



Weather Variables and Climatic Influence on the Epidemiology of Cerebrospinal or Meningococcal Meningitis

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ABSTRACT: Meningococcal infection has been recognized as a serious problem for almost 200 years. Cerebrospinal or Meningococcal meningitis (MCM) can occur as a sporadic case, institution based focal outbreak, or a large epidemic year. The distribution of serogroups causing meningococcal disease (A, B, C, Y, W-135) varies over time and by geographic location and spatial distribution of the disease indicate close linkage of it and weather climatologies. The highest burden is in sub-Saharan Africa, where it occurs in a cyclic mode. The paper while drawing facts on the disease history, African Meningitis Belt, causative agents, disease statistics, risk factors, and overall etiology, compares the information on cases and deaths of MCM from various literature reports, noted the linkage between regional climate variability/ seasonal variation and MCM. Efficient management of the risks of cerebrospinal meningitis requires a multi-sectoral approach, which brings information about how climate interact with the various elements of society, environment and economy. Strong partnerships and high-quality data are needed from all sectors involved. Climate services, that include climate advisories, predictions, warnings and alerts, could also help societies take advantage of the opportunities associated with climate events.

Keywords: Region, Weather variability, Meteorology

REVIEW ARTICLE

INTRODUCTION

Outbreaks involving disease are inherently more sensitive to climate and weather events. Bacterial meningitis is a disease involving the inflammation of the meninges, layers coating the brain and the spinal cord (CDC, 1998). It is a contagious disease, which has proved to be a major public health burden. It has three disease causing agents, and manifests itself in different forms depending on the type of infection agent. Acute purulent meningitis is the predominant form of meningitis under non-epidemic conditions. Meningococcal meningitis or cerebrospinal meningitis is the more common type, typically found under epidemic conditions. Meningococcal septicemia, in which bacteria are found in the blood stream, is less common, but more fatal. Despite the interventions that have been implemented, bacterial meningitis is yet a major source of morbidity and mortality. Meningococcal meningitis (MCM) has affected Sahelian Africa for centuries and became endemic over the past 25 y. During the 1980s, the World Health Organization (WHO) registered between 25,000 and 200,000 disease cases per year, with about 10% of them resulting in death, and with the highest infection rates observed in younger children (WHO, 2000). MCM became, therefore, a public health concern in the poorest regions in the world following the severe drought at the end of the 1970s (Sultan et al., 2006).

Every year West African countries within the Sahelo-Sudanian band are afflicted with major meningococcal meningitis (MCM) disease outbreaks, which affect up to 200,000 people, mainly young children, in one of the world's poorest regions. The timing of the epidemic year, which starts in February and ends in late May, and the spatial distribution of disease cases throughout the "Meningitis Belt" strongly indicate a close linkage between the life cycle of the causative agent of MCM and climate variability. However, mechanisms responsible for the observed patterns are still not clearly identified (Sultan et al., 2006). Recent findings concerning the population dynamics of some infectious diseases have clearly identified the importance of climate as a major driver (Paschual et al., 2000; Rodo et al., 2002). With evidence of the impact of large-scale meteorological phenomena such as El Niño on infectious disease patterns, modern epidemiology is now confronted with a scale problem in the identification of the spatiotemporal scales that might be relevant to explain patterns and processes (Guegan et al., 2005). The cyclic nature of disease and changing distribution of serogroups, combined with frequency of travel worldwide, underscore the need for a prevention strategy that incorporates all major serogroups.

This paper drawing on extensive studies conducted by various authors in Africa and the world, reports on the

world historical epidemiology of cerebrospinal meningitis, African meningitis belt, disease causing agents and statistics, and modes of infection as well as the symptoms. It considers the impact of weather climatology on the epidemiological trajectory of the disease, as well as the risk factors and tentative preventive measures of it.

