

# NUTRITIONAL INTERVENTION IN ACUTE DIARRHEA: IS A LACTOSE-FREE FORMULA ESSENTIAL?

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**Abstract.** A prospective study was done to determine the incidence of disaccharide intolerance among 3-36 month-old patients with acute watery diarrhea who were on breast feeding and/or lactose-containing formula. The effect of feeding intervention on the outcome was investigated. Significant disaccharide intolerance was defined as one with (1) biochemical derangements: stool pH <6.0 (Riedel de Haen pH paper) and reducing substances  $\geq 0.5$  mg% (Clinitest) on two consecutive determinations and (2) clinical evidence: high purging rate ( $>10$  gm/kg/hour) and reappearance of dehydration and/or weight loss while on a lactose containing milk.

Seven of 92 patients (7.8%) had biochemical evidence of disaccharide malabsorption on admission. Subsequent monitoring of the study population showed absence of disaccharide intolerance. Despite a lactose containing formula, a significant ( $p < 0.05$ ) reduction in stool output from the first to the second day ( $145.85 \pm 130.26$  vs  $115.43 \pm 95.65$  g/kg admission weight) was noted. Likewise, weight gain from admission to discharge ( $4.56 \pm 3.44\%$ ) was observed. The mean total duration of illness ( $4.75 \pm 2.84$  days) was well within the usual course of five to seven days.

This study supports the current recommendation of continued breast feeding and/or use of lactose containing formula during acute watery diarrhea.

## INTRODUCTION

Acute diarrheal illness continues to be a major public health problem in developing countries. Global estimates show that there are 1 billion episodes of diarrhea occurring annually with four to five million resulting in diarrheal deaths (Gold, 1988). In the Philippines, the illness consistently ranked among the top ten causes of infant mortality and morbidity over the past twenty years. It is presently the second cause of morbidity and the eighth cause of mortality in our country (Philippine Health Statistics, 1988).

The advent of oral rehydration therapy showed beneficial effects in terms of reduction in hospitalization and deaths from diarrheal dehydration. After oral rehydration, however, adequate nutritional support is mandatory in order to compensate for the increased metabolic demands and nutrient loss secondary to the diarrheal illness.

To date, most clinical studies have evaluated continued feeding during the maintenance phase

of acute diarrhea (Brown *et al*, 1988). A nutritionally significant proportion of the diet, particularly that of carbohydrates, fats and proteins has been proven to be partially absorbed during an enteric infection. Current recommendations on feeding during acute diarrhea include foods that are easily digestible, culturally acceptable, locally available, affordable and with a high nutrient density (Committee on International Nutrition Programs, 1985). Further studies are still needed however to formulate guidelines on the types of food that should be given during acute diarrhea.

One area that needs to be examined and explored is the use of a lactose containing food during acute diarrhea. Several studies have advocated the use of a lactose-free formula to speed the recovery of the intestinal mucosa (Harrison *et al*, 1976; Dagan *et al*, 1984; Chow *et al*, 1988; Placzek and Walker-Smith, 1984; Noah, 1979). It has been known that acute diarrhea is associated with a disturbance in the absorption of carbohydrates, particularly lactose, brought about by a temporary deficiency of lactase in the mucosa of the small intestine. The

disturbance in carbohydrate absorption may in turn contribute to the duration and severity of the diarrhea since undigested lactose produces an increased osmotic gradient in the lumen of the small intestine.

Recent reports, however, have contested the use of a lactose-free formula in acute diarrhea. A study done by Davidson *et al* (1984) in 104 well-nourished Australian children two weeks to five years of age with acute enteritis has shown an incidence of lactose intolerance in only 33% of patients. Moreover, this study demonstrated that not all children with acute enteritis need to be given a lactose-free diet. The incidence of lactose intolerance was higher among patients with rotavirus enteritis. Lactose intolerance was defined as the inability to tolerate a lactose-containing formula as provided by clinical observation regardless of the breath hydrogen test.

A breath hydrogen test however can not be employed on all patients with acute diarrhea. The examination needs sophisticated equipment aside from the fact that patients have to be fasted for eight hours before the examination. A simple bedside examination that could be done is the determination of the pH of and reducing substance in the stool. A stool pH of  $< 6.0$  and reducing substance of  $\geq 0.5\%$  are presumptive evidence of disaccharide malabsorption and indicative that the patient should be monitored more closely for clinical signs of intolerance (Townley, 1966; Soeparto *et al*, 1972; Davidson and Mullinger, 1970). These clinical signs include a high purging rate ( $> 10$  gm/kg/hour) and appearance of dehydration and weight loss on intake of a lactose containing formula (MEDIAC, 1988).

