Hepatitis E Virus Infection, Papua New Guinea, Fiji, and Kiribati, 2003–2005

To the Editor: We report hepatitis E virus (HEV) infection rates in 3 South Pacific island countries—Papua New Guinea (PNG), Fiji, and Kiribati-determined from results of HEV IgG testing. During 2003–2005, specimens were collected from volunteers as part of a study of the epidemiology of viral hepatitis (1,2). Participants recruited were apparently healthy adults in the community and mother-infant pairs (specifically infants who were receiving, or had recently completed, their vaccinations). No specific inclusion/exclusion criteria were applied. Samples were collected from outpatient clinics and hospitals in PNG from Port Moresby, Goroka, Mt Hagen, Madang, and Daru. Samples from Fiji were collected in Suva from outpatient clinics and hospital wards. A proportion of samples from children were taken from nonjaundiced inpatients in PNG and Fiji. In Kiribati, samples were collected from participants at village preschools,

vaccination clinics, and outpatient clinics on North Tarawa and North Tabiteuea (2). These were convenience samples and therefore might not be nationally representative cohorts. We obtained ethics permission for the study from appropriate national agencies. Signed informed consent was obtained from each participant or, for children, from a parent or guardian.

From this sample pool, in a time sequential manner, the first serum samples were assayed: 545 from PNG (48 Goroka, 99 Mt Hagen, 87 Daru, 47 Madang, 156 Port Moresby), 265 from Fiji, and 238 from Kiribati. We evaluated samples using the Wantai (PE2) HEV IgG ELISA kit (Wantai Pharmaceutical Enterprise, Beijing, China), which detects IgG for all 4 known strains of human HEV. The assays were used according to the manufacturer's instructions, and repeat equivocal results were defined as negative (3,4). HEV IgG positivity was highest in PNG (15.2%), followed by Kiribati (8.8%) and Fiji (2.2%) (Table). IgG positivity did not differ significantly between adults and children (<16 years of age) (PNG: 16.1% vs. 11.4%, p = 0.23; Kiribati: 6.0% vs. 13.3%, p =0.06; and Fiji: 1.7% vs. 3.3%, p = 0.42[Fisher exact test]).

To investigate potential parentchild transmission, we tested mother/ child (MC); father/child (FC); and when possible, mother/father/child (MFC) sets. We found no transmission association: In PNG, we tested 88 sets (67 MC, 2 MFC, and 19 FC); in Fiji, 29 sets (20 MC and 9 MFC); and in Kiribati, 65 sets (59 MC and 6 MFC); of the 11 PNG, 1 Fijian, and 8 Kiribati HEV IgG-positive children, none had IgG-positive parents. Because these samples were tested retrospectively, ascertaining the HEV IgG status of other family members was not possible.

The high percentage of HEV-seropositive children <5 years of age in PNG and Kiribati implies active viral circulation in these countries. This is an unusual finding, compared with findings from seroprevalence studies in developing countries where IgG prevalence increases with age (3). The reason for this difference remains to be determined. It is unlikely to relate to acute HEV infection in hospitalized children sampled because the Wantai assay measures IgG, not IgM. The finding that these young seropositive children commonly have seronegative parents suggests that parentchild transmission is not the primary

	Papua New Guinea†		Fiji†		Kiribati†	
Age group, y	All	HEV IgG+, % (95% CI)	All	HEV IgG+, % (95% CI)	All	HEV IgG+, % (95% CI)
Children	12/105‡	11.4 (6.6–18.9)	3/91‡	3.3 (1.1–9.2)	12/90	13.3 (7.8–21.2)
<1	0/21	0	1/23	4.3	1/8	12.5
1-<2	3/26	11.5	1/19	5.3	2/18	11.1
2-<5	6/24	25	1/35‡	2.9	6/48	12.5
5-<10	1/23‡	4.3	0/10‡	0	0/10	0
10-<16	1/6	16.7	0/3‡	0	0/2	0
Unknown	1/5	20	0/1	0	3/4	75
Adults	71/440	16.1 (14.2–21.3)	3/174	1.7 (0.6–4.9)	9/148	6.0 (3.2–11.1)
16–19	7/24	29.2	0/8	0	0/2	0
20-29	19/162	11.7	2/76	2.6	2/61	3.3
30–39	18/143	12.6	0/41	0	3/53	3.7
40-49	13/70	18.6	1/28	3.6	2/19	5.3
50-59	5/22	22.7	0/14	0	2/9	22.2
60–69	3/7	42.9	0/5	0	0/2	0
70–79	0/4	0	0/0	0	0/2	0
Unknown	0/9	0	0/1	0	0/0	0
Total	83/545‡	15.2 (12.5–18.5)	6/265‡	2.3 (1.0-4.9)	21/238	8.8 (5.8–13.1)

^{*}An expanded version of this table that includes sex data is available online (wwwnc.cdc.gov/EID/article/20/7/13-0562-T1.htm). HEV, hepatitis E virus; +, positive.