World historical epidemiology and control of cerebrospinal meningitis

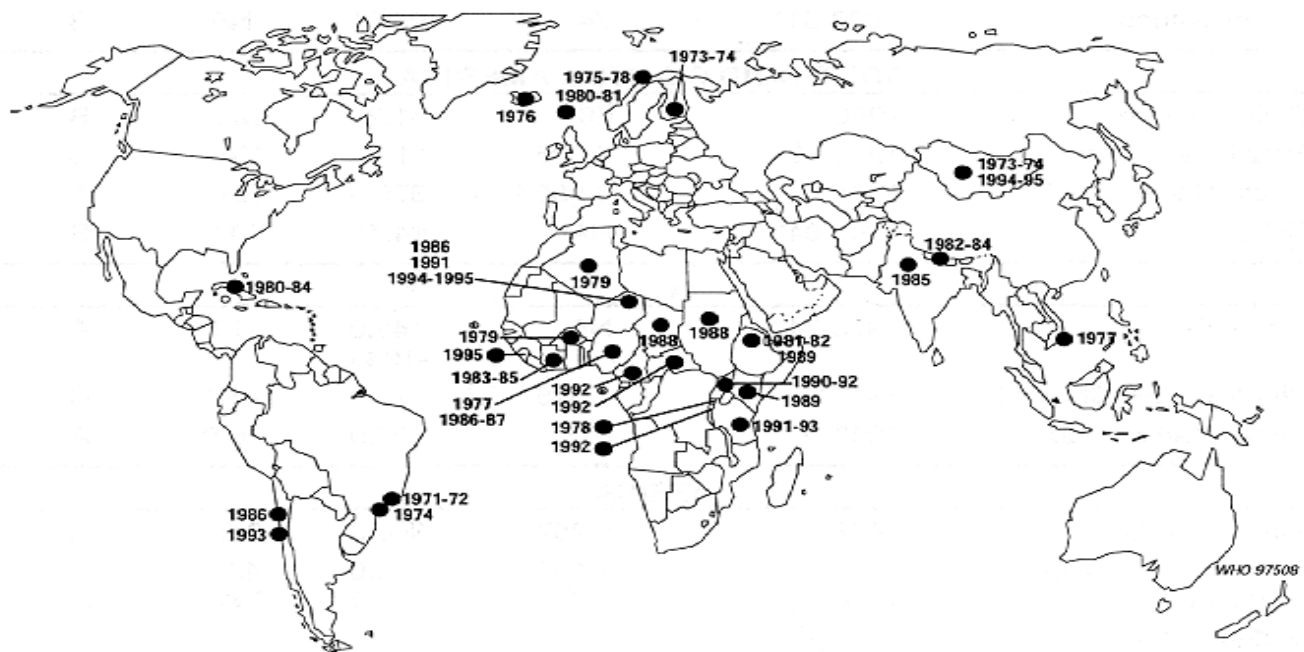
Clinical meningococcal disease was described as early as 1805 during an outbreak in Switzerland; however, *Neisseria meningitidis* was not identified as the causative agent of bacterial meningitis until 1887. The disease probably appeared in northern Africa in the 1880s; however cerebrospinal meningitis has been present in Sudan for much longer. The African continent has been vulnerable to epidemic outbreaks since at least 1909

(WHO, 1997a). Major outbreaks were also observed during World War I and II. Group A *Neisseria meningitidis* seldom caused epidemics in the temperate regions of the developed world after the Second World War; however, it has continued to wreak havoc in Sub-Saharan Africa.

This region experienced approximately 340,000 cases and 53,000 deaths between 1951-1960 alone. In describing epidemics throughout the world (see Figure 1), it is useful to keep in mind the degree of impact a particular incidence may have for a given country. European epidemics have rarely experienced epidemics as large as those in Africa. Therefore, epidemic conditions should be defined as that incidence rate which requires emergency control measures in a given country (WHO, 1997a).

Map 2. Major outbreaks of meningococcal meningitis, 1971–1997

Carte 2. Principales épidémies de méningite à méningocoques, 1971–1997



Norwegian outbreaks occurred in 30-40 year intervals (WHO, 1998). In the 1960s, meningitis was still considered a permanent public health problem in tropical zones but was no longer thought to be a threat in Europe and North America. However, epidemics have continued to occur throughout the world since the 1970s. Several countries in North and South America, Asia, and Europe experienced recurrent epidemics and persistent, sporadic infection. Epidemics again spread throughout India, Nepal, and Africa in the 1980s (WHO, 1998). The wave in the African continent extended beyond the meningitis belt to West Africa. The movement of the disease outside its traditional boundaries could reflect any number of changes in climate, increasingly mobile populations (either voluntary movement or refugee displacement), or the introduction of new strains into susceptible populations (WHO, 1998). The Eastern Mediterranean region of Africa experienced a meningococcal epidemic in 1988 after the return of pilgrims in 1987. Outbreaks have reduced since the early 1990s, after some countries began vaccinating high-risk groups (WHO, 1998).

