for urinary tract infections and urine cultures were only obtained when treatment failed, this would have increased the probability of detecting FQ-R *E coli* relative to fluoroquinolone susceptible (FQ-S) *E coli*. It is unknown (but would not be surprising) if diagnostic and treatment behaviour were correlated. This second mechanism might endanger the study validity, as acknowledged by the authors. As FQ-R *E coli* infections are largely outnumbered by susceptible ones, a small decrease in such infections could explain the relatively large increase in FQ-R *E coli*. If so, this would also have led to overestimation of the indirect effects. It would, therefore, be interesting to compare timing of urine culture collection with antibiotic treatment initiation between neighbourhoods to test this hypothesis.

Finally, as with many other infectious diseases, the results of this study appear to be driven by a subpopulation with substantially higher fluoro-quinolone use than the population average. If we assume that the mean duration of a fluoroquinolone treatment course is 5 days, the mean of 1·5 defined daily dose per 1000 days in the population implies that at most, 11% of the population received fluoroquinolones in the last year. Reported fluoroquinolone use was 20% among patients with FQ-S *E coli*, 26% among patients with sterile cultures, and 58% among patients with FQ-R *E coli*. Consequently, fluoroquinolone use was at least twice the average among patients in which urine samples were tested.

This study emphasises both the power of using large datasets for population-based studies and the consequences of using fluoroquinolones. The observed association between population density and the occurrence of FQ-R *E coli* suggests that direct transmission between humans has contributed to the findings. This has been shown before for *E coli* producing extended-spectrum β-lactamases in Dutch household settings.[2,3] In both studies, the estimated basic reproductive number was, despite documented transmission, far below one, suggesting that further transmission within, and certainly outside, the household setting is unlikely. The observed two to one ratio between direct and indirect acquisition of FQ-R *E coli* in Israel suggests that, on average, direct acquisition through antibiotic use does not lead to many indirect acquisitions, suggesting that transmission in the open population will not lead to epidemic spread of these bacteria. The results of the study from Low and coworkers hopefully inspire others to do detailed population-based studies to quantify transmission of antibiotic resistant bacteria to better understand this potentially important, but still largely unknown, element in the complex system of antibiotic resistance epidemiology.

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The genesis of the Ebola virus outbreak in west Africa

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The 2013–16 Ebola virus outbreak in west Africa was purported to have begun in the Guinean village of Meliandou in December, 2013.[1] Authorities recorded 11 cases of Ebola virus disease (EVD) at this "index site" (where the virus is believed to have first spilled over into the human population), with 100% case fatality. In *The Lancet Infectious Diseases*, Joseph WS Timothy and colleagues[2] present a brilliant piece of epidemiological sleuthing. By combining classic field investigations with an assay that can measure Ebola virus antibodies in oral fluid, the authors have improved our understanding of the early development of the outbreak in Meliandou. They show that there was almost double the number of individuals infected with Ebola virus (21 cases *vs* the 11 cases previously reported), and the case fatality was 55·6%.

The non-invasive assay used in this study reportedly has high specificity,[3] although the scarce number

of negative controls (almost all UK-based), change in cutoff from previous studies, and imperfect orthogonal corroboration restricted to symptomatic participants (as opposed to those deemed minimally symptomatic) mean that further validation of the assay would be welcome. In our previous study[4] in Sierra Leone, we found—albeit with a different assay—that European and American negative controls provide much less noise and thus prompt lower optical density cutoffs (the threshold for determining whether someone is a case or not) than do controls in west Africa, potentially due to cross-reactivity of our assay with closely related pathogens circulating in the region.

Timothy and colleagues corroborate emerging evidence that suggest minimally symptomatic infections were common in the 2013–16 outbreak in west Africa and that a substantial portion of Ebola transmission events might have been undetected during the outbreak.[4,5] Indeed, even WHO has admitted that the true toll of the epidemic "was certainly greater" than the 28 616 suspected, probable, and confirmed cases of EVD that were reported.[6]

Notwithstanding the minor limitation of recall bias, most will agree that this well designed study yields important insights into the genesis of the 2013–16 Ebola virus outbreak in west Africa; however, we must be wary of leaving epidemics to the epidemiologists. It is important to consider that the real genesis of this preventable outbreak is the combined effect of the legacies of slavery (ie, Maafa[7]), exploitative colonialism, purposeful underdevelopment, structural adjustment, resource extraction, illicit financial flows, poverty, gender violence, and enabled civil war.[8,9] When viewed through this lens, even the revised case fatality of 55·6% is misleading: we estimate the true case fatality of EVD to be less than 10% when patients have access to a high-level intensive care unit—notably, all repatriated Americans infected with Ebola virus survived.[10] In summary, although highly skillful work—like that of Timothy and colleagues—is integral to improved understanding of viral transmission dynamics, such virtuosity should not obscure recognition of the structural determinants of epidemics.[11]

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Endemic Zika virus transmission: implications for travellers

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In 2016, a Zika virus epidemic swept across the Americas and resulted in the declaration of a Public Health Emergency of International Concern because of the potentially devastating consequences of congenital infection.[1,2] Globally, Zika virus transmission occurs at a much lower level today than in 2016; however, cases have been reported in 2017 and 2018 in Africa and India, and serological studies suggest nearly 10% of children