controversy. Previous trials have demonstrated that chlorhexidine or povidone-iodine preoperative showers are effective at quantitatively reducing skin colonization.[8-10] However, other trials have reported that preoperative antiseptic showers did not influence the incidence of surgical site infections.[11] Skin colonization plays an important role in surgical site infection.[9] Minimizing the risk of such infection is imperative, particularly in plastic surgery, since even minor infections can complicate the healing process and harm the cosmetic result. Given that the expected infection rates after clean elective procedures are 2% or less, our sample size did not allow us to determine the influence of preoperative chlorhexidine showers on the rate of infection.[2,10] However, considering the statistically significant reduction in skin colonization, we stand by our statement that "chlorhexidine showers before clean plastic surgical procedures involving the trunk...should be seriously considered in clinical practice."[2(p79)]

ACKNOWLEDGEMENTS

Potential conflicts of interest. All authors report no conflicts of interest relevant to this work.

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Infect Control Hosp Epidemiol 2009; 30:812-813

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Emerging Evidence for Seasonality of Gram-Negative Bacterial Infections

To the Editor—We read with great interest the article written by Perencevich and colleagues describing summer peaks in the incidence of hospital- and community-acquired infections due to several gram-negative bacteria. Specifically, higher rates of infection were described for *Acinetobacter baumanii*, *Pseudomonas aeruginosa*, *Enterobacter cloacae*, and *Escherichia coli* during summer months compared to winter months at the University of Maryland Medical Center. The authors reported increases in the incidence rate of infections in summer ranging from 12% for *E. coli* to 46% for *E. cloacae*. Increases in temperature were also independently associated with increases in the incidence of infection due to several organisms. The incidences of infection with *A. baumanii* and with *P. aeruginosa* were shown to increase by 17% with each 10°F increase in temperature during summer months. During winter months, *E. cloacae* infections increased by 9% with each 10°F increase in temperature.[1] The findings of this study are similar to those of a recently published study by Anderson et al describing seasonal variation in bloodstream infections (BSIs) due to *Klebsiella pneumoniae*.[2] This latter study included hospitals located on 4 continents (North America, Australia, Europe, and Asia) and reported increased rates of BSI due to *K. pneumoniae* during the warmest months of the year, ranging from 41% to 49%. Multivariable analysis revealed that both temperature and dew point (a marker for relative humidity) were predictive of increasing rates of *K. pneumoniae* BSI. In light of the findings by Anderson et al, we are writing to inquire whether Perencevich and colleagues examined rates of infection due to *K. pneumoniae*.

Perencevich et al studied all isolates recovered from wounds, respiratory specimens, and urine in calculating incidence rates of infection. It is likely that many of these isolates were colonizers rather than pathogens causing infection at these sites. Thus, the seasonal increases in incidence rates reported by Perencevich et al are likely to be overestimates of the true seasonal increases in clinically significant infection.

It would be interesting if the authors could reexamine seasonal incidence rates of infection with *E. coli*, *E. cloacae*, *A. baumanii*, and *P. aeruginosa* confining their analysis to blood culture or "clinically significant" isolates. In the study by Anderson et al, incidence rates of BSI caused by *Enterobacter* spp. were examined, but, in contrast to the findings of Perencevich et al, no seasonal variation was found. One explanation for this difference may be that seasonal increases in the incidence of BSI are associated only with proportionally larger increases in asymptomatic colonization and nonbacteremic infection. Colonization has been shown to precede clinical infection by *K. pneumoniae*.[3] Hospitalized patients who are colonized with *K. pneumoniae* have been shown to have a 4-fold increased risk of developing clinical infection.[4] If, as seems likely, similar observations also apply to *E. cloacae*, then the study by Anderson et al may have been underpowered compared with the study by Perencevich et al to detect a relatively small seasonal increase in the incidence of BSI with *Enterobacter* spp. We assume that the seasonal increase in BSI due to *K. pneumoniae* observed by Anderson et al would have been associated with a proportionally large seasonal increase in the rate of colonization and nonbacteremic infection with this species. It would be interesting to reexamine seasonal incidence rates of infection by *K. pneumoniae*, both by applying the approach used by Perencevich et al and also by confining another analysis to blood culture isolates only.

Ideally, future studies of seasonal changes in the incidence of gram-negative infections should be multicenter in design. We agree with Perencevich et al that such studies should assess the effects of environmental variables such as temperature and humidity. The studies by Anderson et al and Perencevich et al showed independent associations between physical environmental variables (such as temperature) and rates of infection. These findings suggest that seasonal variations in exposure to organisms present in the environment may explain seasonal changes in infection rates. Both Perencevich et al and Anderson et al cited studies that reported increases in the density of environmental contamination during summer for certain species of gram-negative bacteria.[5,6]

If seasonal increases in rates of infection are indeed a consequence of seasonal increases in the incidence of colonization,[7] seasonal changes in colonization rates may, in turn, result from seasonal fluctuations in exposure to these organisms in the environment. Potential factors leading to seasonal changes in environmental exposure include seasonal changes in human behavior[8,9] (eg, dietary habits), seasonal changes in the environmental ecology of gram-negative bacteria[5,6] (eg, increased concentrations in food and water sources), or a combination of these factors.

The observations made by Perencevich et al should be a stimulus for future research. For example, future studies could examine seasonal variation in the fecal carriage rate of *K. pneumoniae*, *A. baumanii*, *P. aeruginosa*, and *E. cloacae* and compare the genetic characteristics of colonizing strains to those of strains causing infection. Fecal strains of these organisms could also be compared to strains recovered from the environment. Furthermore, seasonal variations in fecal concentrations of these organisms could be correlated with seasonal changes in environmental concentrations and changes in temperature and humidity. Although the methods required to undertake such studies are tedious and imperfect, the implications could be far reaching. For example, if specific gram-negative infections fluctuate seasonally due to seasonal fluctuations in bacterial concentrations present in specific environmental reservoirs, this understanding could lead to the design of future infection prevention initiatives.

ACKNOWLEDGMENTS

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article.

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Infect Control Hosp Epidemiol 2009; 30:813-814

 0899-823X/2009/3009-0025$15.00. DOI: 10.1086/597506

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