and about 15% of survivors pain from long period neurological symptoms. the main cause of large meningococcal disease infections in Europe regions return to serogroup A through first and second world wars, and also in the African regions meningitis infections range, serogroups named W, C, and X were prevalent and still responsible for local epidemics and sometimes causes for more popular epidemic waves and have a relevant influence in morbidity and mortality [15]. Epidemic outbreaks have appeared more lately during the 21st century due to serogroups W and Y. Besides, age classes that are affected by disease have a change, with increasing occurrence of serogroup Y in older age and decreasing of serogroup C in teenagers. There is unchanged in the epidemiological trend of disease infections in Africa, as class A is most extensive [4]. Figure 5.

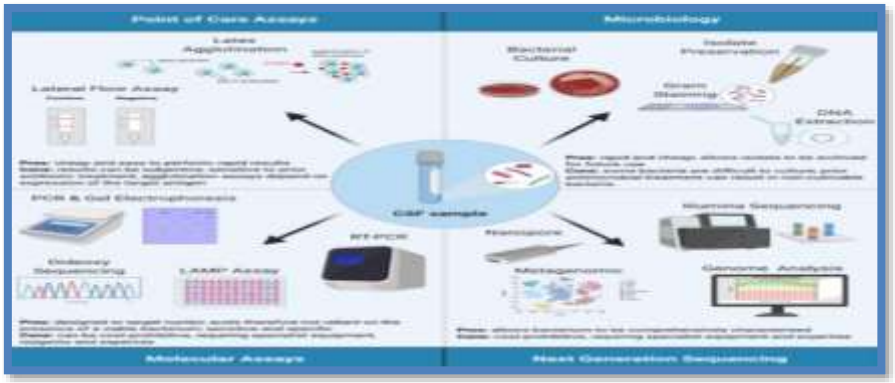

Figure 5: Diagnosis approaches of meningitis from cerebrospinal fluid [16].

2.1 Carrier state of *Nesseria* and interaction with the host:

Neisseria. meningitidis are human infectious agents, sometimes residing within the bodily cavity. Human higher tract a stable ecological niche and meningococci will be usual elements of the microbic flora in buccal tissue layer, rectum, urethra, system tissue layer, and plaque. About 8-25% of subjects were transported at pharyngeal level ; this implies many million individuals within the world, adolescents being the foremost relevant cluster. Uncommon manifestations of meningococcal infection and antimicrobial resistance of *Nesseria* (chloramephnicol high resistance) are found **[17, 18]**. Recent reports indicate a rise in cases of meningitis caused by *Neisseria meningitidis* in Iraq, particularly in the Kurdistan Region. The number of recorded cases has reached approximately 190, mostly among children, with no deaths recorded so far. However, this outbreak is causing widespread concern among locals, given the contagious nature of the disease and its potential severity.Neisseria meningitidis is one of the most dangerous causes of bacterial meningitis, as it can lead to death within 24 hours if not treated promptly. Densely populated areas such as refugee camps, schools, or military barracks also increase the risk of the disease spreading. Epidemics are usually classified based on the number of cases per 100,000 population per week, with an outbreak being considered if this threshold is exceeded. In Iraq's neighboring countries, there are no clear reports of widespread outbreaks currently, but the disease remains a global threat, especially in areas with poor living conditions or a lack of vaccination coverage. The World Health Organization recommends vaccination against Neisseria meningitidis as an effective preventive measure to reduce the spread of the disease **[19,20]**.

2.2 Mainfestations of meinigococcal infection:" symptoms of meningitis"

Mningococcal disease can be varied in their clinical manifestations from transient fever, chilliness, myalgias ,vomiting, the purpuric classic rash with or without bullae formation and bacteremia to fulminant disease with death ensuing within hours of the beginning of clinical symptom. Disease Meningococcal is accented with remarkable morbidity , limb damage, hearing lack, cognitive impairment, weakness visual, difficulties for educational, motor nerve deficits, disorders of seizure, and behavioral problems **[14]**.

The quick estimation and understanding the signs of meningococcal disease are importance to survival for patients. Prompt administration of effective treatment of parenteral antimicrobial and early estimation of the severity of meningococcal disease, including raised intracranial pressure and shock are serious of increase outcomes patient. Shock in meningococcemia is due

to multiple reasons such as capillary leak, loss of vasomotor tone, impaired myocardial function , maldistribution of intravascular volume and impaired function of cellular. A quick realization of sudden shock is important as it causes speedy entrances and improved results. The only quick sign of the first disease phase is tachycardia and should be to mandate fluid resuscitation [20,21].