However, the use of these parameters in identifying a patient with disaccharide intolerance during acute enteritis has not been extensively studied. If these parameters could be employed, the judicious use of a lactose-free formula during an acute episode of diarrhea could be implemented. Furthermore, standard guidelines for feeding during and after a diarrheal episode could be established.

This prospective study was undertaken to determine:

- (1) the incidence of clinically significant disaccharide intolerance among hospitalized patients with acute diarrhea.

- (2) the effect of lactose containing feeds (breast-milk and/or formula) on the severity of the illness, as measured by the duration of diarrhea and stool output.
- (3) if lactose containing feeds will lead to positive weight gain despite the episode of diarrhea.

## MATERIALS AND METHODS

From January to December 1989, male pediatric patients who were 3 to 36 months of age with a history of three or more watery stools in the past twenty-four hours prior to admission of not more than five days duration and with signs of dehydration (Appendix 1) were admitted into the study. Excluded were those patients who had systemic or surgical illness, severe malnutrition, with a history of diarrhea within two weeks before the present episode, grossly bloody stools and continuous use of antibiotics for 48 hours or more before hospitalization. Patients with a history of intolerance to cow's milk or on a lactose-free formula were likewise excluded.

On admission to the study a complete history was taken, taking into account the patient's general data : symptomatology with special reference to the onset of diarrhea, character of stools, presence of associated symptoms and feeding history prior to illness. A complete physical examination was performed with special attention to the vital signs, nude weight and signs of dehydration.

Baseline laboratory examinations included stool pH and reducing substances of the first stool passed on admission (Cole, 1984). The stools were collected in plastic-lined diapers and were tested immediately after collection. The stool pH was taken by dipping a pH paper (Reidel de Haen, pH 0 - 14) in the liquid stool and the reaction was compared with the color chart provided. The presence of stool reducing substances was determined using a Clinitest tablet (Miles laboratory, Ames Division). To test for the presence of disaccharide malabsorption, acid hydrolysis using 1 N HCl was routinely done prior to administration of Clinitest tablet in each fresh stool specimen. The reaction was likewise compared with a color chart. The finding of a stool pH  $< 6.0$  and  $\geq 0.5\%$  reducing substance in the stool was considered

abnormal and suggestive of disaccharide malabsorption.

The stool was likewise examined for ova and parasites. Stool cultures for bacterial pathogens (*E. coli*, *Shigella*, *Helicobacter pylori*, *Vibrio cholerae*, *Salmonella*, *Aeromonas hydrophila*, *V. parahaemolyticus*) utilizing standard microbiological techniques and ELISA for rotavirus antigen were also performed.

Patients were rehydrated with the standard oral rehydration salts (ORS) formulation following the recommendation by the World Health Organization (WHO). The ORS was given by mouth or nasogastric tube as necessary (if the child refused the ORS or developed vomiting of more than three episodes in an hour during the hospital stay). Only ORS was given during the first six hours.

During the maintenance phase, feedings were resumed in the form of breastmilk and/or milk formula. Exclusively breastfed infants received only breastmilk. For patients who were bottlefed, a cow's milk, whey-predominant, lactose containing formula was used consisting of 20 calories per ounce (Appendix 2). Patients were given half-strength formula for the first twenty four hours, then full strength thereafter on demand basis.

Plain water was offered on demand. ORS was given on a volume per volume replacement of stool output until diarrhea stopped. Periodic weighing of the patient was done after rehydration (6th hour) and every twenty-four hour interval and on discharge. The nude weight of the patient was taken using a pediatric weighing scale (SECA, West Germany) calibrated to the nearest 0.1 g. All intake and output were carefully monitored and entered into appropriate recording forms. Fluid intake was measured using a calibrated glass or feeding bottle and reported to the nearest 1 ml. The stool output was determined by getting the difference in weights between the soiled and unsoiled disposable diapers. These were measured in a triple beam balance (OHAUS, USA) sensitive up to 0.1 g. Only male patients were entered into the study to ensure that stool output could be accurately measured by separating it from the urine output. The urine was collected in a pediatric urine bag and the volume was measured using a calibrated cylinder accurate up to 1 ml. The stool pH and reducing substances were determined every

twenty-four hour interval until cessation of passage of watery stools.