[†]No. HEV IgG-positive persons/total no. persons in each group.

[‡]In these groups.

mechanism of infection in the population studied, which is in accord with published data (4).

To investigate whether HEV seropositivity was higher in certain areas, we partitioned the data into regions defined by participant's place of birth and tribal ethnicity. In Fiji and Kiribati, no significant region association was detected. However, in PNG, the proportion of HEV antibody—positive specimens was greater among participants from highland communities (altitude >1,500 m) than from lowland communities (20.4% vs. 9.7%, p = 0.01). (Port Moresby has a mixed immigrant population and was excluded from this analysis).

The reason for the higher proportion of HEV IgG-positive specimens among participants in highland than lowland communities is unclear but might be explained by increased zoonotic transmission. In highland regions, pigs are more frequently kept, and the animals are kept closer to home (5). HEV genotypes 1 and 2 are hyperendemic to many developing countries and typically cause waterborne outbreaks of acute hepatitis in humans (6). Genotypes 3 and 4 are endemic to industrialized countries and are known to be a porcine zoonosis (7). Genotype 3 has been found in pigs in New Caledonia (8).

In our investigation of 3 developing nations in Oceania, we found that HEV IgG positivity varies substantially between, and within, countries; it is high in PNG (15.2%) and low in Fiji (2.2%). By using the same sensitive, diagnostic assay, the seroprevalence of HEV in blood donors in New Zealand was reportedly 4% (9). In New Caledonia, 1.7% of 351 military recruits tested positive (10). In our study, the sampling method limits the applicability of the data to the general population. Nevertheless, our findings suggest HEV infection should be considered in cases of unexplained hepatitis.

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References

 Harrison A, Lemey P, Hurles M, Moyes C, Horn S, Pryor J, et al. Genomic analysis of hepatitis B virus reveals antigen state and genotype as sources of evolutionary rate variation. Viruses. 2011;3:83–101. http://dx.doi.org/10.3390/v3020083

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- Harrison GLA, Pryor J, Malani J, Supuri M, Masta A, Teriboriki B, et al. Infection frequency of hepatitis C virus and IL28B haplotypes in Papua New Guinea, Fiji, and Kiribati. PLoS ONE. 2013;8:e66749. http://dx.doi.org/10.1371/ journal.pone.0066749.
- Labrique AB, Zaman K, Hossain Z, Saha P, Yunus M, Hossain A, et al. Epidemiology and risk factors of incident hepatitis E virus infections in rural Bangladesh. Am J Epidemiol. 2010;172:952–61. http://dx.doi.org/10.1093/aje/kwq225
- Somani SK, Aggarwal R, Naik SR, Srivatsa S, Naik S. A serological study of intrafamilial spread from patients with sporadic hepatitis E virus infection. J Viral Hepat. 2003;10:446–9. http://dx.doi. org/10.1046/j.1365-2893.2003.00458.x
- Hide R. Pig husbandry in New Guinea.
 A literature review and bibliography.
 Canberra (Australia): Australian Center for International Agricultural Research, Canberra; 2003.
- Kamar N, Bendall R, Legrand-Abravanel F, Xia NS, Ijaz S, Izopet J, et al. Hepatitis E. Lancet. 2012;379:2477– 88. http://dx.doi.org/10.1016/S0140-6736 (11)61849-7
- Dalton HR, Bendall R, Ijaz S, Banks M. Hepatitis E: an emerging infection in developed countries. Lancet Infect Dis. 2008;8:698–709. http://dx.doi. org/10.1016/S1473-3099(08)70255-X
- Kaba M, Davoust B, Cabre O, Colson P. Hepatitis E virus genotype 3f in pigs in New Caledonia. Aust Vet J. 2011;89:496– 9. http://dx.doi.org/10.1111/j.1751-0813. 2011.00848.x
- Bendall R, Ellis V, Ijaz S, Ali R, Dalton H. A comparison of two commercially available anti-HEV IgG kits and a re-evaluation of anti-HEV IgG seroprevalence data in developed countries. J Med Virol. 2010;82:799–805. http://dx.doi.org/ 10.1002/jmv.21656
- Bauduceau O, Berlioz A, Buisson Y. Hepatitis B, C, and E in New Caledonia. Seroepidemiologic study in military recruits. Med Trop (Mars). 2000;60:167–70.

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