2.3 "Molecular characteristics":

The genome of *Neisseria meningitidis* is relatively small ranging between 2.0 to 2.3 megabases depending on the strain. The genome is contained within a single circular chromosome and lacks plasmids in most cases. Some strains. Genome Composition GC content of *N. meningitidis* is approximately 51-53%, which is characteristic of many *Neisseria* species. It contains around 2,000 to 2,200 protein-coding genes, covering essential functions such as metabolism, regulatory processes, and virulence. Mobile Genetic Elements: Insertion Sequences (IS elements): These sequences promote genome rearrangements and horizontal gene transfer (HGT). Phage-related regions: Some strains harbor prophages that contribute to genetic diversity ,Genomic Islands: Large DNA segments acquired via HGT, some of which carry virulence or antibiotic resistance genes. Organization of genes in *N. meningitidis* reflects its adaptation to the human host and its ability to evade immune responses.

A. Core genome includes essential genes for DNA replication (*dnaA, gyrA, gyrB*), Transcription (*rpoA, rpoB, rpoC*), Translation (*rpl, rps genes for ribosomal proteins*), Metabolism (*ptsG, pykA for glycolysis; sucC, sucD for TCA cycle*) [22].

B. Virulence *N. meningitidis* is a highly adapted human pathogen, and its virulence genes are regulated Capsule Biosynthesis Genes The polysaccharide capsule is a key virulence factor, encoded by the *capsular operon* (*sia* or *css* genes). Different capsule types define the major serogroups (A, B, C, W, Y). cilia and Adhesion Proteins: Type IV pili genes (*pilE, pilT, pilC*) mediate adhesion to human epithelial cells and enable DNA uptake for transformation. Outer Membrane Proteins (OMPs):*PorA , PorB*: Porins involved in immune system evasion .*Opa , Opc*: Adhesion molecules that facilitate host cell invasion .*TbpA, TbpB*: Iron acquisition proteins, allowing survival in iron-limited environments Lip oligosaccharide (LOS) Biosynthesis Genes: The *lgt* genes encode enzymes for LOS synthesis, which mimic human cell surface molecules to evade immune recognition. [16,23]

C. Regulatory Systems: *N. meningitidis* possesses multiple regulatory networks that control gene expression in response to environmental conditions: Two-component regulatory systems

(TCSs):*MisR /MisS* : Regulates membrane integrity and stress responses. *PhoP /PhoQ*: Controls genes involved in antimicrobial resistance. Phase Variation Genes: Many virulence genes (*opa* , *pilC*) undergo phase variation via simple sequence repeats, allowing immune evasion.

D. Horizontal Gene Transfer (HGT) and Genomic Islands Pathogenicity Islands (PAIs): These are clusters of virulence genes acquired via HGT, meningococcal disease-associated island (MDAI), which contains genes involved in colonization and immune evasion Antibiotic Resistance Genes Strains can acquire resistance genes through transformation, recombination, or conjugation. Mutations in *penA* confer resistance to β-lactam antibiotics [24,25].

A preferable understanding of the epidemiology of the meningococcus was resulted from advances in molecular typing. Genetic diversity of meningococcus is highly structured according to Rouphael and Stephens [1].

According to the structural differences in the capsule contain polysaccharide meningococci There are five types out of 12 serotypes that cause meningitis A, B, C, Y, and W .

Specific bactericidal antibodies induced from capsular polysaccharides benefits of the capsule in Neisseria meningitidis Protection from the immune system Prevents phagocytosis by immune cells such as macrophages and neutrophils, Contributes to immune evasion ,Some serogroups, such as B, C, and Y, have a capsule that mimics molecules found in human cells, reducing the immune system's response, Phase variation helps alter the composition of the capsule, making the bacteria more capable of evading antibodies, The capsule facilitates colonization of the nasopharynx, the primary site for N. meningitidis growth before spreading throughout the body ,It protects the bacteria during its bloodstream travel, allowing it to reach the meninges and cause meningitis , Contributing to Antibiotic Resistance, The Importance of the Capsule in Vaccines Because of its essential role in virulence, the capsule serves as the basis for the development of anti-meningococcal vaccines, such as polysaccharide-based or protein-conjugated vaccines that target the major pathogenic serotypes (A, C, W, and Y). and thus, applied for manufacter vaccine excepting the serotype B-polysaccharide regarded as poorly immunogenic. The potential to exchange between meningococci, is considered an interesting phenomenon that is present in meningococci. So, serogroup B has ability to turn to C or vice versa. Neiserria meningitidis represent two various classes of pili , that differed in antigenically and structurally way. Through disease infection, pili endure speedy section shifts and matter change. Adherence to animal tissue and epithelium cells have occurred through pili, and they

transmit tissue response. The electricity repulsion between the charged tissue layer surfaces is control by pili. Additionally, additionally have a job within the gain of symmetric and asymmetric DNA from surroundings [26,27,28]. The DNA of Neisseria meningitidis consists of a single, circular, double-stranded chromosome without a true nucleus, as is the case in most bacteria, Size: 2.0–2.3 megabases (Mb). GC content: 51–53%, which is average compared to other bacteria. It contains approximately 2,000–2,200 genes, organized into functional groups that include metabolism, regulation, and virulence [29].

