

tributors to the decline in the number of cases of acute diarrhea in children after the tsunami. Although out-migration of island residents or a reduction in case detection after the tsunami also could have contributed to the observed decline in cases of diarrhea, no large-scale migration was reported during the period, and disease surveillance systems were in fact strengthened after the tsunami and further strengthened with the introduction of the Integrated Disease Surveillance Program.

In summary, we found that the incidence of acute diarrhea among children of the Andaman Islands decreased within months after the 2004 tsunami. This result highlights the importance of public health and sanitation measures after a natural disaster.

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Population-Attributable Risk Estimates for *Campylobacter* Infection, Australia

To the Editor: Many industrialized countries have a high incidence of *Campylobacter* infections. An estimated 250,000 cases of *Campylobacter* infection occur annually in the United States (1), and several sequelae compound the impact of these infections. The incidence of *Campylobacter* infections is also important to policymakers—in the United Kingdom it

is used to assess foodborne disease-reduction strategies (2)—and governments worldwide rely on the findings of epidemiologic and microbiological studies on *Campylobacter* infection to shape their food-safety policies.

Population-attributable fractions provide added value in case-control studies by helping researchers identify the most important risk factors for a condition on the basis of risk association and frequency of exposure. In an analysis of data from a previous case-control study of *Campylobacter* infection (3), Stafford et al. (4) used population-attributable fractions to estimate the annual number of *Campylobacter* infection cases among Australians ≥ 5 years of age that were attributable to each risk factor from that study. Using this technique, they estimated that 50,500 cases annually can be attributed directly to eating chicken.

Population-attributable fractions have been defined as “the proportion of disease cases over a specified time that would be prevented following elimination of ... exposure [to the specified risk factors]” (5). Therefore, removing exposure to factors not associated with disease risk will not affect disease incidence. Stafford and colleagues implicitly acknowledge this in their methods: “We calculated PARs [population-attributable risks] ... for each variable that was significantly associated with an increased risk for infection.” It is surprising, therefore, that they subsequently included consumption of cooked chicken in their extrapolation, even though this exposure was not significantly associated with illness (adjusted odds ratio 1.4, 95% confidence interval 1.0–1.9, $p = 0.06$). Because they attributed 35,500 of the 50,500 cases of *Campylobacter* infection to the consumption of cooked chicken, I believe that Stafford et al. overestimated the role of chicken consumption in cases of *Campylobacter* infection by a factor of 3.4.

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In Response: Gillespie (1) questions whether we should have treated consumption of cooked chicken as a contributing factor in estimating population-attributable risk (PAR) for *Campylobacter* infection because it was not a significant risk factor (adjusted odds ratio [aOR] 1.4, 95% confidence interval [95% CI] 1.0–1.9, $p = 0.06$). Although on strict statistical grounds Gillespie is correct, we believe that consumption of cooked

chicken warrants consideration as a risk factor on the basis of biological plausibility, possible misclassification of the “cooked” status of chicken eaten by study participants, and previous empirical evidence.

Many case-control studies have demonstrated that consumption of any chicken is a risk factor for *Campylobacter* infection (2,3), although others have shown that only the consumption of undercooked chicken or chicken eaten outside of the home is a risk factor (4,5). We acknowledged in our study that some misclassification of exposure is likely in any study of reported food-consumption habits and that such misclassification is a major limitation of case-control studies. Because of the difficulty of determining whether previously eaten chicken had been thoroughly cooked or recontaminated after having been cooked, particularly if it was purchased outside of the home, at least some study participants who reported eating cooked chicken could have acquired their infection from the chicken meat. Moreover, as noted above, results from other case-control studies showing an association between disease risk and consumption of chicken or poultry have been based on study participants' reported consumption of both undercooked and cooked chicken.

In our study, we included chicken consumption in our multivariable models as either “cooked chicken” or “undercooked chicken” but not as “chicken.” However, in our univariate analysis, consumption of any chicken (i.e., cooked or undercooked) was significantly associated with illness (OR 1.6, 95% CI 1.2–2.1), and it was also significantly associated with illness when included in a multivariable model as a single variable (aOR 1.7, 95% CI 1.2–2.3). Results of a univariate assessment of various types of cooked chicken meat showed that consumption of chicken fillet (OR 1.2), chicken kebabs (OR 1.7), and bought barbe-

cued chicken (OR 1.2) was each associated with increased risk for illness. Other studies have similarly shown consumption of cooked, fried, or barbecued chicken to be significantly associated with risk for *Campylobacter* infection (3,4).

