Correspondence

Commentary on Fatalities in the 1918–1919 Influenza Pandemic

To the Editor—After the first pandemic in the age of modern virology in 1957, a symposium entitled “Virus Virulence and Pathogenicity” that was devoted chiefly to influenza was held at the Ciba Foundation headquarters in London in 1959. At this meeting, I presented a paper entitled “The Severity of Influenza as a Reciprocal of Host Susceptibility” [1], in which I described the experience at New York Hospital, where most fatalities occurred either in patients previously compromised by cardiopulmonary problems related to rheumatic heart disease or physiologic changes in the third trimester of pregnancy or in patients with secondary bacterial pneumonia [2]. This experience was similar to that of other teaching hospitals at the time. I was struck by the fact that, in my laboratory, influenza virus isolated from persons with fatal cases differed in no discernible way—including virulence in mice—from virus recovered from those with nonfatal cases of brief duration [3].

The best documented fact of 1918–1919 is that all but a few who died of “influenza” died with, if not of, secondary bacterial pneumonia. I wrote at the time, “The virus of 1918 was undoubtedly uniquely virulent, although most patients experienced symptoms of typical influenza with a 3- to 5-day fever followed by complete recovery. Nevertheless, although diagnostic virology was not yet available, bacteriology was flourishing and many careful post-mortem examinations of patients by academic bacteriologists and pathologists disclosed bacterial pathogens in the lungs” [4, p. 9]. In fact, it was a time of high bacterial prevalence and epidemicity, during which army camp fatalities in patients with measles were not uncommon.

In their recent report in the Journal, Morens et al. [5] returned to 1918–1919 lung tissue samples from patients with fatal influenza and found “severe changes indicative of bacterial pneumonia” (p. 962). In essence, this is confirmatory of the histopathology described by skilled pathologists at the time. Although in the present-day analyses bacteria in large numbers were seen in some lung sections, apparently none were cultivatable and definitively identifiable. Nevertheless, the authors have provided a valuable service in allowing us to stand on firmer ground in predicting the need for adequate stockpiles of antibiotics in anticipation of future pandemics. The main lesson of the 1957 pandemic should be stressed as well—that influenza virus infection without bacterial coinvaders can kill hosts who are physiologically compromised.

Because my earlier published studies [1, 4] provide additional evidence of influenza fatalities in the absence of bacterial coinfection, they should be added to Morens et al.’s bibliography of 95 publications in guiding future action in pandemic planning.

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References


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Reply to Kilbourne

To the Editor—We thank Dr. Kilbourne [1] for emphasizing that our findings on the bacterial causes of death in the 1918–1919 influenza pandemic [2] are consistent with his published findings from the 1957 pandemic. Indeed, Dr. Kilbourne’s important body of work, as well as private discussions held over several decades’ time, have greatly influenced each of us. We thus prominently cited in our article one of Dr. Kilbourne’s most important publications from the 1957 pandemic (our reference 59 and reference 2 in his letter) and included the others in the associated online bibliography [2].

In both pandemics, the great majority of persons who experienced symptomatic influenza virus infection (97–99%) had mild and uncomplicated clinical courses in no way different from those of seasonal influenza seen today. Dr. Kilbourne refers to a unique viral virulence of the 1918–1919 influenza strain. We used the term “pathogenicity” and believe that the pathogenicity of the 1918 virus was expressed in its ability to potentiate the