Genes encoding Neisseria meningitidis proteins Outer Membrane Proteins (OMPs):such as PorA, PorB, Opa, and Opc, which play a key role in adhesion to the host and evasion of the immune system.Pilus-associated proteins (PilE and PilC), which help bacteria adhere to host cells and transfer genetic material.Proteins related to capsule production, such as siaA, siaB, siaC, and siaD, which are responsible for forming the capsule that protects bacteria from the immune system.Proteins related to toxin formation, such as NadA, NHBA, and fHbp, which contribute to disease-causing pathogenesis by disrupting host cells. The Neisseria meningitidis genome contains approximately 2.2 megabases, and the genes encoding important proteins are located in specific locations on the bacterial chromosome. Some key sites: Genes for surface proteins are often located within genetic islands that control antigenic variation. Capsule genes are located within cassette-like structures that are tightly regulated by specific transfer enzymes. Genes responsible for toxins and colony-associated proteins are often located within pathogenic islands (PAIs). Possibilities for genetic changes in protein genes include Gene conversion, Exchange of genetic material occurs between similar genes, leading to the generation of diversity in surface proteins.[30]

The trimeric proteins gift within the meningococci outer membrane offers porins, permitting the spreading minimum nutrients of the outer membrane which contribute to adhesion to host cells and evasion of the immune system. Neiserria meningitidis represent 2 porins together: Category I protein, Por A and the two category II or III protein, Por B. Por A is known as cation-selective, while category II or III, Por B, is known as anion-selective. Between category I and II proteins antigenic variations which are used for meningococci serotyping. Opacity protein is found in the external membrane of *N. meningitides*. Class V protein has five variants and represented by a single strain. Proteins opacity will endure substance difference throughout natural disease infection and simplify the attachment of bacterium with the tissues [24]. Approximately five hundredth of the external membrane includes Lipooligosaccharide (LOS), a modified form of liposaccharide (LPS), which helps

activate the immune response and causes inflammation which having a core of lipoid A and short chains of sugar. The Lipooligosaccharide sugars bear high-frequency section and substance difference throughout the infection disease and area unit once more needed for immunotyping of the meningococci. Vesicles that exist as bubbles, area unit shed by the neisserial external membrane incessantly. Bubbles include polymer, protein, and high contents of Lipooligosaccharide, together with lipoid A. The Lipooligosaccharide within these bubbles is involved in the beginning of sudden shock once bacterium entry into blood circulation [31]. **Figure 6**.

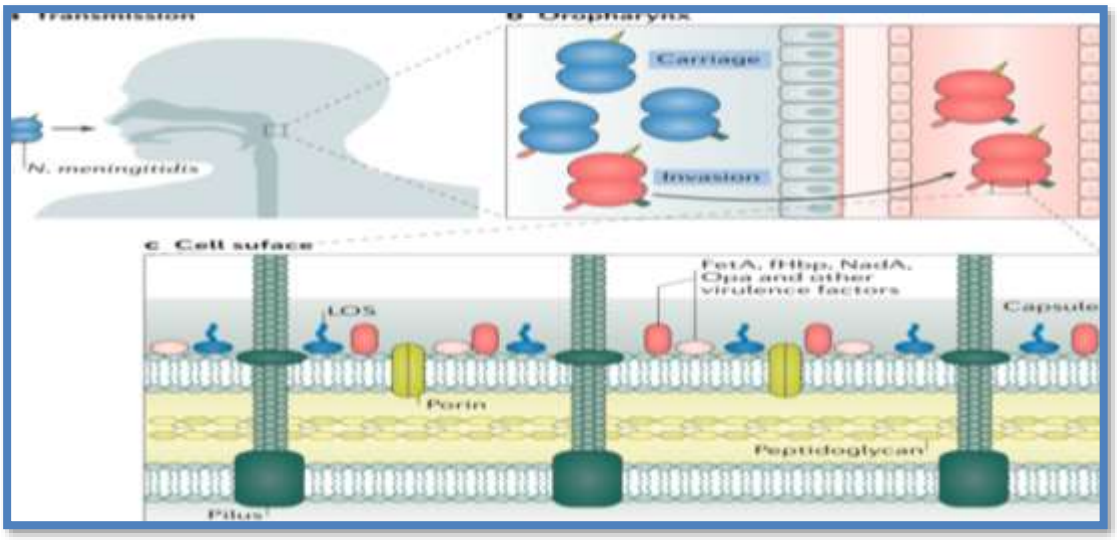

Figure 6: Virulence Factors of *Neisseria meningitidis* [32]

National Health Institute in some countries performs a passive monitoring of *N. meningitidis* the monitoring network system for the precipitating causes of meningitis in the agenda of the American Health Organization. The goal of the program is to receive isolations from Laboratories of Public Health round the state. investigation activities distinguish isolates according to antimicrobial condition, serogroup, and subtyping, as some previous research, like analysis on the rise within the spreading of serogroup Y isolates, and characterization of carriage isolates [33]. Worldwide, resistance to antibiotics especially penicillin has been reported because the appearance of mutations in the penicillin-binding protein and decreasing in production of penicillinases. The appearance of penicillin resistance in *Neisseria* makes researchers think of the possibility of developing resistance in *N. meningitides* [34].

