tor by pulling it down to see, talk, or breathe comfortably in such a way as to reduce its efficacy. Because HEPA respirators are reusable, they need to be stored for subsequent use, thus creating storage problems. Concerns and confusion about using the same HEPA respirator on different patients were raised. HCWs also find it confusing to use HEPA respirators for tuberculosis protection and masks for other types of respiratory isolation. Moreover, the safety, efficacy, and patient-care impact of HEPA respirators still remain controversial.[2,4-7]

At our 390-bed community teaching hospital, we decided to switch from the use of HEPA respirators to the N-95 respirators in 1996. Our criteria for selecting respirators were safety, efficacy, cost-effectiveness, user acceptance, and effect on patient care.

In the past, regulatory agencies and experts have not agreed on which respirator is most appropriate. They now agree that the N-95 respirator meets the Centers for Disease Control and Prevention (CDC)'s criteria for the minimum level of respiratory protection for tuberculosis.[8,9] Our policy of adapting the use of N-95 respirators meets the CDC's criteria. The switch to the N-95 respirator also resulted in an annual savings of 60% for our hospital. Compliance also is increased due to the fact that N-95 respirators are disposable, not bulky, and easier to wear. The use of one type of respirator for all types of respiratory isolation also eliminates confusion among HCWs. Rates of purified protein derivative skin-test conversions remain the same since the N-95 respirator adoption.

The switch from the use of HEPA respirators to N-95 respirators for PPP at our hospital not only meets CDC criteria for tuberculosis protection but also resulted in a 60% annual savings in purchase of respirators and increased HCW compliance.

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The author replies.

We find it ironic, if not a bit perverse, that we have been cast as the defenders of the high-efficiency particulate air (HEPA) respirator, because we were among the first to register our concern.[1] We thought we had spoken our piece in the "Discussion" section of our article, where we commented that "healthcare workers find the HEPA respirators difficult to wear for extended periods and often must leave the room to remove the device and 'catch their breath' before returning into respiratory isolation." We also noted that we had switched to the N-95 because of overwhelming worker preference with the more comfortable device.

However, the point and the tone of the letter from Zafar, Poropatich, and Nguyen suggest that the conclusions of our article may have been unclear. They comment that, at their hospital, the HEPA respirator was cumbersome, unpopular, and essentially unsuitable for human use. After shifting to an N-95 respirator, worker compliance with the program improved, and equipment costs decreased 60%. Without knowing the details of tuberculosis control at Columbia Arlington Hospital, such as number of cases of tuberculosis annually, number of respiratory isolation days annually, and so on, it is difficult to ascribe the savings strictly to cheaper equipment. Also, their "concerns and confusion about using the same HEPA respirators on different patients" suggests a fundamental problem in understanding how best to use these units; however, we are happy that the N-95 respirator is cheaper and seemingly as effective at their hospital.

Our article was meant to serve as a counterpoint to the claim that the cheaper N-95 devices would, as advertised,[2] save "millions" of dollars. As we showed, in our tuberculosis-heavy hospital at least, the HEPA program got progressively cheaper over time, leading us to wonder if a shift to cheaper per-unit equipment would necessarily result in money saved. Simply stated, we think that wearing one $4 HEPA respirator for a month may be cheaper than wearing 10 to 15 N-95 masks (at $0.50 per unit) a month.

For once and for all, we did not, and do not, endorse the HEPA respirator as preferable and feel, as we noted, that "the best respirator is the respirator that people will wear."

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Susceptibility of Vancomycin-Resistant Enterococci to Environmental Disinfectants

To the Editor:

I read with great interest the report on four strains of *Enterococcus faceium,* two sensitive and two resistant to vancomycin, which were challenged with several classes of hospital disinfectants. There was no difference in susceptibility to disinfectants between *E faecium* sensitive or resistant to vancomycin.[1]

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When vancomycin-resistant *E faecium* is grown for 12 hours with one-half minimum inhibitory concentration of vancomycin, large cells 2 to 4 µm in length consisting of individual enterococci connected by wide and fibrous cross walls result.[2] Considering their size and the fact that most constitutive individual cells are shielded from the environment by these wide cross walls, it was conceivable that they could be more resistant to disinfectants than *E faecium* of normal structure.

