

7. Waldenström J, Broman T, Carlsson I, Hasselquist D, Achterberg RP, Wagenaar JA, et al. Prevalence of *Campylobacter jejuni*, *Campylobacter lari*, and *Campylobacter coli* in different ecological guilds and taxa of migrating birds. *Appl Environ Microbiol.* 2002;68:5911–7. DOI: 10.1128/AEM.68.12.5911-5917.2002
8. Dingle KE, Colles FM, Wareing DR, Ure R, Fox AJ, Bolton FE, et al. Multilocus sequence typing system for *Campylobacter jejuni*. *J Clin Microbiol.* 2001;39:14–23. DOI: 10.1128/JCM.39.1.14-23.2001
9. Dingle KE, Colles FM, Ure R, Wagenaar JA, Duim B, Bolton FJ, et al. Molecular characterization of *Campylobacter jejuni* clones: a basis for epidemiologic investigation. *Emerg Infect Dis.* 2002;8:949–55.
10. Manning G, Dowson CG, Bagnall MC, Ahmed IH, West M, Newell DG. Multilocus sequence typing for comparison of veterinary and human isolates of *Campylobacter jejuni*. *Appl Environ Microbiol.* 2003;69:6370–9. DOI: 10.1128/AEM.69.11.6370-6379.2003

Address for correspondence: Jonas Waldenström, Section for Zoonotic Ecology and Epidemiology, School of Pure and Applied Natural Sciences, Kalmar University, SE 391, 82 Kalmar, Sweden; email: jonas.waldenstrom@hik.se

## Acute Diarrhea in Children after 2004 Tsunami, Andaman Islands

**To the Editor:** The Andaman Islands, population  $\approx 350,000$ , are a territory of India located in the Bay of Bengal, northwest of Indonesia. On December 26, 2004, these islands were struck by an earthquake measuring 9.1 on the Richter scale (1) and by the ensuing Great Asian Tsunami (2). The fault slip, which caused permanent land subsidence of several meters (3) and ingression of sea water, resulted in the displacement of most survivors, many of whom were forced to live in temporary camps on higher ground for periods of more than a year. About

80% of the water supply lines were broken (4) and so were most sewage lines, making the situation ideal for transmission of water-borne diseases.

Because an outbreak of cholera had occurred in the Andaman and Nicobar Islands in 2002 (5), we were apprehensive about outbreaks of infectious diseases after the tsunami, particularly among children, who are less immune to most infections; therefore, we increased our efforts to identify and contain these possible outbreaks as quickly as possible. However, except for a cluster of cases of rotaviral diarrhea (6), no major infectious disease outbreak occurred among residents of the Andaman Islands in the year that followed the tsunami.

Although the incidence of severe cases of diarrhea among children admitted to G.B. Pant Hospital in Port Blair, the only referral hospital in the Andaman Islands, varied greatly from month to month during 2001–2007, the incidence began decreasing after 2005, as indicated by the 12-month moving average (Figure). The mean number of cases per year fell from 361.4 during 2001–2005 to only 255.0 during 2006 and 2007 ( $p = 0.00025$ ).

The estimated annual incidence of acute diarrhea per 100,000 children in the Andaman and Nicobar Islands was 609 in 2001, 580 in 2002, 595 in 2003, 601 in 2004, 571 in 2005, 370 in 2006, and 420 in 2007. For these incidence estimates, the population at risk during the years 2002–2007 was calculated by extrapolating from the

2001 census population on the basis of an annual population growth rate of 1.53% (the average for 1991–2001) and assuming that children  $\leq 15$  years old constituted 36.2% of the total population each year (as they did in 2001). The reduction in the number of acute cases of childhood diarrhea began several months after the tsunami, when the water and sewage systems of the islands had been repaired and renovated in many areas.

According to official reports, the cost of the restoration and renovation of the water and sewage systems after the tsunami was 389.9 million rupees,  $>2\times$  the projected cost of work on the water and sanitation systems (172.9 million rupees) prior to the tsunami (4). In the aftermath of the tsunami, 52 km of new pipelines were laid and 12.5 km of old pipelines were replaced. Water supplies were augmented in 49 areas (4). The revamped water and sewage systems eliminated many sources of fecal contamination.

Moreover, by the middle of 2005, post-disaster assistance had been provided by voluntary organizations, missionaries, nongovernmental organizations, and government agencies from mainland India and abroad. This assistance resulted in further improvements in the area's public sanitation infrastructure and hygiene, particularly in the temporary shelters that displaced residents were living in; it also raised awareness among island residents about the threat of water-borne diseases. All of these factors were likely con-

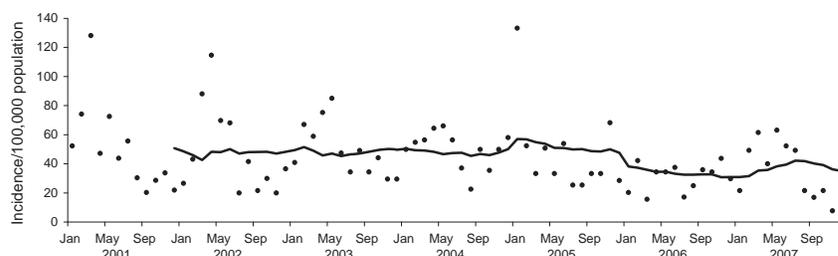


Figure. Estimated monthly incidence (black dots) of acute diarrhea among children  $\leq 15$  years of age in the Andaman Islands and 12-month moving average of the monthly incidence (black line), 2001–2007. Data based on cases of disease among children admitted to G.B. Pant Hospital, Port Blair, Andaman and Nicobar Islands, India.

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.

#### 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%201.10.08\(Sept\).ppt](http://www.and.nic.in/Latest%20Updates/TRP_CS/TRP_Presentation/PPP%20at%20PC%201.10.08(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/fdm054

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](mailto: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 policy-makers—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  $\geq 5$  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.