2.4 Coagulopathies:

Coagulopathy has resulted too multifactorial. Shocks increase damage of endothelial and disseminated intravascular coagulation. Coagulation disorders associated with meningococcal

disease are not affected with therapies. Mild clotting abnormalities can be tolerated and able to not be treated. In severe coagulopathy there are recommendations to use fresh frozen plasma. In some studies, vitamin K was used intravenous and fresh frozen plasma which give good results. Currently, the optimal management of shock is the best treatment choice for meningococcal-related coagulopathy [35].

2.5 Prevention of the disease:

Strategies to monitor meningitis tend to enter specific programs, as there is a range of pathogens responsible for increasing and presence of meningitis. The strategies in the epidemiology reflect the variation of agents etiologic, the restricted synthesis of antigenic and coverage of the helpful vaccines, the complication of primary, secondary prevention of modalities, the cost of setting big scale programs. Antibiotics used in some epidemic situations to inhibit bacterial meningitis infections in regions within one week from the diagnosis of the status. Current recommendations often call for cephalosporins to tolerate and face drug-resistant strains [26]. Mothers having colonies with group B are routinely given antibiotics to inhibit causing disease for infants. Nevertheless, the most effective means of preventing causes of bacterial meningitis disease, like measles and mumps, is vaccination. Improvement vaccines against encapsulated bacteria remains through the early twenty-first centuries although vaccines are authorized steady during the late twentieth [36]. Health care makers must look to the full features of clinical disease caused by N. meningitis at creating important decisions about treatment or vaccination. a wide range of clinical syndromes caused by meningococci as bacteremia septicemia, and localized infections such as arthritis. Vaccine policy of meningococcal must address the potential for incidence severe outbreaks that may deny sufficient getting of antibiotics in a rapid way to cure patients and to reduce the diffision of disease infection through the community. Drug resistance may influence decision therapy [13, 37].

Antimicrobial resistance of Nesseria:

Some previous research have reported that during the treatment of disease any exposure of *Neisseria* spp to antimicrobial agents can cause the chosen of resistant strains as a result of involuntry genetic mutations or also due to the gain of all or some parts of resistance genes.Other research have found mutations isolates, reported that the resistance mechanisms might entered in the development of the resistance to this antibiotic, such as mutations presents in the *rpsJ* gene and the presence of the *TetM* gene [38, 39].

Chloramphenicol high resistance of *Nesseria*:

Chloramphenicol-resistant strain of *N. meningitidis* was detected in Laos lab in year 2017 and thus resulted in a review for revealed documents of chloramphenicol resistance in *N. meningitidis* and display of chloramphenicol status between meningococcal isolates within the lab network. Even though no standard medical treatment for meningococcal infectious disease were applied, chloramphenicol is usually employed in beta-lactam intolerant patients and is usually recommended as another agent in worldwide guidelines in available therapy of meningococcal infectious disease , and thus resistance until now still a difficulty of clinical importance [33,40]. Chloramphenicol resistance in *N. meningitidis* seldom were known and reported, about eleven isolates were initially delineated in year 1998, thanks to the existence of antibiotic drug enzyme cistron (catP) which probably derived from a bioweapon permutable component [41]. Two additional Chloramphenicol resistance *N. meningitidis* isolates were later delineate in Australia. A lot of just recently, chloramphenicol resistance isolates were described in many countries as Brazil and in Vietnam an extra extremely resistant isolate was reported during year 2014. in spite of historical using only one dose oily chloramphenicol for outbreak management of meningococcal infectious disease, we have a tendency to notice no documents of chloramphenicol resistance strains in Africa,as details about African isolates is confined [22].

Advances in the development of vaccine against *Neisseria*:

Although two centuries have passed since Vieusseux delineated epidemic meningococcal unwellness, *Neisseria* meningitidis remains the primary reason behind infectious disease and infection. Overwhelming meningococcal unwellness will develop rapidly and lead to mortality rates prodigious 100%. Thus, efforts to regulate the unwellness have centered on vaccination. In the past, vaccines against meningococcal unwellness did not offer immunogenicity and long protection in infants, UN agency square measure at greatest risk [42,43]. Meningococcal ill health occurring has attenuated throughout the last decade due to the presence of safty programs as connected with vaccines against serogroups A, C, Y, W. lately, the novel antigen named *4CMenB* were presented to decrease the ocuuring of the ill health by serogroups, the predominant disease-causing isolate in industrial countries [1]. To control short lived protection problem versus the meningococcus, conjugation of polysaccharides to a protein carrier has been applied, resulting in hanging T-cell on immunity and a memory response [44, 45].

