

Less diarrhoea but no change in growth: 15 years' data from three Gambian villages

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Abstract

Aims—To review diarrhoea presentations and nutritional status in young rural Gambian children over a 15 year period as a test of an earlier hypothesis that reduced diarrhoea prevalence would lead to improved growth and a reduced prevalence of malnutrition.

Subjects and methods—Growth and morbidity were documented routinely in 1190 children under 2 years of age attending the Dunn Nutrition Group clinic at Keneba between 1979 and 1993.

Results—The numbers of presentations with diarrhoea (1069 in 1979; 220 in 1993) and the proportion of clinic attendees with diarrhoea (30% in 1979; 8% in 1993) fell steadily between 1979 and 1993. However, at both 1 year old, mean weights (Z scores: 1979, -1.8; 1993, -1.8) and mean lengths (Z scores: 1979, -1.3; 1993, -1.7), and at 2 years old, mean weights (Z scores: 1979, -2.0; 1993, -1.9) and mean lengths (Z scores: 1979, -2.0; 1993, -2.1) did not change noticeably over the 15 year period.

Conclusion—Major progressive reductions in clinic presentations with diarrhoea have not been associated with improved nutritional status in this population of young rural Gambian children.

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Keywords: diarrhoea; growth; nutrition

Diarrhoeal disease is widely recognised as a leading cause of morbidity and mortality among young children, particularly in developing countries. More than 880 000 children under 5 years of age died of diarrhoeal illness in sub-Saharan Africa in 1990.¹ Despite continuing high mortality in the developing world, the outlook for diarrhoea has improved over the past 20 years. Bern and colleagues² reviewed studies in the decade to 1990 and estimated a similar incidence for diarrhoeal disease (2.6 episodes/child/year) as 10 years previously,³ but a lower mortality; worldwide, 3.3 million children died of diarrhoeal disease each year compared with 4.6 million 10 years previously. More widespread knowledge and use of oral rehydration solution (ORS) are likely to account for at least some of the improvement in mortality rates.^{4 5}

For more than 30 years, the concept that malnutrition and acute infections interact to the disadvantage of young children has provided an explanation for persistent undernutrition and high young child mortality in many disadvantaged and/or developing societies.⁶

Among acute infections, diarrhoeal diseases are those considered most likely to lead to malnutrition.^{7 8} The children most at risk of persistent diarrhoea seem to be those already malnourished. Black and colleagues⁹ showed that Bangladeshi children developing diarrhoea had more prolonged illness if they were already undernourished. This concept of negative interaction between malnutrition and diarrhoea is enhanced by clinical experience. For example, in the late 1970s, regression analysis of data on disease incidence in young children attending the MRC Dunn Nutrition Group clinic in Keneba indicated a close relation between growth faltering and the incidence of diarrhoea.⁸ The data appeared to suggest that if the incidence of diarrhoeal disease could be reduced to low levels, then rates of growth in early childhood could be expected to follow normal centiles, except during the annual hungry season (during the rains).

The relevance of diarrhoeal illness to persistent growth faltering in early childhood among children in developing countries has been challenged. Briend *et al.*,¹⁰ studying children aged 6-35 months in Bangladesh, found that the medium to long term effects of diarrhoea on growth—both length and weight—were insignificant. Deficits acquired during acute diarrhoeal illness were transient and no longer apparent a few weeks later. Their conclusion was that efforts to control diarrhoea would be unlikely to improve children's nutritional status. Studies of young Indonesian children in the late 1980s also showed no cumulative effect of disease on growth performance.¹¹ Nevertheless, the concept of an important interaction between diarrhoea and growth faltering remains seminal to much public health planning.

We present prospectively collected data on diarrhoeal illness, which have enabled us to re-examine the interaction between this and early childhood growth in our three Gambian "study villages".

Methods

SOCIAL BACKGROUND

Since 1974 the Medical Research Council Dunn Nutrition Centre has had a field station in Keneba, The Gambia, studying nutrition in childhood, pregnancy, and lactation. All children under 2 years of age from Keneba and two neighbouring villages have been part of the unit's research cohort since that time. Morbidity has been documented through attendance at daily clinics, well baby (call) clinics, and emergency presentations. The nearest alternative health centre to Keneba is 15 km away, and not readily accessible by public transport.

