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Weather conditions and cerebrospinal meningitis in Benin (Gulf of Guinea, West Africa)

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Abstract. Over a 28-year period cerebrospinal meningitis, in sporadic as well as epidemic situations, mainly affected the Beninese territory from November to March, April or sometimes May. On the average, the acme occured in February–March. A regression analysis confirmed that 14 to 34.5% of the temporal variability of the disease was due to the northern trade wind (harmattan) and a low absolute humidity in the northern areas, which constitute the main epidemiological pole of the country. On the contrary, cerebrospinal meningitis and climate turned out to be fully independent one from the other in the southernmost areas, where the harmattan is seldom experienced although the meningitis belt is at the present time spreading southwards. But the case-fatality ratio was especially high in the coastal region and during the off season, i.e. when endemic meningitis predominantly affected small children aged under one year. In any way, the climate-meningitis relationship proved to be weaker than is sometimes assumed, perhaps because this relationship is partly overshadowed by both anthropic effects (vaccination campaigns) and latency before disease outbreak.

Key words: Absolute humidity, Cerebrospinal meningitis, Climate impact assessment, Harmattan, Seasonal pattern

Introduction

Cerebrospinal meningitis (CSM) causes a major public health problem in many parts of the world [21, 25, 27, 29]. It is almost everywhere one of the diseases included in the emergency notification system. In Africa the incidence is of such magnitude in the savannah region south of the Sahara and north of the equator that Lapeyssonnie [13] has referred to this area as the meningitis belt. This belt [23] extends some 4,200 km from Ethiopia to Senegal, and an average of 600 km from north to south between the 300-mm and 1,100-mm rainfall lines, with a southward digitation going deep into Benin. Across this Sudano-sahelian area, endemo-sporadic infections occur annually during the dry season (October–May), while large epidemics appear at longer intervals [11]. The mean annual incidence must be around 70 cases per 100,000 persons, yet attack rates during epidemics range from 100 to 1,000 per 100,000 inhabitants per year.

CSM is known in Mali as *finyabana*, literally 'wind illness'. In other countries, people have also noted that the outbreak of the epidemics nearly always coincides with the setting in of the harmattan, a north to east wind, blowing from the Sahara towards the Gulf of Guinea [5, 8, 9, 29]. The harmattan, described as forming part of the trade wind circulation [3], is most prevalent from November till February, when the barometric gradient between the subtropical anticyclonic cell and the equatorial trough is most marked, although the wind can be experienced in some locations as late as May. Temperatures during this period are low (sometimes 16–19 °C at 06:00 h), but they rise sharply towards the end of the season. The inhaled air is so dry that children and adults have badly chapped lips and nasopharyngeal mucosa become irritated. However, presumably the most distinctive characteristic of the harmattan is the dust it carries – almost impalpable particles of quartz and clay, colloids or fine mika flakes which can remain airborne for days and which occasionally reduce visibility below 200 metres for up to forty-eight hours. Therefore, the spread of CSM during the harmattan season was empirically recognized, as was the belief that meningitic activity wans at the onset of the rains. Nonetheless, the reasons for this pattern remain unclear. The present study attempted to describe a few of the epidemiological features of CSM at various locations around Benin, and to consider in more detail the question of the space/time characteristics of seasonal reinforcement of endemic as well as epidemic meningitis. Such a study ought to be of particular interest in a country where dif-

ferent latitudinal patterns shape the landscape and have shown the evidence of different geographical features along a sharp climatic gradient [1, 2].

Materials and methods

Two types of data were used, namely health records and meteorological ones.

Health records. Monthly numbers of CSM cases and deaths were obtained, partly from the Beninese Ministry of Public Health, partly from the World Health Organization (WHO). All these morbidity and mortality data were available for the 28-year period 1965–1992, for Benin as a whole and for its main administrative units. As laboratory facilities for diagnosis by latex agglutination, counterimmunoelectrophoresis or meningococcal antigen assay were uncommon, the included cases had to fulfil one of the minimum criteria of CSM given by the WHO, that is either a positive culture of *Neisseria meningitidis* from the cerebrospinal fluid or Gram-negative intracellular diplococci morphologically identical with *N. meningitidis* in smears of cerebrospinal fluid, even when the macroscopically turbid cerebrospinal fluid was sterile on culture. However, it may be assumed that these records were incomplete, especially in the north, where laboratory facilities were not easily available. So we have separately collected the cases diagnosed from 1981 to 1992 on clinical grounds alone, without bacteriological confirmation. While these clinically defined cases are not free of problems (misdiagnosis, mistaking group A meningococcal meningitis for other causes of sporadic meningitis, etc.), they should not be really influenced by such artefacts as variations in supply of laboratory reagents, overcrowding of local health-care facilities by an influx of patients, or enthusiasm of clinicians for performing a lumbar puncture.