Clinical options for patients:

Supplemental oxygen must be secured and also propped by ventilation as needed. As soon as possible parenteral antibiotics must be provided, and particularly when the patient needs to be transfer, the emergency team must provide the first dose of antibiotics during transport. the essential goal is to reduce the period between the arrival at a health facility and the primery dose of antibiotic "door to needle time" [46]. The primary treating of hemodynamic defect in meningococcal malady is using fluids. If it stills unsteady although with using adequate fluid revivification, the vasoactive medicine is required. catecholamine is employed for treating cardiovascular disease infused via main line .In the absence of catecholamine, Dopastat is given. just in case of associated heart muscle pathology, dobutamine is to be used. Principally Dopastat and dobutamine are used as vasopressors with sensible responsents [47].

Conclusions

The highest prevalence of the disease is in children, followed by a lower peak in adolescence, then becoming lower in old age. Neisseria infections have declined with a wide range in Europe and North America. Polysaccharide changes the capsule in the organism by the mechanism of capsule exchange, thus allowing the organism to escape the mechanisms of the human immune system and may lead to an increase in vaccine serotypes. Weakened immunity in children: The immune systems of children, especially infants and young children, are not yet fully developed, making them more susceptible to infections, including Neisseria meningitidis. Early exposure and overcrowding: Children are often in crowded nurseries or schools, which increases the chance of transmission of the bacteria through respiratory droplets. Lack of or delayed vaccination: Vaccination against Neisseria meningitidis is given on specific schedules. Children who are unvaccinated or have not completed their vaccination schedule are more susceptible to infection. Age-related immunity: As we age (in adolescence and adulthood), the body is exposed to bacteria or vaccines and thus develops natural or acquired immunity that protects it. Lifestyle changes: Older adults and adolescents are less exposed to crowded, enclosed spaces that facilitate the spread of infection, compared to children. Existence of widespread vaccination campaigns: In many countries, children and adolescents are vaccinated against meningococcal disease, which has led to a decline in cases among older adults.

Meningococcal disease is considered a severe infection caused by Neisseria meningitidis. The decline incidence of meningococcal sickness was observed with routine use of meningococcal vaccination. Early estimation and treatment of meningococcal disease square

measure essential in rising outcomes. the main point in treatment meningococcal infection is antibiotics, isolation and make contact with precaution, management of coagulopathies, and distinguishing those in danger might have gotten infected. Neisseria meningitidis DNA exhibits high genetic flexibility, enabling it to adapt to different environments, evade the immune system, and develop resistance to antibiotics. This makes it a dangerous bacterium that requires close monitoring and the continuous development of treatments and vaccines.

References

[1] Rouphael NG, Stephens DS. Neisseria meningitidis: biology, microbiology, and epidemiology. Neisseria meningitidis: advanced methods and protocols. 2012:1-20. https://doi.org/10.1007/978-1-61779-346-2_1

[2] Gabutti G, Stefanati A, Kuhdari P. Epidemiology of Neisseria meningitidis infections: case distribution by age and relevance of carriage. Journal of Preventive Medicine and Hygiene. 2015 Aug;56(3):E116.

[3] Mustapha MM, Harrison LH. Vaccine prevention of meningococcal disease in Africa: Major advances, remaining challenges. Human Vaccines & Immunotherapeutics. 2018 May 4;14(5):1107-15. https://doi.org/10.1080/21645515.2017.1412020

[4] Abio A, Neal KR, Beck CR. An epidemiological review of changes in meningococcal biology during the last 100 years. Pathogens and global health. 2013 Oct 1;107(7):373-80. http://dx.doi.org/10.1080/10426914.2011.552021.

[5] Hu JL, Tao H, Li JX, Dai WM, Song B, Sun JF, Liu P, Tang J, Liu WY, Wang SY, Zhu FC. Safety and immunogenocity of a novel combined Haemophilus influenzae type b–Neisseria meningitidis serogroups A and C-tetanus-toxoid conjugate vaccine in healthy Chinese children aged 6 months to 5 years old. Human Vaccines & Immunotherapeutics. 2015 May 4;11(5):1120-8. https://doi.org/10.1080/21645515.2015.1033592

[6] Charles-Orszag A. Cellular and molecular mechanisms of human endothelial cell plasma membrane remodeling by Neisseria meningitidis (Doctoral dissertation, Université Sorbonne Paris Cité).