Antibiotics were administered in patients with amebiasis or giardiasis on stool microscopy and in culture proven cholera and *Shigella* cases, consisting of the following:

1. Cholera - Tetracycline 50 mg/kg/day × 3 days
2. Shigellosis - Trimethoprim 10 mg/kg/day and sulfamethoxazole at 50 mg/kg/day × 5 days
3. Amebiasis - Metronidazole 30-50 mg/kg/day × 5-10 days
4. Giardiasis - Metronidazole 30-50 mg/kg/day × 5-10 days

A patient was discharged from the study if no stool motion occurred for twelve hours or there was passage of formed stools on two consecutive occasions. The duration of diarrhea was defined as the time in hours, from admission to the study up to the time of the last abnormal stool which was the last stool passed prior to the 12 hour interval or the stool immediately preceding the passage of formed stool. However, if the duration of diarrhea was less than 24 hours, the patient was still retained in the study until completion of the 24th hour of observation. During this time, intake and output were continuously monitored.

Patients found to have disaccharide intolerance were considered as treatment failures. Significant disaccharide intolerance was defined as one with (1) biochemical derangements: stool pH < 6.0 and  $\geq 0.5\%$  reducing substances on two consecutive determinations and (2) clinical evidence: presence of high purging rate (> 10 g/kg/hour) and reappearance of dehydration and/or weight loss during the time that the patient is on a lactose containing formula or breastmilk. These patients were shifted to a soy-based disaccharide-free milk formula.

Standardization of the procedure and methodology was undertaken prior to the conduct of the study. There were four research assistants (medical technologists) employed who were directly involved in the weighing, monitoring, feeding and performance of laboratory procedures. A pilot study was conducted prior to the trial proper to offset interobserver bias.

Percentage weight gain was derived as follows:

$$\% \text{ weight gain} = \frac{\text{weight on discharge} - \text{weight on admission}}{\text{admission weight}}$$

The data were analyzed by taking into account the number of cases who developed clinically significant disaccharide intolerance. The characteristic clinical and laboratory features of these patients were described. Student's *t* test was used for comparison of the means of the stool output and percentage weight gain for each of the specified time intervals. All significance testing was performed at  $p < 0.05$ .

## RESULTS

A total of ninety-two patients were included in the study. The mean age of patients was  $11.27 \pm 6.72$  months, with a majority of patients being less than one year of age (58.7%).

Table 1 presents the clinical profile of the study population on admission. Only thirteen (14.1%) of the patients had normal weight for their corresponding age with most of the patients classified as mildly under weight (53.3%). As can be gleaned

Table 1  
Characteristics of patients prior to intervention.

	No. of cases (percentage)	Mean $\pm$ SD
1. Age		$11.27 \pm 6.72$
3-11 months	54 (58.7)	
12-23 months	32 (34.8)	
24-36 months	6 (6.4)	
2. Mean admission weight (kg)		$7.26 \pm 1.59$
3. Weight for age (% of NCHS norms)		$78.06 \pm 10.78$
Normal (90%)	13 (14.1)	
Mildly underweight (75-89%)	49 (53.3)	
Moderately underweight (60-74%)	30 (32.6)	
4. Duration of diarrhea before admission (hrs)		$58.84 \pm 34.15$
5. Mean frequency of stool motions 24 hours before admission		$8.79 \pm 4.92$
6. History of vomiting before admission (hrs)	60 (65.2)	
7. Duration of vomiting before admission (hrs)		$37.42 \pm 29.18$
8. History of fever before admission	54 (58.7)	
9. Duration of fever before admission (hrs)		$28.72 \pm 24.27$
10. Feeding prior to illness		
Exclusively breastfed	9 (9.8)	
Exclusively on artificial milk formula	14 (15.2)	
Mixed feeding	65 (70.6)	
Solid foods	4 (4.4)	
11. Degree of dehydration on admission		
Mild	79 (85.9)	
Moderate	13 (14.1)	

Table 2

Stool pH and reducing substances on admission, 24th, 48th and 72nd hours.

Stool pH	0 (n=92)	24 (n=81)	48 (n=40)	72 (n=9)
<6	45	27	13	2
>6	47	54	27	7

  

Reducing substance	0 (n=92)	24 (n=81)	48 (n=40)	72 (n=9)
<0.5	85	84	40	9
≥0.50	7	0	0	0

from the table, a majority (70.65%) of the patients were on mixed feeding (breastmilk and/or artificial milk formula and semi-solid foods) prior to the illness. There were 28 (30.4%) patients who were still breastfeeding prior to the illness with nine patients (9.8%) exclusively breastfed. Patients were predominantly mildly dehydrated (85.9%). There was no patient admitted with severe dehydration.