Meteorological records. Meteorological measurements of air temperature (°C), relative humidity (%), vapour pressure (hPa), and dust haze (yes/no) for the corresponding time period were collected from the Agency for Airline Safety in Africa and Madagascar (ASECNA). One weather station was chosen to represent each region, namely Cotonou Airport for the Atlantic, Ouémé and Mono, Savé for Zou, Natitingou for Atacora, and Kandi for Borgou.

Two meteorological indices were used to investigate whether weather conditions could explain the spatiotemporal variations of CSM.

Firstly, the harmattan ambiance was identified and numerically characterized on a 10-day scale. The definition of this weather pattern was, to a certain extent, an arbitrary matter, but it was based on a long experience of the country and it had already proved to be relevant in climatopathological studies [3, 20]. To get an expression of harmattan exposure, we identified a specific meteorological situation when three conditions were simultaneously satisfied, namely a minimal mean temperature of no more than 20 °C, a mean relative humidity of no more than 40%, and the presence of at least three days of dust haze. Bearing in mind the plausible hypothesis that the recurrence of a weather event gradually increases the effect it produces, we finalized an index of harmattan intensity (H). This index consisted in assigning 0 to a period of ten days that did not satisfy the three criteria above, 1 to a period that fulfilled them for the first time, 2 to the second consecutive period of ten days that fit the requirements, 3 to the third period that met them, and so forth up to a theoretical maximum of 36, which would have implied a whole year under the influence of the harmattan. Conversely, towards the end of the season, the chain was deemed to be broken: the index reset at zero as soon as there was a 10-day period in which the criteria were not met. The mean obtained values are given in Figure 1 for two characteristic stations.

Secondly, as several authors saw in it the critical climatic variable [4, 8], absolute humidity U in grams per cubic metre of air ($g.m^{-3}$) was calculated at a 10-day scale from vapour pressure P_V (hPa) and temperature T (°C) using the equation:

$$H = 216.5\ P_V/T + 273.$$

Statistical analysis. All results were coded and a computerized database was set up to facilitate analysis. Relationships between CSM cases or deaths (dependent variables) with H or U values (independent variables) were assumed by means of time series simple linear regression analyses, successively for the whole chronological series and for each of the 12-month periods, so that one could detect which years the random parts were mainly related to. In all cases, the square of the Bravais–Pearson's coefficient r (determination coefficient R^2) was used to assess the amount of variation of the dependent variable accounted for by the selected independent variable, with $p < 0.05$ as the level of significance.

Results

Most of Benin, extending roughly from 6 to 12° north latitude, lies within the meningitis belt, and nowhere else in sub-Saharan Africa does this belt lie further south. It had been thought that the disease probably did not appear in the country prior to 1905 [28], but since then the epidemiological situation has been characterized by an endemic state with large outbreaks developing at variable intervals.

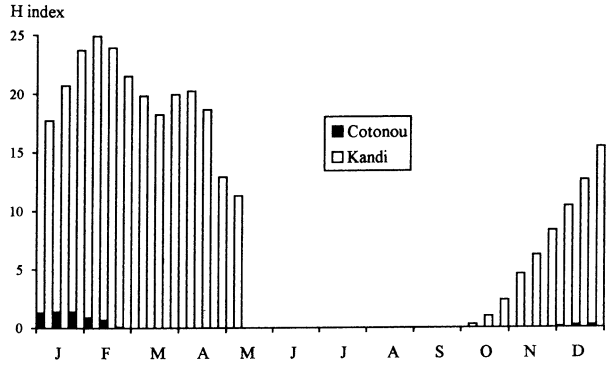

Figure 1. The Harmattan index (H) in two representative Beninese stations (mean of all the yearly indices over the period 1965–1992). See the text for the further explanation on the determination of H. The location of Cotonou and Kandi is indicated in Figure 4.

