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Commentary

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Since the exciting observation in Melbourne by Bishop et al.1 that rotavirus is a significant aetiologial agent for childhood gastroenteritis, this virus has been reported in the stools of many such children in developed countries, particularly during the winter.2-4 However, reports from the developing world have varied greatly both for the prevalence of rotavirus and for the prevalence of conventional enteropathogenic serogroups of E. coli (EPEC). For example, Taylor et al.5 found rotavirus in 45% of children with diarrhoea in a rural treatment centre in Bangladesh, whereas Robins-Browne et al. in Johannesburg, in the above report, found EPEC in 43% of black infants with acute summer diarrhoea. It seems unlikely that this difference in prevalence of aetiological agents is due to the method of detection, and most probably it does represent a genuine fact. Past experience in developed countries may be helpful. It is obvious that in Britain there has been a major change in the aetiology of gastroenteritis, and in the pattern of prevalence. Summer diarrhoea in the east end of London was recorded at the Queen Elizabeth Hospital for Children from 1885 to 1937 and there was a mortality of 40-50% for inpatients aged under 2 years, but the pattern of summer outbreaks ceased in 1937 and did not recur.6 The 1953 MRC multicentre trial of prophylactic antibiotics in infantile gastroenteritis reported a 37% incidence for the serotypes of E. coli, O111 and O55, the only two known at that time. By 1967 the incidence of these two serotypes had declined to 4% of infantile gastroenteritis in Manchester,7 and during the year August 1976-7 at Queen Elizabeth Hospital for Children, only 4% of 544 children with gastroenteritis had one of the larger number of currently recognised strains of EPEC identified in their stools. Indeed now in east London, instead of a summer peak for gastroenteritis there is a winter peak7 and the rotavirus has been recognised as a significant and important aetiological agent during this winter peak, with 50% of stools examined during the winter months containing this virus. In Sydney, the winter peak in gastroenteritis admissions was first observed in 1964, having not been present in the immediately preceding years (D Dorman, 1972, personal communication). Thus major changes over the years have taken place in Australia and Britain, both in the pattern of prevalence and in the aetiology of infantile gastroenteritis. Social and environmental factors have obviously played a major role in producing these changes.

While there is little doubt that improved nutrition, education, and living conditions do lead to great improvement in mortality and prevalence of childhood gastroenteritis, yet in east London such improvements have not led to a disappearance of gastroenteritis, although the prevalence of E. coli enteritis has fallen so strikingly and 'summer diarrhoea' has gone. Indeed the emergence of a winter peak in admissions in east London and in other Western communities has been unexpected. There is a clear need to study the present epidemiology of gastroenteritis in childhood throughout the world and within the UK itself to take account of the geographical variations in prevalence, and the implication such variations may have both for aetiology and prevention. Thus it would seem probable that social and environmental factors are important in the difference between Bangladesh and Johannesburg. An investigation of these factors is urgently needed.

References