

results have been obtained in blind assays of samples with either the elaborate extraction (as used in the paper) or simple methods such as boiling or even 4 freeze/thaw cycles to liberate HPV before PCR. The consistency of these results with different extraction methods indicates that inhibitors are not a difficulty, and that routine testing for high levels of HPV by PCR could soon become practicable.

Imperial Cancer Research Fund,
PO Box 123,
London WC2A 3PX, UK

Department of Chemical Pathology,
University College London

Department of Medical Microbiology,
University College London

Department of Obstetrics,
City Hospital, Nottingham

Department of Pathology,
University Hospital, Nottingham

JACK CUZICK

GEORGE TERRY

LINDA HO

TONY HOLLINGWORTH

MALCOLM ANDERSON

Population movements and cholera spread in Cordillera Province, Santa Cruz Department, Bolivia

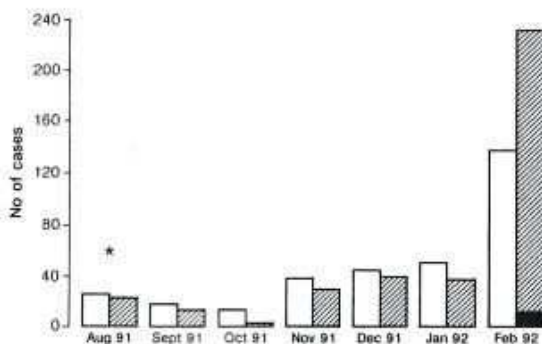
SIR,—The Latin-American cholera epidemic started on the Pacific coast of Peru in January, 1991, and spread rapidly throughout the continent. The causative agent is *Vibrio cholerae* O1, biotype El Tor, serotype Inaba (*Wkly Epidemiol Rec* 1991; 66: 47, 1992; 67: 33–39). By July, 1991, cholera had not yet reached Bolivia. We began a surveillance system for episodes of diarrhoeal diseases to detect promptly any cholera case in Cordillera Province, Santa Cruz, Bolivia.

Cordillera Province occupies 86 245 km² in south-eastern Bolivia. It borders with the Department of Chuquisaca, Tarija, and Paraguay, and is about 150 km from Argentina. 62 231 inhabitants (0.7/km²) were registered in 1987. There are a district hospital in Camiri town, nine area hospitals, and several "health places". Camiri is connected with Santa Cruz and the Argentine border by Carretera Panamericana, one of the most important South American routes.

A report form for episodes of diarrhoea was delivered to each of the ten hospitals at the end of July, 1991. The form, including the numbers of new cases by age, province, and outcome, was returned fortnightly to Camiri district hospital.

From August, 1991, to January, 1992, the number of diarrhoea cases in Camiri district hospital stayed fairly constant, the increase in November to January probably being related to the beginning of the rainy season (figure). The number of cases among children under six years of age was higher than that among adults. A similar trend of diarrhoea cases was seen in the nine area hospitals. No cholera cases were reported in this period.

In February, 1992, the total number of diarrhoea cases increased four-fold compared with January. The number in adults was 1.6 times that in children. The first cholera case in this area was confirmed on Feb 12, 1992 by the bacteriology unit of



New diarrhoea cases in Camiri area, Cordillera Province, Bolivia. <5-years-old (open), >5-years-old (hatched), and cholera-confirmed cases (black). *first cholera case in Bolivia, Aug 27, 1991.

CENETROP, where all fecal samples from suspected cases had been promptly dispatched. 9 further cases of cholera in the Camiri area and 6 cases from four other areas of the district were confirmed during February. The first cholera cases were detected in patients coming back from a two day meeting organised to commemorate the centenary of the last Guarani native indians' genocide. The first cholera case was reported in Bolivia in August, 1991, in Rio Abajo Region, and the epidemic was limited to the north of this country (*Wkly Epidemiol Rec* 1991; 66: 263). But it was impossible to postpone the gathering. About 6000 indians and others from Guarani areas of Argentina, Paraguay, and from northern Bolivian cholera-infected areas participated in the meeting in Kuruyuki, 65 km south of Camiri on Jan 28, 1992.

Santa Cruz Department was included in the list of newly infected areas on March 20, 1992, 6 months after the first notified cholera case in Bolivia (*Wkly Epidemiol Rec* 1992; 67: 87–88). The introduction of the cholera epidemic into Cordillera Province seems to have been favoured by the gathering of Guarani indians in Kuruyuki. Our data underline the role of large population movements and the gathering of people in overcrowded places as risk factors in the spread of cholera, and emphasise the importance of surveillance during the early phase of the epidemic to obtain descriptive information and define intervention priorities for high-risk groups.

Istituto di Malattie Infettive,
University of Siena,
I-53100 Siena, Italy

Academic Department of Infectious Diseases,
University of Florence

Unidad Sanitaria Santa Cruz,
District of Cordillera, Bolivia

Centro Nacional de Enfermedades Tropicales
"CENETROP", Santa Cruz de la Sierra

Laboratory of Medical Bacteriology and Mycology,
Istituto Superiore di Sanità,
Rome

Department of Epidemiology and Biostatistics,
Istituto Superiore di Sanità,
Rome

Academic Department of Infectious Diseases,
University of Florence

P. GUGLIELMETTI

A. BARTOLONI
M. ROSILLI

H. GAMBOA

D. J. ANTUNEZ

I. LUZZI

F. ROSMINI

F. PARADISI

Predisposition and ascaris infection

SIR,—In your May 23 editorial (p 1264), you emphasise the need to control intestinal helminth infections in children, and support the strategy of mass chemotherapy on the basis of the epidemiology and modes of transmission of such infections. There is, however, an important aspect of the editorial that deserves comment.

The term predisposition applied to helminth infections implies that individuals with heavy worm burdens are more likely to reacquire heavy infections after successful treatment, whereas lightly infected individuals tend to become lightly reinfected. This relation has been shown in schistosomes, *Opisthorchis*, and all the major geohelminth infections; it has been detected at all ages although schistosomes in adults seem to be an exception;¹ and it persists for several cycles of treatment.^{2,3} Predisposition has been noted in family units and in specific age-classes of a community,⁴ and does not imply that each individual maintains a consistent position in a ranking of susceptibility to helminth infections—merely that correlation analyses indicate that a significant proportion of individuals retain the same rank of intensity of infection before and after treatment and reinfection.⁵ The fact that some people do not reach the same ranked position is believed to be a result of errors of measurement, factors of chance that affect exposure to infective stages, and heterogeneity in factors that determine the establishment of worms.^{6,7} These consistent observations, supported by much evidence, were confirmed by our study reported in the same issue (p 1253). We therefore disagree with your editorial statement that the reported study "raises questions about the validity of . . . predisposition". Recorded patterns of the intensity of reinfection raise the interesting issue of what factor or combination of factors generates predisposition; this is the subject of much research at present.