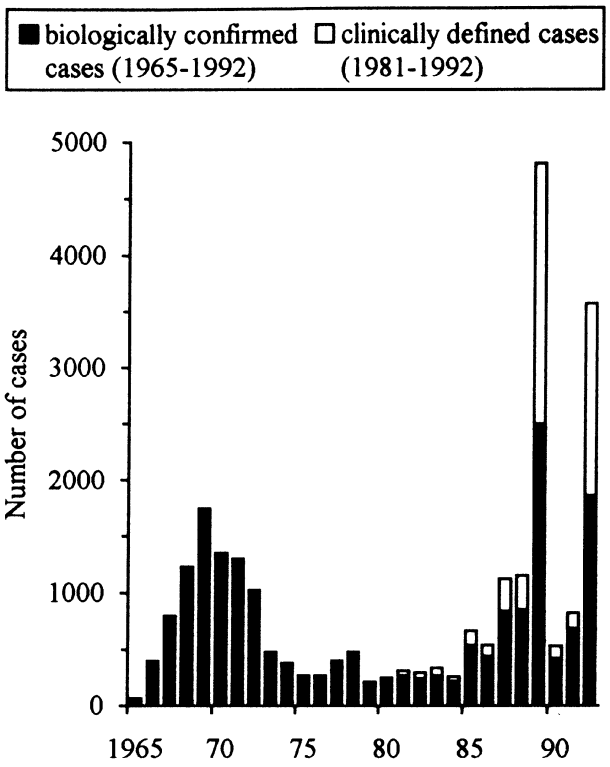

Figure 2. Reported cases of cerebrospinal meningitis by year, Benin. Biologically confirmed cases over the period 1965–1992, clinically defined cases over the period 1981–1992 only.

Morbidity

Figure 2 indicates that the record of biologically confirmed cases (black columns), with both sexes and all age groups pooled, varied greatly from year to year, with extremes being 68 and 2,501 cases over the period 1965–1992. During these 28 years, Benin experienced two (if not three) major epidemic cycles. The incidence of CSM began to rise in 1966, to reach a first peak in 1969. Then incidence fell rapidly. The whole period 1973–1984 was characterized by a very low annual number of cases. After that CSM again appeared in epidemic form in 1985 and the number of cases increased until 1989. Next CSM returned to low levels in 1990, thanks to a mass immunization campaign. However, despite expansion of the vaccination programme, a new wave of epidemics broke out (unless it was the same that went on after a short break) in 1991, and developed in 1992. Unfortunately, the available series was not long enough to bear out the circadecennial periodicity (8- to 14-year cycles) that has been observed for epidemics in bordering countries [11, 22, 23, 26, 28].

The cases notified only on clinical grounds (white columns) were very rare during endemic years (1981–1984), but become almost as numerous as the previous ones during epidemic years (1989, 1992).

The mean monthly occurrence of cases is plotted in Figure 3. CSM was present year-round, but with a quite regular cyclic pattern. The *averaged* seasonal distribution resulted in an increase when dry and dusty weather set into the area (particularly in February–March), while few cases occurred throughout the rainy season (especially in August and September). To sum up, approximately 75% of the biologically confirmed cases and 85% of the clinically based ones were seen during the five months of December through April, with the result that the seasonal variation in incidence reached highly significant limits ($p < 0.01$). Nevertheless, incidence *in any one year* was not necessarily consonant with the averaged seasonal rhythm: for January 1990, only one CSM case was notified for the whole of Benin, and another was clinically diagnosed, while in the same year there was an enormous epidemic outburst in December; similarly, an unexpected rainy-season peak occurred in 1976 and 1991.

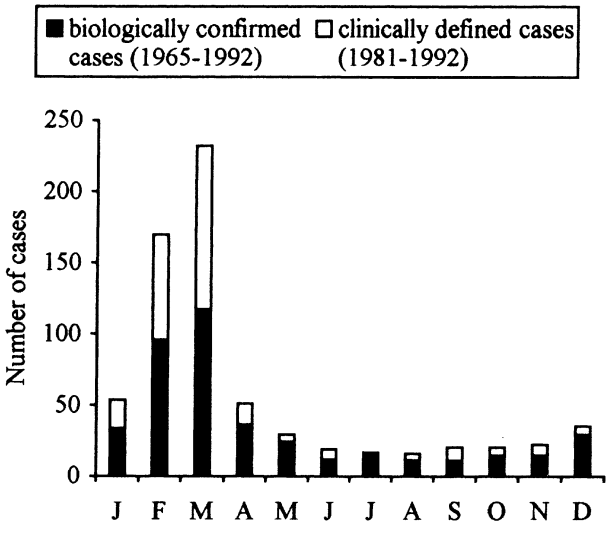

Figure 3. Mean monthly occurrence of cerebrospinal meningitis, Benin. Biologically confirmed cases over the period 1965–1992, clinically defined cases over the period 1981–1992 only.