The African meningitis belt:

The highest burden of meningococcal disease occurs in sub-Saharan Africa, which is known as the “Meningitis Belt”, an area that stretches from Senegal in the west to Ethiopia in the east, with an estimated total population of 300 million people (Figure 2). This hyperendemic area is characterized by particular climate and social habits. During the dry season, between December and June, because of dust winds and upper respiratory tract infections due to cold nights, the local immunity of the pharynx is diminished increasing the risk of meningitis. At the same time, the transmission of *N. meningitidis* is favoured by overcrowded housing at family level and by large population displacements due to pilgrimages and traditional markets at regional level. This conjunction of factors explains the large epidemics which occur during this season in the meningitis belt area. Due to herd immunity (whereby transmission is blocked when a critical percentage of the population had been vaccinated, thus extending protection to the unvaccinated), these epidemics occur in a cyclic mode. *N. meningitidis* A, C and

W135 are now the main serogroups involved in the meningococcal meningitis activity in Africa (WHO, 2003). In major African epidemics, attack rates ranges from 100 to 800 per 100 000 population, but individual communities have reported rates as high as 1000 per 100 000. While in endemic disease the highest attack rates are observed in young children, during epidemics, older children, teenagers and young adults are also affected. In 1996, Africa experienced the largest recorded outbreak of epidemic meningitis in history, with over 250 000 cases and 25 000 deaths registered. Between that crisis and 2002, 223,000 new cases of meningococcal meningitis were reported to the World Health Organization. The countries most affected countries have been Burkina Faso, Chad, Ethiopia and Niger; in 2002, the outbreaks occurring in Burkina Faso, Ethiopia and Niger accounted for about 65% of the total cases reported in the African continent. Furthermore, the meningitis belt appears to be extending further south. In 2002, the Great Lakes region was affected by outbreaks in villages and refugees camps which caused more than 2,200 cases, including 200 deaths (WHO, 2003).



Figure 2. African Meningitis Belt.

Source: World Health Organization (WHO, 1997b). Control of Epidemic Meningococcal Disease. WHO Practical Guidelines, Second Edition. WHO: Emerging and other Communicable Disease, Surveillance and Control.

Disease causing agents and statistics

Over 80% of bacterial meningitis can be traced to three disease causing agents: *Neisseria meningitidis*, *Streptococcus pneumoniae* and *Hemophilus influenzae* type b (WHO, 1998). Before the 1990s, *H. influenzae* was the predominant disease causing agent in the United States, accounting for 45% of cases, with an incidence of 2.9 cases per 100,000 populations (CDC, 1998). In the past few years, however, with the development of the new conjugate vaccine to *H. influenzae*, the incidence of disease has dropped from 421 cases per 100,000 populations to less than 0.7 per 100,000 populations (CDC, 1998). Today in the US, the most common cause of meningitis is *S. pneumoniae*, accounting for 30-50% of the disease, with an incidence of 0.6-1.2 per 100,000 populations. *N. meningitidis* follows next, accounting for 15-40% of the cases, with an incidence of 0.5-1 per 100,000 populations (CDC, 1998). *Neisseria Meningitidis*

has three serogroups: A, B and C. Serogroup A is the major cause of epidemic meningococcal disease all over the world (Figures 3 and 4). Serogroup B and serogroup C cause systemic disease. But the serogroup classification of meningococcal is done depending on the antigenicity of capsular polysaccharides (Stephens et al., 2005). At least 13 subtypes of *N. meningitidis* have been described: A, B, C, D, E, H, I, K, L, W-135, X, Y and Z. Of these, five serogroups (*N. meningitidis* A, B, C, Y and W-135) are recognized to cause epidemics and are responsible for >90% of cases (Gondim et al., 2005). There are several factors, which determine the virulence of the bacteria including (Stephens et al., 2005), Capsular polysaccharide, Outer-membrane proteins, Lipooligosaccharide (LOS), Bacterial pili, Opacity associated proteins (Opa, Opc proteins), Amount of blebbing or vesiculation of outer membrane, Specific nutrient acquisition factors (e.g. iron acquiring mechanism) (Pettersson et al., 1997).

