# Evaluation of the meningitis epidemics risk model in Africa

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(Accepted 2 December 2005, first published online 14 February 2006)

#### SUMMARY

Meningitis epidemics have a strong environmental component in Africa with the most severe epidemics occurring in the Sahelian region known as the Meningitis Belt. The objective of this study is to evaluate an ecological model based on absolute humidity and land cover type to predict the location of these epidemics. The risk model is evaluated prospectively based on epidemics occurring in Africa from January 2000 to April 2004. Seventy-one epidemics occurred during this time period affecting 22% of continental African districts. The model predicted their location with a sensitivity of 88%. The evaluation also suggests that epidemics may be extending south of the Sahel, which is consistent with environmental changes in the region. This model could be used to select priority areas for the introduction of the newly developed conjugate meningococcal vaccines. Further studies are needed to enhance our understanding of the complex relationship between meningitis epidemics and the environment.

#### **INTRODUCTION**

The Meningitis Belt is a region comprising mostly of areas in the Sahel in Sub-Saharan Africa [1, 2]. Countries within this belt experience the highest endemicity and epidemic frequency of meningococcal meningitis in Africa, although other areas in the Rift Valley, the Great Lakes and southern Africa are also affected [3]. These epidemic-prone areas have common ecological characteristics and a model based on the absolute humidity profile and land cover type within a district [3], was able to predict the location of epidemics occurring before 2000 with reasonable sensitivity (83%) and specificity (67%). Here, we

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report a prospective evaluation of this model based on meningitis epidemics occurring since January 2000.

#### METHODS

Data on meningitis epidemics occurring in Africa from January 2000 to April 2004 reported by the WHO's surveillance websites [4], the WHO Regional Office for Africa [5], the OFDA/CRED International Disasters Data Base [6], ProMED-mail [7] and in PubMed [8] were included. Papers included in scientific databases were searched using the keywords 'meningitis/meningococcal AND epidemic/outbreak AND Africa'. Epidemic events were accepted when an author reported an 'outbreak' or an 'epidemic' regardless of whether they stated if the WHO epidemic/alert threshold had been reached [9]. Unlike

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Country	Epidemic years	Predominant serogroup	Cases
Angola	2000, 2001, 2002, 2003	A, A, NA, NA	530, 332, 356, 171
Benin	2000, 2001, 2002, 2003	NA, A, NA, A&W135	1328, 8998, 502, 357
Burkina Faso	2001, 2002, 2003, 2004	A&W135, W135, A&W135, A&W135	13 039, 12 587, 7720, 2783
Burundi	2002, 2003	A, A	934, 40
CAR†	2000, 2001, 2003, 2004	NA, A, NA, A	2629, 2052, 13, 43
Chad	2000, 2001, 2002, 2003, 2004	NA, A, A, NA, A	7636, 5780, 686, 468, 19
Cameroon	2000, 2001, 2004	NA, A, NA	334, 2036, NA
Côte d'Ivoire	2002, 2004	NA, A	244, 100
DRC‡	2001, 2002	A, A	378, 1142
Ethiopia	2000, 2000-01, 2001-02, 2003, 2003-04	A&C, A, A, A&C, A	1004, 6964, 4191, 250, 2400
Gambia	2001, 2002	NA, A	137, 50
Ghana	2001, 2002, 2003, 2004	NA, A, A, NA	1278, 1407, 1393, 306
Guinea	2002	A&C	123
Mali	2002, 2003	A, A&W135	382, 840
Mauritania	2002	NA	26
Namibia	2000, 2001	NA, A	92, 24
Niger	2000, 2001, 2002, 2003	NA, A&C&W135, A, A	13 873, 7906, 3518, 7953
Nigeria	2001, 2002, 2003, 2004	NA, NA, A&W135, NA	340, 100, 3569, 500
Rwanda	2000, 2002	A, C	487, 683
Sierra Leone	2001–2002	NA	50
Somalia	2001-2002	Α	237
Sudan	2000, 2001, 2002, 2003, 2004	NA, A, A, A, A	2549, 3155, 1288, 153, 20
Tanzania	2002	Α	269
Togo	2001, 2002, 2003	NA, A, A	1195, 589, 313
Uganda	2002, 2002–2003, 2004	NA, NA, NA	10, 290, 40

Table 1. Meningitis epidemics in Africa (January 2000 to April 2004)\*

\* A more detailed version of this table can be viewed online on the Journal's website.

