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Detection of Meningitis Epidemics in Africa: A Population-Based Analysis

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Portions of sub-Saharan Africa are subject to major epidemics of meningococcal meningitis that require early detection and rapid control. We evaluated the usefulness of weekly meningitis rates derived from active surveillance data in Burkina Faso for detecting a meningitis epidemic. By analysing the rates of disease in 40 x 40km^2 areas within a study region of Burkina Faso, we found that a threshold of 15 cases/100 000/week averaged over 2 weeks was 72–93% sensitive and 92–100% specific in detecting epidemics exceeding 100 cases/100 000/year. During epidemic periods, the positive predictive value of this threshold approached 100% for detecting local epidemics. Additionally, meningitis incidence was proportional to village size, with villages greater than 8000 having the highest disease rates during a major group A meningococcal epidemic in 1983–1984. Despite the rudimentary nature of surveillance data available in many developing countries, these data can be used to detect the early emergence of meningitis epidemics. Additional studies are needed to determine the relevance of this approach for detecting epidemics.

Epidemic group A meningococcal meningitis is a major health problem in sub-Saharan Africa. Since the polysaccharide meningococcal vaccine has a limited duration of protection in young children,[1] routine meningococcal vaccination is not effective in preventing epidemics. However, widespread vaccination can control meningococcal disease if implemented early in the course of an epidemic.[2,3] Epidemic control requires that an epidemic be detected early and that vaccination efforts be organized rapidly.[4] Because vaccination campaigns are expensive and divert resources from ongoing health programmes, mounting a campaign in the absence of an epidemic can have an adverse impact on health-care delivery in developing countries. A simple and reliable method of detecting emerging epidemics in developing countries is clearly needed.

Although epidemics occur in 8–14 year cycles in the African 'meningitis belt', predicting the exact timing and location of an epidemic is not currently possible. Several methods have been used to predict the occurrence of epidemics based on disease patterns from previous years.[5,6] These methods may be useful for determining the likelihood of an epidemic during a given year, but further documentation of their predictive value is needed. In the meantime, early detection of an emerging epidemic based on meningitis surveillance data is a reasonable objective.

In most nations of the African meningitis belt, meningitis is one of the diseases included in the emergency notification system and is reported to public health agencies. These surveillance data, however, are often rudimentary and have not been rigorously evaluated as to whether they are a useful measure of disease activity. To detect an emerging epidemic, surveillance must be sensitive to changes in meningococcal meningitis incidence. Since laboratory facilities are uncommon in areas that are severely affected by epidemics, meningitis cases are generally diagnosed on clinical grounds alone without laboratory confirmation of infection. This clinical case definition does not differentiate group A meningococcal meningitis from other causes of sporadic meningitis. While meningococcal

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meningitis epidemics are easy to identify after the fact, it is unclear whether surveillance can reliably detect the early emergence of epidemics.

One method for predicting the emergence of an epidemic is to determine whether or not weekly meningitis incidence thresholds exist that are generally exceeded during epidemics, but not during endemic periods. Developing a weekly or biweekly threshold rate that accurately predicts the ultimate occurrence of an epidemic may allow the early mobilization of resources to combat the outbreak. Currently, no criteria exist for determining when to initiate vaccination campaigns based on ongoing surveillance data. We conducted an active review of population-based meningitis surveillance data from 1979 to 1984 in Burkina Faso to determine the utility of using such data for the early detection of epidemics. Using weekly and biweekly disease rates from surveillance data, we developed threshold meningitis-rate guidelines that were both sensitive and specific for detecting the eventual emergence of an epidemic. In addition, we used these data to investigate the relationship between village size and meningitis incidence in Burkina Faso.

METHODS

Meningitis Surveillance

We reviewed records at local health-care facilities and public health centres for clinically defined meningitis cases in a central region of Burkina Faso from July 1979 to June 1984. Meningitis surveillance records were collected from community health centres, dispensaries and hospitals in the four central provinces (Sissili, Sanguie, Bulkiemde, and Passore) making up the study region (Figure 1). This region had a population of 890000 (mid-1981 estimate) distributed over 656 known villages and towns.