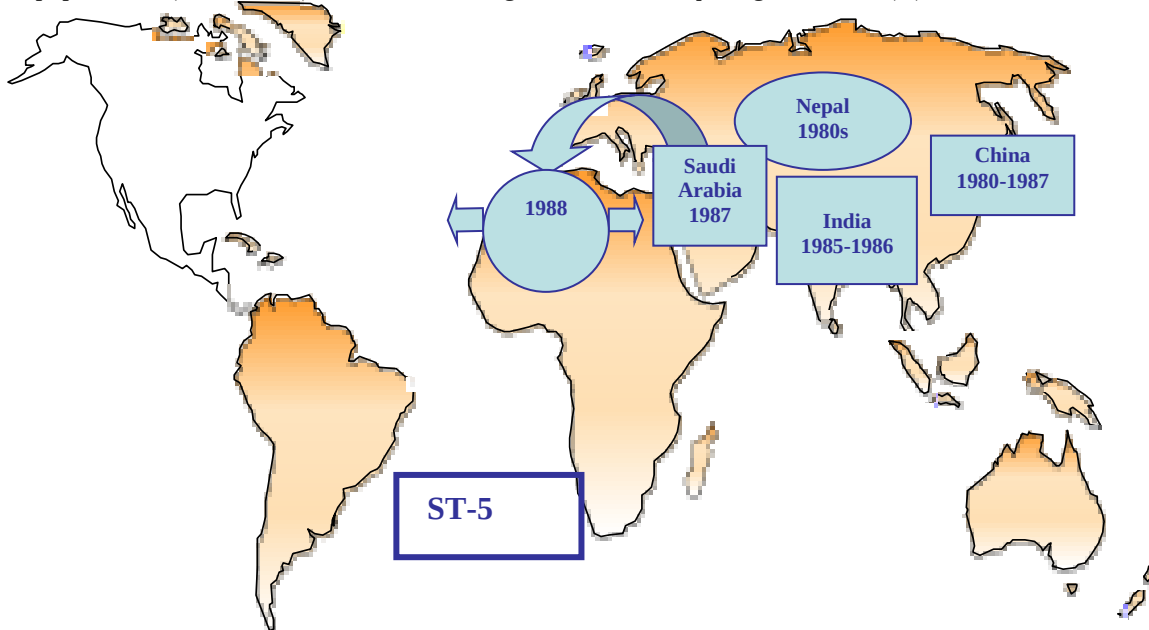


Figure 3. Global expansion of serogroup A meningococcus ST-5 complex
Source: adapted from Pierre Nicolas, WHO. (Nicolas et al., 2005)

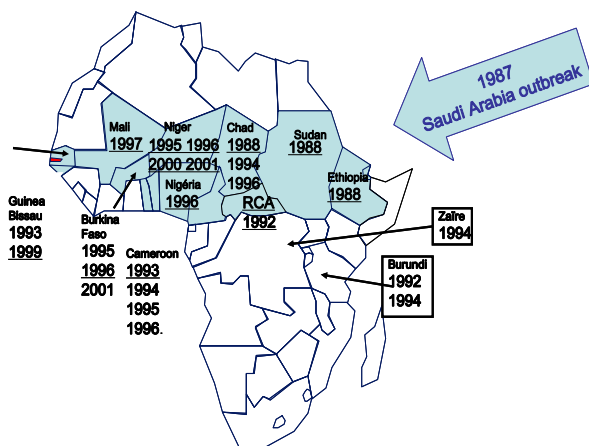


Figure 4. Serogroup A ST-5 expansion in Africa: 1988 – 2001. ST-5 was responsible for the most important epidemic ever seen in Africa in 1996 > 150,000 cases.
Source: Adapted from Pierre Nicolas, WHO: Nicolas et al. (2005). J Clin Microbiol. 2005, 5129-35.

Meningococcal disease can be categorized into two forms of disease: endemic disease and epidemic disease. The major cause of endemic disease is the meningococcus, *Neisseria meningitidis*, a Gram-negative bacteria. The endemic disease manifests itself in small clusters or sporadically. The incidence of endemic meningococcal disease range from 1 to 5 per 100,000 population in Europe and North America (WHO, 1998).

