Contributors 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.

Acknowledgments

We are grateful to the medical superintendent at G.B. Pant Hospital, Port Blair, for sharing data, and to D.R. Guruprasad for his help in data collection.

This study was carried out with the internal funds of the Regional Medical Research Centre (Indian Council of Medical Research).

Subarna Roy, Debdutta Bhattacharya, S.R. Ghoshal, K. Thanasekaran, A.P. Bharadwaj, M. Singhania, and A.P. Sugunan

Author affiliations: Indian Council of Medical Research Regional Medical Research Centre, Port Blair, Andaman and Nicobar Islands, India (S. Roy, D. Bhattacharya, S.R. Ghoshal, K. Thanasekaran, A.P. Sugunan); and G.B. Pant Hospital, Port Blair (A.P. Bharadwaj, M. Singhania)

DOI: 10.3201/eid1505.081096

References

1. Ishii M, Shearer PM, Houston H, Vidale JE. Extent, duration, and speed of the 2004 Sumatra–Andaman earthquake imaged by the Hi-Net array. Nature. 2005;435:933–6.

2. Kohl PA, O'Rourke AP, Schmidman DL, Dopkin WA, Birnbaum ML. The Sumatra–Andaman earthquake and tsunami of 2004: the hazards, events, and damage. Prehosp Disaster Med. 2005;20:355–63.

3. Lay T, Kanamori H, Ammon CJ, Nettles M, Ward SN, Aster RC, et al. The great Sumatra–Andaman earthquake of 26 December 2004. Science. 2005;308:1127–33. DOI: 10.1126/science.1112250

4. Andaman & Nicobar Administration. Presentation on the Tsunami Rehabilitation Programme as of September 2008 [cited 2009 Mar 27]. Available from http://www.and.nic.in/Latest%20Updates/TRP_CS/TRP_Presentation/PPP%20at%20PC%2001.10.08(Sept).ppt

5. Sugunan AP, Ghosh AR, Roy S, Gupte MD, Sehgal SC. A cholera epidemic among the Nicobarese tribe of Nancowry, Andaman, and Nicobar, India. Am J Trop Med Hyg. 2004;71:822–7.

6. Sugunan AP, Roy S, Murhekar MV, Naik TN, Sehgal SC. Outbreak of rotaviral diarrhoea in a relief camp for tsunami victims at Car Nicobar Island, India. J Public Health (Oxford). 2007;29:449–50. DOI: 10.1093/pubmed/dnm054

Address for correspondence: A.P. Sugunan, Regional Medical Research Centre, Indian Council of Medical Research, Port Blair, Andaman and Nicobar Islands, India; email: sugunanap@icmr.org.in

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.
Iain Gillespie
Author affiliation: Health Protection Agency
Centre for Infections, London, UK
DOI: 10.3201/eid1505.081553

References
1. Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C, et al. Food-related illness and death in the United States. Emerg Infect Dis. 1999;5:607–25.
2. Adak GK, Long SM, O’Brien SJ. Trends in indigenous foodborne disease and deaths, England and Wales: 1992 to 2000. Gut. 2002;51:832–41. DOI: 10.1136/gut.51.6.832.
3. Stafford RJ, Schluter P, Kirk M, Wilson A, Unicomb L, Ashbolt R, et al. A multicentre prospective case–control study of Campylobacter infection in persons aged 5 years and older in Australia. Epidemiol Infect. 2007;135:978–88. DOI: 10.1017/S0950268806007576.
4. Stafford RJ, Schluter PJ, Wilson AJ, Kirk MD, Hall G, Unicomb L. Population-attributable risk estimates for risk factors associated with Campylobacter infection, Australia. Emerg Infect Dis. 2008;14:895–901. DOI: 10.3201/eid1406.071008.
5. Rockhill B, Newman B, Weinberg C. Use and misuse of population-attributable fractions. Am J Public Health. 1998;88:15–9. DOI: 10.2105/AJPH.88.1.15

Address for correspondence: Iain Gillespie, Health Protection Agency, Division of Gastrointestinal, Zoonotic and Emerging Infections, 61 Colindale Ave, London NW9 5EQ, UK; email: iain.gillespie@hpa.org.uk

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 barbecued 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.

Russell J. Stafford, Philip J. Schluter, Martyn D. Kirk, and Andrew J. Wilson
Author affiliations: Queensland Health, Brisbane, Queensland, Australia (R.J. Stafford, A.J. Wilson); University of Queensland, Brisbane (R.J. Stafford, P.J. Schluter, A.J. Wilson); Auckland University of Technology, Auckland, New Zealand (P.J. Schluter); and OzFoodNet, Canberra, Australian Capital Territory, Australia (M.D. Kirk)
DOI: 10.3201/eid1505.090080

References
1. Gillespie I. Population-attributable risk estimates for Campylobacter infection, Australia. Emerg Infect Dis. 2009;15:850–1.
2. Evans MR, Ribeiro CD, Salmon RL. Hazards of healthy living: bottled water and salad vegetables as risk factors for *Campylobacter* infection. Emerg Infect Dis. 2003;9:1219–25.

3. Studahl A, Andersson Y. Risk factors for indigenous *campylobacter* infection: a Swedish case-control study. Epidemiol Infect. 2000;125:269–75. DOI: 10.1017/S0950268899004562

4. Eberhart-Phillips J, Walker N, Garrett N, Bell D, Sinclair D, Rainger W, et al. Campylobacteriosis in New Zealand: results of a case-control study. J Epidemiol Community Health. 1997;51:686–91. DOI: 10.1136/jech.51.6.686

5. Effler P, Ieong MC, Kimura A, Nakata M, Burr R, Cremer E, et al. Sporadic *Campylobacter jejuni* infections in Hawaii: associations with prior antibiotic use and commercially prepared chicken. J Infect Dis. 2001;183:1152–5. DOI: 10.1086/319292

Address for correspondence: Russell James Stafford, Queensland Health, GPO Box 48, Brisbane, Queensland 4001, Australia; email: russell_stafford@health.qld.gov.au

The opinions expressed by authors contributing to this journal do not necessarily reflect the opinions of the Centers for Disease Control and Prevention or the institutions with which the authors are affiliated.