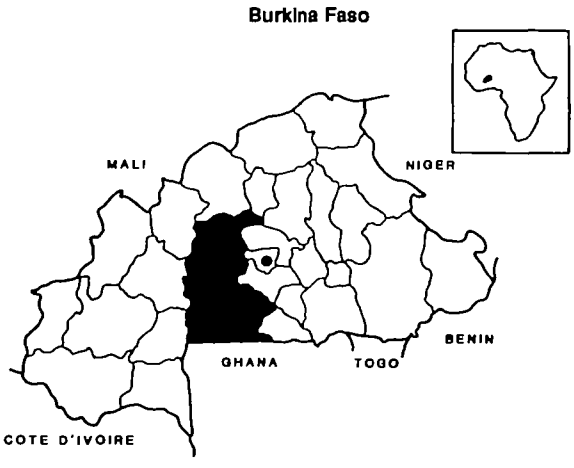

FIGURE 1 *Study area in central Burkina Faso.*

For the purposes of this study, all patients seen at treatment centres with a diagnosis of meningitis were considered to be cases and the date of presentation to the centre was considered to be the date of onset. Because most of the treatment centres did not have laboratory facilities, meningitis cases were primarily diagnosed by the characteristic signs and symptoms of meningitis (fever, headache, nuchal rigidity and petechial rash). This diagnosis was often confirmed by visual examination of cerebrospinal fluid for turbidity. Although meningitis cases included in our study probably represent a mixture of meningococcal, *Haemophilus*, pneumococcal, and nonbacterial meningitides, meningococcal meningitis is the only cause of epidemic meningitis in this region.

Meningitis Incidence Calculations

Since the meningitis season in Burkina Faso extends from December to May, annual incidence rates were calculated from 1 July to the following 30 June in order to include the entire meningitis season within 1 year. Village populations in the study region were determined from a 1975 census and adjusted for a 2.3% annual growth rate (World Bank population estimates, 1983). Incidence calculations were made using the SAS software program (Statistical Analysis Systems, Cary, NC) by dividing the number of cases reported in a given village by the annually-adjusted population of the village. Village and health centre locations were determined by digitizing census maps provided by the Ministry of Public Health (MoPH) using a HP 9845 C desktop computer (Hewlett-Packard Co., Sunnyvale, CA). Because the location, population, and number of meningitis cases for each village was known, disease rates in a given area could be calculated. For example, the entire study region could be divided into thirty-two 40 × 40km^2 grids and the meningitis rate determined for each 1600km^2 area using the overall meningitis rate for the villages within each area grid.

Meningitis Incidence and Village Size

To determine the relationship between village size and annual meningitis rate, all 656 villages in the study region were divided into three broad population categories (<2000, 2000–8000, and >8000 people). Average incidence rates weighted by village population size were determined for each of the three population categories for each year. To assess the influence that access to medical care had on surveillance data, meningitis rates also were examined in a subset of 37 villages with active medical dispensaries or hospitals.

Determination of Weekly Threshold Rates

To detect impending epidemics within the study area, the region was divided into 40 × 40km² areas. Of the 32 areas in the study region, nine with initial populations less than 10000 were excluded from the analysis to avoid fluctuations due to small populations. Weekly meningitis rates were examined throughout the 5-year period in the remaining 23 areas and compared with annual meningitis rates for each of the 40 × 40km² areas.