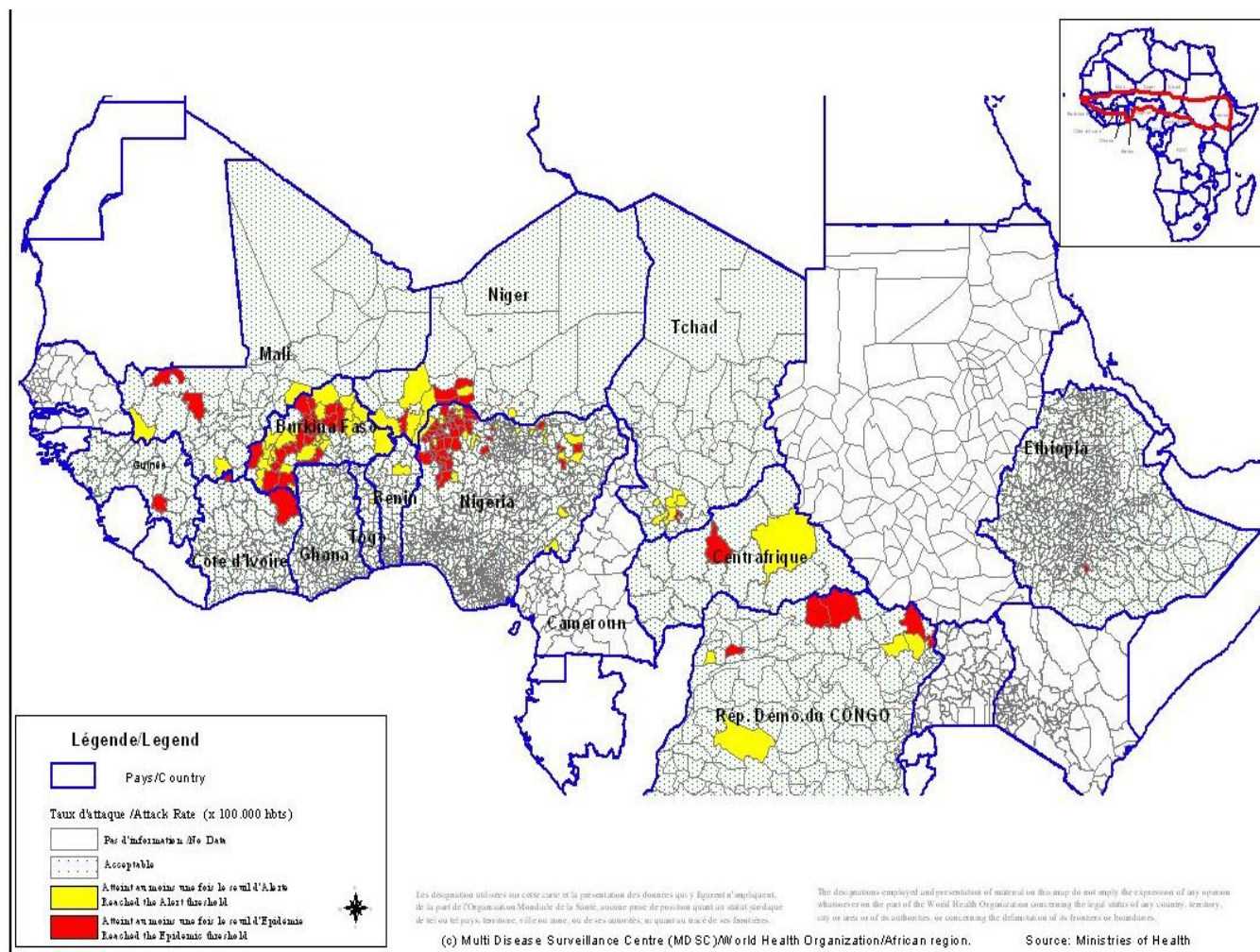


Figure 5. Alert and epidemic districts in African meningitis belt: Weeks 1-26, (2008).

Source: Adapted from Stéphane Hugonnet, WHO. In developing countries the incidence rates varies from <10 to >20 per 100,000 population (WHO, 1998).

Cerebrospinal meningitis or meningococcal meningitis caused by *Neisseria meningitidis* is the only disease that occurs in epidemic form. In developing countries, most often epidemic disease is indistinguishable from endemic disease because of the lack of adequate laboratory resources to clearly diagnose the disease (Figure 5). Thus meningococcal meningitis is grouped with the other endemic forms of bacterial meningitis. In addition to the epidemic cases, at least 1.2 million other cases of bacterial meningitis are reported, 135,000 of them being fatal. Under non-epidemic conditions children are more prone to bacterial meningitis, and 50-60% of cases occur in children between 3 months to 5 years of age. In countries within the meningitis belt, children between the ages of 5-10 are most susceptible to the disease (WHO, 1998).

Modes of infection and symptoms:

Meningococcal meningitis requires person-to-person contact. It is spread efficiently when the respiratory

droplets of the infected person are spread to other individuals. Common routes of transmission are: Coughing, Sneezing, Kissing, and Sharing of utensils. Humans presenting with meningococcal meningitis are the predominant carriers of the organisms. Asymptomatic individuals who carry the organism in the nasopharynx are the second most common reservoir (WHO, 1997a). The infection begins when the bacteria colonize the nasopharynx. The organisms have Pili on their surface to assist in adhering to nasopharyngeal mucosal receptors. Upon colonization; the organism infects neighboring tissues, and ultimately makes its way into the blood stream. After entering the bloodstream, the organism successfully penetrates the blood-brain barrier and invades the subarachnoid space. The host is unable to clear the infection because of the relatively low levels of complement activity and antibodies in the cerebrospinal fluid (CD, 1998). Most people present symptoms by three to four days (Philips et al., 1998). Bacterial meningitis is characterized by a range of symptoms (see Figure 6).

