

Dietary supplementation and rapid catch-up growth after acute diarrhoea in childhood

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Diarrhoea is a major cause of short-term growth faltering in children of the developing world. If catch-up weight gain is delayed by inadequate dietary intake, or by further bouts of diarrhoea, progressive growth failure occurs. To test the hypothesis that early refeeding is as effective as later feeding after acute diarrhoea with weight loss, we measured the effects of a timed dietary intervention on weight gain after acute diarrhoea in underweight Gambian children. Thirty-four children aged 4–22 months with weight loss following acute diarrhoea were given a high-energy–protein supplement for 14 d beginning either immediately after rehydration or a fortnight later. With a 50% increase in energy intake and a 100% increase in protein intake there was a rapid and highly significant ($P < 0.001$) gain in weight within a fortnight whether the supplement was given immediately or 2 weeks after presentation. Rates of weight increase were similar whether supplementation was provided early or late, but over the full 28 d (of intervention and non-intervention) children who received late supplementation had greater overall weight gain ($P < 0.02$) than those supplemented early. Vigorous and early feeding with a high-energy–protein supplement should be central to the management of malnourished children with acute diarrhoea in developing countries, and may be as important as control of diarrhoea in preventing malnutrition and growth failure. This may be achieved in the community using locally available foods, in the face of continuing diarrhoea.

Diarrhoea: Growth: Children: Dietary supplementation

There has been much debate concerning the relationship between childhood diarrhoea and malnutrition, and whether control of diarrhoea or improvement of dietary intake should be regarded as the intervention most likely to prevent and reverse the growth faltering so commonly seen in children of developing countries (Briend *et al.* 1989; Briend, 1990; Black, 1991).

Diarrhoea is a major cause of acute growth faltering in Gambian children, and by the age of 2 years most are well below their expected weight-for-age. Growth faltering begins with the introduction of weaning foods at 3–6 months, and does not improve until the third year (Rowland *et al.* 1977).

Although there are many adverse factors that contribute to poor growth (dietary, gastrointestinal, metabolic, immunological, microbiological; Lebenthal, 1989), the capacity of the undernourished child to show catch-up growth after diarrhoea is likely to be influenced primarily by the quality and quantity of dietary intake (Jackson, 1990; Jackson & Wootton, 1990). The timing of increased intake after acute weight loss may be important

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and this has led to the concept of a short-lived 'anabolic window', present immediately after acute weight loss, when growth potential may be maximized with sufficient and appropriate dietary intake. Countering this is the incapacity of the digestive system, compromised by acute gastroenteritis, to make full use of nutrients, resulting in malabsorption and continued diarrhoea.

The aim of the present study was to test the hypothesis that in children with weight loss following acute diarrhoea, short-term dietary supplementation with a high-energy-protein food promotes rapid catch-up weight gain, and that the timing of increased intake is important.

SUBJECTS

This prospective community study was carried out at the Medical Research Council (MRC) Dunn Nutrition Unit in the rural Gambian village of Keneba (Lamb *et al.* 1984), and ran from June 1990 to February 1991, the period of peak diarrhoeal prevalence.

Forty children, aged 4–22 months, with diarrhoea with or without vomiting for at least 48 h and 5% weight loss were studied with the consent of their parents and with permission of the local Ethical Committee. They were enrolled at the MRC clinic and thereafter managed at home and at an out-patient feeding centre. Diarrhoea was defined as the passage of two or more loose stools per 24 h and was confirmed by examination of a stool sample on presentation. Vomiting was defined as regurgitation of a volume of 10 ml or more. Children who required intravenous rehydration, nasogastric tubes or antibiotics were excluded. All were under regular medical supervision throughout.

Six children left the study after enrolment, three because of antibiotic requirements (two with lower respiratory tract infections, and one with severe skin sepsis) and two because of failed oral rehydration. One child died from a septicaemia. Thirty-four children completed the study.

