

fections. However, we are seriously concerned that the lack of molecular methods to identify the different genospecies of *Acinetobacter* in this study makes the conclusions doubtful.

A. baumannii is just one genospecies of the group *Acinetobacter calcoaceticus*–*A. baumannii* complex (ACB complex), which also includes genospecies *A. calcoaceticus*, *A. nosocomialis*, and *A. pittii*.² As they are phenotypically similar and difficult to distinguish using routine laboratory methods, they have been proposed as a group. After the introduction of molecular methods to accurately identify each genospecies, the clinical characteristics of each have been clarified, and it has been better realized that every genospecies has its own distinct features. For example, in the latest study by Lee et al.,³ patients with *A. baumannii* pneumonia were more likely to have abnormal hematological findings, lobar pneumonia, significantly higher Acute Physiology and Chronic Health Evaluation II scores, and higher mortality than those with *A. nosocomialis* pneumonia. Thus, they concluded that *A. baumannii* and *A. nosocomialis* nosocomial pneumonia are 2 distinct clinical entities.

In conclusion, we suggest that molecular methods to precisely identify the ACB complex should be conducted in studies of *A. baumannii* infections.

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Shih-Yang Su, MD;¹ Chien-Ming Chao, MD²

Affiliations: 1. Department of Emergency Medicine, Tainan Municipal Hospital, Tainan, Taiwan; 2. Department of Intensive Care Medicine, Chi Mei Medical Center, Liouying, Tainan, Taiwan; and Department of Nursing, Min-Hwei College of Health Care Management, Tainan, Taiwan.

Address correspondence to Chien-Ming Chao, MD, Department of Intensive Care Medicine, Chi Mei Medical Center, No. 201, Taikang, Taikang Vil., Liouying Dist., Tainan City 736, Taiwan (R.O.C.) (ccm870958@yahoo.com.tw).

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Reply to Su and Chao

To the Editor—We appreciate the interest of Su and Chao¹ in our study.² As *Acinetobacter calcoaceticus*, *Acinetobacter baumannii*, *Acinetobacter* genomic species 3, and *Acinetobacter* genomic species 13TU are very closely related, it has been proposed to refer to these species as the *A. calcoaceticus*–*A. baumannii* complex (ACB complex). We agree with the comment that molecular methods to precisely identify the ACB complex should be conducted in studies of *A. baumannii* infections. Since the clinically relevant members of the ACB complex cannot be separated by currently available commercial identification systems, such as the Vitek 2, Phoenix, and Microscan systems, *A. baumannii* isolates in our study represent the ACB complex.² Species identification with commercial identification systems that are currently used in clinical microbiology laboratories remains problematic, and molecular methods have been developed and validated for identification of *Acinetobacter* species.³

Given that *A. baumannii*, *Acinetobacter* genomic species 3, and *Acinetobacter* genomic species 13TU share important clinical and epidemiological characteristics, the need for species identification of the ACB complex in clinical microbiology laboratories is questionable.^{3,4} Moreover, *A. calcoaceticus* is the environmental species that has frequently been recovered from soil and water, and the designation “ACB complex” may be misleading and not appropriate if used in a clinical context.³ The majority of studies that have addressed epidemiological and clinical issues related to *Acinetobacter*, including ours, have not employed identification methods for the ACB complex.^{2,3} However, further clinical studies using proper methods for species identification of *Acinetobacter* are warranted to increase our knowledge of the epidemiology, pathogenicity, and clinical implications of the various species of this diverse genus.

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Cheol-In Kang, MD;¹ Seung Ji Kang, MD²

Affiliations: 1. Division of Infectious Diseases, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea; 2. Department of Internal Medicine, Chonnam National University Medical School, Gwangju, Republic of Korea.

Address correspondence to Cheol-In Kang, MD, Division of Infectious Diseases, Samsung Medical Center, Sungkyunkwan University School of

Medicine, Irwon-ro 81, Gangnam-gu, Seoul 135-710, Republic of Korea (collacin@hotmail.com).

