the first step in providing meaningful microbiological data and, ultimately, good patient care.

Saying that the diagnosis of UTI requires correlation of clinical presentation with laboratory results may summarize the unifying concept alluded to by Dr. Johnson [1] and expressed in our article [2]. The role of the laboratory is to provide accurate results of urine cultures, with quantification of bacterial growth and antimicrobial-susceptibility testing when applicable. In this vein, laboratorians use previously established criteria, however imperfect, to determine the extent of work up required for urine culture isolates. Ultimately, clinicians should determine the likelihood that UTI will require therapy on the basis of clinical and laboratory data.

Acknowledgment

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References

No Evidence for the Effectiveness of ClO2-Generating Gloves

Str—In his recently published article, Barza [1, p. 861] claimed that ClO2-generating gloves “are able to reduce counts of S. aureus, L. monocytogenes, E. coli, and S. Typhimurium substantially and quickly on the surface of the gloves” (emphasis ours). We regard this as a misleading claim that is based on insufficient experimental evidence and inappropriate statistical analysis. In fact, the small amount of experimental evidence presented by Barza [1] seems, rather, to indicate only a clinically insufficient reduction in bacterial counts, even after unrealistically long waiting times and the additional requirement of light exposure. Unfortunately, Barza’s unjustified claim has already found its way into local newspapers, such as the Hannoversche Allgemeine Zeitung (Hannover, Germany) [2].

To substantiate our appraisal of Barza’s claim, we refer the reader to the left-hand half of table 1 in his article [1], which gives log counts of bacteria for 2 control and 2 ClO2-generating gloves after various waiting times. Apparently, results in the same column refer to counts obtained at different times but on the same glove, and therefore the results cannot be regarded as being statistically independent. This invalidates the use of the Wilcoxon signed rank test or any other comparable test for analysis of these data.

Any such statistical test could be legitimately applied to the observations at a single fixed waiting time (e.g., 1 min), but that would not lead to a statistically significant result because of the tiny sample size. Furthermore, the observed reductions in counts of ~3 logs, whether statistically significant or not, are clinically insufficient, because alcohol-based hand hygiene already yields a reduction in the count of 5 logs after 30 s [3]. We fear that Barza’s suggestion could lead to very risky health care practices, because use of these special gloves can result in a false sense of security. Sufficient evidence for the use of alcohol-based hand rubs exists not only for laboratory outcomes, but also for clinical outcomes [4–6]. Therefore, the use of alcohol-based hand hygiene is the just recommendation [7].

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References

Reply to Mattner et al.

Str—Hand hygiene in clinical and food-handling environments always should be approached using complementary practices that include thorough washing of hands, use of alcohol-based hand rubs, and wearing of single-use gloves. At no time should only one of these practices be used alone. The ClO2-generating gloves described in my earlier article [1] could augment currently recommended practices and should provide additional pro-

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tection in situations in which users are not fully adherent to these recommendations. The gloves are not intended to replace standard hand hygiene but, rather, to supplement it.

Mattner et al. [2] are mistaken in inferring that the measurements over time given in table 1 (and in the other tables) were successive measurements of the same glove. As stated in the table notes, each measurement is the result for a single glove; no glove is represented more than once in the data. Accordingly, the Wilcoxon signed-rank test was appropriate for statistical analysis.

The determination of the “sufficiency” of the magnitude and rapidity of the effect of these gloves must be related to the environment of use. The experiments reported demonstrate the ability of the gloves to rapidly (i.e., within 2 min of donning) and significantly reduce high levels of contamination. That the technology is triggered by light is part of its beauty: the ClO₂ will be dissipated not while the gloves lie in the box but only when they are worn.

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Reference


Outbreak of Clostridium difficile Infection and Gatifloxacin Use in a Long-Term Care Facility

Sir—we read with interest the report by Gaynes et al. [1] describing a gatifloxacin-associated “epidemic” of Clostridium difficile-associated diarrhea (CDAD) in the Atlanta, Georgia Veterans Affairs Medical Center Long-Term Care Facility (LTCF). The report by Gaynes et al. [1] reminds the reader that CDAD is associated with several different antibiotics, not the least of which are the fluoroquinolones [2, 3]. However, to suggest that there is a higher incidence of CDAD with gatifloxacin therapy than with levofloxacin, on the basis of a retrospective analysis, is a conclusion made with greater confounding variables than supporting evidence. In addition, these findings are not consistent with previously published reports demonstrating the association of levofloxacin therapy with increasing incidences of CDAD [4, 5]. In fact, a recent analysis of cases of C. difficile infection at our institution found a statistically significant increase in the incidence of CDAD associated with the use of levofloxacin and third-generation cephalosporins, but not other fluoroquinolones [6]. Replacement of levofloxacin in the formulary with ciprofloxacin and gatifloxacin in 2000 resulted in a significant decline in the observed rates of C. difficile infection at our institution. Likewise, Changela et al. [7] conducted a cohort-controlled study to review the risk factors associated with CDAD at a Veterans Affairs medical center in Illinois, and they found antibiotic use to be significantly associated with C. difficile infection, with levofloxacin use being significantly with CDAD, compared with the cohort-control group. This is not to suggest that levofloxacin is the sole culprit causing CDAD; there are reports of CDAD that identify moxifloxacin use and ciprofloxacin use as causes, as well [8, 9].

We do know from the study of Gaynes et al. [1] that “a generalized cleaning of the LTCF was performed with a hypochlorite disinfectant during the period of 9–12 June 2002” just before switching the unit back to levofloxacin. Perhaps the decline in the rate of C. difficile infection was directly related to the sterilization of the LTCF and other infection control procedures implemented, rather than to the switch in antimicrobial therapy. To do a reasonable comparison between the fluoroquinolone “study periods,” the authors should have included a case-control study during the levofloxacin dosing period, as well, and a thorough review of all concomitant antibiotics each patient received during each study period. It also appears that the rate of C. difficile infection in the acute-care facility was increasing (there were no data before January 2001) and the rate of CDAD associated with gatifloxacin use in the LTCF was actually lower than the rate with levofloxacin reported in the acute-care facility at the same time (~1.3 vs. ~1.9 cases per 1000 patient-days, respectively). Because these patients were likely being transferred to and from the acute-care facility—certainly the LTCF patients shared diagnostic facilities with patients in the acute-care facility—the problem with C. difficile may have existed in the hospital and been spread to the LTCF, or vice versa. It would be important to note whether there was a trend in the incidence of CDAD at the both sites prior to January 2001 to determine whether it was indeed increasing?

Lastly, Gaynes et al. [1] do report that 25 (55%) of 45 of the isolates from the acute care facility and 2 (50%) of 4 of the isolates from the LTCF were the same type and were all resistant to fluoroquinolones. Does this finding imply that this indeed was an outbreak of a single strain? What methods were utilized to determine that all strains with identical susceptibility patterns were indeed all type A? Perhaps a more in-depth molecular analysis would have addressed many of the questions regarding the source of the strains and how infection-control and sterilization proce-