Children were randomly assigned to an early nutritional intervention group (group E: n 18, eleven males, seven females) or to a late nutritional intervention group (group L: n 16, five males, eleven females). When clinically rehydrated with a World Health Organization-recommended oral rehydration solution (ORS), children in group E were given a high-energy-protein supplement daily for 2 weeks, administered under supervision by the mother or a trained nursemaid, whereas children in group L returned home to their normal diet (Fig. 1). After 14 d supplementation the children of group E returned to their home diet and those of group L received the high-energy-protein supplement from day 15 to day 28. Day 0 (d0) was the day that the child presented at the clinic with diarrhoea, and day 1 (d1) was the first day of study after rehydration. Each child was studied for 42 d. The study was accompanied by an active health and food education programme.

Rehydration was carried out in the feeding centre where intake of ORS was measured. Thereafter ORS was freely available and mothers were advised to give it to their children whenever they passed loose stools. Breast feeding was encouraged throughout the study. To ensure maximal intake of the supplement, parents were requested not to give home foods to their children during the intervention periods.

Table 1 shows the characteristics of the thirty-four children studied. All were clinically rehydrated within 24 h of entry to the study. None had, or developed, oedema, passed dysenteric stools, or had fever for more than 24 h. The mean weight of children in group E on d1 was lower than that of group L ($P < 0.05$). Mean weight-for-age (WFA), weight-for-height (WFH) and height-for-age (HFA), derived from National Center for Health Statistics standards (Hamill *et al.* 1979) were not significantly different.

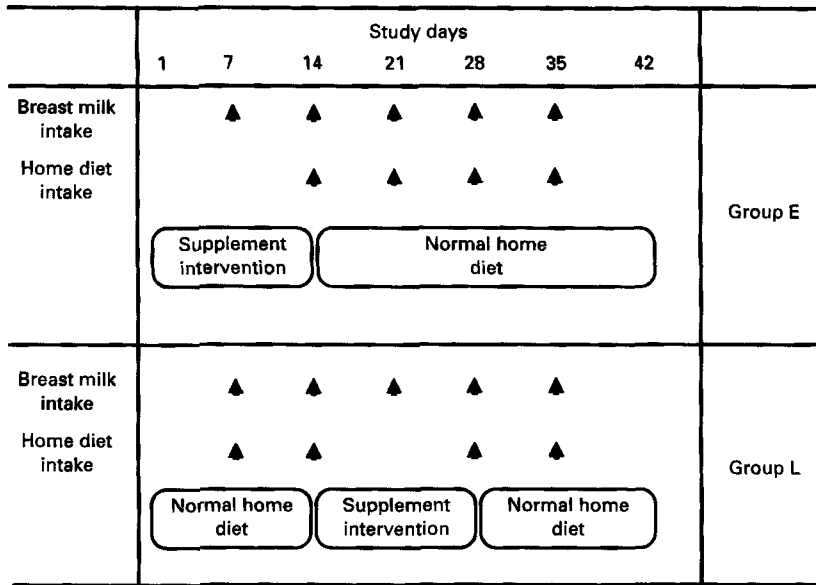


Fig. 1. Summary of study protocol. Nutritional supplementation was provided on days 1–14 for the early (E) group and from days 15–28 for the late (L) group. (▲), Measurements of breast milk and home food intake.

Table 1. Characteristics of subjects in early (E) and late (L) intervention groups at enrolment

(Mean values and standard deviations)

	Group E (n 18)		Group L (n 16)	
	Mean	SD	Mean	SD
Age (months)	10.8	3.2	13.0	4.4
Wt (kg) at presentation (day 0)	6.74	0.80	7.64*	1.27
Wt (kg) after rehydration (day 1)	6.88	0.79	7.76*	1.19
SD score† (WFA) (day 1)	-2.43	0.79	-1.82	0.91
SD score (WFH) (day 1)	-1.58	0.83	-1.27	0.82
SD score (HFA) (day 1)	-1.68	0.79	-1.23	1.02
Duration of diarrhoea before entry to study (d)	4.1	4.3	4.8	4.3

WFA, weight-for-age; WFH, weight-for-height; HFA, height-for-age.