† Central African Republic.

‡ Democratic Republic of Congo.

NA, Not available.

the previous model, areas reported as reaching the alert threshold without evidence of a subsequent outbreak were also included. This modification would have increased the sensitivity of the method to identify epidemics and would test the model with a more rigorous set of criteria. Data extracted included year, districts/regions affected, number of cases and predominant serogroups. Where multiple reports of epidemics with conflicting data occurred, the highest number of cases was used and all reported areas were included.

The locations of the epidemics were mapped using ArcView 3.2a (ESRI, Redlands, CA, USA) with the same administrative boundaries used by Molesworth *et al.* [3, 10]. Epidemics reported using modified district boundaries were mapped as if they had affected the original district areas to maintain comparability. Towns, villages or new districts were located from the *Travel Journal*'s location lists [11], the World Gazetteer [12] or maps provided in the reports.

The model output of Molesworth *et al.* [3, 10] consisted of the probability of a district having experienced an epidemic and several risk cut-off levels can be used for prediction. We selected the risk cut-off of  $\ge 0.4$  for this evaluation, which was found to optimize the model's performance retrospectively [3]. In addition, given the recent displacement of epidemics to more southern locations (Molesworth *et al.*, unpublished observations), we tested the hypothesis that extending the areas at risk by a 100-mile (176 km) buffer south of the current model's predicted risk areas would increase its sensitivity.

The locations of the districts experiencing epidemics from January 2000 to April 2004 were compared with the distribution in the previous 150 years [10] and with the areas identified previously as being at risk of





**Fig. 1.** Meningitis epidemics occurring between 1841 and April 2004. Shaded areas depict epidemics reported before 2000. Dotted areas depict districts with epidemic reports from 2000.

epidemics by the model [3]. The model's performance was then assessed by calculating its sensitivity (i.e. the ability to identify areas which had experienced epidemics) and specificity (i.e. the ability to identify areas not affected by epidemics).

#### RESULTS

A total of 71 meningitis epidemics were reported in 25 countries from January 2000 to April 2004 affecting 721 (22%) of the 3281 continental African districts (Table 1). Serogroup A meningococci were predominant in most epidemics (>70% of epidemics with serogroup reported), although a substantial number of group W135 and group C cases were reported in eight and five of the outbreaks respectively. The majority of epidemics (55, 77%) affected districts in and around the Sahel region and 42 (59%) occurred in countries within the classical Meningitis Belt where the geometric mean number of cases was 1143 (range 19-13873). This was significantly higher than the number of cases in the 29 epidemics located in countries outside this belt (geometrical mean 195, range 10-2629, P<0.001). An updated map of all the districts affected by meningitis epidemics from 1841 until April 2004 is shown in Figure 1. Whilst epidemics have continued to occur in the districts

Fig. 2. Districts predicted to have meningitis epidemics (risk  $\geq 0.4$  plus a 100-mile buffer south of the Sahel, see text for explanation). Dotted areas denote distribution of meningitis epidemics (January 2000 to April 2004).

previously affected, some epidemics occurred in places not reported to have experienced epidemics before 2000. Most of these 'new' epidemic districts, however, were located in areas geographically contiguous to districts previously affected by epidemics.