The result of the stool pH and reducing sub-

stance determinations of the patients on admission and each of the 24 hour interval are shown in Table 2. Forty-nine (49%) percent of patients had acidic stools (pH < 6.0) on admission but only seven of these showed significant stool reducing substances. At the 24th hour of observation, there were 81 patients who were still diarrheic and a repeat examination could be performed. Thirty-three percent of the patients (n=27) had acidic stools but none of them showed signs of malabsorption. Subsequent determinations at the 48th and 72nd hours likewise showed 15 patients having acidic stools but with nonsignificant levels of stool reducing substances.

The characteristics of patients with laboratory-proven disaccharide malabsorption on admission are presented in Table 3. Five of them were still being breastfed prior to the illness with two patients exclusively breastfed. All patients had mild dehydration initially. On the 24th hour from admission, repeat determinations of the stool pH and reducing substance were no longer suggestive of malabsorption. Five patients were discharged while two patients were still diarrheic at the end of the 48th hour. The latter still did not show evidence indicative of a biochemical or clinically significant disaccharide malabsorption at the 48th hour post admission. Three of the seven patients had rotavi-

Table 3

Characteristics of patients with disaccharide malabsorption by biochemical parameters.

Pt No	Age (months)	Feeding prior to illness*	Stool pH/reducing substances						Stool isolate
			Admission		24th hour		48th hour		
1	8	3	5	0.5	7	0.25			No growth
2	6	2	4	0.5	8	0.0	5.0	0.0	Rotavirus
3	4	1	5	0.5	6	0.25			No growth
4	5	2	5	0.5	6	0.0			No growth
5	22	3	4	0.5	7	0.0			No growth
6	6	3	5	0.5	5	0.25	5.0	0.0	Rotavirus
7	9	1	5	0.5	5	0.0			Rotavirus

\* 1 = Exclusively breastfed

2 = Breastfeeding and semisolids

3 = Artificial infant formula and semisolids

Table 4

Effect of nutritional intervention on the outcome of the diarrheal illness.

	No. of cases	Mean $\pm$ SD
1. Mean percentage of weight gain		
0 - 6 hours	92	2.87 $\pm$ 2.36
0 - 24 hours	92	3.99 $\pm$ 3.04
24 - 48 hours	55	4.66 $\pm$ 3.72
48 - 72 hours	34	4.50 $\pm$ 4.44
0 - discharge	92	4.56 $\pm$ 3.44
2. Diarrheic stool output, g/kg admission weight		
0 - 6 hours	92	40.02 $\pm$ 44.52
0 - 24 hours*	92	145.85 $\pm$ 130.26
24 - 48 hours	55	115.43 $\pm$ 95.65
48 - 72 hours	34	115.10 $\pm$ 97.85
0 - discharge	92	257.18 $\pm$ 246.94
3. Duration of diarrhea after the start of interventions (hrs)	92	54.08 $\pm$ 34.58

\*Significant difference at  $p < 0.05$ , student's *t*-test

rus infection.

The mean percentage of weight gain and stool output (g/kg admission wt) on each of the specified time interval is shown in Table 4. Fifty-five patients (59.8%) were still under observation at the end of the forty-eighth hour, while 34 (37%) patients were still confined at the end of the seventy-second hour. A comparison of the mean of the stool output showed a statistically significant decrease on the second day as compared with the first day of observation. The purging rate of patients whether on breastfeeding or on a cow's milk-based lactose containing formula never exceeded 10 g/kg/hour. There was no patient in the study who had a reappearance of dehydration.

The mean duration of diarrhea from admission to the last abnormal stool was 54.08  $\pm$  34.58 hours. The longest duration of diarrhea after the start of intervention was 188 hours. This was in a 16 month old patient who came in with a 30 hour history of illness prior to consult. This patient likewise did not exhibit any sign of disaccharide intolerance.

As to etiologic agents (Table 5), stool pathogens

Table 5

Enteropathogens isolated from stools of patients.

