Taneja *et al*. raised an important issue about the decontamination of any medical equipment within an area. The dry gas process has been used for many years on various types of electronic equipment, but it will depend on the equipment. For example, electrochemical sensors will be affected by any oxidizing agent. On the other hand, we have successfully fumigated computer and other electrical equipment. Equipment within a room should be considered during a risk analysis prior to fumigation. As for liquid or wet processes, I have a personal reservation about using any liquid on any electrical equipment.

Regarding a recommendation for a 'safe' level of environmental contamination, this work is ongoing and will depend on the use of the area (e.g. routine patient ward or intensive care area), the pathogen and the level of contamination. Some environmental disinfection studies have shown lack of efficacy of disinfectants tested, although these results may reflect a lack of adequate efficacy in the time, dilution and formulation type of disinfectants used. There is a need for well-controlled environmental disinfection studies and their impact on infection rates within hospitals. We and others are investigating this and process optimization for healthcare applications. It is clear that this will not only include fumigation processes but also guidelines on the effective use of routine disinfectants in hospital practice.

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Available online 6 December 2005

Editorial comment

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doi:10.1016/j.jhin.2005.09.006

Prospective survey of digestive tract colonization with enterobacteriaceae that produce extended-spectrum β-lactamases in intensive care units

Sir,

Enterobacteriaceae that produce extended-spectrum β-lactamases (E-ESBLs) are an increasing problem. E-ESBLs are important agents of nosocomial infection and are responsible for outbreaks that occur predominantly in intensive care units (ICUs).[1] ESBLs are most commonly produced by isolates of *Klebsiella pneumoniae* and *Escherichia coli*, but infection and colonization involving other ESBL-producing organisms such as *Morganella morganii*, *Enterobacter* spp., *Citrobacter* spp. and *Proteus* spp. have been reported.[2] In Belgium, over the last 15 years, nosocomial infections with E-ESBLs have gradually increased. ESBLs have mainly been studied from isolates of *Enterobacter aerogenes*, the species of greatest concern in Belgian hospitals.[3]

Normal intestinal microflora is the major source of common hospital-acquired infections such as urinary tract infections.[4] Most of these infections are caused by faecal aerobic Gram-negative bacilli.[5] As the occurrence of bacterial resistance has increased, interest in the identification of specific reservoirs of bacterial resistance has grown.

The aim of the present letter is to report and discuss the results of a prospective study performed in three ICUs over an 18-month period in order to assess the importance of digestive tract colonization with E-ESBLs. For this purpose, freshly passed stool specimens were taken on admission and twice per week from each patient until discharge from the ICU. They were inoculated on selective Drigalski agar (Biorad, Marnes la Coquette, France) containing ceftazidime. Clinical isolates of enterobacteriaceae were identified using the API 20E® system (BioMérieux, Marcy l'Etoile, France).

Detection of ESBLs was performed using a double disc diffusion test. On Muller-Hinton agar (BioMérieux, Marcy l'Etoile, France) inoculated with a 0.5 McFarland suspension of the isolate, discs of ceftazidime (30 μg), cefepime (30 μg) and cefotaxime (30 μg) (Becton, Dickinson and

Co.,Sparks, MD, USA) were applied 25 mm away from a disc containing amoxicillin-clavulanic acid (30 μg) (Becton, Dickinson and Co.). After overnight incubation at 35 °C, the test was considered to be positive when the zone of growth inhibition for at least one cephalosporin had a 'champagne cork' aspect.[6]

Six hundred and ninety-two stool specimens from 224 patients (148 men, 76 women, mean age 61 years) were cultured on the ceftazidime-containing medium during the study period. One hundred and eighty yielded enterobacteriaceae and ESBLs were detected in 98 isolates (14.16%) originating from 32 patients (14.29%). The most frequent species producing ESBLs was *E. aerogenes* (50%), followed by *E. coli* (21.43%) and *K. pneumoniae* (9.18%). In several Belgian[7] and French hospitals, it has also been observed that *E. aerogenes* has replaced *K. pneumoniae* as the predominant producer of ESBLs.[8] Among the 32 patients colonized with E-ESBLs, 14 were identified on admission or within 48 h of admission to the ICU. For the remaining 18 patients, the mean number of days between admission and colonization with E-ESBLs was 6.78 (range 3-21 days).

Of the 32 patients who were colonized by E-ESBLs in the digestive tract, 68.75% ($N=22$) were also colonized or infected by E-ESBLs at another body site. In contrast, among the 192 patients with no faecal colonization during their ICU stay, only 23 (12%) were infected or colonized with E-ESBLs at another body site ($P \leq 0.001$). Consequently, faecal carriage of E-ESBLs seems to be an important risk factor for colonization or infection with E-ESBLs at other sites. For this reason, rectal swabs should be collected for all 'high-risk' patients on admission to the ward and repeated every week until the patient's discharge from the ICU. Screening on admission would certainly be necessary because, in this study, 44% of the patients were E-ESBL positive within 48 h of admission to the ICU. The origin of this carriage is not documented: had these patients been hospitalized previously in the last few months or was this a cross-contamination? The first hypothesis seems more plausible because of the hospital ecology of this type of enterobacterium and because of rather persistent carriage. For 18 new cases, the acquisition of E-ESBLs could be due to colonization by an endogenous strain selected from the patient's own flora or patient-to-patient transmission via healthcare personnel.