In addition, CSM was not uniformly distributed throughout the country, mean morbidity being much higher in the northernmost regions (Atacora: 63.7 cases per 100,000 inhabitants; Borgou: 45.3) than in the central and southern areas (8.9/100,000 in Zou, 6.7 in the Atlantic region, 4.3 in Ouémé, 2.5 in Mono). This latitudinal gradient is shown in Figure 4. Even if the partition into six administrative units is not sufficient to give a faithful reproduction of all internal differences of a 112,600 km^2-wide country, it is clear that the south of Benin, exposed to a Guinean type subequatorial climate, is not really part of the meningitis belt, just as it is not really part of the harmattan domain (with high absolute humidities throughout the year, including January–February). However, this assertion should be qualified with two points. On one hand, the spatial distribution was affected by a strong interannual variability. The mean pattern illustrated in Figure 4 was rather characteristic of endemic years, and it might be subjected to enormous alterations during an outbreak. Thus, while Atacora was mainly affected by the 1989 epidemic, the one of 1992 (whose initial focus was in Borgou) quickly spread out over the whole country, until it reached the coastal areas. On the other hand, it has been thought since the great Sahelian drought that Lapeyssonie's belt was reportedly spreading southwards. Thus, the three southern Beninese regions, containing two thirds of the total population, recorded 16.4% of all CSM cases in 1984 but their contribution rose to 44% in 1991. It is unlikely that better reporting of cases resulted by itself in this geographical shift. But attention must be drawn to the fact that in the south the epidemic outbursts occured from July to September (in 1991, 47 cases in August and 30 in September in the Mono; in 1992, 101 cases in the Atlantic region and 142 in the Ouémé during the months of July and August). On a year by year basis there was not any significant correlation between disease activity in the two northern areas and in the three southern ones (r = 0.11; $p > 0.05$): such a result evidences the strength of the barrier that climate differences across the country present.

Mortality

In many respects, deaths from CSM led to diametrally opposite patterns, because mortality was continually higher in summer than in winter (Figure 5a), and in southern Benin than in the northern areas (case-fatality ratio as high as 21.0% in the Atlantic region, or 16.0% in Ouémé, versus 11.1% in Atacora and 10.1% in Borgou). Furthermore, the mean case-fatality ratio (Figure 5b) culminated in September (24.4%) and fell to more tolerable levels in December (6.9%). Thus, CSM appeared to be proportionally the most fatal in the areas and during the periods that experienced the lowest incidence.

Meteorological conditions

During our study period, harmattan blew only a few days in December and January at Cotonou (6° N),

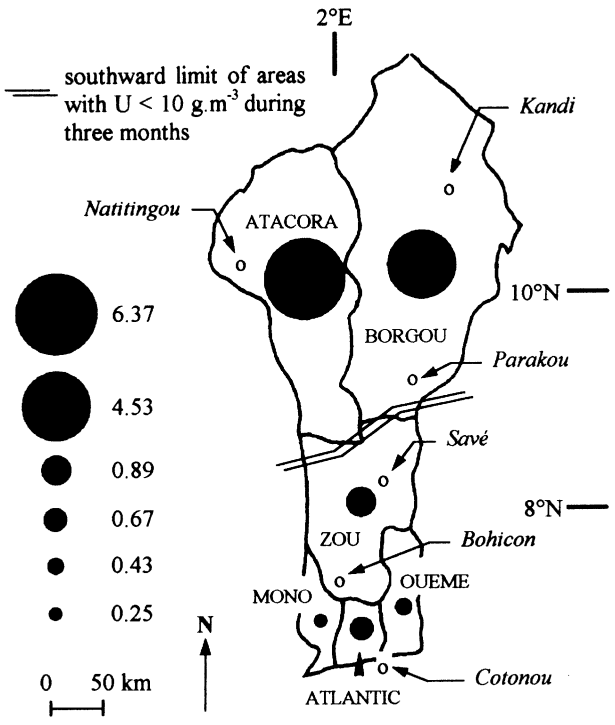

Figure 4. Spatial distribution of cerebrospinal meningitis, Benin, 1965–1992 (incidence rate per 100,000).