The strength of our approach is that we were able to estimate the number of cases of campylobacteriosis attributable to chicken consumption each year and to assess the uncertainty of these estimates. Because we were unable to calculate CIs for the estimated number of cases not detected by surveillance, we used computer simulation to generate an overall distribution of the number of cases that could plausibly be attributed to chicken consumption; the estimated range was 10,000–105,000 cases annually. This represents a conservative approach to assessing the contribution of chicken consumption to *Campylobacter* infections in Australia. Thus, although we concur that the inclusion of cooked chicken as a risk factor for *Campylobacter* infections was debatable on strictly statistical grounds, we believe that including cooked chicken in our estimates of the PAR for and community incidence of these infections was reasonable.

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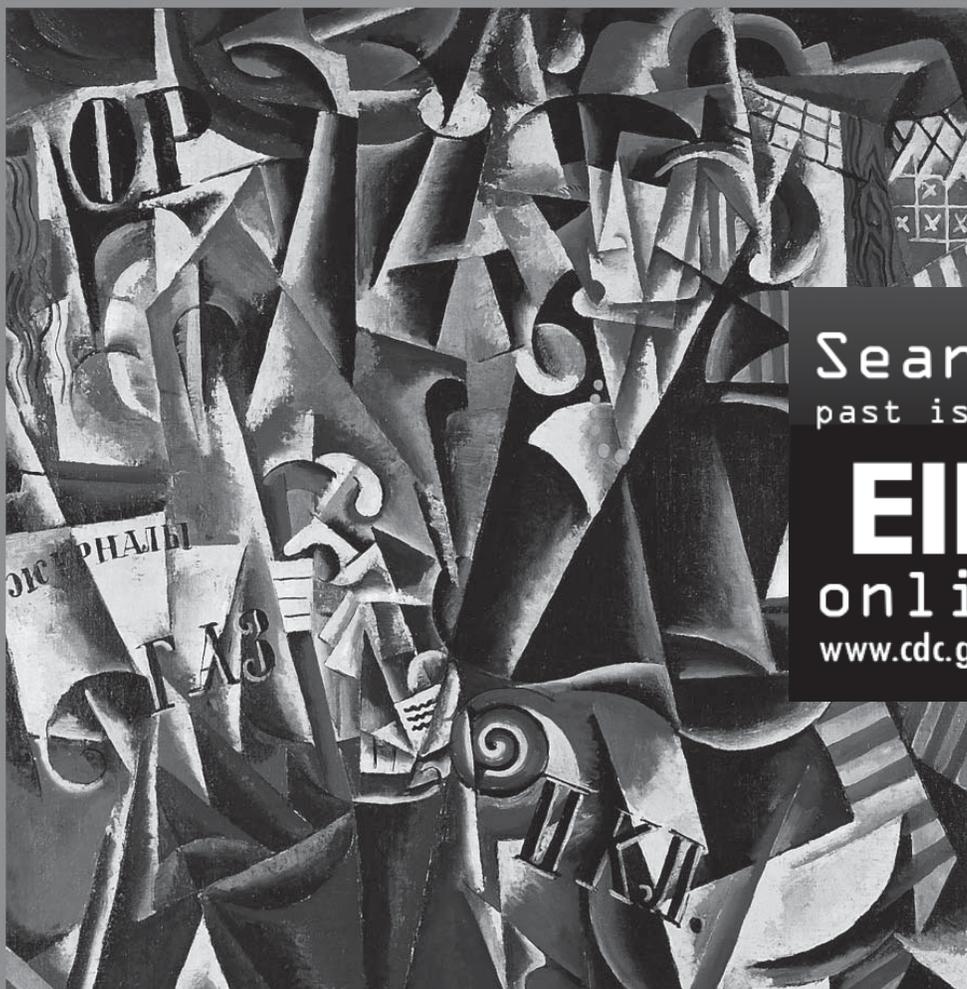
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