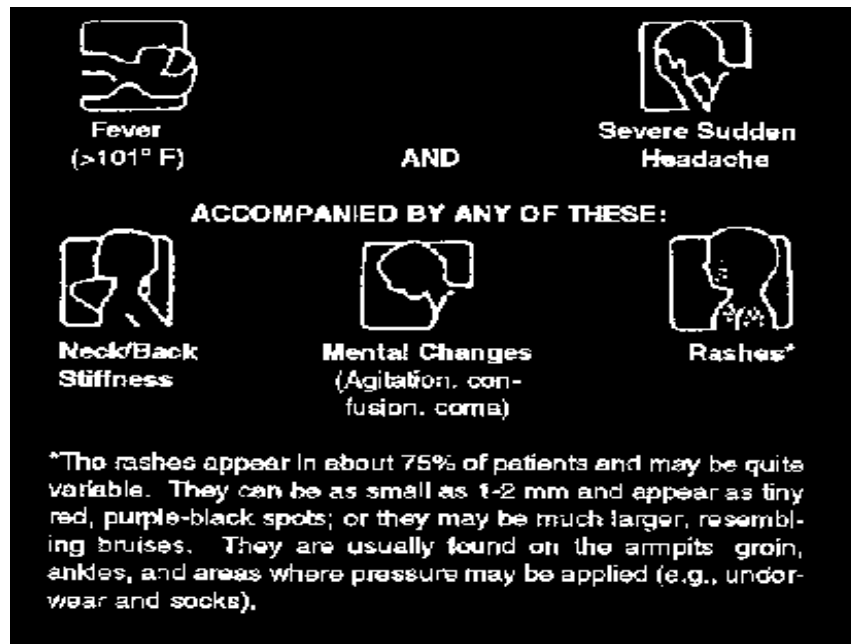


Figure 6. The usual signs and symptoms of meningococcal meningitis. Source: McKinley Health Center (1996).

More on Symptoms: Below are the symptoms of cerebrospinal meningitis as given by Rhode Island Department of Health: Patients may experience a severe, sudden headache, fever, a stiff neck or back, nausea and vomiting, and possibly a rash. 50-60% of adult and adolescent patients will present with signs of swelling such as hypertension and bradycardia.

Patients may develop neurological symptoms such as lethargy, delirium, coma, or convulsions. About 75% of patients experience petechial or purpuric rashes that range from tiny, reddish-purple spots to bruise-like marks. Usually these rashes are found on the armpits, groin, and ankles. Cerebrospinal fluid (CSF) samples will appear cloudy. Infants and young patients: Infants may present with a bulging fontanelle (soft spot), fever, irritability, vomiting, or lethargy (WHO, 1997a). CSF may or may not appear abnormal in such young patients (Rhode Island Department of Health). Elderly patients, Elderly patients may or may not demonstrate signs of inflammation such as fever, but may experience an altered level of consciousness and experience confusion or notice that senses have become less acute (Rhode Island Department of Health).

Infection due to *N. meningitidis* (from McKinley Health Centre, 1996). 10-20% of those individuals infected with *N. meningitidis* will develop septicemia. Septicemic patients will have a fever, rash, and hypotension. They may experience seizures, and even coma. CSF may or may not be cloudy. Septicemic patients progress rapidly, approximately 30% die because of they responded poorly to antibiotics.

Climatic influences and weather variables:

Among favorable conditions for the resurgence and then dispersion of the disease, climatic conditions may be important as environmental forces inducing periodic fluctuations of disease incidence. Recent findings concerning the population dynamics of some infectious diseases have clearly identified the importance of climate as a major driver (Rodo et al., 2002; Paschual et al., 2000). MCM outbreaks in West Africa usually start at the beginning of February, and then disappear in late May. The geographical distribution of disease cases is called the “Meningitis Belt” and is roughly circumscribed to the biogeographically Sahelo-Sudanian band (Cheesbrough et al., 1995; Lapeyssonnie, 1963). This Sahelo-Sudanian region has a dry winter, dominated by northern winds, called the Harmattan, followed by a wet season starting in spring with the monsoon. The co-occurrence in both space and time of MCM disease cases and climate variability within the Sahelo-Sudanian area suggests that the occurrence of MCM might be directly related to climate. So far, very few studies have tried to quantify the potential linkages that could exist between climate and MCM outbreaks. Meningitis in Africa is largely, though not entirely, confined to regions with a defined dry season. Meningitis epidemics always occur in the dry season. It is culturally associated with dust, which is seasonal (in fact, in many languages the name for meningitis is “sand disease”). Meningitis epidemics end abruptly with the start of the rainy season (Adams-Fogor et al., 2008). See Figures 7 and 8. The winter climate causes damage to the mucous membranes of the oral cavity through dry air and

strong dust winds, and creates propitious conditions for the transmission of the bacteria responsible for cerebrospinal meningitis; low absolute humidity and dust may enhance

meningococcal invasion by damaging the mucosal barrier directly or by inhibiting mucosal immune defenses (Figure 9).