To perform these calculations, an annual rate in a 40 × 40km² area of 100 cases/100000 population or greater was arbitrarily defined as an epidemic—a rate that is comparable to the epidemic definition used by other authors.[5] Areas with annual rates exceeding 100 cases/100000 in any given year were retrospectively considered to have experienced an epidemic, and areas with annual rates below this cutoff were considered to have been nonepidemic. Weekly rates between 5 and 30 cases/100000 population were then investigated as potential thresholds for predicting the eventual emergence of an epidemic during that same year. Each area exceeding a given threshold weekly rate at any time during the year was examined to see if the same area subsequently exceeded the 'epidemic' rate of 100 cases/100000/year. By determining whether or not each grid exceeded both the given weekly threshold rate and the epidemic annual rate, the sensitivity, the specificity, and the positive predictive value of a weekly threshold rate to predict epidemic annual rates could be calculated. Threshold rates averaged over 2 consecutive weeks were also calculated by dividing the biweekly rate by 2 to determine the average weekly meningitis rate. This analysis was performed in an attempt to improve the accuracy of the detection method, since it requires that a given threshold rate be maintained on average for at least 2 weeks in order to surpass the threshold.

Because a major group A meningococcal meningitis epidemic occurred in Burkina Faso during 1984, the 5-year observation period was divided into separate endemic and epidemic periods for the purposes of this study. The endemic period consisted of the 4 years from 1 July 1979 to 30 June 1983, and the epidemic period was the single year from 1 July 1983 to 30 June 1984. The endemic period had 92 grid-year observations (23 grids over 4 years) and the epidemic period had 23 grid-year observations (23 grids over 1 year).

Vaccination

To determine the effect of meningococcal vaccination on meningitis rates, vaccine distribution records were obtained from the MoPH. No other sources are known to have distributed meningococcal vaccine during the study period, although limited amounts of vaccine were also commercially available. The vaccination rate for 40 × 40km² areas was determined because it was not possible to determine the vaccination rate within individual villages. Because meningococcal vaccine efficacy remains high for several years in children over 4 years old and adults, cumulative vaccination rates were compared with meningitis rates for each year for each 40 × 40km² area. It was assumed that all doses were administered within the area containing the vaccine distribution centre and that no one was vaccinated twice. These assumptions tend to overestimate the amount of vaccine administered within each area. The influence of pneumococcal and *Haemophilus* vaccination was considered to be negligible since these vaccines have not been widely distributed in Burkina Faso.

RESULTS

From July 1979 to June 1984, 5591 cases of meningitis diagnosed on clinical grounds were recorded in health centres throughout the study region. Of these cases, the home village of 5478 (98%) of the patients was recorded. The meningitis season in a given year typically lasted from 17 to 33 weeks during the study interval (Figure 2). During the 4-year period from July 1979 through June 1983, annual endemic meningitis rates ranged from a low of 30.2 cases/100000 in 1979–1980 to a high of 94 cases/100000 in 1980–1981.

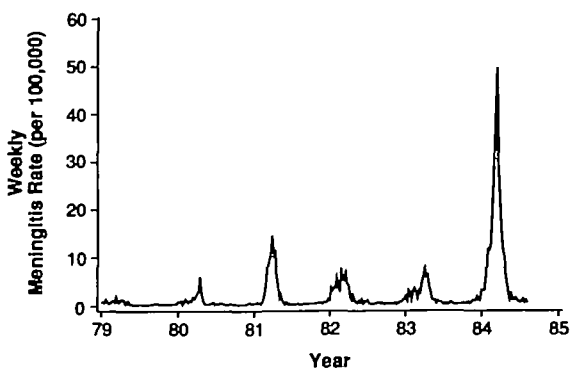

FIGURE 2 *Weekly meningitis rates in the study area.*

In 1983–1984, a major group A meningococcal meningitis epidemic with 2965 cases occurred in the study region, resulting in an annual incidence rate of 318 cases/100000. During this epidemic, high rates were not uniformly distributed throughout the study region. When the study region was divided into thirty two 40 × 40km² areas, 17 (53%) of these areas had incidence rates greater than 100 cases/100000/year, and 12

(38%) of these areas had rates exceeding 200 cases/100000/year. In contrast, 15 (47%) areas, primarily located in the sparsely populated province of Sissili, did not exceed the epidemic threshold of 100 cases/100000/year during the 1983–1984 epidemic. During the endemic period 1979–1983, local outbreaks often exceeded the 100 cases/100000/year threshold, but were not widespread enough to increase the level of disease throughout the study region to epidemic proportions.

