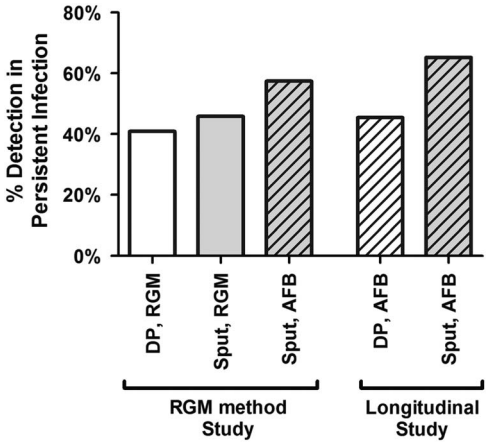

FIGURE 1. Frequency of detection of NTM by different samples and methods. Recovery of NTM in patients with persistent infection was assessed in deep pharyngeal (DP, white bars) or sputum (Sput, grey bars) using either the RGM culture method (solid bars) or AFB cultures (hatched bars) using data from two different studies: an assessment of the RGM culture method[10] and a longitudinal evaluation of NTM infection in CF.[4] Recovery of NTM from DP samples was generally lower than in sputum, although differences did not reach statistical significance.

why the ~45% recovery rate of *M. abscessus* was less in this subset of patients relative to the 65%–75% sensitivity for the RGM method estimated for the group as a whole.[10]

Notably, the results from this study are specific for rapidly growing mycobacteria from the *M. abscessus* complex and do not apply to the slower-growing pathogens from the *M. avium* complex. *Mycobacterium avium* complex bacteria cannot be recovered by the RGM culture method, nor have we observed *M. avium* from AFB cultures of deep pharyngeal swabs. While a limitation, *M. abscessus* and related pathogens appear to be more closely linked to negative clinical outcomes,[3,4] and recent studies suggest that these pathogens are increasing in frequency in CF.[1,2]

These findings suggest a role for deep pharyngeal swabs in the management of *M. abscessus* respiratory disease in CF, particularly in conjunction with the RGM culture method. The relative ease with which the RGM culture method can be implemented within regular microbiology work flows suggests that this approach could be utilized in routine screening for *M. abscessus* in children too young to produce sputum, for whom there are currently no effective screening methods. However, this approach should not be utilized to rule out NTM disease when it is clinically suspected given the lower sensitivity relative to other sample types and the inability to recover *Mycobacterium avium* complex pathogens.

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High Counts of Carbapenemase-Producing *Enterobacteriaceae* in Hospital Sewage

To the Editor—Carbapenemase-producing *Enterobacteriaceae* (CPE) are an increasing problem worldwide.[1] Because they are

TABLE 1. Characteristics of Carbapenemase-Producing *Enterbacteriaceae* (CPE) in Sewage and Clinical Samples

Institution	CPEs in Sewage	CFU/mL	No. of Clinical Isolates of CPE[a]	Predominant CPEs in Clinical Isolates	No. of Isolates[a]
Hospital A	*Enterobacter cloacae* bla_{NDM}	80,000	34	*Klebsiella pneumoniae* bla_{KPC}	10
	Citrobacter freundii bla_{NDM}	10,000		*Klebsiella pneumoniae* $bla_{OXA-48\ type}$	5
	Aeromonas caviae bla_{IMP}	1,000		*Escherichia coli* bla_{KPC}	4
Hospital B			14	*Klebsiella pneumoniae* bla_{NDM}	7
Site 1	*Enterobacter cloacae* bla_{KPC}	80,000		*Klebsiella pneumoniae* $bla_{OXA-48\ type}$	2
Site 2	*Aeromonas caviae* $bla_{OXA-48-type}$	30,000		*Escherichia coli* bla_{NDM}	2
Hospital C	*Escherichia coli* bla_{NDM}	1,000	9	*Klebsiella pneumoniae* bla_{IMP}	2
Hospital D	*Enterobacter asburiae* bla_{KPC}	70,000	1	*Escherichia coli* $bla_{OXA-48\ type}$	1
	Enterobacter kobei bla_{KPC}	10,000			
Hospital E	0		2	*Serratia marcescens* bla_{NDM}	1
				Klebsiella pneumoniae bla_{NDM}	1
Hospital F					
Site 1	0		0	…	…
Site 2	0				
Community	0		…	…	…

