2010 had distinct random amplified patterns that differed from those generated from isolates A–G (Figure).

A previous study in Taiwan showed that the incidence (no. cases/100,000 inpatients and outpatients) of all pulmonary disease caused by NTM increased significantly from 2.7 (1.26) in 2000 to 10.2 (7.94) in 2008 (*6*). The most common organism in localized pulmonary infection and disseminated infection was *Mycobacteriam avium* cellular complex, and *M. abscessus* predominated in skin and soft tissue infection and lymphadenitis (*6,8*). The rise in pulmonary infections or colonization by NTM over recent decades, particularly among immunocompromised populations, is reported to be partly associated with the increased use of showers (*3–5,9*). Recently, a few studies have shown a link between pulmonary *M. avium* complex infections and home showerhead water microbiology (*3,4*). Although pulmonary disease caused by *M. abscessus* did not develop in the patient reported here, multiple respiratory specimens showed evidence of pulmonary colonization. The fact that cultures of the swabs taken from the interior surface of 4 showerheads were positive for *M. abscessus* but that cultures of the shower water were negative for the organism support previous findings that assemblages of NTM can occur inside biofilm that forms on the interior surface of showerheads (*5*). The same strains of *M. abscessus* isolated from different showerheads suggested the possibility of contamination in the environment by the aerosolized microorganism from respiratory secretions of the patient.

The mechanisms of susceptibility to mycobacterial infection in the patient with Sjögren's syndrome remain unknown (*1,2*). Previous studies suggest that toll-like receptor 2, dectin-1, tumor necrosis factor–α, interferon-γ, leptin, T-cells, and possibly neutrophils are major components in the host defense of HIV-noninfected patients against rapidly growing mycobacterial infections, including those caused by *M. abscessus* (*10*).

In summary, we report a case of bacteremic lymphadenitis caused by *M. abscessus* in a patient with Sjögren syndrome. Our data provide evidence that the interior surface of showerheads may serve as a source of infection by this waterborne and aerosolized microorganism.

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Antimicrobial Drug Resistance in *Corynebacterium diphtheriae mitis*

To the Editor: *Corynebacterium diphtheriae* is the agent of pharyngeal and cutaneous diphtheria. We did a retrospective analysis of the antimicrobial drug susceptibilities of 46 *C. diphtheriae* isolates sent during 1993 through 2010 to the French National Reference Centre of Toxigenic Corynebacteria. The isolates came from metropolitan France and French overseas departments and territories. Only 1 isolate, *C. diphtheriae* biovar *mitis*,

FRC24, expressed the following antimicrobial drug susceptibility profile: susceptible to penicillin, amoxicillin, ciprofloxacin, clindamycin, erythromycin, gentamicin, imipenem, kanamycin, rifampin, tetracycline, and vancomycin and resistant at an uncommonly high level to trimethoprim, sulfamethoxazole, and co-trimoxazole with Etest (bioMérieux, Marcy l'Etoile, France) MICs of >32, >1,024, and >32 mg/L, respectively.

This FRC24 isolate was isolated in 2008 from a cutaneous wound on a vaccinated 11-month-old child in Mayotte, an overseas department located in the Indian Ocean. Cutaneous carriage of *C. diphtheriae* is frequent in tropical countries where cutaneous diphtheria is endemic; cutaneous carriage represents a common mode of transmission of the bacterium. FRC24 was identified by using the API Coryne strip (bioMérieux). FRC24 is a toxigenic isolate; toxigenicity was confirmed by both *tox* gene detection and Elek test (*1*). Multilocus sequence typing was performed, and the sequence type (ST) of the isolate is ST91. This ST contains only this isolate and is part of lineage II, as are all *mitis* and *gravis* biovars (*2*).

To date, resistance to trimethoprim, sulfamethoxazole, or co-trimoxazole seems to be rare among the *C. diphtheriae* species, but few data are available (*3*). As trimethoprim resistance is often encoded by integron-driven *dfr* determinants, we looked for integrons. Integrons are bacterial genetic elements able to capture and express antimicrobial drug resistance gene cassettes (GCs) (*4*). GC movements are catalyzed by an integron-encoded integrase IntI. GCs, mainly promoterless, are usually expressed through a common Pc promoter (*5*). Only rare GCs contain their own promoter (*cmlA, qac, ereA1*). Three main classes of integrons have been described and are involved in the dissemination of antimicrobial drug resistance; class 1 is the most widely found in clinical isolates. Integrons have been mainly described among gram-negative bacteria; only a few studies have reported integrons in *Corynebacterium* spp. (*6,7*).

