

**Table: Prevalence of hepatitis B surface antigen in different tribes in India**

State	Tribe	Population sampled (n)	HBsAg (n.; %)	Ref.
Andaman and Nicobar Islands	Andamanese	27	1 (3.70)	1
	Onges	58	18 (31.00)	
	Nicobarese	1144	267 (23.30)	
	Shompens	37	14 (37.80)	
Madhya Pradesh	Jarawas	64	42 (65.60)	2
	Baiga	91	4 (4.40)	3
	Halbas	67	2 (3.00)	4
	Gonds	123	16 (13.00)	
	Kawars	58	6 (10.30)	
	Oraons	118	10 (8.50)	
	Bhils	397	73 (18.40)	
	Bhilals	122	23 (18.90)	
	Barelas	429	76 (17.70)	
	Bhils	536	55 (10.26)	5
Rajasthan	Raj Gond	450	126 (28.03)	5
	Kolam	106	15 (14.15)	
	Naik Gond	42	4 (9.52)	
Maharashtra	Pradhan	43	3 (6.98)	
	Not mentioned	296	25 (8.50)	6
Arunachal Pradesh	Lambada	890	46 (5.20)	7
Andhra Pradesh	Tribes of Kolli hills	161	3 (1.86)	8
Tamil Nadu				
Ladakh				
(Jammu and Kashmir)	Ladhakis	144	14 (9.72)	9

suggested the possibility of blood-sucking arthropods in the transmission of the infection.<sup>2</sup>

The *chi* square for heterogeneity among the studies was significant, indicating that the studies were heterogeneous. Another limitation of the present study is the fact that HBsAg prevalence in some of the tribes was assessed more than 25 years earlier. However, in absence of any vaccination program, it does not appear likely that the HBsAg rates could have changed significantly over the years.

Our study indicates that the prevalence of HBsAg among the scheduled tribes of India is much higher than that in the general population. As the tribes comprise a sizeable proportion of the population in different States in India, hepatitis B vaccine should be included in the immunization program for tribal children.

M V Murhekar, S P Zodpey\*

National Institute of Epidemiology, Chennai 600 031, and \*Department of Preventive and Social Medicine, Government Medical College, Nagpur 440 004

### References

1. Murhekar MV, Murhekar KM, Das D, Arankalle VA, Sehgal SC. Prevalence of hepatitis B infection among the primitive tribes of Andaman and Nicobar Islands. *Indian J Med Res* 2000;111:199-203.

2. Murhekar MV, Murhekar KM, Arankalle VA, Sehgal SC. Alarming prevalence of hepatitis B infection among the Jarawas, a primitive Negrito tribe from Andaman and Nicobar Islands, India. *J Viral Hepatitis* 2003;10:232-3.
3. Reddy PH, Tedder RS. Hepatitis virus markers in the Baiga tribal population of Madhya Pradesh, India. *Trans R Soc Trop Med Hyg* 1995;89:620.
4. Joshi SH, Gorakshakar AC, Mukherjee M, Rao VR, Sathe MS, Anabhavane SM, *et al.* Prevalence of HBsAg carriers among some tribes of Madhya Pradesh. *Indian J Med Res* 1990;91:340-3.
5. Mukherjee M, Joshi SH, Rao VR, Gorakshakar AC, Sather MS. Prevalence of hepatitis B surface antigen (HBsAg) among some tribes of Madhya Pradesh, Rajasthan and Maharashtra. *J Indian Anthropol Soc* 1990;25:68-72.
6. Prasad SR, Rodrigues FM, Dhorje SP, Ramamoorthy CL. Prevalence and subtypes of hepatitis B surface antigen in the tribal population of Arunachal Pradesh, India. *Indian J Med Res* 1983;78:300-6.
7. Chandra M, Khaja MN, Farees N, Poduri CD, Hussain MM, Aejaz Habeeb M, *et al.* Prevalence, risk factors and genotype distribution of HCV and HBV infection in the tribal population: a community based study in south India. *Trop Gastroenterol* 2003; 24:193-5.
8. Kalaivani V, Rajendran P, Thyagarajan SP, Rajesh PK, Hari R, Selvakumar C, *et al.* Seroprevalence of hepatitis B and C viruses and the associated risk factors in the Kolli hills tribal population of Tamil Nadu. *Biomedicine* 2001;21:7-13.
9. Dutta RN, Sen S. A study of Australia antigen, cold antibodies and ABO blood group frequencies in Ladakhies. *Indian J Med Res* 1975;63:1635-40.
10. Lau J, Ioannidis JPA, Schmid CH. Quantitative synthesis in systematic reviews. *Ann Intern Med* 1997;127:820-6.
11. Fleiss JL. *Statistical Methods for Rates and Proportions*, 2nd ed. New York: J Wiley. 1981: p. 161-5.
12. Murhekar MV, Murhekar KM, Arankalle VA, Sehgal SC. Epidemiology of hepatitis B infection among the Nicobarese, a mongoloid tribe of the Andaman and Nicobar Islands, India. *Epidemiol Infect* 2002;128:465-71.