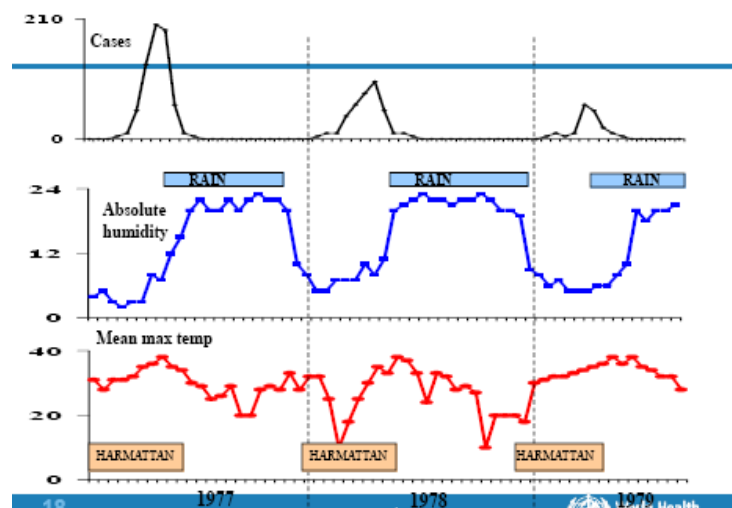


Figure 7. Season and Meningococcal Epidemics

Source: Greenwood et al. (1979), Lancet 1979; 73: 557-62 In: Sultan et al. (2006)

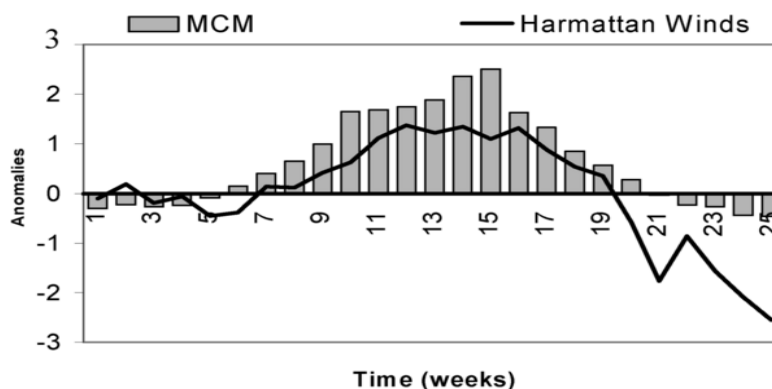


Figure 8: Temporal Patterns of Epidemics and Climate.

Weekly means of the Harmattan wind index over the 1994–2002 period and mean seasonal pattern of the number of cases of MCM (in standardized anomalies). Source: Sultan et al. (2006).

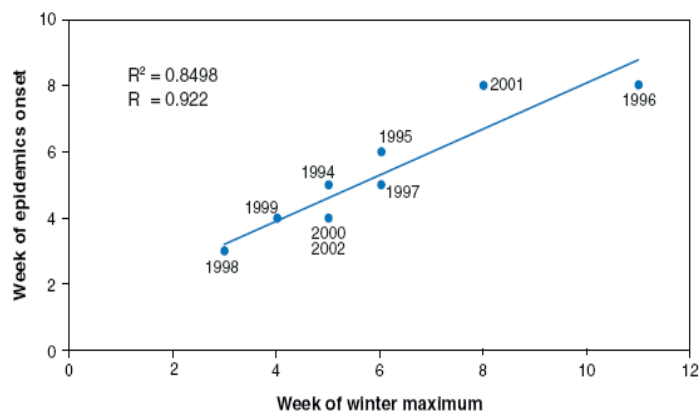


Figure 9. The Onset of Epidemics and the Winter Maximum.

Seasonal onset of cases may be triggered by climate. Scatter plot of the week of epidemic onset and the week of winter maximum over the 1994–2002 period shows a linear relationship with the timing of maximum Harmattanwinds. Source: Adams-Fogor et al. (2008); Sultan et al. (2006).

In contrast, higher humidity during both the spring and summer seasons strongly reduces disease risk by decreasing the transmission capacity of the bacteria (Molesworth et al., 2002; Besancenot et al., 1997), and MCM epidemics generally stop with the onset of rainfall (UCAR, 2008). In addition to the seasonal cycle, the link between climate and meningitis has also been documented at the interannual scale in northern Benin, where Besancenot et al. (1997) have suggested a positive

relationship between low absolute humidity and interannual variability in meningitis.