Village Size and Meningitis Rate

The annual meningitis rate was strongly associated with village population size during both endemic and epidemic years. The meningitis rate for villages with populations over 8000 was two- to four-fold higher than in villages with populations under 2000 during the endemic years 1979–1983 (Figure 3a). This trend was marked during the 1983–1984 epidemic; the annual incidence rate in villages over 8000 in population was 888 cases/100000 compared with 219 cases/100000 for villages with under 2000 population. During this year, the largest two cities in the study area had populations greater than 15000 and a combined annual meningitis incidence rate of 1096 cases/100000, or approximately 1.1% of the citywide population for 1983–1984. Although only 13.9% of the population in the four provinces lived in villages with 8000 population or greater, 38.7% of 1983–1984 meningitis cases were reported from these villages.

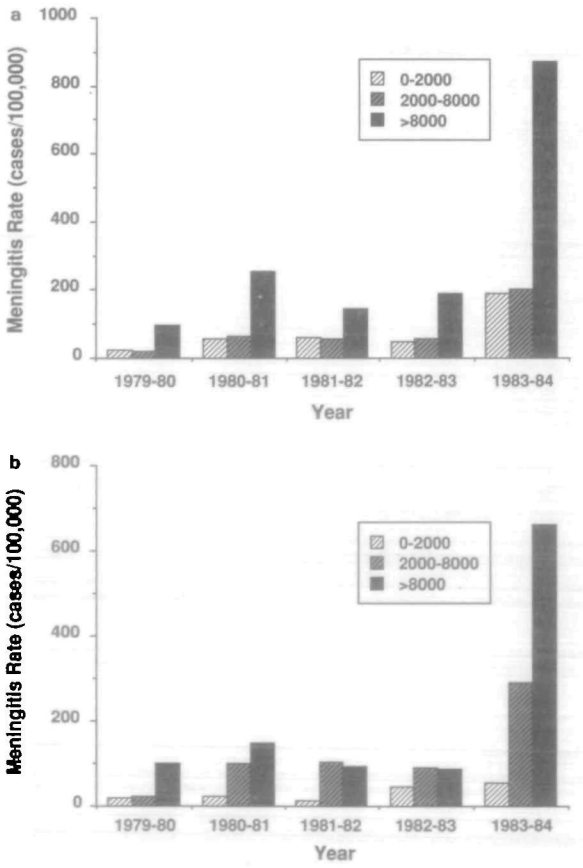

FIGURE 3a *Annual meningitis rates in villages with <2000 population, 2000–8000 population and >8000 population.* b. *Annual meningitis rates in the 37 villages in the region with health centres.*

The relationship between meningitis rate and village size could result from patients in larger villages having better access to health care and being more likely to be seen at a health facility. However, an examination of only those villages with health facilities suggests that this was not due to a reporting artifact. When only the 37 villages with health-care facilities were examined, meningitis rates remained highest in large villages throughout the study period (Figure 3b). During the epidemic year 1983–1984, annual meningitis rates were 662 cases/100000 in villages over 8000 inhabitants with health facilities compared to only 55 cases/100000 in villages under 2000 inhabitants with health facilities.

Use of Weekly Rates to Detect Epidemics

To examine the utility of predicting epidemics from weekly threshold rates, the study region was divided into thirty-two 40 × 40km² areas. Nine areas with populations less than 10000 were excluded from the analysis (to avoid fluctuations due to small numbers), leaving twenty-three 40 × 40km² areas for analysis. Threshold rates for the endemic years 1979–1983 and the epidemic year 1983–1984 were examined separately.

Overall, the sensitivity of any threshold rate for detecting local epidemics was high for epidemic years (Figure 4a), but declined for endemic years as the stringency of the threshold increased. In contrast, higher threshold values increased the specificity of epidemic detection. This trend is seen in Figure 4b for both epidemic and endemic periods; specificity progressively increases as the threshold increases from 5 to 30 cases/100000/week.