* Mean values were significantly different from those of group E, $P < 0.05$.

† SD (Z) scores, based on measurements after rehydration (day 1), were derived from National Center for Health Statistics standards (Hamill *et al.* 1979).

METHODS

The high-energy-protein supplement consisted of a maize-soyabean flour (8 parts), vegetable oil (7 parts), dried skimmed milk (8 parts), sugar (7 parts), and water (66 parts) cooked and thinned using a locally prepared amylase (*EC* 3.2.1.1)-rich flour made from germinated maize seeds (Editorial, 1991). Local fruits and honey were used as flavourings to reduce food monotony. Vitamins and minerals were added to the feeds so that daily intake met or exceeded the recommended dietary intake for developing countries (Truswell, 1983). The supplement (Table 2) was offered to the children every 2 h from 07.30 to 19.30

Table 2. *Composition of the high-energy-protein supplement**

Nutrient	Concentration (per 100 g)
Carbohydrate (g)	16.6
Fat (g)	7.7
Protein (g)	4.5
Energy (kJ)	669
Vitamin A (μg)	1240
Vitamin D (μg)	10
Vitamin C (mg)	53
Folate (μg)	2500
Thiamin (mg)	1.0
Calcium (mg)	137
Iron (mg)	37.5
Zinc (mg)	2.0

* Provided (% energy): carbohydrate 41, fat 47, protein 12.

hours. Intake was calculated by weighing, and losses from vomiting were estimated using weighed bibs. Nude weight was recorded on a digital integrating electronic balance accurate to 5 g (SECA Ltd, Birmingham, West Midlands) from d1 to d28 and on d35 and d42. Length was measured at weekly intervals from d1 to d42 using a calibrated length-board (Holtain Ltd, Crymych, Dyfed) accurate to 1 mm. All measurements were made, blind, by the same two trained field workers.

Frequency of breast feeds was recorded daily from d1 to d28 by maternal recall and corroborated using a 'bean-in-the-bag' method, where a bean was discarded from a known number in a purse worn by the mother, each time the baby nursed. Breast-milk intake was measured on days 7, 14, 21, 28 and 35, by weighing the child before and after each breast feed during the 12 h period 07.30 to 19.30 hours. The 24 h intakes were obtained by doubling this value, and the energy density of breast milk was estimated from calculated values for Gambian village women (Prentice *et al.* 1986).

Intake of home-prepared food was estimated by weighing the ingredients before cooking, at serving, and by re-weighing any remaining food at the end of the meal. Four 24 h measurements were made at weekly intervals during the non-intervention periods for both groups. Gambian food tables were used to estimate energy and protein intake, after analysis of duplicate samples from all meals for water content (Hudson *et al.* 1980).

A prospective, longitudinal study design was chosen in which early nutritional intervention was compared with later nutritional intervention. Because it is well-known that significant catch-up weight gain does not occur without adequate nutritional intake, in The Gambia (Rowland *et al.* 1977), as elsewhere, a third, control, group that received no nutritional supplementation was not included. Enrolment to the two groups was done using a random permuted block system. Unless otherwise stated, results are given as means and standard deviations (SD). Comparison of means was made using *t* tests. Changes in height during the short duration of the study were small and changes based on them have not been further analysed here. WFA standard deviation (*Z*) scores were used in the analyses to take into account the effects of sex and age on the distribution of weights of the subjects.

Table 3. *Weights and weight changes (δW) (kg) of children in early (E) and late (L) intervention groups \ddagger*
(Mean values and standard deviations)

Day	Group E		Group L	
	Mean	SD	Mean	SD
1	6.88	0.79	7.76	1.19
14	7.33***	0.90	7.86	1.22
δW	0.44	0.33	0.09	0.44
15	7.38	0.94	7.92	1.25
28	7.35	0.94	8.65†††	1.44
δW	-0.03	0.30	0.72	0.35

*** Mean value was significantly different from that for day 1, $P < 0.001$.

††† Mean value was significantly different from that for day 15, $P < 0.001$.