Meanwhile, although the global influence of climate is quite clear, the effects of climatic variability on MCM population dynamics are still only partially known because of the mixing of different processes acting at different spatial hierarchical scales, and the interactions between disease outbreaks and medical, demographical, and socioeconomic conditions.

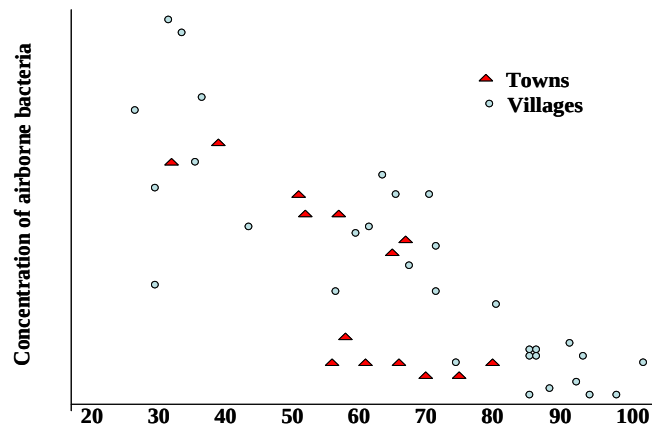


Figure 10. Humidity and meningococcal concentration. Source: Adams-Fogor et al. (2008).

The epidemics usually end with the onset of the summer rainy season. Researchers are uncertain why dry and dusty conditions are correlated with the disease. Some theorize that it may have to do with the mucous linings in people's respiratory systems becoming irritated by the dusty conditions. Others suspect changes in social behavior: residents tend to stay indoors during the dusty season, facilitating the spread of the disease. People are also at increased risk in winter as bad weather and dark evenings mean we tend to spend more time indoors and in closer proximity to others, which means germs are spread more easily (UCAR, 2009).

Risk factors and prevention:

Personal Risk Factors (from McKinley Health Centre, 1996): Damage to normal mucosal barriers, Possibly associated with concurrent upper respiratory infection, Crowded living conditions/poor ventilation, Low socio-economic status, Possibly associated with nutritional status. Prevention on a personal level (from McKinley Health Centre, 1996 and Rhode Island Department of Health): Carrier immunity, Immunization/vaccination and/ or chemoprophylaxis. Balanced diet, adequate rest and sleep, exercise, low stress (all help to maintain a strong immune system). Avoidance of upper respiratory tract illnesses and cigarette smoke inhalation, Covering mouth when coughing or sneezing, washing hands frequently. Epidemic Risk Factors (from WHO, 1997a; c; McKinley Health Centre, 1996).

Gathering of susceptible people in concentrated area/ migration, Nasopharyngeal carriage, Transmission of virulent strain, Low rainfall and humidity/dry season, Dust storms, Control and Prevention of Epidemics (from WHO, 1997a,c; McKinley Health Centre, 1996): Herd immunity/population immunity, Surveillance to monitor communicable disease and early outbreak detection, Establishment of emergency preparation and response committee to monitor and respond to rising levels of infection, Public education about early symptoms/ dispelling misconceptions regarding transmission, Policy guidelines for use of vaccine- preparation to ensure timely protection, Mass vaccination, Simplified treatment protocols due to potential shortages, logistical difficulties, and high incidence.

DISCUSSION

Meningococcal disease is a serious illness that can progress rapidly, resulting in substantial morbidity and mortality even with appropriate treatment. Despite progress in our understanding on the seasonality, and weather and climate dependency of cerebrospinal meningitis, societies, institutions and government still remain unprepared in disseminating the possible risk factors and easily achieved preventive mechanisms to the public in reducing the outbreak. Efforts to improve our ability to manage climate-dependent health risks cannot be addressed by a single institution. Efficient management of the risks of cerebrospinal meningitis requires a

multisectoral approach, which brings information about how climate and /or weather variables interact with the various elements of society, environment and economy. Strong partnerships and high-quality data are needed from all sectors involved. Importantly, enhanced climate services, that include climate advisories, predictions, warnings and alerts, could also help societies take advantage of the opportunities associated with climate events. It will aim to do this by improving the gathering and sharing of climate/ observations and products; encouraging interdisciplinary, targeted research to develop climate predictions, international services and tools to support their applications; encouraging the development of policies that support the application of climate services; and building national, regional, and institutional capacities to generate and apply climate services.

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