The positive predictive value (i.e. the per cent of areas exceeding the threshold rate that actually had an epidemic annual rate) also increased with higher threshold values (Figure 4c). As anticipated, the positive predictive value was also higher at any given threshold value in 1983–1984 than in 1979–1983 because the high prevalence of local outbreaks during the major meningococcal epidemic in 1983–1984.

Overall, a threshold rate of 15 cases/100000/week was very sensitive, but only moderately specific, in

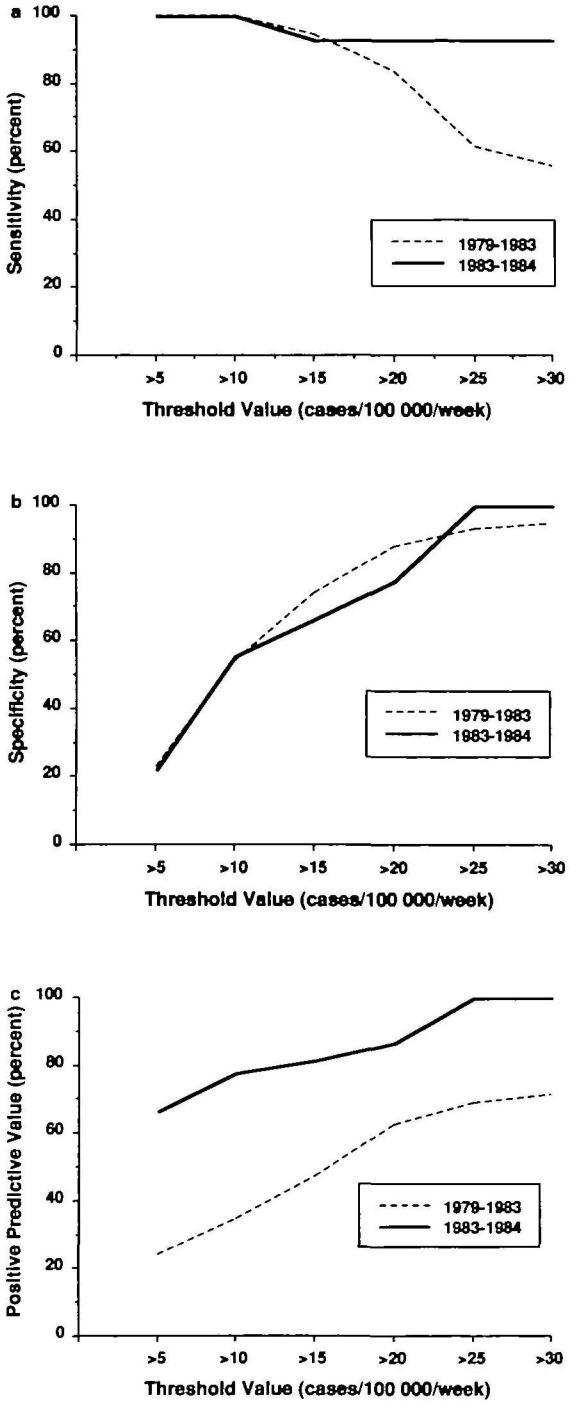

FIGURE 4 *Use of weekly meningitis rates as thresholds to predict annual meningitis rates greater than 100 cases/100 000/year in twenty-three 40 × 40km² areas.* a. *Sensitivity of thresholds during 1979–1983 and 1983–1984.* b. *Specificity of thresholds during 1979–1983 and 1983–1984.* c. *Positive predictive value of thresholds during 1979–1983 and 1983–1984.*

predicting annual disease rates greater than 100 cases/100 000. To improve the specificity of this threshold, this meningitis rate was averaged over 2 weeks (equivalent to 30 cases/100 000 per 2-week period) and compared with the single-week threshold (Figure 5). As anticipated, using the weekly rate averaged over 2 weeks improved specificity but moderately reduced the sensitivity of detecting an epidemic compared with the 1-week threshold.