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Table 1 Annual clinic attendances (all clinics) and attendances with diarrhoea ("incidence") for children less than 2 years old, 1979–93

| Year | Total attendances | Diarrhoea attendances | |
|-------|-------------------|-----------------------|------|
| | | Number | % |
| 1979 | 3537 | 1069 | 30.2 |
| 1980 | 4216 | 907 | 21.5 |
| 1981 | 3836 | 948 | 24.7 |
| 1982 | 3761 | 846 | 22.5 |
| 1983 | 3826 | 825 | 21.6 |
| 1984 | 3843 | 795 | 20.7 |
| 1985 | 3776 | 700 | 18.5 |
| 1986 | 3895 | 574 | 14.7 |
| 1987 | 3178 | 566 | 17.8 |
| 1988 | 3493 | 526 | 15.1 |
| 1989 | 3300 | 565 | 17.1 |
| 1990 | 3268 | 407 | 12.5 |
| 1991 | 2860 | 281 | 9.8 |
| 1992 | 2895 | 263 | 9.1 |
| 1993 | 2893 | 220 | 7.6 |
| Total | 52577 | 9492 | 18.1 |

Adults, particularly men, visit traditional healers readily, but young children are taken to traditional healers only very rarely and then usually when Western medicine is deemed to have failed. Thus, we have almost complete documentation of morbidity in children under 2 years of age resident in the three study villages.

Typically, young children in this part of The Gambia have a low average birth weight compared with international standards (mean, 2700–2900 g, depending on season), but there is rapid catch up growth in the first 3–4 months of exclusive breast feeding, so that 3 month old babies have average weights close to those of international standards.¹² Commonly, after 4 months, there is a faltering in weight and length increments, which persists until around 21 months, when growth gradually assumes velocities more or less equivalent to international standards.¹²

CLINICAL DATA COLLECTION

Clinics are held at Keneba morning and evening, seven days a week, and medical advice is available for emergencies at any time of the day or night. Children under 2 years are seen by one of the unit paediatricians or by one of two experienced nurses/midwives, and diagnosis and treatment are recorded at each attendance. Diagnostic criteria are based largely on

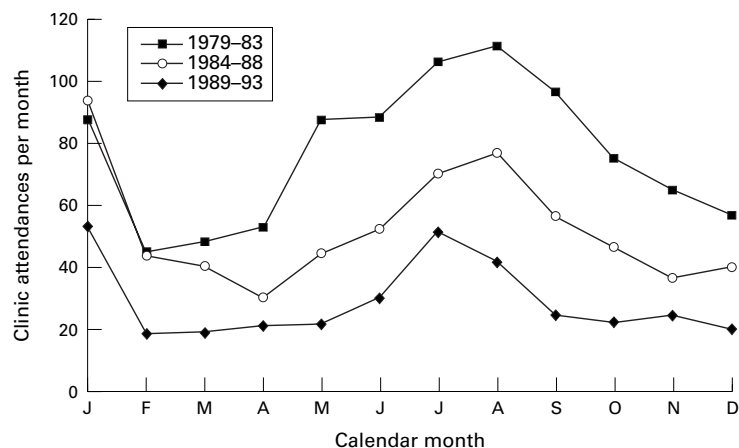


Figure 1 Five year averages for clinic attendance with diarrhoea in children under 2 years of age for the periods 1979–83, 1984–88, 1989–93.

clinical findings because microbiological services are limited.

All newborns are visited by a paediatrician in the 1st few days of life so dates of birth are known precisely. Children are "called" to well baby clinics for immunisation and growth/development assessment nine times in the 1st year of life and four times in the 2nd year of life. Children failing to attend these "call" clinics are followed up so that all are likely to be reviewed regularly, provided they remain resident in one of the three study villages.

The same basic clinical criteria have been used over the years to record diagnoses. This paper is confined to presentation of diarrhoeal disease, which has been defined as four or more stools/day and/or a significant change in stool consistency. Other diseases commonly recorded in the clinic are upper and lower respiratory tract infections, eye and skin infections and, during the rainy season and shortly after, malaria.