After bacterial genomic DNA extraction (DNeasy Blood & Tissue Kit; QIAGEN, Courtaboeuf, France), a multiplex Taqman-based quantitative PCR approach able to detect the 3 main classes of integrons was performed (*8*). We found that FRC24 harbored a class 1 integron. Analysis of the GC array showed that this integron harbored 2 GCs: *dfrA16* of 588 bp conferring resistance to trimethoprim and *qacH* of 511 bp conferring resistance to quaternary ammonium compounds (GenBank accession no. FR822749). To our knowledge, this GC array has not been previously reported, even among reports of other gram-negative isolates. Moreover, a *qac* determinant has been found only once in a *Corynebacterium* species, *C. pseudogenitalium* (which harbors a *qacH* variant in the chromosome [GenBank accession no. ABYQ02000013]), but not in an integron background. GC arrays were followed by the *qacEΔ1* (which also confers resistance to quaternary ammonium compounds), *sul1* (resistance to sulfamethoxazole), and *orf5* determinants as found in most class 1 integrons (*4*). In class 1 integrons, 13 Pc variants have been described (*5*). In the FRC24 integron, the *dfrA16* expression was mediated through a strong Pc variant (PcW_{TGN-10}) (*5*) that enables the high-level resistance observed for trimethoprim. As previously demonstrated, the *qacH* GC possessed its own promoter (*9*).

Trimethoprim is a commonly prescribed antimicrobial agent used in combination with sulfamethoxazole (co-trimoxazole) for the treatment of diarrheal diseases. This antimicrobial drug might have selected the emergence of such a strain expressing trimethoprim resistance. Furthermore, the FRC24 integron contains the antiseptic (quaternary ammonium compounds) resistance gene *qacH*. As cutaneous carriage of *C. diphtheriae* is frequent in tropical countries such as Mayotte, this bacterium could be exposed to quaternary ammonium compounds contained in disinfectants, hygienic hand washes, and cosmetic products. These products exert a selective pressure, which might play a role in selecting *qac*-containing strains, as has been suggested for *Staphylococcus* spp. (*10*). For staphylococci, the MICs of quaternary ammonium compounds are ≥2 mg/L. With FRC24, we tested for the MIC of cetyltrimethylammmonium bromide and found a MIC of 4 mg/L, suggesting that *qacH* is expressed in FRC24.

The sequencing of the genetic environment of this integron showed that it was framed by 2 copies of the insertion sequence IS*6100* disrupting at the left-hand side the *intI1* integrase gene (online Appendix Figure, wwwnc.cdc.gov/EID/article/17/11/11-0282-FA1.htm). IS*6100* has been described in a wide spectrum of host organisms, including *Corynebacterium* spp. (*6,7*), thus enabling this integron to be efficiently transferred to various bacteria.

Our findings show that *C. diphtheriae* is able to harbor integrons, which is of clinical relevance. Indeed, this genetic feature would give the isolates the capacity to easily acquire new GCs, such as *ere* GCs encoding resistance to erythromycin, which is one of the antimicrobial drugs recommended for diphtheria treatment.

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Transfusion-transmitted Syphilis in Teaching Hospital, Ghana

To the Editor: Transfusion-transmitted syphilis, which is caused by *Treponema pallidum* subspecies *pallidum*, is one of the oldest recognized infectious risks of blood transfusion (*1*). Routine screening of blood donors and refrigeration of donated blood before its use has resulted in only 3 reported cases of transfusion-transmitted syphilis over the past 4 decades (*2*–*6*).

The World Health Organization recommends screening all donated blood for syphilis (*7*), but doing so is challenging for many developing countries. Many blood banks in low-income countries, including Komfo Anokye Teaching Hospital in Kumasi, Ghana, do not screen donated blood for syphilis.

This study was conducted at Komfo Anokye Teaching Hospital. The purpose of this study was to determine the prevalence of syphilis among blood donors and whether seroconversion occurred in transfusion recipients. The study was approved by the ethics committees in Kumasi, Ghana, and Liverpool, UK.

Pretransfusion plasma samples from 200 conscious transfusion recipients in adult, pediatric, and obstetric inpatient departments and samples of their transfused blood were tested for syphilis. A positive initial result by enzyme immunoassay (EIA) (Bioelisa Syphilis 3.0; Biokit, Barcelona, Spain) was confirmed by using a *T. pallidum* hemagglutination assay (TPHA) (Syphagen; Biokit). A rapid plasma reagin (RPR) assay (RPR Reditest; Biokit) was used to determine whether seropositivity was caused by recent infection. Seronegative recipients who had received seropositive blood were retested 30 days posttransfusion to identify seroconversions. All donors and recipients with recent infections were offered counseling and treatment in accordance with national guidelines.

A total of 145 (73%) blood donors were male, and 109 (57%) units of blood had been stored for <4 days. Sixteen units (8%, 95% confidence interval [CI] 4.3%–11.7%) were seropositive for syphilis by EIA and TPHA. Of these units, 7 (44%) were RPR reactive, which indicated a prevalence of recent infections of 3.5% (95% CI 1.0%–6.0%) (Table). Twenty-six transfusion recipients (13%; 95% CI 8.3%–17.7%) were seropositive by EIA and TPHA. Of these recipients, blood samples from 9 (35%) were RPR reactive, indicating a prevalence of recent infection of 4.5%.

One recipient, an 8-year-old girl with severe malarial anemia (recipient 10), showed seroconversion after receiving an RPR-reactive unit of blood that had been refrigerated for only 1 day before being issued for