they reported an amplified effect of direct antibiotic use in terms of an odds ratio of 2.26 (95% CI, 1.71–2.97); this analysis was based on a risk metric (see Table 1). We believe that this amplification can be explained analogously by a competing risk analysis. As in our analysis, we expect that patients with direct antibiotic use remain at risk longer in the hospital (ie, a reducing effect of antibiotic treatment on the discharge hazard occurs without HAI).

As correctly stated by Brown et al, when analyzing cohort studies with time-fixed or time-dependent exposures using the corresponding Cox proportional hazard model (approaches 1 and 2 in Brown et al[1]), patients were technically considered censored if they experienced discharge or death without infection. This analysis is valid, but we argue that it is incomplete if the impact of the exposures on discharge or death without infection is not studied. Therefore, an additional analysis regarding the competing events is necessary. This is done by performing additional Cox proportional hazard models with the same exposures but for the competing events as the outcome. Patients who acquire a HAI are then censored at the time of infection onset.[4]

Such competing risk analyses are not only very informative, they might also explain phenomena due to the 2 metrics. We believe that competing risk analyses are necessary since ignoring the potential effect of exposures on the competing events can easily lead to incorrect conclusions. For instance, a rate metric analysis showed no effect of burns on HAI in African children but a simple risk metric analyses showed a 3 times higher risk of HAI because children with burns remain at risk much longer in the hospital.[3] The type of metric highly matters and influences the conclusion. Thus, only the use of both metrics can provide a complete picture in multivariate analyses of HAI risk factors.[4,5] However, in the presence of time-dependent exposures, the rate metric approaches are very suitable,[6] but risk metric approaches have still challenging limitations in their interpretation.

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Martin Wolkewitz, PhD

Affiliations: Center for Medical Biometry and Medical Informatics, Institute for Medical Biometry and Statistics, Medical Center University of Freiburg, Germany.

Address correspondence to Prof. Dr. Martin Wolkewitz, Stefan-Meier-Str. 26, 79104 Freiburg, Germany (wolke@imbi.uni-freiburg.de).

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Reply to Wolkewitz: When to Use Cumulative Risk-Based Versus Rate-Based Approaches in the Analysis of Hospital-Acquired Infection Risk Factors? That Depends on the Question

To the Editor—We thank Dr. Wolkewitz for his thoughtful comments and clear breakdown of cumulative risk-based and rate-based measures of association in hospital acquired infection (HAI) research. We agree that a thorough understanding of the distinction between rate-based and cumulative risk-based metrics is essential for researchers performing studies of HAI risk factors.

Another way of thinking about this distinction is through the lens of the study question, which is often either etiologic or prognostic in nature.[1] The objective of an etiologic research question is to assess the causal association between a risk factor and a given outcome. That is, if a given exposure were introduced experimentally, would a given patient be more or less likely to experience the outcome.[2] On the other hand, prognostic research aims to predict the probability that a patient subgroup experiences an outcome on or before a given time point in a hospital stay, irrespective of whether a given risk factor caused an increased rate of disease.

In HAI research, patient subgroups with longer hospital stays may be more likely to develop an HAI during a given stay *only* because of the longer average duration of their stay. Whereas a risk-based approach would capture this as a difference in cumulative risk of HAI, a rate-based approach would find that the rate of HAI is no different. Epidemiologists interested in questions regarding etiology may be more likely

to gravitate toward rate-based approaches, which have been subject to criticism by advocates of causal inference methods,[3] while clinicians interested in prognostic questions may be more likely to veer toward cumulative risk-based approaches. Because of our primary interest in developing strategies for the prevention of HAI, which exclude modifying length of hospital stay, our research has tended to focus on rate-based approaches.[4,5]

In certain circumstances, a comparison of each approach may certainly be useful, while in others, it may not be worth the additional analytic burden. What is important is to understand and interpret the insights derived from cumulative risk-based and rate-based approaches correctly; to conflate the 2 approaches is to muddy the epidemiologic waters.

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Kevin A. Brown, PhD;[1,2]
Nick Daneman, MD, MSc;[3]
Vanessa Stevens, PhD;[4,5]
Tom H. Greene, PhD;[6]
Paul Arora, PhD[2,7]

Affiliations: 1. Public Health Ontario, Toronto, Canada; 2. Division of Epidemiology, Dalla Lana School of Public Health, University of Toronto, Canada; 3. Sunnybrook Health Sciences Center, University of Toronto, Canada; 4. VA Salt Lake City Health Care System, Salt Lake City, Utah, United States; 5. Department of Pharmacotherapy, University of Utah, Salt Lake City, United States; 6. Division of Epidemiology, University of Utah, Salt Lake City, United States; 7. Center for Global Child Health, The Hospital for Sick Children, Toronto, Canada.

Address correspondence to Kevin A. Brown, Public Health Ontario, Toronto, Canada (kevin.brown@oahpp.ca).

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Surveillance for Ventilator-Associated Pneumonia: Can We Apply Centers for Disease Control and Prevention–National Healthcare Safety Network 2013 Definitions for All Settings?

To the Editor—In the present journal, we read the article by Greene et al[1] about the influence of Centers for Disease Control and Prevention–National Healthcare Safety Network (CDC-NHSN) 2015 definitions for catheter-associated urinary tract Infection surveillance with great interest. Here, we share our experience of using CDC-NHSN 2008 and 2013 criteria for ventilator-associated pneumonia (VAP) surveillance at different intensive care unit (ICU) settings.

VAP is one of the most important problems in ICUs. Adequate surveillance of VAP is critical in order to introduce effective control measures early. The surveillance criteria for VAP was released by CDC-NHSN in 2008 and 2013.[2,3] The 2013 criteria was based on the worsening pulmonary functions. The worsening oxygenation—as increase in positive end-expiratory pressure and fraction of inspired oxygen—was termed ventilator-associated condition. When an abnormal temperature or white blood cell count and new antibiotics are added to a ventilator-associated condition, this condition is described as infection-related ventilator-associated complication. Diagnosis of possible VAP requires detection of specific microbioogic etiology in addition to an infection-related ventilator-associated complication. From January 1, 2013, through March 30, 2015, we adapted CDC-NHSN 2013 definitions for VAP surveillance; however, we observed a huge difference between the number of patients with a clinical diagnosis of VAP versus the VAP rate detected by CDC-NHSN 2013 criteria, pariticularly in the surgical ICUs. Then, we decided to use CDC-NHSN 2008 criteria for VAP surveillance to understand the role of definitions in the rate of the rapidly changed VAP rate.

Our hospital is a 700-bed tertiary center. A patient-based infection control program has been set up more than 20 years. An infection control nurse daily visits all patients hospitalized in the ICU to detect ICU-acquired infections. All surveillance data are periodically discussed with the infection control doctor, who is an infectious diseases physician. There are 3 surgical ICUs with 42 beds (general surgery with 9 beds, cardiothoracic surgery with 22 beds, and neurosurgery with 11 beds), and a medical ICU (MICU) with 9 beds in our adult hospital. In surgical ICUs, there are no intensivists and all patients are followed by individual surgical teams who operate on the patients. Owing to lack of adequate number of staff, ventilator parameters are not recorded properly and even sometimes cannot be managed according to the actual clinical condition of the patients. Diagnostic tests such as complete blood count or cultures from respiratory tract and blood are delayed because of shortage of well-trained staff in surgical