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Burkholderia pseudomallei Sequence Type 562 in China and Australia

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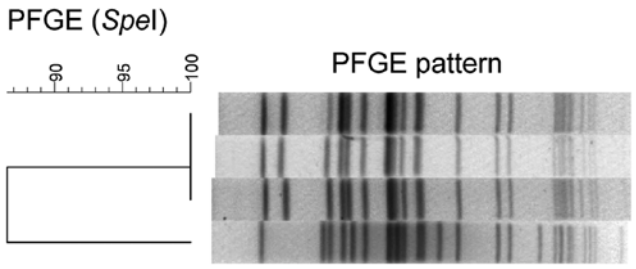
To the Editor: Melioidosis is increasingly being recognized in tropical and subtropical areas worldwide; the world's 2 major endemic foci are Thailand and northern Australia (1,2). Phylogenetic analyses of Burkholderia pseudomallei isolates, performed by using multilocus sequence typing (MLST) (3), have led to phylogeographic associations that can be used to track melioidosis epidemics (4). However, in contrast to the previous separation of B. pseudomallei into 2 phylogenetic groups (Australia and Southeast Asia/rest of the world) (5), we report an MLST sequence type (ST) that seems to be present in northern Australia, Taiwan, and southern China.

In mainland China, melioidosis was first reported in 1990 (*6*) and is now known to be endemic to several tropical provinces, including Hainan, a southern island province close to Southeast Asia. Since 2008, cases of melioidosis in Hainan have escalated; from July 2008 through July 2012, a total of 110 cases were microbiologically diagnosed at 2 general hospitals (Sanya People's Hospital and Haikou Municipal Hospital).

We characterized clinical isolates of *B. pseudomallei* from the 110 cases by using MLST, pulsed-field gel electrophoresis (PFGE), and 4-locus multilocus variable-number tandem-repeat analysis (MLVA-4) (*3*,*7*,*8*). MLST revealed 40 STs, 39 of which were consistent with STs from Southeast Asia, as evident from the global *B. pseudomallei* MLST database (http://bpseudomallei.mlst.net/). A single ST, ST562, which accounted for 3 cases in Hainan, was previously described on the global database as being from Australia; the 20 isolates from humans and 10 isolates from the environment deposited until September 1, 2014, all from Australia, had been isolated from 2005 through 2012. Although not deposited in the global MLST database, ST562 has also recently been reported from Taiwan (*7*). Among the 253 isolates of *B. pseudomallei* collected in Taiwan during 2004–2010, 1 clinical isolate and 9 environmental isolates were described as being ST562. Moreover, these 10 ST562 isolates displayed a unique PFGE pulsotype, distinct from that of other *B. pseudomallei* strains from Taiwan (*7*).

Of the 3 patients from Hainan from whom ST562 strains were isolated, 2 resided in the city of Sanya and 1 in the neighboring city of Lingshui (online Technical Appendix, http://wwwnc.cdc.gov/EID/article/21/1/14-0156-Techapp1.pdf); all denied a history of foreign travel, they shared no common risk factors, and all survived the infection. Further analysis of ST562, performed by using eBURST-based (http://eburst.mlst.net/) population analysis of the MLST dataset, showed that ST562 is a single-locus variant of ST167, which is represented on the MLST dataset by multiple human and environmental isolates from Thailand and to date by 1 human isolate from Cambodia. ST167 accounted for 1 of the 110 *B. pseudomallei* strains from Hainan. The *narK* locus of ST167contains allele 3 instead of allele 29, as seen in ST562; 3 base differences are found in allele 3: C72T (C→T position 72), C126T, and A435G. According to PFGE, the 3 ST562



Isolate source	Time of isolation	STs	MLVA-4 profile
Patient A	2008.07	562	11, 8,15,7
Patient B	2012.07	562	11, 8,17,7
Patient C	2012.06	562	10,10, 11,5
Patient D	2011.12	167	6, 5,14,8

Figure. Pulsed-field gel electrophoresis (PFGE) patterns for 3 sequence type (ST) 562 and 1 ST167 *Burkholderia pseudomallei* strains isolated during 2008–2012, Hainan, China. The isolate source, isolation time, ST, and 4-locus multilocus variable-number tandem-repeat analysis (4-MLVA) profiles are indicated for each strain. Scale bar indicates percentage similarity.

isolates from Hainan displayed a single pulsotype, and the other 107 isolates from Hainan belonged to distinct and diverse pulsotypes, similar to those observed in Taiwan. The uniformity of PFGE patterns in the Hainan and Taiwan isolates supports the possibility that ST562 might be a recently emerging clone. PFGE patterns of Hainan ST562 exhibited 86% similarity with ST167, differing by 6 bands (Figure).

Hainan ST562 isolates were further analyzed by using MLVA-4 (*8*), which divided 3 isolates (from patients A, B, and C) into 3 distinct MLVA-4 types (Figure). The 2008 isolate (MLVA-4 profile 11,8,15,7) and one 2012 isolate (profile 11,8,17,7) exhibited close relatedness, whereas another 2012 isolate (profile 10,10,11,5) was divergent from these, indicating that ST562 isolates in Hainan have been present long enough for some divergence into lineages.