In conclusion, these data confirm that digestive tract colonization with E-ESBLs is relatively common and that faecal carriage of E-ESBLs can be a good marker for colonization or infection with E-ESBLs at another body site. Consequently, rectal swabs should be collected for all 'high-risk' patients on admission to the ward and repeated every week until the patient's discharge from the ICU. This screening of E-ESBLs has two aims: to limit the cross-transmission of E-ESBLs by applying contact precautions for patients who have screened positive; and to establish empirical antibiotic treatment covering E-ESBLs in case of infection of a patient with prior carriage of E-ESBLs.

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Available online 5 December 2005

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doi:10.1016/j.jhin.2005.09.014

Hospital work as a major risk factor for *Helicobacter pylori* infection

Sir,

In a paper recently published in the *Journal of Hospital Infection*, Mastromarino *et al*. concluded that hospital work involving direct contact with patients seems to constitute a major risk factor for *Helicobacter pylori* infection.[1] The finding is of potential relevance for knowledge of risk factors for infection and practical implications. However, some methodological flaws in the study make interpretation of the data difficult.

Three groups of workers at a university teaching hospital were compared: 92 staff from gastrointestinal endoscopic units (Group A), 105 general medical staff (Group B), and 52 staff from laboratories and other units (Group C). The total number of workers considered for the study was not given, so it is not known how many eligible workers did not participate or why. From the above reported numbers, it seems likely that the vast majority of the target population did not participate in the study, thus introducing a potential self-selection bias. Furthermore, Group A workers were probably over-represented compared with Group B and C workers.

Another potential bias may derive from the higher percentage of workers with a high level of education carrying a lower risk of *H. pylori* infection[2] in Group A (50%) compared with Group B (30%).

A history of gastritis was reported in 26% of Group A workers, 23% of Group B workers and 17% of Group C workers. As the diagnosis of gastritis is histological, this means that a relevant proportion of workers with a mean age of 43 years had undergone an upper gastrointestinal endoscopy in the past. This high proportion of endoscopic examinations is extremely unusual in this age group, and strongly points towards self-selection bias.

Seventeen percent of Group C workers had a past diagnosis of gastritis; however, only 2% of them had a history of *H. pylori* infection. As *H. pylori* is the cause of the vast majority of cases of gastritis,[3] it is very surprising that only a small fraction of subjects with gastritis had infection in this group, and suggests that Group C had rather unusual clinical characteristics.

Mastromarino *et al*. reported that 2% of Group C workers (i.e. one worker) had a history of *H. pylori* infection. However, six members of this group had received therapy aimed at *H. pylori* eradication, meaning that five workers had received pointless *H. pylori* eradication therapy. Alternatively, it may be argued that collection of data was inaccurate in this regard. In any case, considerations by the authors on the re-infection rate in Group C seem to be based on unreliable data.

It is unclear why Mastromarino *et al*. believed that 30% of the *H. pylori* positivity observed in Group B and C workers who underwent previous eradication treatment was due to re-infection; treatment failure is far more likely. Indeed, re-infection in adults is quite rare,[4] whereas the success rate of eradication therapy has declined in the last years due to antibiotic resistance, and 30% treatment failure is by no means an unexpected finding.[5]

Mastromarino *et al*. found a higher prevalence of infection in older subjects belonging to Group A, and no age effect was observed on prevalence in Groups B and C. They speculated that safer working habits among young personnel working in endoscopy units may be responsible for this finding. However, an increase in the prevalence of infection with age is generally found in the Western world, due to the well-known cohort phenomenon.[6] Therefore, what is unusual is the lack of an age effect in Groups B and C, probably due to self-selection bias.

Mastromarino *et al*. reported a correlation between 'gastrointestinal' symptoms and *H. pylori* infection in Group A patients, without distinguishing between upper abdominal symptoms (i.e. dyspepsia), which may have a relationship with infection, and symptoms relating to the lower abdomen (i.e. irritable bowel syndrome), which bear no relationship to infection. Furthermore, they made a curious classification of symptoms, recognizing abdominal pain, dyspepsia and nausea as distinct entities. Indeed, according to the universally accepted Rome criteria,[7] upper abdominal pain and nausea are part of dyspepsia.

No significant difference was found in the prevalence of infection between workers exposed to oral or faecal secretions and non-exposed workers. As the putative mechanism of *H. pylori* transmission is through oral and faecal secretion,[8] this finding suggests that infection was acquired outside the working environment.