Medical personnel usually spend two years at the unit. Some variation in coding of diagnoses as a result of individual diagnostic and therapeutic practices has been inevitable over the years, but we have found no evidence to suggest that changes in the frequency of diagnoses are significantly related to the residency of particular clinicians.

We present data on the number of attendances with diarrhoea at all clinics as proxy for the incidence of diarrhoea at any one time. We make an estimation of the duration of diarrhoea by reviewing the next attendance of each case at the clinic and whether he or she complained of diarrhoea at the second attendance. We use the documentation of diarrhoea at call clinics as proxy for prevalence of diarrhoea because children were brought to the clinic on those occasions for review and did not attend specifically for treatment of symptomatology.

ANTHROPOMETRY

Weight unclothed was recorded in kilograms (to two decimal places) by trained technicians each time a child attended the clinic. In the early years of the study, manually operated baby scales (Todd Scales, Cambridge, UK) but, later, Seca electronic baby scales (CMS, London, UK) were used. Lengths were measured on Holtain infantometer (Holtain, Crymmych, UK) but more recently on Kiddimetre (Raven Equipment, Great Dunmow, Essex, UK). Lengths were recorded only at call clinics. Weight and lengths were related to international standards¹² to develop Z or standard deviation scores.

Results

POPULATION

The total population of the three villages increased from 1500 in 1979 to 2700 in 1993. Annual births averaged 98 in the quinquennium 1979–83; 92 in 1984–88; and 108 in 1989–93. Overall, 1190 children aged 0–2 years are involved in our analysis.

Table 2 Annual call clinic attendances and attendances with diarrhoea ("prevalence") for children less than 2 years old, 1979-93

| Year | Total attendances | Diarrhoea attendances | |
|-------|-------------------|-----------------------|------|
| | | Number | % |
| 1979 | 2532 | 871 | 34.4 |
| 1980 | 2707 | 706 | 26.1 |
| 1981 | 2572 | 733 | 28.5 |
| 1982 | 2675 | 694 | 25.9 |
| 1983 | 2793 | 693 | 24.8 |
| 1984 | 2800 | 665 | 23.8 |
| 1985 | 2766 | 595 | 21.5 |
| 1986 | 2973 | 508 | 17.1 |
| 1987 | 2531 | 506 | 20.0 |
| 1988 | 3342 | 518 | 15.5 |
| 1989 | 2403 | 493 | 20.5 |
| 1990 | 2238 | 343 | 15.3 |
| 1991 | 1797 | 235 | 13.1 |
| 1992 | 1778 | 231 | 13.0 |
| 1993 | 1743 | 180 | 10.3 |
| Total | 37650 | 7971 | 21.2 |

MORTALITY

Infant mortality has not changed appreciably over the 15 year period (1979-83, 44/1000; 1984-88, 39/1000; and 1989-93, 48/1000).

DIARRHOEAL DISEASE

Table 1 shows the total number of clinic attendances for children under 2 years over the period 1979-93, together with the number and

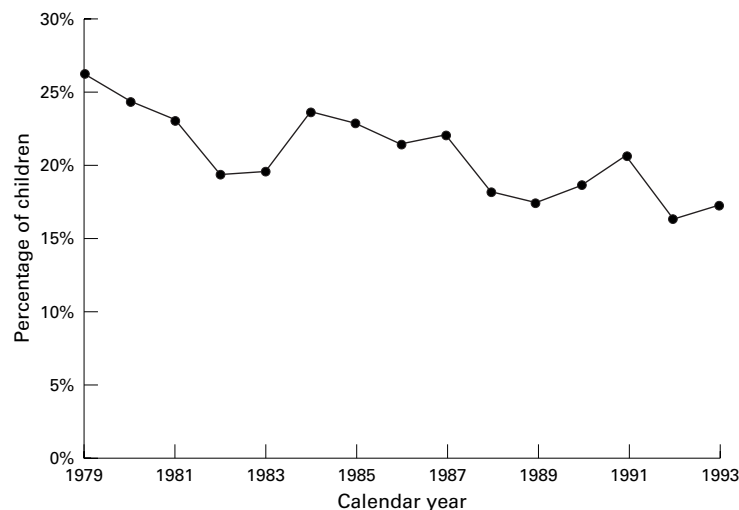


Figure 2 Percentage of children under 2 years of age returning to clinic within 10 days of first presentation still suffering from diarrhoea, 1979-93.

