Correspondence to: O. Joseph Bienvenu, MD, PhD, Johns Hopkins University School of Medicine, 600 N Wolfe St, Meyer 115, Baltimore, MD 21287; e-mail: jbi@jhu.edu

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Physical Activity vs Psychomotor Activity
Prognostication of COPD

To the Editor:

We read with interest the recent article by Nguyen et al1 in CHEST (July 2013), which demonstrated novel findings. However, we would like to draw attention to certain conceptual issues that could effectively question the crux of those findings. As the authors mentioned, objectively measured physical activity is an excellent predictor of prognosis for patients with COPD. However, physical activity is distinct from psychomotor activity. Psychomotor activity is defined as motor/physical activity that is secondary to or dependent on a psychic component and is mostly non-goal-directed. For example, manic, psychotic, and anxious patients would demonstrate increased psychomotor activity. This is generally state-dependent, that is, it lasts during the course of psychiatric symptoms and normalizes on effective treatment.

Furthermore, comorbid anxiety symptoms have never been implicated as a good prognostic factor in either COPD or any other chronic illnesses, to our knowledge. If anything, mild anxiety symptoms predict positive outcome negating a sedentary lifestyle; it has to be trait anxiety symptoms that refer to those individuals with anxious predisposition or temperament from adolescence. Considering that the Nguyen et al1 study used a cross-sectional design, state and trait anxiety symptoms could have been discerned using an anxiety inventory like the State-Trait Anxiety Inventory. An anxious state resulting in increased step counts per day is expected and logical even in a patient with COPD. This cannot be erroneously interpreted as an increase in physical activity and predictor of good prognosis in patients with COPD. The authors propose no strong hypothesis to explain the better prognosis of mild anxiety symptoms. In fact, we would consider anxiety symptoms as a confounding factor in assessment of physical activity in patients with COPD. A longitudinal study of physical activity in patients with COPD accounting for anxiety or use of an anxiety inventory like the State-Trait Anxiety Inventory (additionally in a cross-sectional study) would better clarify the picture.

Physical Activity vs Psychomotor Activity
Prognostication of COPD

To the Editor:

We read with interest the recent study by Cillóniz et al1 in CHEST (September 2013), in which several associations between the cause and outcome of community-acquired pneumonia (CAP) were reported in patients > 65 years of age, studied over a period of 12 years. The authors mention that the nonhomogeneous assessment of microbial cause is a potential limitation. In our opinion, this is an understatement of the bias that may have resulted from this approach, and this limitation precludes the conclusion that was reached.

The most important finding of the study was that the presence of comorbidities was associated more with potential multidrug-resistant (MDR) pathogens as a cause of CAP than was age. Thus, the authors concluded that “comorbidities rather than age should be considered in the selection of antibiotic treatment.” However, the outcome (in this case, a microbial cause) was not assessed uniformly in all included patients, which is a well-known cause of bias in predictive research. Apparently, microbial testing was left to the discretion of the treating physician. This is at least suggested by the pattern of microbial testing: serologic tests in 1,537 patients (44%), sputum cultures in 1,913 patients (54%), and blood cultures in 2,753 patients (78%). By ignoring the fact that microbial tests could be different across patients, the authors implicitly assumed that the tests were missing at random. However, in clinical practice, the choice for microbial testing is often influenced by patient and disease characteristics. Therefore, more extensive diagnostic testing in patients with comorbidities may well explain the higher prevalence of potential MDR pathogens in this patient group. Demonstration of comparable diagnostic procedures in patients with and

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Predicting Community-Acquired Pneumonia Etiology

To the Editor:

We read with interest the recent study by Cillóniz et al1 in CHEST (September 2013), in which several associations between the cause and outcome of community-acquired pneumonia (CAP) were reported in patients > 65 years of age, studied over a period of 12 years. The authors mention that the nonhomogeneous assessment of microbial cause is a potential limitation. In our opinion, this is an understatement of the bias that may have resulted from this approach, and this limitation precludes the conclusion that was reached.

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Affiliations: From the Department of Psychiatry (Dr Gnanavel), All India Institute of Medical Sciences, New Delhi; and Physiology and Cardiopulmonary Rehabilitation (Dr Robert), Vallabhbhai Patel Chest Institute.