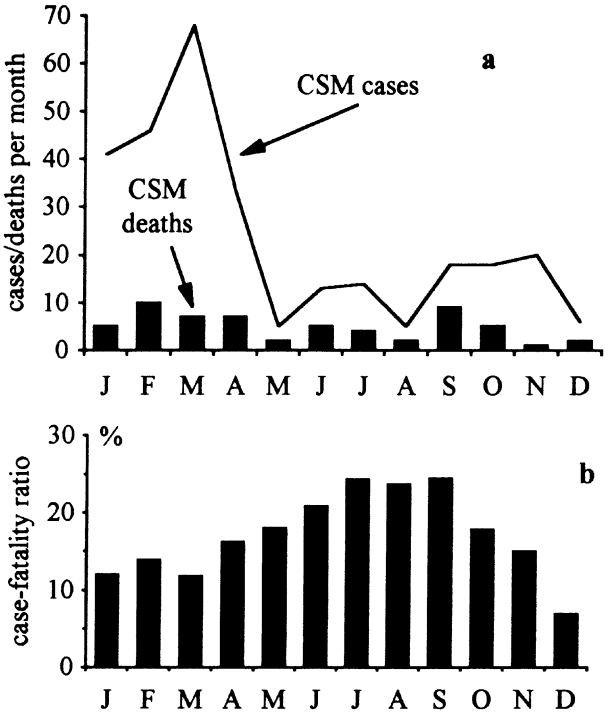

Figure 5. Morbidity, mortality (a) and mean case-fatality ratio (b) from cerebrospinal meningitis in Benin, 1965–1992.

on average seven years out of ten, while its mean relative frequency reached 2–3% of the year at Bohicon (7° N) in December and January, nine years out of ten, 10.5% at Savé (8° N) from November through February, every year, 27.2% at Parakou (9° N) from November through February– March, 21.5% at Natitingou (10° N) and 37.2% at Kandi (11° N), from October through April. Absolute humidity reached its nadir during harmattan and in northern Benin, where it remained for up to three consecutive months below 10 $g.m^{-3}$, but averaged values as high as 19 and 24 $g.m^{-3}$ (respectively in the north and in the south) were recorded in wet season. From year to year, H showed a strong correlation with U ($r = 0.94$; $p < 0.001$) in both Atacora and Borgou, while the correlation did not make any sense in the coastal areas because of harmattan being very infrequent and absolute humidity following a different annual rhythm (Figure 6): at Cotonou, for example, U regularly peaked in February (24.0 $g.m^{-3}$) and reached its lowest value in August, even if this minimum was as high as 20.5 $g.m^{-3}$.

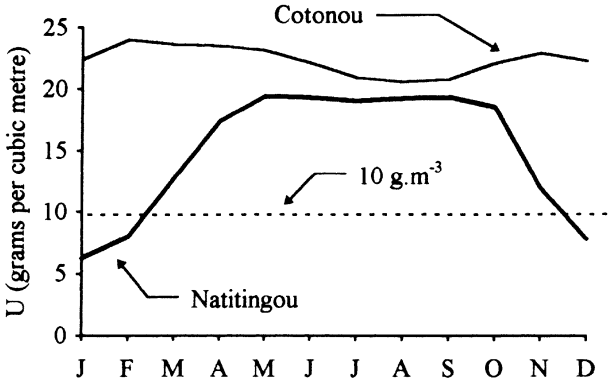

Figure 6. Mean absolute humidity ($g.m^{-3}$) in two representative Beninese stations, 1965–1992. The location of Cotonou and Natitingou is indicated in Figure 4.

Morbidity-weather relationship

The analysis of the effects of harmattan on CSM prompted us to distinguish between two main geographical sectors, northern versus southern Benin, on either side of the line (approximately 8°30′ N in lowlands) which limits southward the area with mean absolute humidity remaining during three consecutive months below 10 $g.m^{-3}$ (Figure 4).