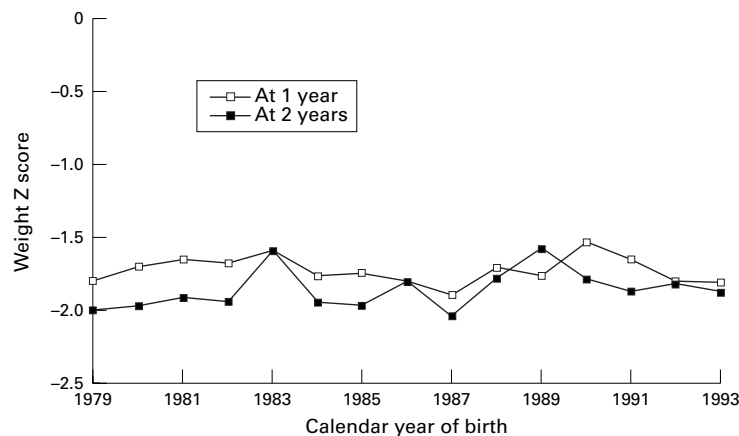


Figure 3 Mean weight Z score for children at 1 year and 2 years of age, 1979-93.

percentage of all attendances in this age group caused by diarrhoea. It is obvious that there have been large changes in both sets of figures. However, the distribution of diarrhoea over the years follows similar patterns (fig 1), with consistent peak levels in the rainy season (July to September) and a small peak early in the year, previously shown to be caused by rotavirus epidemics.¹³ This seasonality is consistent with an overall reduction of diarrhoeal illness, rather than a reduction in particular epidemic pathogens.

Table 2 shows a very similar pattern for diarrhoea among call clinic attendees, suggesting that both the incidence of diarrhoea presenting at the clinic and the prevalence have declined. Figure 2 shows that diarrhoea is less prolonged. In 1979, 26% of children returned within 10 days still complaining of diarrhoea, whereas in 1993 the same figure was 17% of children, and most returned in the early days of the disease.

WEIGHT AND LENGTH

Figure 3 shows mean weight Z scores at 1 and 2 years of age according to the year of birth, based on an annual average of 80 weights at 1 year and 55 at 2 years. There is some movement away from the villages as mothers may "transfer" after childbirth to live with their husbands in urban areas, and in the 2nd year, as children are weaned from the breast and sent to grandmothers. Despite a dramatic reduction in the incidence and prevalence of diarrhoeal disease, there was no improvement in weight gain in the study children over the first 2 years of life for the period 1979-93. Mean weight Z scores (SD) at one year were -1.8 (0.9) in 1979 and -1.8 (1.1) in 1993. At 2 years, these figures were -2.0 (0.7) in 1979 and -1.9 (0.9) in 1993. Likewise, length measurements showed no significant change over this period. Mean length Z scores at 1 year were -1.3 (1.0) in 1979 and -1.7 (1.0) in 1993. Corresponding mean length Z scores at 2 years were -2.0 (0.9) in 1979 and -2.1 (0.9) in 1993. The similarity in SD of the Z scores over the survey period rules out the possibility that the lack of change in mean Z scores is a result of some children doing better, but others doing very badly yet surviving, during the latter part of the study, whereas they might have died in the early years of study.

Discussion

The fall in the number of children presenting to our clinics since 1979 with symptoms of diarrhoea is impressive. It could be argued that this reflects greater ability of parents to manage diarrhoea at home since the introduction of ORS,¹⁴ with its availability from the unit or from individuals within the villages altering the management of diarrhoea. However, the decline in the number of children attending with diarrhoea has been fairly steady over the years, rather than occurring in the early 1980s when the introduction of ORS might have been expected to have had an impact. Thus, we believe that the fall in cases cannot be fully explained by the use of ORS. Furthermore, we continue to see most children with diarrhoea in

the first days of illness, whereas if most cases were managed at home, we would expect that only those whose home treatment had failed would attend the clinic. In this case, presentation would be later into the illness and diarrhoea more likely to be protracted. Our evidence is that not only have the numbers of presentations with diarrhoea fallen but so have the number of days children are ill with each episode of diarrhoea. Moreover, diarrhoea prevalence as suggested by call clinic data also indicates that diarrhoea is less common than in the late 1970s.