\ddagger For details of subjects and procedures, see pp. 480-482.

RESULTS

Weight changes

Table 3 shows the mean weights of the children on days 1, 14, 15 and 28. There were highly significant increases in mean weight in both groups during the intervention period ($P < 0.001$). During non-intervention periods no significant weight gain occurred in either group.

Fig. 2 shows the relationship between weight gain, expressed as mean change in WFA Z-score from d1 (δZ), and dietary intervention. In group E, an initial decrease in mean δZ during the first 3 d was followed by a linear increase to d15. In group L there was a decrease in mean δZ to d6 followed by return to baseline by d14. Thereafter there was a linear increase in mean δZ to d28. Children of group E re-achieved their baseline weight 3.5 times faster than those of group L ($P < 0.001$).

There was no significant difference in δZ between the two groups during the periods of linear increase in weight: mean δZ was $+0.46$ (SD 0.39) from d3 to d15 (0.038 SD/d) in group E, and was $+0.68$ (SD 0.41) from d13 to d28 (0.045 SD/d) in group L (NS). Mean δZ from d1 to completion of supplementation and non-supplementation periods (d1-d28) was $+0.22$ (SD 0.42) for group E and $+0.62$ (SD 0.48) for group L ($P < 0.02$).

During the non-intervention periods (d15-d28 in group E and d1-d14 in group L) there was no significant δZ in either group. From d28-d42, mean δZ was $+0.04$ (SD 0.23) in group E (NS) and was $+0.09$ (SD 0.37) in group L (NS). Mean δZ in group L was significantly greater on d35 (0.55 (SD 0.42) and 0.25 (SD 0.35); $P < 0.05$), but by d42 there was no significant difference between the two groups (0.52 (SD 0.58) and 0.26 (SD 0.47)).

Dietary intake

There was no significant difference in mean intake of the food supplement between the two groups when calculated as daily intake, or as total 14 d intakes during the supplement period (group E: 1047 (SD 88) g supplement/kg body weight; group L: 1023 (SD 88) g supplement/kg body weight).

The energy intakes of the two groups are shown in Fig. 3. In group E mean total energy intake on d7 (mid-point of intervention) was 644 (SD 255) kJ/kg per d, decreasing to 406 (SD 138) kJ/kg per d on d21 (mid-point of the first 2 weeks of non-intervention) ($P < 0.001$). Energy intake in group L rose from 410 (SD 109) to 623 (SD 146) kJ/kg per d from