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Correspondence to: Sundar Gnanavel, MD, Department of Psychiatry, All India Institute of Medical Sciences, New Delhi, Ansari Nagar, New Delhi 110029, India; e-mail: sundar221103@yahoo.com

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without comorbidities would reduce the likelihood of bias, although even that would not rule out bias completely. It is hardly possible to adjust for this analytically, even when information on why certain microbial tests were (or were not) obtained was available.

Because of global aging and the corresponding increase in the number of comorbidities, which will also occur in patients with CAP, a recommendation to include MDR antibiotic coverage based on comorbidities inevitably increases antibiotic use. In an era of increasing antimicrobial resistance and a high prevalence of nosocomial Clostridium difficile infection, such recommendations should be based on unbiased results. Because the cause of CAP cannot be reliably determined by clinical data, empirical antibiotic therapy should be guided by local epidemiologic data, the site of admission, and prior microbial culture results, rather than by the presence of comorbidities.

Douwe F. Postma, MD
Cornelis H. van Werkhoven, MD
Jan Jefriok Oosterheert, MD, PhD
Marc J. M. Bonten, MD, PhD
Utrecht, The Netherlands

Affiliations: From the Julius Center for Health Sciences (Drs Postma, van Werkhoven, and Bonten), the Department of Internal Medicine and Infectious Diseases (Dr Oosterheert), and the Department of Medical Microbiology (Dr Bonten), University Medical Center Utrecht.

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Correspondence to: Douwe F. Postma, MD, University Medical Center Utrecht, Julius Center for Health Sciences, PO Box 85590, 3508 GA Utrecht, The Netherlands; e-mail: df.postma@umcutrecht.nl © 2013 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details. DOI: 10.1378/chest.13-1756


Response

To the Editor:

We appreciate the interest of Dr Postma and colleagues in our recently published article in CHEST.1 In the Discussion section of our article, we already acknowledged that a potential limitation of the study is not having 100% microbial etiologies. This study comes from our database, with information collected prospectively over 12 years from patients with community-acquired pneumonia (CAP) who had a well-defined diagnostic and treatment protocol from the very beginning; we disagree with the suggestion that “apparently, microbial testing was left to the discretion of the treating physician.”

We are not aware of any CAP study having >75% etiologies. This is impossible for the following reasons: (1) Blood cultures are poorly sensitive, (2) sputum is often unavailable or contaminated, (3) bronchoscopies cannot be performed in many patients, (4) patients are lost in follow-up (for serologies), and (5) polymerase chain reaction techniques are still not available for all microorganisms and all hospitals. Having said that, we believe that our results are very representative of CAP microbiology in patients older than 65 years of age, given the high number of patients included (2,149).

We do not think that our recommendation will increase the number of antibiotics administered. In fact, we believe the opposite will occur because we restricted our recommendations to (and only included) patients older than 65 years of age.

Finally, we doubt sincerely that site of care is the best approach for empirical treatment in CAP. In fact, patients in the ward who should be admitted to the ICU (particularly with Pneumonia Severity Index V) from the beginning can frequently be observed. Instead, we believe that we have to move forward to treat patients empirically according to severity scales.2 We thank Dr Postma and colleagues again for their interest in our article.

Catia Cillóniz, PhD
Antoni Torres, PhD, FCCP
Barcelona, Spain

Affiliations: From the Department of Respiratory Diseases (Drs Cillóniz and Torres), Instituto del Tórax, Hospital Clínico de Barcelona, Institut d’Investigacions Biomèdiques August Pi i Sunyer, Universitat de Barcelona; and Centro de Investigación Biomédica en Red Enfermedades Respiratorias (Drs Cillóniz and Torres).

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Correspondence to: Antoni Torres, PhD, Department of Pneumology, Hospital Clinic of Barcelona, Calle Villarroel 170, Barcelona, Spain; e-mail: aторres@clinic.ub.es © 2013 American College of Chest Physicians. Reproduction of this article is prohibited without written permission from the American College of Chest Physicians. See online for more details. DOI: 10.1378/chest.13-1832

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Sarcoidosis, Fatigue, and Sleep Apnea

To the Editor:

We read with interest the article by Patterson et al1 in a recent issue of CHEST (June 2013) that demonstrated a high proportion of daytime sleepiness using the Epworth Scale in patients with sarcoidosis. The authors concluded that sleepiness could be a contributing factor to fatigue in some of these patients. The authors are careful to point out that daytime sleepiness is not the same as fatigue, as emphasized by Brown2 in the accompanying editorial. We recently published a double-blind, placebo-controlled, crossover study examining the treatment of sarcoidosis-associated fatigue

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Correspondence