The trend towards less severe diarrhoea fits in with more widespread surveys. The overall decline in diarrhoea incidence is less well documented. As mentioned above, Bern and colleagues² found the prevalence of diarrhoea worldwide to be about the same as 10 years before, but the overall morbidity appeared significantly less. It would be heartening to feel that the decline in diarrhoea was a reflection of greater resistance to the complications of diarrhoeal infections owing to improved nutrition. Clearly, with our children this cannot be substantiated. We have to accept that the position we adopted in the 1970s,⁸ that removal of diarrhoea from the population of young children would result in almost normal growth, is no longer tenable. Despite an ~75% drop in diarrhoeal illness and a reduction in the number of days ill with diarrhoea, the average weights and lengths at 1 and 2 years of age in our population have not increased over the years 1979–93.

The change in the number of children presenting with diarrhoea is dramatic. Why has this occurred? Poor quality water sources and inadequate sanitation are often considered as contributing to the prevalence of diarrhoea.^{15–16} Have changes in these accounted for the changes in the incidence of diarrhoea in our villages over the 15 years of study? In the early days of the Dunn Nutrition Group in Keneba, most water was obtained from hand drawn wells on the compounds.¹⁷ There were fewer latrines and specific areas of the bush were often used for sanitary purposes. Now one village has four taps delivering water from a borehole and all villages have covered hand pumped wells. Open wells on the compounds have largely run dry owing to a falling water table and lack of use because of other water sources. Most villagers have shallow pit latrines on their compounds. These changes have all been present for the past 10 years and do not adequately explain the progressive fall in diarrhoea. A study here in 1990, involving a health promotion programme which stressed the importance of hand washing, showed decreased skin and eye infections and reduced rates of diarrhoea but no decrease in respiratory tract infections in the children of families receiving the hand washing advice.¹⁸ However, this project cannot account for the decline in diarrhoeal incidence and prevalence before 1990. After 1990, the decline seems to continue a steady downward, rather than sudden stepwise, trend. Studies elsewhere have suggested that it is only with substantial

Key messages

In a rural West African community:

- Diarrhoea is less common in young children than 15 years ago
- Episodes of diarrhoea are shorter
- Reduction in diarrhoeal disease has not been associated with improved young child growth

changes in sanitation and water sources (flush toilets as well as tapped water supply) that falls in diarrhoea prevalence are associated with water/hygiene developments.^{19–20}

We are disappointed by the lack of association between reduction in diarrhoea cases and nutritional status of the children. However, our findings are not unique in this respect.^{10–11} A recent retrospective study of Aboriginal children in north west Australia²¹ from 1969 to 1993 demonstrated that declines in the incidence of low birth weight, mortality, and infectious disease were not accompanied by improvements in the growth patterns of children 0–60 months old. Reductions in the incidence and prevalence of diarrhoea are undoubtedly important for child health and morbidity; but it is clear that policies to reduce diarrhoea cannot alone be expected to achieve the hoped for improvements in growth and in rates of malnutrition in young children.

It could be interpreted that our study shows the importance of inadequate dietary intake, as opposed to infection, in leading to growth faltering and failure of catch up growth in poor village children. Yet we have shown previously that communal macronutrient supplementation of village weanlings does not improve overall growth.²² Catch up growth has been induced in our young child population after diarrhoea by ensuring energy intakes 50% in excess of recommendations and protein intakes 100% more than recommended.²³ But such intakes only achieved rapid catch up growth during the period of supplementation and even then the rates of growth fell far below the catch up expected in recovery from acute severe protein energy malnutrition: kwashiorkor and marasmus.²⁴ That catch up growth can be induced with these very high intakes is not in itself evidence that poor growth is caused by intakes below recommended levels. Energy and protein supplementation can induce improved growth in conditions such as cystic fibrosis²⁵ and in high output congenital heart disease.²⁶ This does not indicate that affected children are necessarily having intakes less than recommended, but that extra food can overcome some of the consequences of illness.