Two strains of *E faecium* resistant to 400 µg/mL vancomycin were incubated for 12 hours with 200 µg/mL vancomycin to produce the large cells. A Gram stain confirmed the presence of large cells. Suspensions of approximately 10^6 colony-forming units (CFU)/mL of large cells, as well as organisms grown without vancomycin (control), were challenged by the suspension technique with disinfectants or with saline as a control.

The organisms were exposed to 70% isopropyl alcohol for 5 and 10 seconds, diluted in trypticase soy broth, and planted on blood agar. They also were exposed to povidone iodine 1:10 in water for 30 and 60 seconds, neutralized with 1% sodium hyposulfite, and planted on blood agar. Colony counts were done after 48 hours of incubation. Exposure of large cells for both strains for 5 seconds to 70% isopropyl alcohol or 30 seconds to povidone iodine 1:10 produced growth of 70 and 90 CFU/mL, respectively. Exposure for 10 seconds to the alcohol and for 60 seconds to povidone iodine resulted in no growth. The controls, not exposed to disinfectants, produced growth ranging from 10^6 to 4×10^6 CFU/mL.

In conclusion, the large cells of *E faecium* that resulted from exposure to vancomycin, and the cells of normal structure grown without vancomycin, were highly and equally susceptible to alcohol or to povidone-iodine.

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Natural History of Colonization With Vancomycin-Resistant *Enterococcus faecium*

To the Editor:

We would like to add our observations on gastrointestinal colonization with vancomycin-resistant enterococci (VRE) in cancer patients to those of Montecalvo et al.[1] At the University of Maryland Cancer Center (now the Greenebaum Cancer Center), VRE have been isolated from rectal surveillance cultures of 51 patients during a 3-year period (March 1993-February 1996). We describe the pattern of colonization on weekly inpatient follow-up cultures and the influence of antibiotic use, specifically vancomycin, on the pattern of colonization.

Fifty-five percent of these patients had acute leukemia; 25%, other hematological malignancies; 14%, solid tumors; and 6%, other diagnoses (sickle cell anemia, cryoglobulinemia, aplastic anemia). Their mean age was 55 years (range, 23-84). The mean length of stay prior to the first VRE isolation was 45 days (range, 1-156). Seventy-one percent died during the follow-up period. The mean number of days survived in those who died was 214 (range, 1-736).

Of the 51 patients, there was sufficient follow-up information on 36 (70%) to define three patterns of VRE follow-up in patients. Forty-four percent had a persistent pattern of colonization: two or more cultures over at least 2 weeks were consecutively positive for VRE until death or end of study period. Thirty-three percent had a clearing pattern of colonization: two or more cultures over at least 2 weeks were consecutively negative for VRE until death or end of study period. Twenty-three percent had an intermittent pattern of colonization: VRE was detected again before death or end of study period after at least three cultures negative for VRE over at least 3 weeks. This is very similar to the patterns that Montecalvo et al describe.

Molecular typing by pulsed-field gel electrophoresis (PFGE) of the VRE isolates also showed similar findings. PFGE on consecutive isolates in patients with persistent colonization demonstrated that two thirds of the patients maintained the same strain over time, whereas the remaining third acquired a different strain. In the patients with intermittent colonization, half the patients maintained the same strain, over periods of 3 to 15 months with negative cultures, while the other half acquired a different strain.

We found an association between vancomycin use and the pattern of VRE colonization in these patients (Table). Patients with a persistent pattern of colonization were more likely to have received vancomycin while hospitalized compared to patients with intermittent or clearing patterns of VRE colonization. Also, patients with an intermittent pattern of colonization were more likely to have received vancomycin while hospitalized compared to patients with a clearing pattern of VRE colonization. Although a similar trend was seen with overall antibiotic use, the effect of vancomycin was more striking.

TABLE
THE USE OF ANTIBIOTICS WHILE HOSPITALIZED IN CANCER PATIENTS WITH VANCOMYCIN-RESISTANT *ENTEROCOCCUS* (VRE) COLONIZATION

	Pattern of VRE Colonization			
	Persistent N=14	Intermittent N=10	Clear N=15	*P**
Days hospitalized, mean	127	47	42	<.01
All antibiotics				
% hospital days on antibiotics, mean	87	75	65	0.37
Specific antibiotics				
% hospital days on vancomycin, mean	41	27	17	0.02

* One-way analysis of variance.

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