[7] Barroso DE, Castiñeiras TM, Freitas FS, Marsh JW, Krauland MG, Tulenko MM, Fonseca ÉL, Vicente AC, Rebelo MC, Cerqueira EO, Xavier AC. Three outbreak-causing Neisseria meningitidis serogroup C clones, Brazil. Emerging infectious diseases. 2013 Nov;19(11):1847. https://doi.org/10.3201%2Feid1911.130610

[8] Halperin SA, Bettinger JA, Greenwood B, Harrison LH, Jelfs J, Ladhani SN, McIntyre P, Ramsay ME, Sáfadi MA. The changing and dynamic epidemiology of meningococcal disease. Vaccine. 2012 May 30;30:B26-36. https://doi.org/10.1016/j.vaccine.2011.12.032

[9] Marshall HS, McMillan M, Koehler AP, Lawrence A, Sullivan TR, MacLennan JM, Maiden MC, Ladhani SN, Ramsay ME, Trotter C, Borrow R. Meningococcal B vaccine and meningococcal carriage in adolescents in Australia. New England Journal of Medicine. 2020 Jan 23;382(4):318-27. https://doi.org/ 10.1056/NEJMoa1900236

[10] Hung MC, Christodoulides M. The biology of Neisseria adhesins. Biology. 2013 Jul 29;2(3):1054-109. https://doi.org/10.3390/biology2031054

[11] McNamara LA, Thomas JD, MacNeil J, Chang HY, Day M, Fisher E, Martin S, Poissant T, Schmink SE, Steward-Clark E, Jenkins LT. Meningococcal carriage following a vaccination campaign with MenB-4C and MenB-FHbp in response to a university serogroup B meningococcal disease outbreak—Oregon, 2015–2016. The Journal of Infectious Diseases.2017Nov1;216(9):1130-40. http://dx.doi.org/ 10.1093/infdis/jix446

[12] Bazan JA, Tzeng YL, Stephens DS, Carter AM, Brown MA, Snyder B, Prince DJ, Turner AN. Repeat episodes of symptomatic urethritis due to a uropathogenic meningococcal clade. Sexually transmitted diseases. 2020 Jan 1;47(1):e1-4. https://doi.org/10.1097%2FOLQ.0000000000001079

[13] Daugla DM, Gami JP, Gamougam K, Naibei N, Mbainadji L, Narbé M, Toralta J, Kodbesse B, Ngadoua C, Coldiron ME, Fermon F. Effect of a serogroup A meningococcal conjugate vaccine (PsA–TT) on serogroup A meningococcal meningitis and carriage in Chad: a community study. The Lancet. 2014 Jan 4;383(9911):40-7. http://dx.doi.org/10.1016/S0140-6736(13)61612-8

[14] Borrow R, Alarcón P, Carlos J, Caugant DA, Christensen H, Debbag R, De Wals P, Echániz-Aviles G, Findlow J, Head C, Holt D. The Global Meningococcal Initiative: global epidemiology, the impact of vaccines on meningococcal disease and the importance of herd protection. Expert review of vaccines. 2017 Apr 3;16(4):313-28. https://doi.org/10.1080/14760584.2017.1258308

[15] Nolan T, Booy R, Marshall HS, Richmond P, Nissen M, Ziegler JB, Baine Y, Traskine M, Jastorff A, Van der Wielen M. Immunogenicity and safety of a quadrivalent meningococcal ACWY-tetanus toxoid conjugate vaccine 6 years after MenC priming as toddlers. The Pediatric Infectious Disease Journal. 2019 Jun 1;38(6):643-50. https://doi.org/ 10.1097/INF.0000000000002334

[16] Diallo K, Feteh VF, Ibe L, Antonio M, Caugant DA, Du Plessis M, Deghmane AE, Feavers IM, Fernandez K, Fox LM, Rodrigues CM. Molecular diagnostic assays for the detection of common bacterial meningitis pathogens: a narrative review. EBioMedicine. 2021 Mar 1;65. https://doi.org/10.1016/j.ebiom.2021.103274

[17] Andrade AL, Minamisava R, Tomich LM, Lemos AP, Gorla MC, de Cunto Brandileone MC, Domingues CM, de Moraes C, Policena G, Bierrenbach AL, Cristo EB. Impact of meningococcal C conjugate vaccination four years after introduction of routine childhood immunization in Brazil. Vaccine. 2017 Apr 11;35(16):2025-33. https://doi.org/10.1016/j.vaccine.2017.03.010

[18] Jannic A, Mammeri H, Larcher L, Descamps V, Tosini W, Phung B, Yazdanpanah Y, Bouscarat F. Orogenital transmission of Neisseria meningitidis causing acute urethritis

in men who have sex with men. Emerging Infectious Diseases. 2019 Jan;25(1):175. https://doi.org/10.3201%2Feid2501.171102

