Stenotrophomonas maltophilia in Salad

To the Editor: *Stenotrophomonas maltophilia* has emerged as an important nosocomial pathogen, especially in debilitated and immunocompromised persons (1). However, comparatively little is known of the epidemiology of this bacterium, and sources and routes of transmission of *S. maltophilia* are not well understood. The bacterium is widely distributed in the environment, including in plant rhizospheres (2). Although environmental sources such as ice-making machines have been implicated in outbreaks of nosocomial *S. maltophilia* sepsis, the source of infection in other outbreaks and in sporadic cases often remains unidentified. Food as a source of the bacterium has not been investigated; given the association of the bacterium with plants, we investigated the prevalence of the bacterium in salad vegetables.

Salads were purchased from reputable supermarkets and transported immediately to the laboratory. Ten grams of salad was homogenized in 90-mL sterile saline in a stomacher for 1 to 2 min. Aliquots (200 µL) of decimal dilutions of the homogenate were plated onto vancomycin-imipenem-amphotericin B (VIA) agar (3) and incubated for 24 to 48 h at 30°C. Occasional imipenem-resistant environmental bacteria, such as *Janthinobacterium lividum* or vancomycin-resistant *Enterococcus faecium*, may grow on VIA, but the medium contains a mannitol/bromothymol blue indicator system, allowing these bacteria, which produce acid from mannitol, to be distinguished from *S. maltophilia*. Putative *S. maltophilia* colonies were further identified by the API 20NE system (bioMérieux, Marcy l'Etoile, France).

Susceptibilities of 9 confirmed isolates to ceftazidime, chloramphenicol, colistin sulfate, gentamicin, minocycline, piperacillin/tazobactam, and trimethoprim-sulfamethoxazole were determined by using a disk diffusion method. *Pseudomonas aeruginosa* (NCTC10662) was used as a control. Because disk diffusion is not a reliable method for determining the susceptibility of *S. maltophilia* to quinolone antimicrobial agents (1), the Etest was used.

S. maltophilia was cultured from 14 (78%) of 18 salads. Numbers ranged from 1.50×10^2 to 1.96×10^5 CFU/g (mean 1.75×10^5 CFU/g, median 7.05×10^3 CFU/g). All isolates were susceptible to ciprofloxacin, colistin sulfate, minocycline, and trimethoprim-sulfamethoxazole. Eight (89%) were resistant to chloramphenicol; 7 (78%) to piperacillin/tazobactam; 5 (56%) to ceftazidime; and 2 (22%) to gentamicin.

All products examined were labeled "washed and ready to eat," and, thus, consumers would be unlikely to wash these products before consumption. The growth characteristics of *S. maltophilia* in products of this type, especially if subject to temperature abuse, are unknown, but in the domestic setting, numbers of the bacterium may increase before use.

All of the 9 isolates examined exhibited resistance to >2 of the 8 agents tested, with 2 isolates resistant to 4 compounds and thus had resistance phenotypes similar to those of strains associated with human infection. These findings are in agreement with those of Berg (2), who reported multiple resistances among isolates of *S. maltophilia* associated with oilseed rape. However, as with most strains of clinical origin, all the isolates tested here remained susceptible to trimethoprim-sulfamethoxazole and minocycline.

Although other investigators have examined salad products for the prevalence of pathogenic bacteria (4), they did not attempt to isolate *S. mal-tophilia.* Another study may have underestimated the prevalence of the bacterium in these products since a selective medium was not used (5). We have shown in a clinical setting that the use of a medium selective for *S. maltophilia* improves the recovery of this bacterium (6). Furthermore, the optimal growth temperature of *S. maltophilia* is 30°C, and incubation at higher temperatures (7) may reduce the likelihood of recovering the bacterium from food products. We recommend that future studies of *S. maltophilia* in food products use a medium such as VIA and that cultures be incubated at 30°C.

Prepackaged, ready-to-eat salads, such as those examined in this study, are washed in chlorinated water before sale. This measure is clearly insufficient to remove *S. maltophilia* from these items, possibly because the bacterium may exist in biofilms in some of the components of these products. *S. maltophilia* is capable of forming biofilms on a number of materials (8). Alternatively, products may become contaminated from environmental sources in production plants after washing.

