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Geographic Expansion of Buruli Ulcer Disease, Cameroon

To the Editor: Buruli ulcer disease (BU) is a necrotizing skin disease caused by Mycobacterium ulcerans that affects mostly children in humid, tropical areas (1). The exact mode of M. ulcerans transmission remains unclear, although the role of water bugs has been supported by various observations and experimental studies (2,3).We report the identification of a new BU-endemic area in Cameroon, the Bankim district, and specify ecologic and clinical characteristics of M. ulcerans infection in this area. These characteristics hint at the possible role of environmental changes (building of a dam several years ago) in the expansion of BU in this area.

Since 1969, only 1 BU-endemic area in Cameroon has been described: the Nyong River basin, where equatorial forest predominates (4). In 2004, clinically suspected cases of BU in the district of Bankim have been reported (5). This region differs from the first BU-endemic area by geography and climate. Representing a transition between forested south and sayanna north, this area has

benefited from the building of a dam on the Mape River in 1989, which created an artificial lake of 3.2 billion m³ capacity.

From January 2007 through June 2009, all cases of skin lesions evocative of active BU were recorded as BU probable cases according to World Health Organization guidelines (6). During this period, 195 clinically suspected cases were reported from the Bankim health district (Figure). The overall median age for these 195 patients was 19.5 years (interquartile range 10-37 years). No significant difference in age was found according to gender, but a significant trend of decreasing overall median age was found (20 years in 2007 to 12 years in 2009. The most frequent type of lesion was ulcer. Since March 2009, the Centre Pasteur of Cameroon has performed laboratory confirmation for suspected BU cases: microscopic examination for acid-fast bacilli, culture, and M. ulcerans DNA detection by PCR (6). From April through June 2009, of 34 consecutive samples tested in the reference laboratory, 10 were positive for M. ulcerans by at least microscopy and PCR

Whether BU is emerging in Bankim or is just a newly recognized preexisting disease is difficult to establish. However, that the incidence of BU in the region is increasing is unquestionable. The decreasing median age of patients since 2007 might be consistent with emergence of BU as a new disease in Bankim. This observation could suggest either an increasing level of acquired immunity in the population, leading to protection correlated with age, or the expansion of risky sites for human infection with *M. ulcerans*.

During 1 week in January 2008, water bugs were collected from the artificial lake and water bodies located within or close to each community. A previously described sampling method was used (2). To detect *M*.

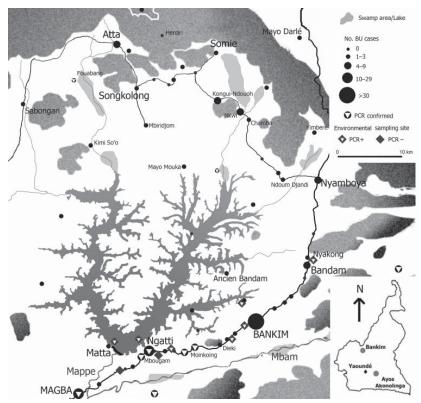


Figure. Distribution of Buruli ulcer (BU) patients reported January 2007–June 2009, and of water bodies with aquatic bugs harboring *Mycobacterium ulcerans*, Cameroon. Inset, Bankim area. A color version of this figure is available online (www.cdc.gov/EID/content/17/3/551-F.htm).

ulcerans DNA, we pooled the insects per family in groups of up to 10. Moreover, 99 members of the families Belostomatidae and Naucoridae were kept alive for saliva collection (2). The DNA of insect pool homogenized tissues and individual saliva samples were purified. We then searched for M. ulcerans molecular signatures (2). Among 1,349 insect specimens, 8 from the aquatic Heteroptera families were identified, and 12 (5%) of 244 insect pools were M. ulcerans positive. M. ulcerans-positive saliva was found in 11 (18%) of 61 insects in the family Belostomatidae and in 3 (8%) of 38 in the family Naucoridae. Water bodies where M. ulcerans-positive insects were collected are shown in the Figure.