We are still left with the question: why do young children in so many underdeveloped rural communities grow so poorly? In Keneba we have studied the effects of providing macronutrient supplementation^{22–23} and micronutrient supplementation^{27–28}; we have succeeded in reducing the prevalence of severe illness and mortality in young children²⁹; we have carried out vigorous programmes of nutrition

education¹⁸; and currently we are in the process of investigating the role of enteropathy in young Gambian children.³⁰⁻³¹ Yet we continue to see patterns of growth that define half the village children as moderately malnourished (< 80% expected weight for age) by the end of their 1st year, despite above average rates of growth in the first 4 months of life.

It is sometimes suggested to us that the "growth faltering" we are studying is "normal growth" for these children. This is not likely, given that they show such good growth in the first 4 months. The shape of the growth curve is not equivalent to any known normal childhood growth pattern. Moreover, within our population there are some children who do pursue expected patterns of growth. We must accept that the average growth pattern of Keneba children is abnormal and pathological.

Again, it could be argued that if these children grow poorly because of malnutrition this might be a natural way of using the limited amounts of food available to best advantage. However, small is not beautiful in terms of childhood nutrition. There is good evidence that even mildly malnourished children are more at risk of death from infection³²; low physical fitness as older children and adults³³; neurodevelopmental impairment³⁴⁻³⁵; and a greater propensity to conditions such as hypertension³⁶ and diabetes³⁷ in later life. We might be unable to explain nutritional stunting in disadvantaged populations, but we believe that it is a problem that we must continue to explore for the benefit of present and future generations.

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- 1 Murray CDL, Lopes AD. *Disease burden expenditure and intervention packages*. Geneva: WHO, 1994.
- 2 Bern C, Martinez J, de Zoysa I, Glass RI. The magnitude of the global problem of diarrhoeal disease: a ten year update. *Bull WHO* 1992;70:705-14.
- 3 Snyder JD, Merson MH. The magnitude of the global problem of acute diarrhoeal disease: a review of active surveillance data. *Bull WHO* 1982;60:605-13.
- 4 Rahaman MM, Aziz KMS, Patwari Y, Munshi MH. Diarrhoeal mortality in two Bangladeshi villages with and without community-based oral rehydration therapy. *Lancet* 1979;ii:809-16.
- 5 Cutting WAM. Oral therapy at home for diarrhoea. In: Gracey M, ed. *Diarrhoeal disease and malnutrition*. Edinburgh: Churchill Livingstone, 1985:158-82.
- 6 Scrimshaw NS, Taylor CE, Gordon JE. Interaction of nutrition and infection. *Am J Med Sci* 1959;237:367-403.
- 7 Mata L. Diarrhoeal disease as a cause of malnutrition. *Am J Trop Med Hyg* 1992;47:16-27.
- 8 Rowland MGM, Cole TJ, Whitehead RG. Quantitative study into the role of infection in determining nutritional status in Gambian village children. *Br J Nutr* 1977;37:441-50.
- 9 Black RE, Brown KH, Becker S, Yunus M. Longitudinal studies of infectious disease and physical growth of children in rural Bangladesh. 1: Patterns of morbidity. *Am J Epidemiol* 1982;115:305-14.
- 10 Briend A, Hasan KZ, Aziz KMA, Hoque BA. Are diarrhoea control programmes likely to reduce childhood malnutrition? Observations from rural Bangladesh. *Lancet* 1989;iii:319-22.
- 11 Kolsteren PWV, Kusin JA, Kardjati S. Morbidity and growth performance of infants in Madura, Indonesia. *Am Trop Paediatr* 1997;17:201-8.
- 12 World Health Organisation. *Measuring change in nutritional status*. Geneva: World Health Organisation, 1983.
- 13 Rowland MGM, Leung TSM, Marshall WC. Rotavirus infection in young Gambian children. *Trans R Soc Trop Med Hyg* 1980;74:663-5.
- 14 Rowland MGM, Barrell RAE. The effect of early glucose electrolyte therapy on diarrhoea and growth in rural Gambian village children. *J Trop Paediatr* 1980;26:54-7.

