Buruli Ulcer Prevalence and Altitude, Benin

To the Editor: Buruli ulcer (BU), caused by Mycobacterium ulcerans, is one of 13 recently classified neglected tropical diseases (1). Little is known about factors influencing its focal distribution. In Benin, altitude may play a role in such distribution of BU.

Incidence, prevalence, and other health-related data are usually reported at national or district levels. These data convey the importance of the disease but do not show the wide variations existing at the village level. Data from the surveillance system (2) and surveys (3–6) in Benin have shown that BU-endemic areas are confined to the southern regions. Substantial variability in endemicity levels have been detected from 1 department to another, at the district and village levels, and from year to year (2–5).

However, some districts (Lalo in the Mono-Couffo Department, Ouinhi in the Zou Department; Zé in the Atlantique Department; and Adjohoun, Bonou, and Dangbo in the Oueme Department) remain the most persistently BU-endemic from year to year. In addition, these BU-endemic districts are all located at the same latitude. A map of these districts can be superimposed on the Lama depression (a median of all located at the same latitude. A map of these districts can be superimposed on the Lama depression (a median of all located 100 meters (10.2/10,000 inhabitants) and that of villages with elevations >100 meters (5.4/10,000 inhabitants) (p = 0.0003; Kruskal-Wallis test).

In addition, we performed a simple linear regression, including all villages (model A) and only BU-endemic villages (prevalence ≠ 0) (model B). Model A showed that at 0 altitude, the expected prevalence of BU was 26.7/10,000 inhabitants. This prevalence decreased by 0.1/10,000 inhabitants for each meter of increase in altitude (correlation coefficient 0.20; coefficient of determination 4%). Model B demonstrated that at 0 altitude, the expected prevalence was 89.6/10,000 inhabitants. This prevalence decreased by 0.7/10,000 inhabitants for each meter of increase in altitude (correlation coefficient 0.50; coefficient of determination 25%). Therefore, we conclude that a low but significant linear relationship exists between altitude and BU prevalence in disease-endemic villages. Thus, altitude may be 1 factor in determining variations in prevalence (4% for all villages and 25% for BU-endemic villages).

The focal distribution of BU was discussed in 1974 by Meyers et al. in Zaire (8). In the Bas-Congo Province, although the concentration of BU in Songololo was high, the nearby broad Bangu plateau, ≈300 meters higher than Songololo, was devoid of BU (D.M. Phanzu, unpub. data). Soil and geologic features (e.g., chemical composition of substrata; vegetation, fauna, and pH of swamps) were raised as environmental factors that might explain this focal distribution (8,9). The focal distribution of BU was also described by Johnson et al., who found an inverse relationship between the prevalence of the disease in Lalo District villages and distance from the Couffo River (4).

Few studies have investigated environmental risk factors (other than water-related) possibly related to the prevalence of BU. In 2008, Wagner et al. suggested that villages with higher prevalence rates were located in areas of low elevation. They associated the high prevalence of BU with farming activities that occurred primarily at low elevations (10). Our results are similar, but we have provided additional quantification of the relationship between prevalence and altitude.

One reasonable explanation for the relationship between altitude and BU prevalence is that because lowlands tend to be wetter than higher grounds, they provide more favorable conditions for the proliferation and spread of the etiologic agent. Furthermore, persons are more apt to frequent these wetter lowlands to plant and tend their crops, thus becoming vulnerable to infectious agents in the area.

An extension of this study to all BU-endemic villages is needed to further refine our results. The endemicity of BU is multifactorial; however, our results suggest that altitude should be included in future analytical models of environmental risk factors for this disease.

Acknowledgments

We are grateful to all the participants in this study, the staff of Centre de Dépistage et de Traitement de l’Ulcère de Buruli d’Allada, and partners who support the Programme National de Lutte contre
3. Sopoh GE, Johnson RC, Chauty A, Dos-
4. Johnson RC, Makoutode M, Sopoh GE,
sou AD, Aguiar J, Salmon O, et al. Bu-
5. Sopoh GE, Adinsi V, Johnson RC, Barogui 
6. Debacker M, Aguiar J, Steunou C, Zinsou 
7. Kolawolé SA, Boko M. Le Benin [French] 
8. Meyers WM, Connor DH, McCullough B, 
9. Portaels F. Epidemiology of mycobacterial 
10. Wagner T, Benbow EM, Brenden OT, Qi J, Johnson RC. Buruli ulcer disease prevalence 

**Vibrio cholerae O1 in 2 Coastal Villages, Papua New Guinea**

To the Editor: Cholera outbreak reports are of international public health interest, especially in areas that were previously cholera free (1). Although many recent cholera outbreaks have originated in coastal areas (2), identifying the source of cholera introduction has been challenging (1). The detection of *Vibrio cholerae* in coastal, brackish and riverine waters in cholera-endemic and cholera-free areas supports the view that autochthonous *V. cholerae* is involved in the introduction of cholera (3,4). To our knowledge, cholera has not been reported in Papua New Guinea, despite social and environmental conditions likely to facilitate transmission and the nation’s close proximity to cholera-endemic countries (5,6).

On August 6, 2009, a physician who visited the coastal village of Lambutina reported an outbreak of acute watery diarrhea that was associated with the death of his father and 4 other persons from this and a neighboring village. The outbreak began in the village of Nambariwa and spread to neighboring Lambutina, Morobe Province. From August 13, multidisciplinary teams worked with the community to reduce the number of deaths through early identification and treatment of case-patients. The teams also worked to limit transmission through improvements to the water and sanitation infrastructure and by encouraging better hygiene practices among the villagers. A suspected case of cholera was defined as acute watery diarrhea or vomiting in a resident of Lambutina or Nambariwa villages since July 22, 2009. In the 2 villages, 77 cases were identified; attack rates were 14% in Lambutina (48/343) and 5.5% in Nambariwa (29/532). The overall case-fatality ratio was 6.5% (5/77); 2 patients died after they were discharged from the referral hospital.

A retrospective frequency-matched case-control study was conducted in Lambutina to identify the risk factors associated with suspected cholera. Neighborhood controls (± 5 years of age) were selected from unaffected households. Univariate and multivariate analyses were conducted with STATA version 10 (StataCorp., College Station, TX, USA).

Of the 48 case-patients in Lambutina, 43 participated in the study with 43 age-matched controls. In addition