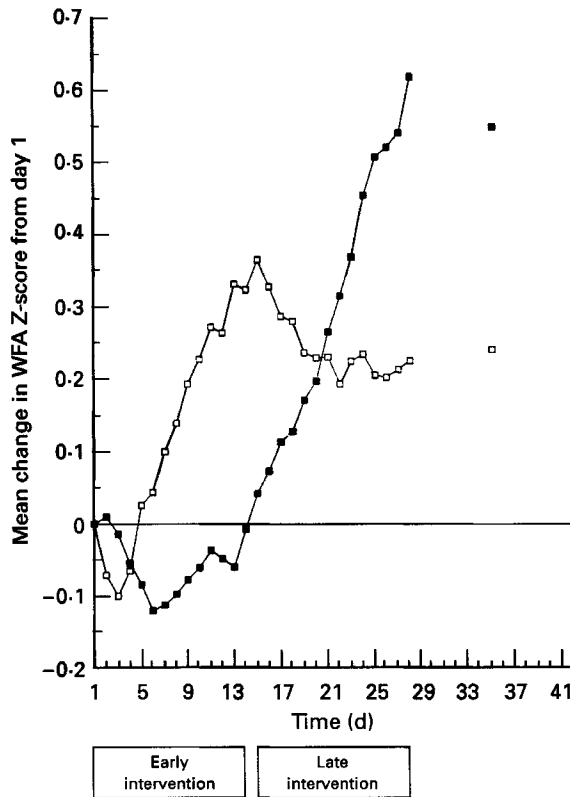


Fig. 2. Mean change in weight-for-age (WFA) Z-score from day 1 for early (□) and late (■) intervention groups. Daily measurements are plotted from day 1 to day 28, with single measurements for each group on days 35 and 42. Periods of supplementation are shown in boxes below the x-axis.

d7 (mid-point of non-intervention) to d21 (mid-point of intervention) ($P < 0.001$). There were no significant differences in energy intake from home diet and supplement between the two groups during periods of intervention and non-intervention.

Protein intake decreased from 3.9 (SD 2.2) g protein/kg per d on d7 to 1.8 (SD 2.0) g/kg per d on d21 for group E ($P < 0.01$). In group L protein intakes increased from 1.7 (SD 1.8) g/kg per d on d7 to 4.0 (SD 2.1) g/kg per d on d21 ($P < 0.01$). Protein intakes between the two groups both during intervention and non-intervention periods were not significantly different. The average proportion of energy supplied by protein was 10.4% during the periods of intervention and 7.2% during non-intervention periods.

In group E mean breast-feed frequency did not differ significantly between intervention (10.0 (SD 4.3) feeds/d) and non-intervention periods (10.4 (SD 3.8) feeds/d). Group L showed a fall in mean breast-feed frequency between non-intervention (12.0 (SD 4.1) feeds/d) and intervention periods (9.6 (SD 3.7) feeds/d; $P < 0.01$) but this returned to the pre-intervention frequency by d28. There were no significant inter-group differences for breast-feed frequency during intervention and non-intervention periods.

Mean breast-milk intake for group E increased from 356 (SD 161) g/d during the intervention period to 601 (SD 258) g/d during the following 14 d of non-intervention ($P < 0.001$). Intakes for group L fell from 567 (SD 167) g/d during non-intervention to 406 (SD 156) g/d during intervention ($P < 0.01$). There were no significant differences in breast-milk intake between the two groups, during either intervention or non-intervention periods.

