In Response: We thank Leung and Cowling (*1*) for taking time to comment on our article (*2*). One problem with the random effects model is the rapid decline in performance of the model as the heterogeneity within studies increases. Extensive heterogeneity for asymptomatic (I^2 = 97%; T^2 = 0.31) and subclinical (I^2 = 97%; T^2 = 0.45) infection was identified. However, the model selected to pool the prevalence estimates—inverse variance heterogeneity—maintains its coverage at the nominal level, even when large heterogeneity is present (*3*).

Regarding inclusion criteria, we elected to review all publications detailing asymptomatic influenza prevalence in humans, as is made clear from the original article's title onward. This method included experimental studies, as well as newly emerging zoonotic strains. We note further that the 2 experimental studies in our review had subclinical influenza infection levels within the range identified in the pooled estimate of the metaanalysis (43.4%, 95% CI 25.4%–61.8%). Also, because antibody titers can vary drastically with technique used and between laboratories, we used the antibody titer threshold defined by each individual study.

The results/conclusions from the study published by Leung et al. (*4*) cannot be compared with those reported in our meta-analysis (*2*) for 2 important reasons. First, the case definition for asymptomatic was different; Leung et al. grouped patients without signs and symptoms (asymptomatic in our metaanalysis) with patients that did not fulfill the criteria of influenza-like illness (subclinical in our meta-analysis). We explained in our article why pooling asymptomatic and subclinical cases is inappropriate and likely to provide spurious results. As an example of how the case definition can affect the results, Pascalis et al. found that in the same group of patients, 30.6% had subclinical infection (not fulfilling criteria for influenza-like illness) but only 1.6% had no symptoms at all (*5*). Second, the number of studies included in the 2 meta-analyses was different: our comprehensive review comprised 55 studies, whereas Leung et al. included a subset of only 30 studies pertaining specifically to seasonal influenza. The different studies included and different meta-analytical methods unsurprisingly yielded different outcomes.

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Mycobacterium lepromatosis Lepromatous Leprosy in US Citizen Who Traveled to Disease-Endemic Areas

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To the Editor: The article by Virk et al. (*1*) highlighted that a person can acquire *Mycobacterium lepromatosis* infection without exposure to a person infected with leprosy or to known vectors during short stays (2 trips of 7 days each over 3 calendar years) in Mexico. The authors then concluded that *M. lepromatosis* lepromatous leprosy is a travel-related hazard for travelers to Mexico or other disease-endemic areas. We note that the exact source of acquiring the *M. lepromatosis* infection by the patient in this study was entirely uncertain, and experimental evidence was not enough to prove *M. lepromatosis* to be a travel-related hazard.

In contrast, Jessamine et al. (*2*) reported *M. lepromatosis* infection and leprosy-like illness in a patient in Canada who had no history of contact or travel to leprosy-endemic areas. Jessamine et al. indicated that transmission dynamics of *M. lepromatosis* infection is complex, and undiscovered mechanisms or unknown reservoir interactions may exist in such areas of nonendemic regions. Previous studies have also reported the roles of subclinical cases and environmental reservoirs in the transmission of leprosy (*3*,*4*). However, Virk et al. have not disentangled other possible sources (existence of unrecognized subclinical cases, contact with

hidden leprosy cases, and environmental reservoir) of prolonged exposure of *M. lepromatosis* to the study patient in his vicinity. Thus, the assertion of Virk et al. (*1*) that United States citizens can acquire *M. lepromatosis* when traveling to Mexico or other leprosy-endemic areas as tourists is misleading and demands extensive research to prove it.

In addition, it is intriguing to note that host genetic determinants can influence the acquisition and onset of leprosy (*5*). Therefore, the inference of a single case study cannot be generalized for all citizens of the United States. The data from these reports suggest that the epidemiologic studies of leprosy in nonendemic areas should consider travel history to delineate this issue.

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Spread of Plague by Respiratory Droplets or Ectoparasites

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To the Editor: Drancourt and Raoult (*1*) have emphasized the risk of overestimation of pneumonic plague contagion by respiratory droplets and hypothesize that only transmission of *Yersinia pestis* by ectoparasites, such as lice and fleas, by close contact with infected humans can sustain outbreaks and epidemics. The outbreak of pneumonic plague in Madagascar in 2017 (*2*) reminds us that plague remains a potential serious threat in locations that are relatively inaccessible or have limited capacity for a robust public health response. Records describe substantial outbreaks of pneumonic plague (*3*) but portray a more dangerous disease than that described by Drancort and Raoult. High rates of transmission are possible (*4*) when pneumonic plague is spreading through social networks, in a way similar to that observed in West Africa during the recent epidemic of Ebola virus disease (*5*). The Ebola virus is not thought to be easily transmitted but is clearly capable of generating a sustained epidemic.

The role of ectoparasites in the transmission of *Y. pestis* should not be dismissed. However, until a substantial epidemic has been documented with this proven etiology, this explanation of plagues, both historical and modern, must remain in the realm of conjecture.

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