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Evaluation of the Reporting Validity of Central Line–Associated Bloodstream Infection Data to a Provincial Surveillance Program

To the Editor—Periodic evaluation of the validity of data submitted to regional and/or national central line–associated bloodstream infection (CLABSI) surveillance programs is crucial to ensure their scientific credibility and to identify methodological problems.^{1–3} In 2003, the Surveillance Provinciale des Infections Nosocomiales–Bactériémies Associées aux Cathéters Centraux (SPIN-BACC) program was launched in the province of Quebec with the purpose of providing provincial benchmarks and data for the planning of provincial infection control interventions.^{4,5} Given its importance, we aimed to evaluate the accuracy of CLABSI reporting to SPIN-BACC.

We included 14 SPIN-BACC intensive care units (ICUs) that had reported 3 or more CLABSIs during at least 11 consecutive 4-week periods between April 1, 2008, and March 31, 2009. The SPIN-BACC surveillance methods have been described in detail elsewhere.^{6,7} This project was approved by the McGill University Institutional Review Board and by the directors of professional services of all participating institutions. Participating ICUs provided a list (data set 1) containing all CLABSIs (see definitions in Table 1) reported to SPIN-BACC for the year under study, as well as a second list (data set 2) of all ICU BSIs that occurred during the same period but were not classified as CLABSI by the local surveillance teams. We selected a random sample of cases from data sets 1 and 2, stratified by ICU and proportional to the number of CLABSIs reported to SPIN-BACC during the study period.

Two previously trained independent researchers (P.S.F. and I.R.) blinded to patients' CLABSI status reported to SPIN-

BACC reviewed the included charts. The reviewers' adjudication of CLABSI status was defined as our reference standard. In case of discrepancies between the 2 reviewers, the opinion of a third researcher (C.Q.), an infectious disease/medical microbiologist specialist with expertise in CLABSI surveillance, was sought.

As measures of validity, we computed sensitivity and specificity and their respective exact binomial 95% confidence intervals (CIs). Sample size (90 charts) was calculated using the width of the 95% CI (75%–95%) that we aimed to obtain for a SPIN-BACC hypothesized sensitivity of 85% (similar to the sensitivity published by the National Nosocomial Infections Surveillance [NNIS] system in 1998).⁸ To achieve the necessary numbers of true positives (45) and true negatives (45), we reviewed a total of 109 charts.

Data sets 1 and 2 included a total of 138 reported CLABSIs (68% of cases reported and 52% of catheter-days in 2008–2009) and 419 non-CLABSI cases, respectively. We randomly sampled 57 reported CLABSI cases and 52 non-CLABSI cases to be reviewed. We identified 5% more CLABSI cases (60) and 6% fewer non-CLABSI cases (49) than were reported. Overall, 21% of the charts (23) needed to be discussed for a consensus to be reached.

Of the 57 CLABSI cases reviewed, only 4 (7%) were classified as false positives. Of the 52 non-CLABSI cases that were reviewed, 7 (13%) were classified as false negatives. False-positive and false-negative cases were equally distributed among hospitals. Calculated sensitivity and specificity were 88% (95% CI, 77%–95%) and 92% (95% CI, 80%–98%), respectively.

Of the 7 false-negative cases, 3 (43%) were found to be CLABSI according to NNIS criterion 2b, 2 (29%) according to criterion 2a, and 2 (29%) according to criterion 1.⁶ Of the 4 false-positive cases, 2 (50%) did not fulfill NNIS criteria for bloodstream infection, and 2 (50%) had another source of infection.

Our study showed that CLABSI data reported by the ICUs participating in SPIN-BACC are valid. Our estimated sensitivity compares to the one reported by the NNIS system (85%) in 1998 and is above the sensitivity reported by KISS (Germany, 66%) and NSIH (Belgium, 59.3%).⁸ However, our specificity is still mildly lower compared with these national programs (92% vs 98.3%–99.4%).

Compared with other jurisdiction-wide programs, SPIN-BACC results are superior. Sensitivity and specificity reported by Backman et al⁷ (Connecticut, United States) were 48% and 99%, respectively, and McBryde et al⁹ (Victoria, Australia) reported 61% and 70%, respectively. In both cases, the low sensitivity was attributed to misinterpretation of NNIS criterion 2b for CLABSI.⁶ Although we used this criterion for CLABSI diagnosis until 2010 and, thus, during the study period, its interpretation was not problematic, as only 4 (10.2%) of the 39 criterion 2b CLABSIs were misclassified.

We believe our results are a reflection of the use of sound surveillance methods, which are based on the NHSN system, the effectiveness of the training offered to the participants, and