North of 8°30′ N, in both Atacora and Borgou regions (U < 10 $g.m^{-3}$ from December to February or March) the regression model showed an undeniable statistical dependency of CSM on the intensity of harmattan weather (Table 1a) and on the absolute humidity (Table 1b). Thus, the H index accounted for about 14% of the morbidity variance in Atacora, and for 34.5% in Borgou. Be that as it may, these percentages showed a noteworthy interannual variability, attested by R^2 values ranging from 0.31 to 0.58 in Atacora (Table 2) and from 0.21 to 0.73 in Borgou (not shown). Some years with the strongest harmattan (1988–1989, on a lesser level 1991–1992) were epidemic years, while others (1982–1983) were not. However, only one or two years did not show any significant influence of harmattan on meningitis incidence (1989–1990 in Atacora, 1985–1986 and 1988–1989 in Borgou).

On the contrary, the data provided in Table 1 established that no relationship could be demonstrated between CSM and H (or U) *in the four southern regions*, including the Mid-Benin. In these areas, the part of variance explained by the harmattan intensity or by absolute humidity was always less than 2%, and never statistically significant, in averaged as well as in individual years.

Mortality-weather relationship

Once again results obtained for mortality were very different from the morbidity pattern. Table 3 indicates that the H index never explained more than 8% of the variance of lethality, this last result being obtained in Borgou.

Discussion

The meteorological factors that cause the transition of CSM from an endemic steady-state to a rapidly evolving epidemic are not yet well understood, since

Table 1. Morbidity-weather relationships: results of the linear regression for the period 1984–1992 as a whole

	a) CSM/H regression			b) CSM/U regression		
Region	r	R^2	Significance	r	R^2	Significance
Atacora	0.375	0.141	$p < 0.05$	0.420	0.176	$p < 0.05$
Borgou	0.587	0.345	$p < 0.05$	0.523	0.274	$p < 0.05$
Zou	0.090	0.008	ns	0.132	0.017	ns
Ouémé	0.126	0.016	ns	0.158	0.025	ns
Mono	0.187	0.035	ns	0.287	0.082	ns
Atlantic	0.065	0.004	ns	0.144	0.021	ns

CSM: cerebrospinal meningitis; H: harmattan index; U: absolute humidity (see the text for further explanation).

Table 2. Morbidity-harmattan relationship: results of the linear regression in the Atacora, for each year during the period 1984–1992 (see the text for further explanation)

Year	*r*	R^2	Significance	Regression equation
1984–1985	0.580	0.336	$p < 0.05$	y = 0.067 H + 3.32
1985–1986	0.659	0.435	$p < 0.05$	y = 0.11 H + 4.09
1986–1987	0.575	0.330	$p < 0.05$	y = 0.45 H + 1.1
1987–1988	0.760	0.583	$p < 0.05$	y = 1.7 H – 1.81
1988–1989	0.659	0.434	$p < 0.05$	y = 8.5 H + 2.56
1989–1990	0.556	0.310	ns	y = 0.32 H + 0.9
1990–1991	0.760	0.585	$p < 0.05$	y = 0.41 H + 6.24
1991–1992	0.659	0.435	$p < 0.05$	y = 1.7 H + 10.8

Table 3. Mortality-harmattan relationship: results of the linear regression with the H index for the period 1984–1992 as a whole

Region	r	R^2	Significance
Atacora	0.265	0.070	ns
Borgou	0.28	0.080	ns
Zou	0.170	0.030	ns
Ouémé	0.161	0.026	ns
Mono	–0.103	0.011	ns
Atlantic	0.095	0.009	ns

the same factors may affect the risk of exposure to a meningococcal carrier, the likelihood that transmission will occur, and the development of invasive disease. However, three major questions emerge from our study. First, does CSM always occur in the wake of the harmattan and, if so, what mechanisms do underlie the association of CSM with the weather? Secondly, could the present-day shift of the meningitis belt be considered as climate-related? Lastly, how can the difference between the patterns of mortality and morbidity be explained?

Does meningitis occur in the wake of the harmattan?

First and foremost, it can be argued that our results are consistent with the earlier findings and reinforce the classical explanatory model of CSM-weather relationship [11]. *In an averaged situation*, the risk increases for seasons and locations where the continental trade wind blows with the greatest regularity and where the absolute humidity reaches its lowest values. In contrast, it is hard to agree with the popular view quoted by J. Samways [24] that meningitis is completely in the harmattan's wake. There are four reasons for this:

1. The major peaks of CSM generally occur *after* and not during the main harmattan season; in March the dry wind blows with a lower regularity, absolute humidity increases (12,8 $g.m^{-3}$ at Natitingou) and temperatures are high both day and night (respectively 37.4 and 22.9 °C).
2. Many dry seasons pass without epidemic outbreaks.
3. Very often individual years depart from the mean model.
4. In any case, the part of total variance explained by the regression model involving the harmattan intensity (or the absolute humidity) remains rather low, with a maximum of 34.5%, and drops to zero along the Atlantic coast.