[19] Mazamay, S. Guégan, J. Diallo, F. N .Bompangue, D. Bokabo, E. J. Muyembe, J. & Broutin, H. "An overview of bacterial meningitis epidemics in Africa from 1928 to 2018 with a focus on epidemics "outside-the-belt". BMC InfectiousDiseases" .2021. 21, 1-13. https://doi.org/10.1186/s12879-021-06724-1

[20] Jafri RZ, Ali A, Messonnier NE, Tevi-Benissan C, Durrheim D, Eskola J, Fermon F, Klugman KP, Ramsay M, Sow S, Zhujun S. Global epidemiology of invasive meningococcal disease. Population health metrics. 2013 Dec;11:1-9.https://doi.org/10.1186/1478-7954-11-17

[21] Acevedo R, Bai X, Borrow R, Caugant DA, Carlos J, Ceyhan M, Christensen H, Climent Y, De Wals P, Dinleyici EC, Echaniz-Aviles G. The Global Meningococcal Initiative meeting on prevention of meningococcal disease worldwide: epidemiology, surveillance, hypervirulent strains, antibiotic resistance and high-risk populations. Expert review of vaccines. 2019 Jan 2;18(1):15-30. https://doi.org/10.1080/14760584.2019.1557520.

[22] Tran TX, Le TT, Trieu LP, Austin CM, Van Quyen D, Nguyen HM. Whole-genome sequencing and characterization of an antibiotic resistant Neisseria meningitidis B isolate from a military unit in Vietnam. Annals of Clinical Microbiology and Antimicrobials. 2019 Dec;18:1-9. https://doi.org/10.1186/s12941-019-0315-z

[23] Juan, X. Yuquan, C. Mengmeng, Y. Jianxing ,Y. Fuyi, H. Li, X. & Shaoa, Z. "Prevalence of *Neisseria meningitidis* serogroups in invasive meningococcal disease in China, 2010 - 2020: a systematic review and meta-analysis." HUMAN VACCINES & IMMUNOTHERAPEUTICS 2022, VOL. 18, NO. 5, e2071077 (8 pages) https://doi.org/10.1080/21645515.2022.2071077

[24] Schipper K., Preusting L , Sorge N and Ende A. "Meningococcal virulence in zebrafish embryos depends on capsule polysaccharide structure." Front. Cell. Infect. Microbiol., 23 September 2022. Volume 12 - 2022 . https://doi.org/10.3389/fcimb.2022.1020201

[25] John,C. Phillips,N. Din,R. Liu, M. Hoiby, E. and Jorvis, G. "Lipooligosaccharide Structures of Invasive and Carrier Isolates of *Neisseria meningitidis* are Correlated with Pathogenicity and Carriage." LOS structural correlates with meningococcal pathology. The American Society for Biochemistry and Molecular Biology, Inc. Journal of Biological Chemistry. 2016 Feb 12;291(7):3224-38. https://doi.org/10.1074/jbc.M115.666214

[26] Cooper B, DeTora L, Stoddard J. Menveo®: a novel quadrivalent meningococcal CRM197 conjugate vaccine against serogroups A, C, W and Y. Expert review of vaccines. 2011 Jan 1;10(1):21-33. https:// doi.org/ 10.1586/erv.10.147

[27] Sridhar S, Greenwood B, Head C, Plotkin SA, Sáfadi MA, Saha S, Taha MK, Tomori O, Gessner BD. Global incidence of serogroup B invasive meningococcal disease: a systematic review. The Lancet infectious diseases. 2015 Nov 1;15(11):1334-46. https://doi.org/ 10.1016/S1473-3099(15)00217-0S1473-3099(15)00217-0

[28] Wang B, Santoreneos R, Giles L, Afzali HH, Marshall H. Case fatality rates of invasive meningococcal disease by serogroup and age: a systematic review and meta-analysis. Vaccine. 2019 May 9;37(21):2768-82. https://doi.org/10.1016/j.vaccine.2019.04.020

[29] Vogel U. Molecular epidemiology of meningococci: application of DNA sequence typing. International Journal of Medical Microbiology. 2010 Nov 1;300(7):415-20. https://doi.org/ 10.1016/j.ijmm.2010.04.017

[30] I. vaškevičienė, J. Silickaitė, A. Mačionienė, R. Ivaškevičius, A. Bulavaitė, V. & Gėgžna,Plečkaitytė M.. "Molecular characteristics of *Neisseria meningitidis* carriage strains in university students in Lithuania." BMC microbiology, 2023. 23(1), 352. https://doi.org/10.1186/s12866-023-031115

[31] Gorla MC, Pinhata JM, Dias UJ, de Moraes C, Lemos AP. Surveillance of antimicrobial resistance in Neisseria meningitidis strains isolated from invasive cases in Brazil from 2009 to 2016. Journal of medical microbiology. 2018 Jun;67(6):750-6. https://doi.org/ 10.1099/jmm.0.000743

