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Chikungunya-related Fatality Rates, Mauritius, India, and Reunion Island

To the Editor: During the epidemic of chikungunya virus infection that occurred on Reunion Island in 2005–06, we reported an overmortality corresponding to the epidemic peak, which was estimated by comparing observed and expected deaths (1). The excess was similar to the number of deaths related to chikungunya infection reported by death certificates (2). The case-fatality rate (CFR) on Reunion Island was estimated to be 1/1,000 population.

According to Beeson et al. (3), the fatality rate attributable to chikungunya infection was much higher on Mauritius: 743 deaths in excess of expected deaths led to a CFR of $\approx 4.5\%$, with 15,760 confirmed or suspected cases for 2005 and 2006 as reported in this letter. A similar CFR of 4.9% can be calculated for the city of Ahmedabad, India, during the 2006 chikungunya epidemic (4).

This 45- to 49-fold difference could be explained by a greater severity of chikungunya infection in Mauritius or Ahmedabad that could be due to a mutating strain, differences in the preexisting conditions of patients, differences in the management of patients, or by coincident deaths in excess from other causes.

However, the most probable explanation can be attributed to the surveillance systems of chikungunya cases. On Reunion Island, surveillance was highly sensitive and relied either on active case finding or on estimates of suspected cases. Results have been assessed by iterative external studies and serosurveys, and the CFR we found is likely consistent.

If we apply this rate to Mauritius, $\approx 60\%$ of the population would have contracted chikungunya infection during this epidemic. If so, the risk of epidemic resurgence could be much lower than previously expected. This point raises the need to conduct seroprevalence studies in those territories, the only way to evaluate the herd immunity level of the population.

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Aquaculture and Florfenicol Resistance in *Salmonella enterica* Typhimurium DT104

To the Editor: In June 2006, the World Health Organization (WHO), the Food and Agriculture Organization of the United Nations (FAO), and the World Organisation for Animal Health (OIE) convened an Expert Consultation to consider the risks to human health represented by the use of antimicrobial drugs in aquaculture. This would, therefore, appear to be an opportune time to reexamine some of the arguments that have been presented with respect to the assessment of these risks.

In their contributions to the debate regarding the risks associated with the use of antimicrobial agents in aquaculture, Angulo (1), Angulo and Griffin (2), Ribot et al. (3), and, more recently, Cabello (4) have argued that the available molecular evidence suggests that the *flo* gene that encodes chloramphenicol and florfenicol resistance in *Salmonella enterica* serovar Typhimurium DT104 (DT104) originally emerged in Japanese aquaculture and may have transferred horizontally from this host to DT104. This argument also appears in the report of the WHO/FAO/OIE consultation (ftp://ftp.fao.org/ag/agn/food/aquaculture_rep_13_16june2006.pdf). These authors (1–4) have based their argument on the assertions that florfenicol was first used in Japan and that *flo* gene-mediated resistance to this agent was first identified in bacteria isolated from Japanese fish farms.

In attempting to identify the date of the emergence of florfenicol resistance in Japanese aquaculture, Angulo and Griffin (2) state that florfenicol had been used in this country since the early 1980s. However, Schering Plough, the manufacturer of florfenicol, reports first marketing this