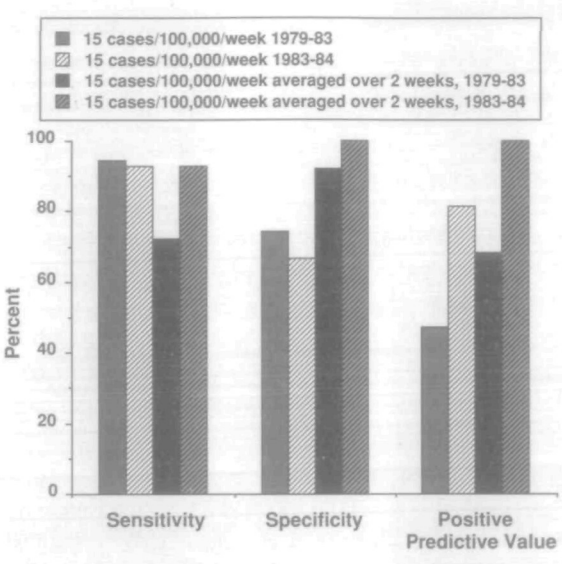

FIGURE 5 *A comparison of the sensitivity, specificity and positive predictive value of the 15 case/100 000/week threshold and the 15 case/100 000/week threshold averaged over 2 consecutive weeks.*

In order to be useful, the threshold for declaring an epidemic should be crossed early enough during an epidemic to allow rapid control. To determine the timeliness of the threshold of 15 cases/100 000/week averaged over 2 weeks, the potential number of cases prevented during the 1983–1984 season was examined (Table 1). If universal vaccination had been completed within 2 weeks after exceeding the threshold rate (assuming 100% vaccine efficacy and an additional 2 weeks to mount an effective immune response), 1727 (58.2%) cases in 1983–1984 could have been prevented by vaccinating in areas that had crossed the threshold value. Using this threshold as a standard for declaring an epidemic, a total of 707 000 people would have been vaccinated in 1983–1984 and 24.4 cases could have been prevented per 10 000 doses of vaccine administered. In comparison, use of the more sensitive threshold (5 cases/100 000/week averaged over 2 weeks) resulted in more potential cases being prevented

TABLE 1 *Meningitis cases potentially preventable if vaccinations had been instituted after surpassing various biweekly meningitis incidence thresholds, 1983–1984*[a]

Meningitis threshold[b]	Population vaccinated (%)	No. of cases potentially prevented (%)	No. of cases potentially prevented per 10000 doses of vaccine
5 cases/100000	782067 (87.6)	2246 (75.8)	28.72
10 cases/100000	747353 (83.7)	1968 (66.4)	26.33
15 cases/100000	706927 (79.2)	1727 (58.2)	24.43
20 cases/100000	706927 (79.2)	1446 (48.8)	20.45
25 cases/100000	706927 (79.2)	1071 (36.1)	15.15

[a] Assuming a 2-week delay after threshold is crossed for implementation of a vaccination campaign and development of immunity.
[b] Cases per 100000 population averaged over 2 weeks.

(75.8%), more cases prevented per 10000 doses of vaccine administered (28.7 cases prevented per 10000 doses), and more total vaccine distributed (782000 doses).

Influence of Vaccination

To evaluate the possibility that prior vaccination might have influenced the incidence of meningitis during the study period, vaccination distribution records were examined for the study area. Table 2 shows the annual amount of vaccine distributed in the study area, the estimated per cent of the population vaccinated, the cumulative vaccination rate, and the correlation between disease rate and cumulative vaccination rate. Vaccination rates were low throughout the study region, with the highest rate of vaccination attained during a major vaccination campaign initiated in 1981–1982. Generally, vaccination rates were not correlated with meningitis rates, suggesting that the low levels of vaccination achieved in the study region did not influence overall meningitis rates. In 1980–1981 and 1982–1983, meningitis and vaccination rates were directly correlated, probably as the result of vaccine administration in areas with high meningitis rates.