**Correspondence to: Dr Murhekar, National Institute of Epidemiology, Mayor V Ramanathan Road, Chetput, Chennai 600 031. E-mail: mmurhekar@yahoo.com**

### Association of asthma and allergic rhinitis with celiac disease

The association of celiac disease (CD) with bronchial asthma and allergic rhinitis has been investigated

previously, with variable results.<sup>1-6</sup> We present our data on such an association in a largely in-bred population of about 400,000 persons residing in the Maltese Islands in the Mediterranean Sea.

Patients previously diagnosed to have CD (based on serological tests and duodenal biopsy) and attending a medical out-patient clinic answered a questionnaire designed to determine whether they had previously been diagnosed to have asthma or allergic rhinitis. They were also asked about symptoms suggestive of asthma; patients with such symptoms but no prior diagnosis of asthma underwent physiological lung tests to look for undiagnosed asthma. All patients provided informed consent. The frequency of asthma and allergic rhinitis in CD patients was compared with data from the International Study of Asthma and other Allergic Conditions in Childhood in the Maltese Islands (ISAAC1997), using *chi*-squared analysis.

All 86 patients (age range 16-69 [median 43] years; 65 female) answered the questionnaire about CD and asthma. They constituted 21% of the 409 patients with CD in the Maltese islands included in a register kept for controlling free prescription of gluten-free foods.

Of 86 respondents, 24 (27.8%; 21 female) had asthma, including 22 with known asthma and 2 with previously undiagnosed asthma; the frequency of asthma in CD patients was higher than that reported in the general Maltese population (11.1%;  $p < 0.00005$ ).<sup>7</sup> In addition, four non-asthmatic patients (one smoker, two ex-smokers, one non-smoker) reported wheezing in the absence of respiratory tract infection in the past; they however had normal pulmonary function tests. Another woman with CD gave history of wheezing and cough after exercise. She was a non-smoker, had family history of asthma, and her lung function tests showed 10% reversibility in FEV1 after the administration of bronchodilator. Another patient had nocturnal cough; he was an ex-smoker, had family history of asthma, and had normal lung function tests.

In 16 patients, asthma preceded CD by 3 months to 39 years (median 20 years). Among these patients, gluten-free diet had led to improvement in asthma in 6 patients, possible improvement in 2 patients, and no change in 8 patients. In the remaining 8 patients, asthma followed CD by 2 to 14 years (median 8). Thirty-one of 86 patients with CD and 11 of 24 patients with CD and asthma gave family history of asthma among first-degree relatives.

Eighty-two patients (62 female) answered the questionnaire about allergic rhinitis. Of these, 36 (44%) suffered from allergic rhinitis; this frequency was higher than that reported in the general Maltese population (32.3 %;  $p < 0.05$ ).<sup>7</sup>

Our findings suggest that asthma and allergic rhinitis are more common in CD patients than in the general population in Malta. In patients with atopic diseases, index of suspicion for CD should be high.

Pierre Ellul, Mario Vassallo, Stephen Montefort  
St. Luke's Hospital, Malta

### References

1. Zauli D, Grassi A, Granito A, Foderaro S, De Franceschi L, Ballardini G, *et al*. Prevalence of silent coeliac disease in atopics. *Dig Liver Dis* 2000;32:780-1.
2. Collin P, Reunala T, Pukkala E, Laippala P, Keyrilainen O, Pasternack A. Coeliac disease: associated disorders and survival. *Gut* 1994;35:1215-8.
3. Bottaro G, Cataldo F, Rotolo N, Spina M, Corazza GR. The clinical pattern of subclinical/silent celiac disease: an analysis on 1026 consecutive cases. *Am J Gastroenterol* 1999;94:691-6.
4. Greco L, De Seta L, D'Adamo G, Baldassarre C, Mayer M, Siani P, *et al*. Atopy and coeliac disease: bias or true relation? *Acta Paediatr Scand* 1990;79:670-4.
5. Hodgson HJF, Davies RJ, Gent AE, Hodson ME. Atopic disorders and adult coeliac disease. *Lancet* 1976;i:115-7.
6. Tarlo SM, Broder I, Prokipchuk EJ, Peress L, Mintz S. Association between celiac disease and lung disease. *Chest* 1981;80:715-8.
7. Montefort S, Lenicker HM, Caruana S, Agius Muscat H. Asthma and other allergic conditions in childhood in the Maltese Islands. *ISAAC* 1997.

Correspondence to: Dr Ellul, 29 Heapfold, Norden, Rochdale, Lancashire, OL 127 NR, England. E-mail: pierre\_ellul@yahoo.co.uk

### Pancreatic pseudocysts in brothers: familial, environmental or incidental?

A 42-year-old non-alcoholic man presented with upper abdominal pain and gradually increasing mass of 2 years and 6 months duration. There was no past history of acute pancreatitis, trauma, gallstone disease, jaundice or tuberculosis. Examination revealed a 6 cm x 7 cm ill-defined intra-abdominal mass in the epigastrium. The rest of the examination was unremarkable. Laboratory investigations and plain radiographs of the chest and abdomen were normal. Ultrasonography revealed a thick-walled cystic mass, 6 cm x 6 cm, in the lesser sac with internal echoes and echogenic debris. The head and body of the pancreas was irregular and appeared shrunken. A diagnosis of chronic pancreatitis with pancreatic pseudocyst was made. He underwent laparoscopic cystogastrostomy and made satisfactory recovery.