The importance of *S. maltophilia* in ready-to-eat salads, which are marketed in a manner that assumes the product does not need washing before consumption, is unknown; nevertheless the presence of the bacterium in these products serves to highlight recommendations that these items should be avoided by severely immunocompromised persons, especially those with neutropenia (9). Recently, Apisarnthanarak et al. (10), in a prospective study of hospitalized oncology patients, identified intestinal colonization with *S. maltophilia* in 4 (9.5%) of 41 patients, which emphasizes that foodstuffs may be a potential source of this bacterium for some patients. This is a preliminary study, however, and further studies are needed, in particular, molecular typing of food and human-associated isolates,

to investigate the hypothesis that intestinal carriage of *S. maltophilia* may follow consumption of contaminated foodstuffs.

Andleeb Qureshi,* Louise Mooney,* Miles Denton,† and Kevin G. Kerr‡§

*University of Leeds, Leeds, United Kingdom; †General Infirmary at Leeds, Leeds, United Kingdom; ‡Harrogate and District National Health Service Foundation Trust, Harrogate, United Kingdom; and §Hull York Medical School, York Campus, Heslington, United Kingdom

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Address for correspondence: Kevin G. Kerr, Department of Microbiology, Harrogate and District NHS Foundation Trust, Harrogate HG2 7SX, UK; fax: +44-1423-553077; email: kevin.kerr@hdft.nhs.uk

Avian Influenza H5N1 and Healthcare Workers

To the Editor: Since January 2004, 35 human cases of avian influenza A virus H5N1 have been reported in Vietnam. Human-to-human transmission of H5N1 is a major concern, particularly because of reported family clustering (1). Two probable cases of human-to-human transmission were recently reported from Thailand (2), and evidence for human-to-human transmission was found in the 1997 Hong Kong outbreak (3). We evaluated healthcare workers exposed to 2 patients (patients 5 and 6 [1], referred to as patients A and B, respectively, in this article) with H5N1 infection, confirmed by polymerase chain reaction (PCR), to determine the potential risk for nosocomial human-to-human transmission of H5N1.

Patient A was admitted to a general ward of a pediatric hospital in Ho Chi Minh City on January 15, 2004, on day 8 of illness; no infection control measures were taken at that time. On January 18, 2004, she was transferred to the intensive care unit (ICU). Eight hours after ICU admission, limited infection control measures were implemented: the patient was transferred to a single room, and healthcare workers were required to use disposable surgical masks and gloves and wear nondisposable gowns. However, because resources were limited, each healthcare worker wore only 1 glove.

On January 23, patient A was transferred to another hospital.

Patient B was admitted to the infectious diseases ward of the pediatric hospital on January 19, 2004, on day 6 of illness; he was transferred to the ICU after 4 hours and stayed there until he died on January 23. Infection control measures were implemented 2 days after ICU admission; these measures were similar to those taken for patient A except that no single room was available.

From January 25 to 27, 2004, a nasal swab specimen and baseline serum sample were collected from healthcare workers at the hospital; each worker also completed a questionnaire. On February 9 and 10, follow-up serum samples were collected. Nasal swab samples were tested by reverse transcription (RT)-PCR to detect the H5 gene (1). Paired serum samples were subjected to enzyme-linked immunosorbent assay (ELISA) (Virion/Serion, Würzburg, Germany) to detect immunoglobulin G against the nucleoprotein of influenza A; samples were also subjected to an H5-specific microneutralization assay (4).

Of 62 healthcare workers involved in caring for patient A, patient B, or both, 60 (97%) provided both samples and questionnaires: 16 who cared for patient A on the general ward, 33 who cared for patients A and B in the ICU, and 11 who cared for patient B on the infectious diseases ward or who were consulted for diagnostic or clinical procedures involving either patient. Characteristics of the workers and their exposures are shown in the Table.

The median time between last exposure and collection of the nasal swab and the baseline serum samples was 7 days (range 2–12 days). The median time between last exposure and collection of the follow-up serum sample was 21 days (range 17–26 days). All 60 nasal swab samples were negative by RT-PCR. Paired serum samples were available from 46