transmission. However, prevention of norovirus infection is often challenging due to several factors: low infectious dose, prolonged survival on dry inanimate surfaces, resistance to commonly used disinfectants, long infectious period, and prolonged shedding.[5–7]

Common foods that can cause norovirus infection include oysters, shellfish, fruits, and vegetables, especially when they are consumed raw without heating. Recently, another norovirus outbreak associated with consumption of green seaweed that was eaten unheated with vinegar seasoning was reported in South Korean schools.[8] However, outbreaks due to shredded, dried, laver seaweed processed for long-term preservation are unprecedented. As such, the current events provide several implications for preventing future norovirus outbreaks.

First, even in a packaged food product that has undergone heat-treatment, norovirus contamination can occur if it is handled by an infected person's bare hands during the manufacturing process. Although no regulations currently govern whether food manufacturers use gloves throughout the entire manufacturing process in Japan, more attention should be given to the risks of contamination. A previous report showed that the contaminated hands of food handlers could transfer infective norovirus even during gloving,[9] so effective hand hygiene including handwashing with soap and water should be emphasized by food manufacturers.

In addition, the integrated and streamlined school-lunch provision system is highly vulnerable to norovirus outbreaks. An appropriate decentralized provision system should be explored, weighing the risks and costs prudently. In the modern society where highly connected distribution routes are well established, a single contaminated product can cause multiple outbreaks in geographically disparate places. The responsible local authorities should share infection information promptly and widely across geographic borders.

Finally, a comprehensive surveillance system including dried processed foods should be established to detect the source of such pathogens as early as possible to avoid further spread of the virus. Notably, an Italian study using a polymerase-chain reaction identification technique revealed the presence of norovirus in semidried tomatoes deemed "ready to eat."[10] In the past, packaged dried foods may have been overlooked as the source of pathogens. In norovirus outbreaks, we should remember to consider this surprising source of infection.

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## Accidental Occupational Exposure to *Burkholderia pseudomallei* in South Korea Did Not Result in Melioidosis

*To the Editor*—Melioidosis is caused by *Burkholderia pseudomallei* and presents a high mortality rate of up to 40%.[1] *B. pseudomallei* is endemic in Southeast Asia and northern Australia. In Korea, there have been several imported cases; however, there have been no autochthonous cases.[2]

*B. pseudomallei* is regarded as a biothreat, and to date, 2 cases of laboratory-acquired melioidosis have been reported.[3] When laboratory workers are exposed to *B. pseudomallei*, postexposure monitoring and/or postexposure prophylaxis (PEP) according to the risk are recommended.[3] However, data are limited regarding the incidence of the development of melioidosis after accidental occupational exposure outside the laboratory.

On June 10, 2016, a 64-year-old Korean man presented with right second-toe pain, cough, sputum, and fever at the tertiary-care center in Ulsan, South Korea. He had been living in Singapore and Indonesia for 20 years. He was a sailor and had recently worked carrying sand in Indonesia. He returned from Indonesia to Korea because of his illness, 1 day before admission. He had been diabetic for the previous 18 years. Magnetic resonance image (MRI) of his foot showed an abscess and osteomyelitis in the second toe, and computed tomography scans of the chest revealed dense consolidation in the left upper lobe and multiple cavitary nodules in both the lungs. We performed incision and drainage of the abscess in the toe multiple times.

On June 15, *Burkholderia cepacia* was observed in the blood culture performed on June 10, which was identified using VITEK 2 (BioMerieux, Lyon, France). It was not identifiable via matrix-assisted laser desorption ionization time-of-flight mass spectrometry (MALDI-TOF MS) (BioMerieux, Lyon, France). However, to differentiate between *B. cepacia* and *B. pseudomallei*, we performed 16S rRNA sequencing, as previously described.[4] Nucleotide BLAST searches (http://blast.ncbi.nlm.nih.gov/Blast.cgi) of the isolate sequences showed the following matches: *B. pseudomallei* strain 982 chromosome 2 (GenBank accession number CP012577.1), 99% (1471 of 1472 bp); *B. pseudomallei* strain 982 chromosome 1 (GenBank accession number CP012577.1), 99% (1471 of 1472 bp). The patient had persistent *B. pseudomallei* bacteremia from June 10 to June 20, and his sputum and pus culture from the wounds also yielded *B. pseudomallei.* We administered ceftazidime, and the patient survived with prolonged antibiotic therapy.

After we identified *B. pseudomallei* infection, we contacted HCWs who were at risk, and classified them as low or high risk, according to the modification of recommendations by Peacock et al.[3] Low-risk exposures included (1) plate sniffing, opening of the lid of an agar plate growing *B. pseudomallei* outside a biological safety cabinet, (2) possible contact with the blood or body fluid (pus or urine) with intact skin or protected body and hands without evidence of aerosols, and (3) spillage of small volumes of liquid culture (1 mL) within a biological safety cabinet. High-risk exposures included (1) a penetrating injury with contaminated equipment, (2) any splash causing contamination of the mouth or eyes, (3) contact with the blood or body fluid (pus or urine) with nonintact skin, and (4) aerosol-generating activities performed outside a biological safety cabinet. Exposure of laboratory workers with certain health conditions, such as diabetes mellitus and chronic liver or kidney disease, in the absence of proper personal protective equipment, was also considered high risk. Serological testing of exposed HCW was performed using indirect hemagglutination assay (IHA) as previously described.[5] Thirty HCWs were exposed during 21 days; 5 were classified as high risk because they had been possibly exposed to the patient's blood and pus through nonintact skin during venipuncture or dressing wounds, and 25 including 2 laboratory workers were classified as low risk, mainly due to contact of blood with intact skin during venipuncture or dressing wounds. Laboratory workers inadvertently opened the lid of an agar plate growing *B. pseudomallei* outside a biologic safety cabinet. High-risk HCWs were offered trimethoprim/sulfamethoxazole for 21 days. All 30 HCWs were monitored to ascertain whether symptoms developed until September 5, and IHA was performed on July 7–11 (2 weeks or 4 weeks after exposure), July 25 (4–6 weeks after exposure), August 5 (6–8 weeks after exposure), and September 5 (11–13 weeks after exposure). None of the HCWs showed seroconversion or had the symptoms of melioidosis.