However, it is probable that the impact of climate could hardly exceed this degree of correlation because two super-imposed cycles interfere with each other, one relating to season and the other relating to new strains of *N. meningitidis* arising from time to time probably combined with the growing up of a new cohort of relatively unexposed group of children and adolescents in the population (which must have an approximately 10-year period).

If the classical model provides a reasonable explanation for several important aspects of the behaviour of epidemic or endemic CSM, it appears to be rather inaccurate in some other respects. A possible reason for this lies in the fact that our data were incomplete and not always accurate. A latent period of 3–8 days usually separates a climatic aggression and the disease outbreak [11]. It follows that monthly compilation of cases or deaths can only give a rough indication of the epidemic trend, and it is probable that meningitis case counts over a 10-day period would have provided more significant results. Similarly, incidence rates at the regional level may not accurately reflect the intensity of the disease at the village or city level since it has been reported that meningitis is never uniform over large areas [17]. It is also questionable whether meteorological data provided by a single station are representative of a larger area around the point of measurement. Last, anthropic action may disturb the natural effect of climate on disease. By way of proof, the worse coefficient of determination obtained in Atacora concerned the harmattan season 1989–1990, which coincided with an extensive vaccination programme covering 227,828 persons. In the same way, in Borgou, CSM was uncorrelated with the harmattan only during the years 1985–1986 and 1988–1989

when a mass immunization campaign was performed. Although it is too early to assess the full impact of vaccination on all aspects of the disease's epidemiology, it is clear that vaccines confer significant short-term protection in certain age groups, so much so that the diffusion of the meningitis is completely disturbed.

Having said that, how climate modulates CSM remains unclear. Much has been written about the dry season prevalence of the disease across sub-Saharan Africa. With a few rare exceptions [4, 6], it is today acknowledged [6, 12, 19, 25] that the overall carriage rate of meningococci does not change significantly from month to month nor correlate with the risk of invasive disease. The results of many studies of nasopharyngeal carriage elsewhere in Africa [7, 9] have not suggested in fact that transmission of *N. megintitidis* decreases markedly with the start of the rains [8], a circumstance indicating that transmission alone is not sufficient to trigger an epidemic [16]. Moreover, even if the number of carriers reaches a peak in places at the height of the wet season [5, 27], no clinical cases of CSM are necessarily observed. These findings suggest that transmission of aerosolized particles containing potentially pathogenic meningococci continues throughout the year but that the proportion of clinical cases to carriers changes with the season, suggesting that climate primilarly influences co-factors necessary to permit invasive disease, such as mucus desiccation, herd immunity [10, 16] or upper respiratory tract infections. Thus, the seasonality of CSM is probably due to increased invasiveness rather than to transmission of meningococci during the dry season.

It is interesting to note that upper respiratory infections and CSM roughly follow the same seasonal rhythm (Figure 7). In particular, throat infections diagnosed from 1987 to 1992 on clinical grounds peaked between January and February in two health care services of Natitingou (Atacora: Ch. S. Houssou, personal communication), while they reached their highest incidence in June or July at Cotonou (J. Gandonou, personal communication). In both locations, the peak of pharyngeal diseases occured about one month before meningitis cases become the most frequent.

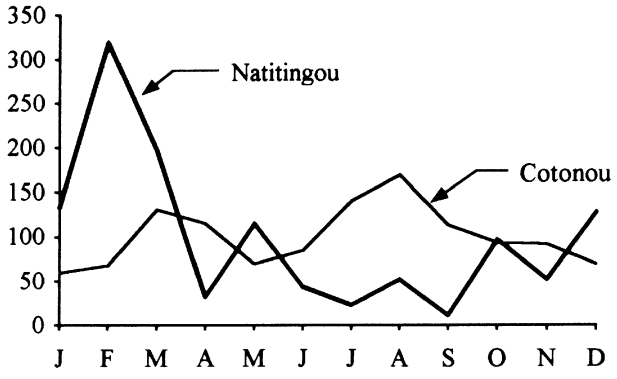

Figure 7. Throat infections diagnosed from 1987 to 1992 on clinical grounds at Natitingou (Atacora: Ch. S. Houssou, personal communication) and at Cotonou (J. Gandonou, personal communication). 100 = yearly mean. The location of both towns is indicated in Figure 4.

