had serum samples that were either positive or equivocal. The low prevalence of antibodies to *C. burnetii* in the participants in our study (3/97) indicates that most were very unlikely to have had contact with the organism. If the results are true positives, the source of the infection was quite likely outside of New Zealand. However, considering the heavy exposures associated with the cultivation and harvesting of RHDV in live rabbits and the known infectivity of Q fever, *C. burnetii* was not likely to have been introduced inadvertently to New Zealand at the same time as RHDV.

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**References**


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**Correction, Vol. 8, No. 10**

In the article, “Antimicrobial Resistance of *Escherichia coli* O26, O103, O111, O128, and O145 from Animals and Humans” by Carl M. Schroeder et al., errors occurred in the figure on page 1412. The corrected figure appears below and online at http://www.cdc.gov/ncidod/eid/vol8no12/02-0070.htm.

We regret any confusion these errors may have caused.

**Figure 2. Comparison of antimicrobial resistance frequencies**

* A. Between *E. coli* (B). Am, ampicillin; Cx, cefoxitin; C, chloramphenicol; Frx, ceftriaxone; Smx, sulfamethoxazole; Cf, cephalexin; Gm, gentamicin; Na, nalidixic acid; Cip, ciprofloxacin; Fur, ceftiofur; Te, tetracycline; T/S, trimethoprim-sulfamethoxazole; A/C, amoxicillin-clavulanic acid; Str, streptomycin.

B. Between *E. coli* (A). In contrast, of isolates from humans, resistance frequencies were generally lower for STEC compared with other *E. coli* (B).

We regret any confusion these errors may have caused.