[32] Caugant DA, Brynildsrud OB. Neisseria meningitidis: using genomics to understand diversity, evolution and pathogenesis. Nature Reviews Microbiology. 2020 Feb;18(2):84-96. https://doi.org/10.1038/s41579-019-0282-6

[33] McGill F, Heyderman RS, Michael BD, Defres S, Beeching NJ, Borrow R, Glennie L, Gaillemin O, Wyncoll D, Kaczmarski E, Nadel S. The UK joint specialist societies guideline on the diagnosis and management of acute meningitis and meningococcal sepsis in immunocompetent adults. Journal of Infection. 2016 Apr 1;72(4):405-38. https://doi.org/10.1016/j.jinf.2016.01.007

[34] Hamed MM, Mir FA, Elmagboul EB, Al-Khal A, Maslamani MA, Deshmukh AS, Al-Romaihi HE, Janahi MA, Abid FB, Kashaf AS, Sher G. Molecular characteristics of Neisseria meningitidis in Qatar. Scientific Reports. 2021 Feb 26;11(1):4812. https://doi.org/10.1038/s41598-021-84262-1

[35] Dass Hazarika R, Deka NM, Khyriem AB, Lyngdoh WV, Barman H, Duwarah SG, Jain P, Borthakur D. Invasive meningococcal infection: analysis of 110 cases from a tertiary care centre in North East India. The Indian Journal of Pediatrics. 2013 May;80:359-64.https://doi.org/10.1007/s12098-012-0855-0

[36] Dretler AW, Rouphael NG, Stephens DS. Progress toward the global control of Neisseria meningitidis: 21st century vaccines, current guidelines, and challenges for future vaccine development. Human vaccines & immunotherapeutics. 2018 May 4;14(5):1146-60. https://doi.org/10.1080/21645515.2018.1451810

[37] Stoddard J, Dougherty N. Universal immunization of infants against Neisseria meningitidis: Addressing the remaining unmet medical need in the prevention of meningitis and septicemia. Human Vaccines. 2010 Feb 1;6(2):219-23. https://doi.org/10.4161/hv.6.2.10330

[38] Unemo M, Nicholas RA. Emergence of multidrug-resistant, extensively drug-resistant and untreatable gonorrhea. Future microbiology. 2012 Dec 1;7(12):1401-22. https://doi.org/ 10.2217/fmb.12.117

[39] Unemo M, Shafer WM. Antimicrobial resistance in Neisseria gonorrhoeae in the 21st century: past, evolution, and future. Clinical microbiology reviews. 2014 Jul;27(3):587-613. https://doi.org/10.1128%2FCMR.00010-14

[40] Harrison OB, Claus H, Jiang Y, Bennett JS, Bratcher HB, Jolley KA, Corton C, Care R, Poolman JT, Zollinger WD, Frasch CE. Description and nomenclature of Neisseria meningitidis capsule locus. Emerging infectious diseases. 2013 Apr;19(4):566., https://doi.org/10.3201%2Feid1904.111799

[41] McMillan M, Chandrakumar A, Wang HL, Clarke M, Sullivan TR, Andrews RM, Ramsay M, Marshall HS. Effectiveness of meningococcal vaccines at reducing invasive meningococcal disease and pharyngeal Neisseria meningitidis carriage: a systematic review and meta-analysis. Clinical Infectious Diseases. 2021 Aug 1;73(3):e609-19. https://doi.org/ 10.1093/cid/ciaa1733

[42] Fitzwater SP, Watt JP, Levine OS, Santosham M. Haemophilus influenzae type b conjugate vaccines: considerations for vaccination schedules and implications for developing countries. Human vaccines. 2010 Oct 1;6(10):810-8. https://doi.org/10.4161/hv.6.10.13017

[43] Cohn AC, MacNeil JR, Clark TA, Ortega-Sanchez IR, Briere EZ, Meissner HC, Baker CJ, Messonnier NE. Prevention and control of meningococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). Morbidity and Mortality Weekly Report: Recommendations and Reports. 2013 Mar 22;62(2):1-28.

[44] Miglietta A, Fazio C, Neri A, Pezzotti P, Innocenti F, Azzari C, Rossolini GM, Moriondo M, Nieddu F, Iannazzo S, D'Ancona F. Interconnected clusters of invasive meningococcal disease due to Neisseria meningitidis serogroup C ST-11 (cc11), involving bisexuals and men who have sex with men, with discos and gay-venues hotspots of transmission, Tuscany, Italy, 2015 to 2016. Eurosurveillance. 2018 Aug 23;23(34):1700636. https://doi.org/10.2807/1560-7917.ES.2018.23.34.1700636

[45] Nadel S, Carcillo J. Treatment of meningococcal disease. Handbook of Meningococcal Disease Management. 2016 Jan 21:75-90. https://doi.org/10.1016/j.jadohealth.2016.04.013