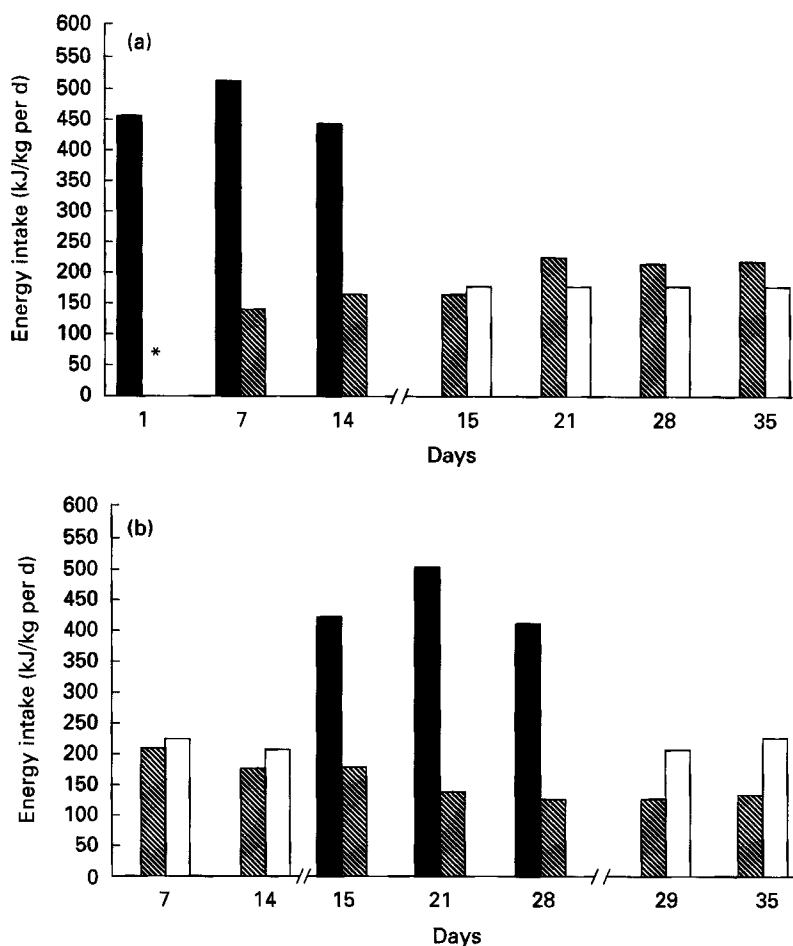


Fig. 3. Mean energy intakes of children receiving a high-energy-protein supplement (■), breast milk (▨) and home weaning foods (□) during (a) early intervention and (b) late intervention to prevent growth faltering following diarrhoea. * Breast milk intake was not measured on day 1.

Diarrhoea and vomiting

The number of days with diarrhoea before entry to the study did not differ significantly between the two groups (Table 1). Diarrhoea and vomiting increased in both groups during the intervention periods (Table 4). When assessed as mean child-days with diarrhoea (child-days = number of children with symptom \times mean duration of symptom) in group E there was a significant decrease when dietary intervention ceased ($P < 0.001$). In group L there was no significant difference in child-days with diarrhoea during intervention compared with the preceding non-intervention period.

There was a marked decline in child-days with vomiting on cessation of the supplement in group E ($P < 0.001$), and an increase when the supplement was started in group L ($P < 0.05$). During intervention, group E had a greater number of child-days with diarrhoea compared with group L ($P < 0.01$) but there was no inter-group difference in child-days with vomiting.

Table 4. *Diarrhoea and vomiting in early (E) and late (L) intervention groups during 14 d periods of intervention (INT) and non-intervention (NINT)*

Day ...	Group E (n 18)		Group L (n 16)	
	INT 1-14	NINT 15-28	NINT 1-14	INT 15-28
No. of children with diarrhoea‡	18	10	16	16
Mean no. of days with diarrhoea (range)	10.0 (5-14)	4.3 (0-12)	5.8 (0-12)	6.6 (0-12)
Total child-days§ with diarrhoea (range)	180.0 (90-252)	43.3***†† (0-120)	94.0 (0-192)	106.0** (0-192)
No. of children with vomiting	17	10	11	14
Mean no. of days with vomiting (range)	7.6 (0-13)	1.5 (0-6)	3.0 (0-14)	6.4 (0-12)
Total child-days with vomiting (range)	130.3 (0-221)	15.5*** (0-60)	33.0 (0-154)	90.1† (0-168)

Mean values within a row were significantly different from INT (days 1-14): ** $P < 0.01$, *** $P < 0.001$.

Mean values within a row were significantly different from NINT (days 1-14): † $P < 0.05$, †† $P < 0.01$.

‡ Two or more loose stools per d.

§ Number of children with diarrhoea/vomiting \times duration of diarrhoea/vomiting (d).

|| Regurgitation of volume of 10 ml or more per d.

DISCUSSION

The present study is relevant to two major controversies: the strength of the relationship between diarrhoeal disease and growth failure (Briend *et al.* 1989; Briend, 1990; Black, 1991), and the optimal management of the child who suffers acute weight loss after an episode of diarrhoea (Brown & MacLean, 1984). That recurrent diarrhoea is a major cause of protracted growth faltering has been challenged (Briend, 1990; Black, 1991; Moy *et al.* 1991), and it has been argued that insufficient dietary intake is a more important causative factor than diarrhoea. If this is correct, nutrition-intervention programmes deserve as much attention as the prevention and treatment of diarrhoea in overall strategies to control childhood malnutrition (Becker *et al.* 1991).