- 15 Wright CE, Alamy ME, DuPont HL, et al. The role of home environment in infant diarrhoea in rural Egypt. *Am J Epidemiol* 1991;134:887-94.
- 16 Alam N, Wojtyniak B, Henry FJ, Rahaman MM. Mothers' personal and domestic hygiene and diarrhoeal incidence in young children in rural Bangladesh. *Int J Epidemiol* 1989;18:242-7.
- 17 Rowland MGM, McCollum JPK. Malnutrition and gastroenteritis in The Gambia. *Trans R Soc Trop Med Hyg* 1977;71:199-203.
- 18 Hoare K. Tackling infant malnutrition in The Gambia. *Health Visitor* 1994;67:102-3.
- 19 Kolsky PJ. Diarrhoeal disease: current concepts and future challenges. *Trans R Soc Trop Med Hyg* 1993;87(suppl 3):43-6.
- 20 Esrey SA. Water, waste and well-being: a multi-country study. *Am J Epidemiol* 1996;143:608-23.
- 21 Rousham EK, Gracey M. Persistent growth faltering among aboriginal infants and young children in North West Australia: a retrospective study from 1969-1993. *Acta Paediatr* 1997;86:46-50.
- 22 Prentice A. Nutrient requirements for growth, pregnancy and lactation: the Keneba experience. *South African Journal of Clinical Nutrition* 1993;6:33-8.
- 23 Hoare S, Poppitt SD, Prentice AM, Weaver LT. Dietary supplementation and rapid catch-up growth after acute diarrhoea in childhood. *Br J Nutr* 1996;76:479-90.
- 24 Ashworth A. Growth rates in children recovering from protein-energy malnutrition. *Br J Nutr* 1969;23:835-45.
- 25 MacDonald A. Nutritional management of cystic fibrosis. *Arch Dis Child* 1996;74:81-7.
- 26 Jackson M, Poskitt EME. The effects of high energy feeding on energy balance and growth of infants with congenital heart disease and failure to thrive. *Br J Nutr* 1991;65:131-43.
- 27 Bates CJ, Prentice AM, Paul AA, Prentice A, Sutcliffe BA, Whitehead RG. Riboflavin status in infants born in rural Gambia and the effects of a weaning food supplement. *Trans R Soc Trop Med Hyg* 1982;76:253-8.
- 28 Bates CJ, Evans PH, Dardenne M, et al. A trial of zinc supplementation in young rural Gambian children. *Br J Nutr* 1993;69:243-55.
- 29 Lamb WH, Foord FA, Lamb CM, Whitehead RG. Changes in maternal and child mortality rates in three isolated Gambian villages over ten years. *Lancet* 1984;ii:912-14.
- 30 Lunn PG, Northrop-Clewes CA, Downes RA. Intestinal permeability, mucosal injury and growth faltering in Gambian infants. *Lancet* 1991;338:907-10.
- 31 Weaver LT. Malnutrition and the mucosal response. In: Auricchio R, Troncone R, eds. *Dynamic nutrition research*. Basel: Karger, 1995:153-60.
- 32 Pelletier DL, Frongillo Jr EA, Schroeder DG, Habicht J-P. The effects of malnutrition on child mortality in developing countries. *Bull WHO* 1995;73:443-8.
- 33 Spurr GB. The impact of chronic undernutrition on physical work capacity and daily energy expenditure. In: Harrison GA, Waterlow JC, eds. *Diet and disease. Society for the study of human biology: symposium no 30*. Cambridge: Cambridge University Press, 1990:24-61.
- 34 Grantham-McGregor SM, Powell C, Stewart ME, Schofield WN. Longitudinal study of growth and development in young Jamaican children recovering from severe malnutrition. *Dev Med Child Neurol* 1982;24:321-31.
- 35 Galler JR, Ross RN. Malnutrition and mental development. In: Suskind RM, Lewinter-Suskind L, eds. *Textbook of pediatric nutrition*, 2nd ed. New York: Raven Press, 1993:173-9.
- 36 Barker DJP, Winter PD, Osmond C, Margetts BM, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. In: Barker DJP, ed. *Fetal and infant origins of adult disease*. London: BMJ Publishing Group, 1992:141-9.
- 37 Barker DJP. *Mothers, babies and disease in later life*. London: BMJ Publishing Group, 1994:80.