TABLE 2 *Correlation between meningitis rate and vaccination rate in twenty-three 40 x 40km² areas with populations exceeding 10000*

Year	Population vaccinated (%)	Cumulative per cent vaccinated[a]	Spearman correlation coefficient, R[b]	*P* value
1979–1980	1.6	1.6	−0.10	0.64
1980–1981	5.9	7.5	0.63	0.001
1981–1982	16.7	24.2	−0.03	0.89
1982–1983	1.4	25.6	0.55	0.01
1983–1984	4.9	30.5	0.12	0.58

[a] Cumulative per cent of study population vaccinated since July 1979.
[b] Correlation between cumulative vaccination rate in each area and annual meningitis rate in each area for the twenty-three 40 × 40km² areas.

DISCUSSION

Despite the difficulties of conducting meningococcal meningitis surveillance in sub-Saharan Africa, our study indicates that surveillance can provide useful information for controlling outbreaks of disease. Despite the potential for misclassification of meningococcal meningitis when a clinical case definition is used, surveillance data can detect the emergence of an epidemic. The reason for this is probably that *Neisseria meningitidis* is the most frequent cause of meningitis during the dry season in the region[7] and the only cause of epidemic meningitis. Therefore, it is likely that increases in meningitis incidence during our study were primarily due to increased meningococcal disease activity.

In our study of meningitis patterns in Burkina Faso, we found a clear correlation between meningitis rates and village size during both endemic and epidemic periods. We were unable to strictly control for urban versus rural access to health care, but an examination of only those villages with health centres suggests that this relationship is not an artifact of underreporting in rural areas. Also, because meningitis surveillance records were obtained directly from health-care and public health centres, failure to report cases to the MoPH was not a source of bias in our data. Differences in meningococcal vaccination coverage for urban and rural areas are unlikely to account for our findings since urban dwellers tend to have higher immunization rates than rural inhabitants in Burkina Faso (personal communication, David Sokal, Family Health International). In our study, cumulative vaccination rates over the 5-year study period were not related to the meningitis rate, presumably due to the low overall level of achieved vaccination coverage.

In contrast to our results, others have not found a relationship between population size and meningitis rate in sub-Saharan Africa. Lepeyssonie[8] found no correlation between provincial population density and meningitis rate in Burkina Faso during meningitis epidemics in the 1950s. However, meningitis rates at the provincial level may not accurately reflect the intensity of disease at the village level since meningitis rates are

not uniform over large areas. In a study conducted during a group A meningococcal epidemic in The Gambia, Greenwood *et al.* did not find a relationship between village size and meningitis rate.[9] Our study differed from the Gambian study in that we examined a number of villages and cities with populations over 2000.

It is not clear that the higher rate of meningitis in larger villages is due to increased crowding. Crowding was not directly assessed in our study, and other socioeconomic and demographic factors, such as transportation routes, nutrition, and sanitation, could play a role in the higher rates of disease in larger villages. In addition to regional population density, household crowding has also been studied as a risk factor for group A meningococcal meningitis; three studies have shown an association with disease,[1,10,11] but another study did not find this association.[9] Household crowding may increase the risk of disease by increasing the transmission of meningococci or coincident upper respiratory infections.[11]

Our findings have implications for vaccination strategies. Although universal vaccination of the population is the preferred strategy during an outbreak,[4] larger villages should be targeted for vaccination during an epidemic if there is limited availability of vaccine. Furthermore, based on our finding that meningitis activity was not uniformly distributed throughout the study region, it is reasonable to concentrate vaccination efforts in those areas showing evidence of epidemic activity.[12]

We have also attempted to predict the occurrence of epidemics using weekly meningitis rates. Given the high sensitivity and specificity of the threshold of 15 cases/100000 population/week averaged over 2 weeks, this rate appears to be an appropriate threshold for initiating investigation and control activities in Burkina Faso. By requiring that this level be maintained over a 2-week period, the chances of unnecessarily instituting vaccination during nonepidemic periods is decreased.