Catch-up growth after acute diarrhoea, without dietary intervention, has been demonstrated in malnourished Bangladeshi and Brazilian children but occurs over many weeks (Briend *et al.* 1989; Schorling & Guerrant, 1991). The rate of catch-up growth depends on the availability and intake of food and on the frequency of further diarrhoeal episodes (Black, 1991). Programmes of nutritional supplementation undertaken over long periods can have positive effects on the growth performance of malnourished children (Heikens *et al.* 1989; Lutter *et al.* 1989; Rivera *et al.* 1991). However, they are expensive, difficult to maintain, not always effective and weight loss may follow when they cease (Feachem, 1983; Kennedy & Knudsen, 1988). Furthermore, they may have adverse effects on parental attitudes to the provision of food for their children, or on traditional food preparation skills, and create dependence on outside agencies (Kennedy & Knudsen, 1988).

There have been few previous studies aimed at assessing the effects of short-term dietary intervention on catch-up weight gain after acute diarrhoea, using locally available foods in a community setting. We have shown clearly that vigorous dietary supplementation promotes rapid catch-up weight gain at a rate 4-5-fold greater than that during the unsupplemented period. However, intakes of about 628 kJ/kg per d and 4 g protein/kg per d were necessary to achieve and maintain such weight gain. These are well in excess of currently recommended requirements for healthy children (Truswell, 1983; World Health

Organization, 1985; Prentice *et al.* 1988). As in other studies, when children returned to their traditional home diet, rapid weight gain ceased.

Of the children under the age of 2 years in this community, 70–80% are underweight during the season of highest diarrhoeal prevalence (Rowland & Whitehead, 1978), despite the availability and provision of food intakes close to those recommended (Truswell, 1983; World Health Organization, 1985; Prentice *et al.* 1988). This is a further indication that intakes approaching recommended levels for normal growth rates are insufficient to produce rapid catch-up growth, although slow catch-up is possible over prolonged periods. The success of the present intervention depended not simply on the availability of a high-energy-protein supplement, but on achieving intakes well above those generally recommended.

There is strong evidence that the malnutrition of Gambian children is due in large part to a small-intestinal enteropathy (Sullivan *et al.* 1991). Persistent abnormalities of intestinal permeability (an indirect measure of enteropathy) account for up to 43% of the observed growth faltering (Lunn *et al.* 1991). We have shown that it is possible to override the effects of acute diarrhoea by supplementing dietary intake, and the effect of an episode of diarrhoea on growth faltering may be relatively insignificant against a background of chronic mucosal damage (Sullivan *et al.* 1992).

Anorexia following diarrhoea is often cited as another cause of poor dietary intake and growth (Hoyle *et al.* 1980; Molla *et al.* 1983) and therefore we encouraged frequent feeding with small amounts of flavoured food to override this potential problem, and ensured that feeding was undertaken by mothers or nursemaids under supervision.

We are not the first to propose early nutritional intervention in the management of the child with weight loss after acute diarrhoea (Chung & Viscorova, 1948). Cautious, graded re-introduction of food has traditionally been advocated, in the belief that the digestive system, 'injured' by acute diarrhoeal disease, must recover before it can cope with a full diet. Secondary lactase (*EC* 3.2.1.23) deficiency, disordered bile salt metabolism and dietary protein intolerance (Lebenthal, 1989; Brown, 1991*a, b*) have been regarded as contraindications to immediate refeeding. On the other hand, it has been argued that the reserve digestive and absorptive capacity of the gastrointestinal tract (Weaver *et al.* 1991) makes it possible to undertake rapid refeeding. This view was proposed long ago (Chung & Viscorova, 1948) and has increasingly returned to favour (Brown & MacLean, 1984).