Commentary

Poskitt *et al* report that, despite a decline in the prevalence and duration of diarrhoeal disease in children younger than 2 years in three Gambian villages served by a single clinic, the prevalence of undernutrition has remained unchanged over 15 years. This paper adds valuable data to the debate, highlighted by Briend,¹ that childhood undernutrition is likely to result as much from poor food intake as from recurrent diarrhoea.

The continuous collection of data on diarrhoeal prevalence through clinic attendance rates, and nutritional status by serial anthropometry, offers a rare opportunity to examine this question, but the absence of data about dietary intake makes it difficult to reach a firm conclusion.

Growth is a function of food intake, energy-nutrient retention, and utilisation, and under-

nutrition must be caused by one or more of a combination of factors—insufficient intake, defective digestion or absorption, increased metabolic demands, or excessive losses (largely from the gastrointestinal tract).² While it may be true that it does not follow that if growth can be achieved by dietary “super-supplementation”³ undernutrition is caused by inadequate intake alone (as the authors assert); their findings focus attention on the “intake” and “utilisation” components of the energy–balance equation.

The enteropathy present in most of these Gambian children⁴ may well lead to suboptimal absorption, and recurrent or chronic infections contribute to a systemic inflammatory response that increases energy and nutrient needs. The incidence of chronic infection is not trivial. We have recently shown in the same population of children that *Helicobacter pylori* infection acquired in early life contributes to the growth faltering.⁵ There is a significant association between *H pylori* and undernutrition,⁶ by 3 years old the vast majority of children are colonised with the microorganism,⁷ and breast milk with high titres of anti-*H pylori* IgA may be protective.⁸ What effect this chronic infection has on gastrointestinal function and growth is the subject of much interest worldwide.^{9 10}

The conclusion of Briend¹ that childhood undernutrition is caused as much by inadequate food intake as to recurrent diarrhoea remains very plausible but unconfirmed. Poskitt *et al*, by

demonstrating that poor growth persists when the burden of diarrhoea lessens, focuses the debate on nutrient intake and utilisation rather than loss, and challenges us to investigate the adequacy of the diet young children receive (not just after episodes of diarrhoea), how that is handled by the gut, and the ability of children—undernourished and with chronic microbial colonisation—to utilise it thereafter.

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- 1 Briend A. Is diarrhoea a major cause of malnutrition among the under-fives in developing countries? A review of available evidence. *Eur J Clin Nutr* 1990;44:611–28.
- 2 Weaver LT. Feeding the weanling in the developing world: problems and solution. *Int J Food Sci Nutr* 1994;45:127–34.
- 3 Hoare S, Poppitt SD, Prentice AM, Weaver LT. Dietary supplementation and rapid catch-up growth after acute diarrhoea in childhood. *Br J Nutr* 1996;76:479–90.
- 4 Lunn PG, Northrop-Clewes CA, Downes RA. Intestinal permeability, mucosal injury and growth faltering in Gambian infants. *Lancet* 1991;338:907–10.
- 5 Dale A, Thomas JE, Darboe MK, Coward WA, Harding M, Weaver LT. *Helicobacter pylori* infection, gastric acid secretion, and infant growth. *J Pediatr Gastroenterol Nutr* 1998;26:393–7.
- 6 Sullivan PB, Thomas JE, Wight DGD, *et al*. *Helicobacter pylori* in Gambian children with chronic diarrhoea and malnutrition. *Arch Dis Child* 1990;65:189–91.
- 7 Thomas JE, Dale A, Harding M, Coward WA, Cole TJ, Weaver LT. *Helicobacter pylori* colonisation in early life. *Pediatr Res*. [In press.]
- 8 Thomas JE, Austin S, Dale A, *et al*. Specific human milk IgA antibody protects against *Helicobacter pylori* infection in infancy [letter]. *Lancet* 1993;392:121.
- 9 Sack RB, Gyr K. *Helicobacter pylori* infection in the developing world. *Lancet* 1993;341:1274–5.
- 10 Weaver LT. *Helicobacter pylori* infection, nutrition and growth of West African infants. *Trans Roy Soc Trop Med Hyg* 1995;89:347–50.