

# Correspondence

## In Memoriam: John P. Quinn, MD

TO THE EDITOR—With great sadness, we mark the passing of our cherished colleague and friend, John P. Quinn, MD (Figure 1). John leaves behind a beloved wife (Dr Maria Virginia Villegas), a daughter (Veronica), 3 sons (John Jr., Robert, and Christopher), and 2 grandchildren. We in the field of infectious diseases will miss his friendship, his smile, his humor, his embrace, and his warmth. His absence will be felt profoundly because it occurs at a time when talent and passion dedicated to the study of drug-resistant organisms are most urgently needed.

John fiercely embodied the qualities we all strive to achieve. He was a gifted clinician and a dedicated and insightful researcher who strove to translate the lessons learned in the laboratory to the patient's bedside. John's contributions led to the increasing recognition of the crisis of antibiotic resistance in gram-negative bacteria. He leaves a great void in our field.

The eldest among 8 brothers, John grew up in Chicago's South Shore neighborhood (the "South Side"). He pursued an academic career at his city's venerable institutions, including Rush Medical College, Loyola University, Michael Reese Hospital, University of Illinois at Chicago, and Cook County Hospital, eventually attaining the rank of Professor of Medicine and Vice-Chairman. Like a true son of Chicago, all his accomplishments were earned the "hard way." He worked evenings and weekends to support his medical school education as he raised a young family.

Very early in his career, John established himself as a pioneer who led the way in the field. Soon after imipenem/cilastatin was introduced in 1985, John presciently reported therapeutic failures in the treatment of *Pseudomonas aeruginosa*. Moreover, he detailed how penetration of imipenem through the outer membrane of *P. aeruginosa* was prevented by changes in the porin protein OprD2

[1, 2]. Later, he also studied carbapenem-resistant *Serratia marcescens* isolates from across the United States that harbored SME, a serine carbapenemase [3]. With these sentinel discoveries, John heralded the emergence of resistance to carbapenems decades before the current era of "nightmare bacteria."

John also observed how resistance during treatment with ceftazidime can emerge in *Enterobacter cloacae*; here, he examined the role of  $\beta$ -lactamase induction [4]. Also, he was quick to grasp the significance of the unusual susceptibility pattern of 2 isolates of *Klebsiella pneumoniae* that demonstrated resistance to ceftazidime. He identified TEM-10 as the mechanism of resistance, which, together with the subsequent report of TEM-26, were 2 of the first occurrences of plasmid-mediated extended-spectrum  $\beta$ -lactamase (ESBL) in North America [5, 6]. Since then, he advocated for the judicious use of expanded-spectrum cephalosporins to limit selective pressure and the spread of ESBLs. After these findings, John earned for himself the moniker of "ESBLman," which he later adopted as his email address.

Working with others, John demonstrated the importance of detecting  $\beta$ -lactamase-mediated resistance to avoid therapeutic failures, and recognized that facilities such as nursing homes can be "reservoirs of resistance"—notions that continue to enjoy currency in the contemporary epidemic of carbapenem-resistant organisms [7, 8]. John also chronicled the eroding reliability of fluoroquinolones in intensive care units due to the emergence of resistance associated with the increased use of this class of drugs [9]. From his vantage point in Chicago, John described the first nosocomial outbreak of *P. aeruginosa* producing



**Figure 1.** John P. Quinn in the heights of Machu Picchu, Peru, 2010. Picture provided by Dr Maria Virginia Villegas.

VIM-2 metallo- $\beta$ -lactamase (MBL) in the United States, revealing that this country is vulnerable to MBLs [10]. He warned about the threat posed by mobile genetic elements containing MBLs, especially because they may not be readily detected. The need for increased vigilance to curb the spread of MBLs is attested by the recent importation of New Delhi metallo- $\beta$ -lactamase-1 (NDM-1) into the United States.

John leveraged his expertise and reputation to establish collaborations with scientists from around the world. For instance, at an early stage, he and investigators at the Peking Union Medical College documented the predominance of CTX-M-type enzymes in China, a key piece in the global epidemiology of ESBLs [11]. More recently, John authored a definite review on carbapenem-mediated resistance in *K. pneumoniae* together with scientists from 14 different countries and 5 continents [12]. The most meaningful of these partnerships was with Dr Villegas from CIDEIM (Centro Internacional de Entrenamiento e Investigaciones Medicas) in Cali, Colombia. Together, they formed a scientific collaboration that extended for more than a decade and led to important insights into the emergence of antibiotic resistance in South America and its significance in the global context [13–16]. At the same time, John and Dr Villegas created research opportunities, scientific capacity, and awareness in antimicrobial resistance throughout the entire region.

At the culmination of his academic career, John joined the pharmaceutical industry in order to seek practical solutions to the crisis caused by drug-resistant bacteria. As a physician, John saw firsthand the impact that these pathogens wrought; he also recognized that, contrary to the past, new molecules were not being added to the therapeutic armamentarium. He believed that the discovery and development of new antibacterial agents was a “moral obligation.” Working for Pfizer and later for AstraZeneca, he was

recognized as someone who could build bridges between scientists and clinicians and play a critical role in the development of new anti-infectives. To Pfizer’s and AstraZeneca’s credit, each recognized John’s intellectual depth, scientific credentials, and clinical talents. John’s vision was to make a difference in the treatment of bacterial infections and to persevere in seeking fundamental answers regarding mechanisms of antibiotic resistance. This was the sentiment he unequivocally voiced in the public television program “Frontline,” broadcast 4 days after his death [17].

Clear lessons emerge from examining John’s life and career, and these constitute his legacy: his keen observations and prepared mind, his interest in forming and sustaining collaborations, his global outlook and reach, his ability to inspire and support young investigators, and the resolution of his beliefs and courage to conduct himself accordingly. Keeping faithful to these lessons will be key in our society’s efforts to preserve the dividend of antibiotics to global health. John had a lasting impact on our field, and we must ensure that his lessons survive him by passing them to future generations. John will always be in our hearts and minds, and we only hope to emulate him by moving forward with fortitude and conviction. A few days before his death, he told one of us, with a smile on his face, “I am ready to go”; he knew his legacy will continue.

## Note

**Potential conflicts of interest.** All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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