Feeding with high-osmolality foods after diarrhoea and vomiting may lead to osmotic diarrhoea and an increase in the frequency of vomiting (Brown, 1991*a*). Nevertheless we have shown that in spite of persistence of diarrhoea during refeeding, weight gain, and therefore net nutrient absorption, took place. The composition of feeds should aim at a compromise between high nutrient content and low osmolality. Because of the likelihood of exacerbating vomiting and diarrhoea, continuing access to ORS should be available. Some authors have stressed the positive effect of ORS on subsequent growth, in addition to its effects on hydration (Hirschorn, 1980).

Another potential negative effect of dietary supplementation is that it may compromise breast-milk intake (Prentice & Paul, 1990). In an earlier study we found a positive correlation between feed frequency and milk intake at all ages after 2 months (Prentice *et al.* 1986), and in the present study there was only a transient decrease in breast feed frequency, followed by rapid recovery when supplementation ceased. We have also shown elsewhere that a prolonged effect on breast-milk intake of this short period of supplementation is unlikely (Downes *et al.* 1992). It is possible that the current measurements have exaggerated the effects of supplementation on milk intake since they were made only during daylight hours when the supplement was being actively given. Suppression of night-time intakes may not have been so great.

During intervention periods mean weight gain was 4.6 g/kg per d (group E) and 6.5 g/kg per d (group L). This 3.5-fold rate of increase in body weight was achieved with a 50% increase in energy intake, 100% increase in protein intake, and 30% increase in the proportion of energy supplied by protein. These values are broadly in accordance with those calculated by other workers, including ourselves (Whitehead, 1977).

The energy and protein requirements for catch-up growth have been reviewed in detail by Waterlow (1992). Although it has long been recognized that the energy density of feeds can be a limiting factor (Whitehead, 1977), protein intake must also be sufficient to meet the needs for catch-up growth, as well as that lost through incomplete digestion and absorption. With an intake similar to that used in the present study, Fjeld *et al.* (1989) recorded weight gains of 11.8 g/kg per d in children recovering from severe protein-energy malnutrition.

Ashworth & Millward (1986) recommended an energy intake of 418 kJ (100 kcal)/kg per d and a protein intake of 2 g/kg per d (protein 8% of energy intake) to achieve a weight gain of 5 g/kg per d during nutritional rehabilitation. We used a more energy-dense supplement, given to children immediately after an episode of acute diarrhoea, which, with continued gastrointestinal losses during intervention, may account for a rate of weight growth more like that reported by Ashworth & Millward (1986).

The maximal rate of weight gain was similar in both groups, and the results suggest that continued supplementation of group E beyond 14 d would lead to continued catch-up growth thereafter. Early feeding led to reachievement of baseline weight faster than late feeding, and late feeding resulted in greater overall weight gain during the 28 d following acute diarrhoea (Fig. 2).

We suggest that it is insufficient diet, and not the underlying mucosal enteropathy, that is the principal factor that limits rapid catch-up weight gain after acute diarrhoea. Slow catch-up growth may eventually occur given a sufficient diarrhoea-free period (Briend *et al.* 1989; Schorling & Guerrant, 1991), but improved dietary intake can have a dramatic effect on weight gain, returning a child to his or her premorbid weight within 14 d. Although diarrhoea may persist during refeeding, and is probably related to a persisting enteropathy that is not rapidly improved during nutritional rehabilitation (Lunn *et al.* 1991), vigorous dietary supplementation should be undertaken as soon as possible after acute weight loss.

Programmes designed to treat diarrhoea, which do not also include dietary supplementation, may be ineffective in decreasing either the prevalence or incidence of childhood malnutrition and growth faltering in the developing world. Short-term nutritional supplementation with a high-energy-protein supplement is essential to promote rapid catch-up growth and nutritional recovery in populations with a high prevalence of childhood diarrhoeal disease and undernutrition. Such treatment can be undertaken successfully outside hospital using locally available, locally prepared foods and locally trained staff, and should be a central part of the care of undernourished children.

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