because bacteria could have been transported to Chile by ballast water from the Northern Hemisphere (*1*,*4*,*6*). As in previous outbreaks, shellfish responsible for this epidemic were harvested near international shipping lanes (*1*,*3*,*4*,*6*). The appearance of *V. parahaemolyticus* O3:K6 in Chile has thus converted the expansion of this strain into a real pandemic because this vibrio is now present in 5 continents. The persistence of *V. parahaemolyticus* in Region X might also have been encouraged by an expansion of finfish and shellfish aquaculture in that area. As in other parts of the world, expansion of these food industries could provide physical and nutritional substrates for vibrios to persist and propagate when growth is triggered by increases in temperature of seawater (*1*,*2*,*8*).

Emergence of *V. parahaemolyticus* in Region X has also coincided with expansion of harmful algal blooms in the same area. These blooms are triggered by increases in seawater temperature and degradation of the coastal environment (*9*,*10*). A connection has been established between algal blooms and the presence of *V. cholerae* and cholera epidemics in the Gulf of Bengal and off the coast of Peru at the start of the Latin America epidemic (*10*). Further research is necessary to ascertain whether persistence of *V. parahaemolyticus* and epidemics are related to algal blooms in this region of Chile.

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Toxoplasma gondii Prevalence, United States

To the Editor: We correct the prevalence of *Toxoplasma gondii* immunoglobulin (Ig) G antibodies published in Emerging Infectious Diseases in 2003 (*1*). An incorrect cutoff value in the computer program used to calculate seropositivity of anti–*T. gondii* IgG antibody resulted in some incorrect prevalence rates. We discovered this error when analyzing more recent National Health and Nutrition Examination Survey (NHANES) data.

The cutoff value for anti–*T. gondii* IgG seropositivity used in the prior publication (*1*) was >6 IU, which is the correct value for NHANES III 1988–1994 data (*2*) but not for NHANES 1999–2000 data. Because of a change by the *T. gondii* test kit manufacturer, the cutoff value for NHANES 1999–2000 seropositivity data was increased to ≥10 IU. This cutoff change from >6 to ≥10 IU does not cause a large difference in the *T. gondii* seroprevalence reported. In addition, it does not change the overall findings of the article or the overall relationship between NHANES III (1988–1994) and NHANES 1999–2000. However, it does produce a borderline change for 2 demographic subgroups (non-Hispanic white per-

Table. Comparison of *Toxoplasma gondii* IgG antibody seroprevalence, NHANES 1999–2000 and NHANES III (1988–1994)*†

	NHANES 1999–2000			NHANES III (1988–1994)		
	N‡	Prevalence	95% CI	N‡	Prevalence	95% CI
Total	4,234	14.3	12.3–16.2	11,132	16.0	14.5–17.5
Sex						
Male	2,013	15.2	12.4–18.0	5,144	16.7	14.8–18.6
Female	2,221	13.4	11.2–15.5	5,988	15.3	13.5–17.0
Race/ethnicity						
Non-Hispanic white	1,293	10.8	8.1–13.6	3,304	14.3	12.5–16.2
Non-Hispanic black	1,027	16.8	13.4–20.3	3,674	18.0	16.1–19.8
Mexican American	1,553	14.2	10.1–18.4	3,661	18.3	16.7–20.0
Age group, y						
12–19	2,105	7.3	4.7–10.0	2,749	8.5	6.4–10.5
20–29	735	11.9	9.5–14.4	3,100	15.2	12.1–18.3
30–39	726	17.0	12.9–21.2	2,960	16.1	14.6–17.6
40–49	668	18.7	15.0–22.3	2,323	22.2	19.4–25.0
Country of birth						
United States	3,211	10.5	8.3–12.8	8,606	14.1	12.7–15.5
Not United States	995	32.0	24.0–39.9	2,493	27.9	24.1–31.7

*IgG, immunoglobulin G; NHANES, National Health and Nutrition Examination Survey; CI, confidence interval.
†Sex, race/ethnicity, country of birth,and total values are age-adjusted to the 2000 census–estimated population using the 4 age categories shown.
‡Totals for the race/ethnicity and country-of-birth categories do not add up to the total number because an "other" race/ethnicity category was included in the totals but not shown in these categories or because persons did not provide a response to country-of-birth questions.

sons and persons born in the United States), for whom the difference from NHANES III to NHANES 1999–2000 data reached statistical significance at $p<0.05$ in the *t* test, but the 95% confidence intervals (CIs) for the prevalence estimates for these groups still overlapped between NHANES III and NHANES 1999–2000 (i.e., the *t* test is a less conservative measure of association than CI).

After this correction, the overall age-adjusted *T. gondii* antibody prevalence according to NHANES 1999–2000 data changed from 15.8% (95% CI 13.5%–18.1%) to 14.3% (95% CI 12.3%–16.2%). The Table shows the overall and stratified seroprevalence rates for NHANES 1999–2000 (corrected) compared with NHANES III (no corrections needed).

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Human Infection with *Schineria larvae*

To the Editor: Myiasis remains prevalent worldwide (*1*,*2*) and is infestation by larvae from fly species of live or dead tissues from vertebrate hosts (*1*,*3*,*4*). In humans, myiasis most frequently causes infection of exposed ulcers or traumatic wounds (*1*). In industrialized countries, most cases occur in tourists returning from tropical and subtropical areas (*5*,*6*), but autochthonous cases still exist. Several bacterial species have been associated with fly larvae, including species of the family *Enterobacteriaceae* and, more recently, *Schineria larvae* (*7*,*8*). *S. larvae*, a gram-negative bacterium, has been grown from larvae of *Wohlfahrtia magnifica*, a fly species responsible for myiasis (*7*,*8*). Its 16S rRNA gene has been amplified from a bacterial community of species involved in aerobic thermophilic bioprocesses (*9*). We report a case of *S. larvae* bacteremia in a man with wound myiasis.

On June 12, 2006, a 76-year-old man who had type 2 diabetes mellitus was examined at the emergency