Once epidemic disease has been detected, however, there is no advantage in using a stringent threshold cutoff for instituting vaccination in adjacent areas. During an epidemic period, the prevalence of epidemic disease in a region is high, and the predictive value of crossing a lower threshold is considerably improved (Figure 4c). Therefore, once epidemic disease is detected in a given locale, it is reasonable to institute vaccination at low threshold values (such as 5 cases/100000 population/week) in nearby areas to prevent unnecessary delay. If this policy is implemented in areas adjacent to epidemic foci, a greater number of cases may be prevented.

In our study, we attempted to avoid wide fluctuations in weekly incidence rates by eliminating from our analysis 40 × 40km^2 areas with under 10000 people. Similarly, since a few cases in a small population may result in an inaccurate estimate of disease activity, it is reasonable to apply these threshold rates to populations of at least 30000 to 50000 people. To estimate the potential number of cases prevented by vaccination after a particular threshold was crossed, we assumed a vaccine efficacy and coverage of 100% for purposes of comparison. During an actual epidemic, however, lower degrees of coverage and efficacy will apply.

Our study was a retrospective review of dispensary records, and therefore, our case detection is likely to be more complete than routine meningitis surveillance for most countries of the meningitis belt. In areas of sub-Saharan Africa where little is known about the sensitivity of meningitis surveillance, these thresholds can only be considered as an initial guideline for epidemic management. Additional studies are needed to determine the relevance of our findings to other settings, where modification of these thresholds may be necessary. Differences in both timeliness and sensitivity of case detection could also affect the reported patterns of disease in other countries. If reporting from health stations is incomplete or delayed, the actual meningitis activity may be much higher at any given time than indicated by surveillance figures. Under these conditions, the threshold of 15 reported cases/100000/week averaged over 2 weeks may be too high to detect an epidemic. Finally, since threshold rates were determined in 40 × 40km^2 areas with an average population of approximately 35000, additional studies are needed to determine whether or not these thresholds are applicable to larger populations, such as entire countries.

Because of a lack of laboratory facilities in the study region, our thresholds were based on reports of purulent meningitis, as is likely to be the case for most developing countries where these guidelines would be appropriate. Although this will result in some misclassification of cases, it is unlikely to cause a false-positive declaration of an epidemic since only *Neisseria meningitidis* causes epidemic meningitis. To ensure that control measures are appropriate, however, it is recommended that bacteriological confirmation be obtained during the initial rapid assessment of any suspected meningitis outbreak.[4]

Our study proposes quantitative threshold meningitis rates to facilitate epidemic disease management. The demonstration that thresholds can be used to detect an impending epidemic should encourage the collection and analysis of meningitis surveillance data

in other sub-Saharan countries. The importance of timely and accurate reporting of meningitis cases in sub-Saharan countries should be emphasized since these factors are critical for an early-warning system based on threshold meningitis rates. While political and operational considerations will also affect meningitis control efforts, surveillance data have potential to guide effective control strategies.

Epidemic meningococcal disease can be effectively controlled if widespread vaccination is implemented early in the course of an epidemic.[2,3] Although the current group A polysaccharide vaccine is highly efficacious, epidemics in the meningitis belt cannot be prevented altogether with this vaccine. Routine childhood administration is likely to be of limited benefit because of the short duration of vaccine-induced protection in children under 4 years old.[1] Although the duration of vaccine protection is longer in older children and adults, routine vaccination of those aged 4 years and older during nonepidemic periods is impractical for many countries and does not provide coverage for young children—a large portion of the total population at risk. In the long term, it is hoped that a new group A meningococcal vaccine will become available that can provide prolonged group A meningococcal immunity when administered to infants during routine immunization programmes. Until such a vaccine is developed, strategies for the early detection and rapid response to epidemic meningococcal disease will remain important for the control of this disease.

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