Can the meningitis shift be interpreted as climate-derived?

Classically, the south of Benin lies outside the meningitis belt. However, health statistics throw light on recent outbreaks in this part of the country, the last one in 1991 and 1992. A key issue arises from this statement: could there be a link between the marked shift of the belt and a possible climatic change, especially the major drought that began in 1968? An argument in support of this hypothesis is that in the Ivory Coast, Soro et al. [26] have put forward partial "sahelisation" that would have obvious climate (intensification of the dry season and southward advance of the harmattan since 1983–1984), landscape (deforestation, soil overuse) and human environmental effects (increasing number of cattle breeders, migrations). In Burkina Faso, Monnier [15] has also mentioned the reckless deforestation that has caused the harmattan to blow with increased force and at ground level. But the problem is far from solved. According to our results, there was in fact no relation at all in southern Benin between the H index or the absolute humidity and the appearance of meningococcal disease. The fundamental reason for not believing that the Monnier's assumption could be transposed to Benin is that during the recent years, namely 1991 and 1992, the most cases recorded in the coastal area occured between July and September, when absolute humidity was around 21.5 $g.m^{-3}$, a value not below but slightly above the seasonal average, that is 20.7 $g.m^{-3}$ for our whole 28-year period. If there is no doubt that meningitis is shifting southwards and that some climatic changes have occurred, the link between the two phenomena is yet to be proved.

Why does CSM lead to high mortality in seasons and areas of low endemicity?

This question is a difficult one to answer because it requires a knowledge of factors such as the virulence of various strains of meningococcus about which little is known. However, the arguments that immediately come to mind are not convincing. Meningitis is so common and symptoms are so suggestive that it would be surprising that health personnel would not immediately identify cases occurring outside of the usual season. Similarly, one could have supposed that the most serious cases were transferred from the northern areas into the Cotonou University Hospital, so that they inflated the mortality figures of the

coastal areas. Yet, more than 94% of the individuals who died at Cotonou from CSM resided in the Atlantic region, and 88% in the city area. It would appear, then, that a supply of non-native patients did not really alter the results. A further hypothesis implicates the potential for misdiagnosis in the laboratory. Were cases of pneumococcal meningitis, which carries a higher mortality and is not so seasonably dependent, said to be meningococcal, it could explain the apparent rise in mortality in the off season. But one finds it difficult to understand why these wrong diagnosis would be more common in the coastal area, where health facilities are by far the most advanced of the whole Benin. A more convincing, though incomplete, explanation might be that the distribution of cases by age group changes from season to season, if not from area to area. During the epidemics in the north, there was a significant ($p < 0.01$) shift toward older groups: for example, in Atacora from December to March 59% of all CSM cases occured among 10- to 15-year olds, and not more than 3,8% among children aged under one year. In contrast no fewer than 56% of the cases diagnosed between June and September affected the 0–12 months age group. Now various studies all over the world indicated that the disease was usually fatal in the neonatal period [14, 19] and had a case-fatality ratio of 5%–25% in older groups. The pattern turn out to be more ambiguous in the Atlantic region, where children from birth to 12 months of age accounted for 10.8% of all CSM cases and 11.7% of all CSM deaths, without any difference from a season to another. Yet the annual rhythm of the disease in the coastal areas differs from the usual pattern: the main epidemic outbreaks, let's not forget, occur outside of the harmattan season, so that the peak of deaths well coincides in this area with the peak of morbidity, adolescents and young adults being the age group most affected.

In conclusion it can be said that CSM mainly affects the northernmost regions of Benin, from November to March, April or sometimes May. On the average, the acme occurs in February–March. Regression analysis confirmed that a notable part of the space/time variability of the disease is due to the harmattan and a low absolute humidity. Mediation of adverse climatic conditions in the infectious phenomenon is therefore indisputable, through both outbreaks of concurrent respiratory tract infections and sudden drops in mucosal barrier immunity. However, the climate-meningitis relationship proved to be weaker than is sometimes assumed, perhaps because this relationship is partly overshadowed by both anthropic effects (vaccination campaigns) and latency before disease outbreak. Lastly, the rather obscure influence of time of year and weather conditions on CSM re-emphasizes the multifactorial nature of the aetiology of this disease.

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