[a]January 1, 2014–May 31, 2014.

often resistant to almost all available antibiotics, treating infections caused by these bacteria is extremely difficult. Recently, concern has also been raised about hospital sewage as a potential source of CPE in the environment.[2] To investigate this possibility locally, we recently sampled sewage from 6 hospitals and a community institution (used as control) in Singapore.

Unconcentrated sewage was streaked out using 1 μL and 10 μL loops onto ChromID CARBA plates (bioMérieux, Marcy l'Etoile, France). After incubation for up to 48 hours at 35°C in air, 3 predominant colony morphotypes resembling *Enterobacteriaceae* per sample were chosen for further investigation. Carbapenemase activity was determined by the Carba NP test and the modified Hodge test.[3,4] Identification of bacteria was done by MALDI-TOF (Bruker Daltonics Pte. Ltd., Singapore). The presence of carbapenemase genes was confirmed by multiplex polymerase chain reaction (PCR).[5] The results of our survey are shown in Table 1 together with the number of CPEs isolated from clinical specimens (not stool surveillance cultures) taken from patients in each hospital.

CPEs were found in the sewage of 4 hospitals. The numbers of isolates of CPE in sewage generally corresponded with the frequency of isolation of clinical isolates in the hospitals. However, there was no direct correlation between sewage and clinical isolates with regard to bacterial species and carbapenemase genes. Clinical isolates were commonly *Klebsiella pneumoniae* and *Escherichia coli*, whereas *Enterobacter* and *Aeromonas* species were more common in sewage. We are uncertain of the reasons for this discrepancy between sewage and clinical isolates; it could be a chance finding resulting from limited sampling. However, this finding does seem to be quite consistent across different hospitals. Another possibility is that it may reflect a difference between the species of CPE that cause disease (clinical isolates) and those that merely colonize the gut (found in sewage) without resulting in infection. This explanation is only partial. During the same period (January 1, 2014–May 31, 2014) in Hospital A, which has the most comprehensive surveillance program for CPE, *K. pneumoniae* (83 isolates) and *E. coli* (47 isolates) remained the most common species isolated from patient stools, with *Enterobacter cloacae* a distant third (27 isolates). No *Aeromonas* species were isolated from stool surveillance cultures. Finally, these results may be the result of an ecological dynamic occurring in the sewage system. *Aeromonas* species are able to survive and proliferate in water distribution systems and may acquire plasmids containing carbapenemase genes from the clinical isolates entering into the system.[6]

This study has several limitations. Data collection was performed at only 1 time point per sampling site, and the methods used for quantifying colony counts were very simple. Nevertheless, we were surprised at the ease with which large numbers of CPEs could be isolated from the unconcentrated sewage of some hospitals. All 6 hospitals that took part in this study already have a stool screening program for CPE in place, but the extent of the screening varies. Most hospitals screen patients who have been hospitalized in the past year. The hospitals with the largest clinical burden of CPE (hospitals A and B) additionally screen patients on at-risk wards like Hematology and Intensive Care. Finally, we did not exclude the possibility that the CPE came from staff because personnel are not routinely screened for CPE carriage.

The results of this study suggest that large numbers of CPEs in hospital sewage may strengthen the case for a screening program if none exists. The microbial ecology of resistant bacteria in the sewage system, the risk of environmental contamination, and the role of *Enterobacter* species and *Aeromonas*

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species in the dissemination of carbapenemase genes in the environment remain to be studied.

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