Two mutually exclusive gene clusters, *B. thailandensis*–like flagellar gene cluster (BTFC) and *Yersinia*-like fimbrial gene cluster (YLF), have been linked to geographic origin and have been suggested for differentiating groups of *B. pseudomallei* (*9*). By PCR we found that ST562 isolates of Hainan were all YLF positive. BTFC predominates in Australian *B. pseudomallei* strains, and YLF predominates in Southeast Asia. Presence of YLF was also observed in strains from Papua New Guinea, possibly reflecting that country's location, intermediate between major foci of melioidosis (*10*).

In conclusion, by using MLST and the online MLST database, we revealed that *B. pseudomallei* ST562 is present in southern China as well as in Australia and Taiwan. The intercontinental character of this ST raises new questions about the epidemiology and control of melioidosis. Given the usual geographic separation of *B. pseudomallei* STs, we suggest that this wide-ranging presence of ST562 might result from more recent spread caused by transmission between regions. Increasing farming exchanges and trade of agricultural products between melioidosis-endemic regions might facilitate breaking of the geographic barrier; clonal introduction of *B. pseudomallei* could potentially occur in new locations. Improved and cooperative surveillance is required for elucidating the current and future global dispersion range of *B. pseudomallei* and for monitoring the consequent melioidosis infections.

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Hemolytic Uremic Syndrome Associated with *Escherichia coli* O8:H19 and Shiga Toxin 2f Gene

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To the Editor: Gastroenteritis caused by Shiga toxin–producing *Escherichia coli* (STEC), associated with hemorrhagic colitis and hemolytic uremic syndrome (HUS), has been identified as a major health problem (*1*). Shiga toxin is essential for the development of HUS (*2*). Shiga toxin can be distinguished into Shiga toxin 1 (Stx1) and Shiga toxin 2 (Stx2). The stx_{2f} STEC variant is a distinct group within STEC (regarding virulence genes) and is known to cause relatively mild disease, although reports of human illness are scarce (*3*).

During autumn 2013, a healthy 9-year-old boy in the Netherlands experienced fever, vomiting, and bloody diarrhea which persisted for days; he was admitted to the pediatric ward of a local hospital because of clinical signs of HUS with renal insufficiency: serum creatinine level 439 μmol/L (reference range 31–68 μmol/L); blood urea nitrogen concentration 34.1 mmol/L (reference range 3.3–5.6 mmol/L); thrombocytopenia (46 platelets/nL); reference range 150–450/nL), and low haptoglobin level. Hemoglobin levels decreased within 48 hours from 7.4 mmol/L to 5.5 mmol/L (reference range 6.9–8.4 mmol/L). His blood pressure was 127/82 (99th percentile for age and height). Renal insufficiency worsened over time, evidenced by maximum urea levels of 57.3 mmol/L and maximum creatinine levels of 744 μmol/L. Vomiting increased, and feeding became difficult. The boy was transferred to an academic nephrology center, where he received erythrocyte and thrombocyte infusions, then peritoneal dialysis. He received 1 prophylactic dose of cefazolin during insertion of the dialysis catheter. After 2 days, he entered a polyuric phase of renal failure; renal function normalized within a few weeks, however. To improve proteinuria, physicians prescribed a 3-month course of angiotensin-converting enzyme inhibitors after discharge.

A fecal sample tested positive for STEC by PCR in a local laboratory. Five isolates were sent to the National Institute for Public Health and the Environment (RIVM) as part of the national STEC surveillance. By using PCR, 1 of the 5 tested positive for the stx_{2f} gene and the attaching and effacing gene (*eae*), and negative for the genes stx_1, $stx_{2a\text{-}e}$, H7, O157, and enterohemorrhagic *E. coli* hemolysin (*hly*). Serotyping identified O8:H19. The other 4 isolates tested negative for all of the above-mentioned genes and were not serotyped.

The family had stayed in a hotel in Turkey and returned to the Netherlands 5 days before onset of illness. The only reported contact with animals was with a parrot in the hotel. On return to the Netherlands, the boy had eaten filet américain, a sandwich spread made of raw beef. The day before disease onset, he attended a party where barbecue was served by a catering company.

Since 2007, besides this reported case, 8 cases of STEC O8 were registered within the STEC surveillance system in the Netherlands: O8:H– (4 cases), O8:H19 (2 cases), O8:H8 (1 case), and O8:H9 (1 case). All 8 isolates were $stx_{2a\text{-}e}$-positive and stx_1-, stx_{2f}-, *eae*-, and *hly*-negative. Disease associated with these cases was relatively mild. During 2007–2010, a total of 13,545 human STEC infections were reported in Europe: 20 were registered as STEC O8; HUS did not develop in these case-patients (*4*). HUS developed in 2 patients infected with STEC O8 (O8:H2, O8:H19) in Germany during 1996–2000 (*5*); these isolates and all other isolates from HUS and non-HUS case-patients in this period tested negative for stx_{2f}. During 2008–2011, 87 stx_{2f} STEC infections were registered in the Netherlands (*3*). These infections were relatively mild; no HUS cases were registered. The virulence genes seen in the isolate of the described case, stx_{2f} and *eae*, but no *hly* or other toxin genes, were also seen in 97% of stx_{2f} STEC infections reported in the Netherlands (*3*). Besides being detected in humans, stx_{2f} STEC has only been detected in pigeons (*6*).