There have been 2 laboratory-acquired cases to date.[3] One occurred after sonication outside a safety hood, and the other after cleaning up a centrifuge spillage of *B. pseudomallei* culture with bare hands. This HCW had an ulcerative lesion on the finger at the time of exposure. During 2008–2013, there were 261 persons at risk for occupational exposure to *B. pseudomallei* while performing laboratory diagnostics in the United States; however, no infection occurred.[6] In addition, a review of work in a clinical laboratory in an endemic area suggests low risk to laboratory workers.[7] Our data suggest that the possibility of development of melioidosis after exposure to patient's body fluid on nonintact skin outside the laboratory may be low.

Notably, not following standard precautions led to many exposures identified in this study. Using appropriate personal protection equipment when performing dressing and venipuncture, expecting to contact patient urine or body fluids, or HCWs having nonintact skin, cannot be overemphasized. In addition, considering potentially false-negative antibody testing, lack of performing longer-term postexposure follow-up is a limitation of our study.

In conclusion, delayed diagnosis of melioidosis and not following standard precaution led 30 HCWs to be exposed to patient's blood or body fluids; however, none developed melioidosis.

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# Determination of a Cutoff Time Point for Prophylactic Exchange of Central Venous Catheters for Prevention of Central Venous Catheter–Related Bloodstream Infections in Patients with Hematological Malignancies

*To the Editor*—Prophylactic exchange of central venous catheters (CVC) for prevention of CVC-related bloodstream infections (CRBSI) in cancer patients is not generally recommended.[1,2] The related studies were conducted in relatively small populations in general and especially in small populations of cancer patients; in addition, thrombocytopenia, which often occurs in cancer patients, was an exclusion criterion in these trials.[3,4] However, prophylactic CVC exchange is sometimes still clinical practice in hematology, even though the optimal time point is unclear. Therefore, we aimed to investigate this question in a larger cohort of patients with hematological malignancies.

For this purpose, pooled data from the prospective Study to Evaluate Central venous Catheter-related Infections in Hematology and Oncology (SECRECY) registry (German Clinical Trial Register No. DRKS00006551)[5] and the prospective Antimicrobial Catheter Securement Dressings for the Prevention of CVC-related Bloodstream Infections in Cancer Patients (COAT) study (ClinicalTrials.gov No. NCT01544686)[6] from 11 centers in Germany were analyzed. SECRECY is an ongoing real-life registry of CRBSI in patients with hematological and oncological malignancies. COAT was a randomized multicenter trial comparing different CVC dressings in terms of CRBSI incidence in neutropenic patients.

In this study, we analyzed CRBSI due to short-term CVC (≥1 day in situ) inserted in the jugular or subclavian vein in patients with hematological malignancies. Only definitive CRBSI (dCRBSI) and the combination of definitive and probable CRBSI (dpCRBSI) according to the 2012 Infectious Diseases Working Party of the German Society for Hematology and Medical Oncology (AGIHO/DGHO) criteria[2] were considered from both data subsets. Using a receiver operating characteristic, CVC duration was used to determine a cutoff time point for CRBSI risk. An area under the curve (AUC) of <0.500 and 0.500–0.700 were considered of no and low predictive significance, respectively.[7]

Altogether, 1,194 CVC patients (median age, 59 years; range, 18–86; 59.2% men) with 20,330 CVC days (median CVC duration, 17 days; range, 1–60) were analyzed. In total, 610 CVC patients (51.1%) were from the COAT study and 584 (48.9%) were from the SECRECY registry. Underlying diseases were acute leukemia in 568 of these patients (47.6%), multiple myeloma in 316 patients (26.5%), and lymphoma in 226 patients (18.9%). The insertion site was the jugular vein in 819 of these patients (68.6%) and the subclavian vein in 375 patients (31.4%). In 890 of these patients (74.5%), chlorhexidine-containing CVC dressings were used from the beginning of the CVC insertion.

In total, 55 dCRBSIs and 137 dpCRBSIs occurred. Definitive CRBSI originated in the jugular vein CVC in 26 of these 55 patients (47.3%); dpCRBSI originated in the jugular vein CVC in 87 of 137 dp CRBSI patients (63.5%). The epidemiological data are summarized in Table 1. The CVC duration was the same for CVC with dCRBSI and dpCRBSI (median, 16 vs 16 days; $P = .62$). No significant difference was detected between dCRBSI onset and dpCRBSI onset (median, 14 vs 13 days; $P = .24$). Comparing dCRBSI onset with dpCRBSI onset in jugular vein CVC, we also found no significant