Figure 2 shows the areas predicted by ref. [3] as having a risk of experiencing epidemics  $\ge 0.4$  and compares the model with the areas affected by epidemics since 2000. The model had a sensitivity of 81% and specificity of 56%, which is in agreement with its retrospective validation (Table 2). When the 100-mile buffer was added to the Sahelian areas, the sensitivity increased significantly to 88% (P < 0.05) with a specificity of 45%.

# DISCUSSION

The pattern of the most severe meningococcal epidemics in terms of frequency and number of cases does not seem to have altered, with most of the outbreaks still occurring in the WHO's extended Meningitis Belt. However, our findings confirm that the areas affected by smaller epidemics are still expanding to new districts, with the southwards extension in the Sahelian region (in Côte d'Ivoire, Togo, the Central African Republic, and Cameroon) particularly apparent. This is consistent

	Observed districts (2000–2004)			
Epidemic experience	Epidemic	Not epidemic	Total	
Model prediction (risk $\ge 0.4$ plus buffer zone)				
Epidemic	632	1405	2037	
Not epidemic	89	1150	1239	
Total	721	2555	3276	
	Sensitivity (95% CI)	Specificity (95% CI)		
Model validation				
Retrospective*	84 % (80-87)	65% (62–69)		
Prospective				
$Risk \ge 0.4$	81 % (78–84)	56% (54–58)		
Risk $\ge 0.4$ plus buffer zone	88 % (85–90)	45% (43-47)		

 Table 2. Performance of the model for predicting epidemics in 2000–2004

CI, Confidence interval.

\* From the model evaluation of Molesworth et al. [3].

with environmental changes in this area such as deforestation [13] and desertification [14] that may have caused the Sahelian areas to expand southwards. This combination of changes in land use and climatic factors may have led to an increase in dust and a reduction in humidity, favouring conditions for the epidemics [15], although the relationships between land degradation, dust, humidity and meningitis outbreaks are complex and still largely unclear.

The interpretation of these findings, however, needs to consider the recent developments in information technology in this field. New online surveillance resources are now becoming more widely available and these provide more detailed information, with most epidemics nowadays being reported to the district level thus being more likely to detect and report smaller events. This is in contrast to academic reports, which have restricted space for publication and are nowadays more concerned with control strategies and novel events such as the W135 epidemics in Burkina Faso. In addition, the prospect of new meningococcal conjugate vaccines for the area has stimulated enhanced surveillance in recent years. The quantitative and qualitative improvement in the information available may have resulted in better reporting of smaller epidemics or those occurring in remote or unusual places and hence the apparent spread of the at-risk area may reflect only the availability of improved datasets.

The prospective evaluation of the model corroborates its retrospective evaluation. Even though the definition of epidemics was modified to include smaller events, the model still predicted the areas at risk with a sensitivity of 81% and the buffer zone increased its sensitivity to 88%. This buffer zone includes densely populated areas and could optimize the sensitivity of the model with a relatively small loss of specificity. Molesworth *et al.* [3] suggested that the model could be improved further by considering nonenvironmental factors such as population movement and epidemic experience of a district in recent years. Our findings support the further development of the model and suggest it could be used to identify priority areas for vaccination when the new meningococcal group A conjugate vaccines become available.

In conclusion, the areas at risk of meningitis epidemics may be expanding, although major epidemics still seem to be confined to the Meningitis Belt. Continued monitoring of the spatial distribution of epidemics in Africa is required to confirm the extension of the areas at risk and to further our understanding of factors triggering these epidemics. The model of Molesworth *et al.* [3] can predict the location of recent epidemics using a cut-off value of  $\ge 0.4$ and the sensitivity of the model increases to 88% by extending the predicted at-risk area south from the Sahel. The model could be used as a tool to guide the selection of priority areas to receive the new group A meningococcal conjugate vaccines until these become widely available.

#### ACKNOWLEDGEMENTS

Financial support was gratefully received from the Meningitis Research Foundation, UK.

None.

## NOTE

Supplementary information accompanies this paper on the Journal's website (http://journals.cambridge.org).

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