The emergence of BU may be a consequence of the marked changes in the environment caused by the building of the dam. Elsewhere, human environmental modifications such as

construction of dams have been linked with increased incidence of BU (1). The main visible environmental effect is the large amount of flooded farmland. According to the seasons, the reservoir margins change the milieu of swamps and meadows. All these modifications affect plant and animal resources in the reservoir area by favoring rapid growth of aquatic macrophyte populations during reservoir filling, thus providing breeding sites for insects and leading to the extinction of area-endemic species and creation of new niches (7). These changes might favor development of M. ulcerans in biofilms on aquatic plants, which are then ingested by herbivorous animals, which are further prey for water bug predators, hosts, and possible vectors of M. ulcerans (8,9). The water bugs that were most frequently trapped and colonized by M. ulcerans (families Belostomatidae, Naucoridae, Nepidae,

Notonectidae) are carnivorous and able to bite humans (10).

Our study confirms expansion of BU in Cameroon. To facilitate detection of new BU foci, and to improve patient treatment (medical, surgical, rehabilitative), health care workers involved in tuberculosis/leprosy control programs should be educated about BU.

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Risk for Mycobacterium celatum Infection from Ferret

To the Editor: Mycobacterium celatum belongs to the group called "mycobacteria other than tuberculosis"; it is characterized by slow growth and a slender, rodshaped form $(0.25-0.5 \times 0.5-13.0)$ μm). The cells are acid fast and do not form cords or branches. The species name, celatum, which means hidden or concealed, refers to the problem of phenotypically distinguishing the species from other mycobacteria, especially M. xenopi. M. celatum was first described in 1993 as a pathogen in persons with AIDS (1). Until now, few cases in humans have been reported; those cases were predominantly mycobacteriosis disseminated immunocompromised patients (mainly those with AIDS), but they have also occurred in immunocompetent persons (1,2). For animals, 1 case of M. celatum infection in a ferret has been described (3). We describe another case in a ferret, with possible transmission to a human.

In 2009, a 3-year-old, neutered male, domestic ferret was examined in a veterinary clinic in Nuremberg, Germany, for a 5-month history of coughing, recent weight loss, reduced general condition, vomiting, and mild diarrhea. A chest radiograph showed multiple nodular densities in the lungs. Because of a poor prognosis, the ferret was euthanized. Necropsy was performed at the Institute of Veterinary

Pathology in Munich. The lungs contained multifocal firm, light brown nodules, 6–10 mm in diameter (Figure, panel A). Spleen and lymph nodes (cervical, retropharyngeal, bronchial, gastric, mesenterial, popliteal) were enlarged. Histologic examination of lung, lymph nodes, spleen, liver, and brain showed granulomatous inflammation with predominantly macrophages, epithelioid cells (in the lung, including bronchioles), and some multinucleated giant cells. Several acid-fast bacilli were visible with Ziehl-Neelsen staining, mainly intracytoplasmically in epithelioid cells (including those of bronchioles) (Figure, panel B).

Conventional mycobacterial culture and PCR were used to look for mycobacteria in the lung, spleen, and lymph nodes. For culture, the material was homogenized, decontaminated, and spread onto solid Loewenstein-Jensen agar and injected into a liquid culture (Mycobacteria Growth Indicator Tube; Becton Dickinson, Heidelberg, Germany) for automated detection of mycobacterial growth.

DNA was extracted from the homogenized tissue by using the QiaAmp DNA Mini Kit (QIAGEN, Hilden, Germany), and a 510-bp fragment at the 5' end of the ribosomal 16S rDNA was amplified as described (4). The amplified fragment of the expected length was sequenced, and data were analyzed by using the Integrated Database Network System (SmartGene Services, Lausanne, Switzerland; www.smartgene.com). The resulting sequence was clearly interpretable and unambiguously assigned to M. celatum; sequence identity to GenBank accession no. Z46664 was complete except for 1 mismatch in bp 490. Minor sequence diversity in the M. celatum 16S rDNA gene has been documented (5). The most closely related species, M. kyorinense, differs substantially, having 11 mismatches within the 16S rDNA gene (6). Species identity was