Enteropathogens	No (%) of cases*
Rotavirus	35 (38.04)
Enterotoxigenic <i>E. coli</i>	
heat stable	3 (3.3)
heat labile	5 (5.4)
Enteropathogenic <i>E. coli</i>	2 (2.2)
Shigellae:	
<i>Shigella flexneri</i>	5 (5.4)
Salmonellae	
<i>Salmonella</i> , non typhoidal	1 (1.1)
<i>Salmonella</i> group B	8 (8.7)
<i>Salmonella</i> sp.	1 (1.1)
Vibrio	
<i>Vibrio cholera</i> non 0-1	2 (2.2)
<i>Vibrio cholerae</i> El tor (Ogawa)	1 (1.1)
<i>Vibrio</i> sp.	1 (1.1)
Aeromonas	
<i>Aeromonas hydrophila</i>	1 (1.1)
<i>Aeromonas caviae</i>	1 (1.1)
<i>Aeromonas</i> sp.	2 (2.1)
<i>Helicobacter pylori</i>	2 (2.2)
No growth	36 (39.1)

\*14 patients had more than one isolate

were isolated in 61% of patients with rotavirus accounting for 63% of all stool isolates (35/56). Fourteen patients had mixed infections on admission, with eleven out of the fourteen patients (78.6%) having a combination of bacterial and viral pathogens. Among the bacterial isolates, *Salmonella* group B (8.7%) and enterotoxigenic *E. coli* (8.7%) were the most frequently seen, followed by *Shigella flexneri* (5.4%).

There was no treatment failure in the study, as no patient exhibited biochemically and clinically significant disaccharide intolerance. All patients were discharged improved and were advised appropriate dietary management, home fluids and follow-up.

## DISCUSSION

Lactose is a disaccharide composed of the

monosaccharides glucose and galactose in a 1,4-beta linkage (Angelides and Davidson, 1985). Following its ingestion, lactose is hydrolyzed in the small intestine by a specific disaccharidase called lactase, which is located in the brush border of the microvillus membrane. When small intestinal mucosal lactase activity is deficient, up to 75% of dietary lactose passes through the small intestine to be metabolized in the cecum and colon by bacteria. The effect on intestinal fluid and gas production results in symptoms of lactose intolerance which include colicky abdominal pain, abdominal distension, increased flatus and passage of acidic watery stools.

There are two types of lactase deficiency: congenital and acquired (Angelides and Davidson, 1985; Silverman and Roy, 1983). Congenital lactase enzyme deficiency connotes the absence of the enzyme at birth, with signs and symptoms appearing after the first milk feeding and the enzyme deficit persisting throughout life. The disease is very rare. It is inherited as an autosomal recessive disorder. The acquired type of lactase deficiency is more commonly encountered. Secondary lactase deficiency may be associated with a variety of conditions, the most common of which follows an acute gastrointestinal infection. This was the reason why, traditionally, lactose-free formula was recommended for patients following a bout of acute diarrhea. The deleterious effects of lactose ingestion have been postulated to comprise an increase in stool water, electrolyte and bicarbonate losses with resultant caloric deprivation secondary to loss of metabolizable carbohydrates. Lifshitz *et al* (1971) demonstrated that the persistence and severity of diarrhea was directly related to the degree of lactose intolerance.

More recently, the need to shift to a lactose-free formula and the gradual reintroduction of full-strength milk over several days have been questioned. Despite the presence of carbohydrate malabsorption, studies have shown that this nutrient is still absorbed within the range of 80-95% (Howard *et al*, 1985; Brown and MacLean, 1984; Brown *et al*, 1979). Moreover, continued intake of lactose containing formula has been shown to be beneficial in the restoration of tolerance to lactose, while breastfeeding was also shown to decrease stool output and the risk of hypernatremia. These have become the basis of specific recommendations of feeding in acute diarrhea

including continued breastfeeding even during the period of rehydration or reintroduction of a half-strength milk formula during the maintenance phase, gradually increasing to full strength after twenty-four hours, depending on type of feeding prior to illness.

A stool pH of  $<6.0$  is said to be characteristic of sugar malabsorption, yet a majority of the stool specimens with acidic pH (pH  $<6$ ) contained nonsignificant amounts of reducing substances (less than 0.5 mg%). The follow-up examinations of the acidic stool specimens were likewise free of sugar. This study confirms previous reports of Soeparto *et al* (1972) that estimation of the stool pH is not a reliable screening test for the diagnosis of sugar malabsorption. Among patients with acute diarrhea, passage of unabsorbed fatty acids from colonic salvage may explain the findings of an acidic stool but with nonsignificant reducing substances. Despite the presence of a compromised intestinal absorption, the gut, particularly the colon has been shown to play an important role in the conservation of malabsorbed nutrients, a condition termed as "colonic salvage mechanism" (Brown and MacLean, 1984). Carbohydrates not absorbed in the small intestine are rapidly degraded by the colonic bacteria through the process of fermentation into short chain fatty acids. However, when the fatty acids produced exceed the absorptive capacity of the colon, osmotic diarrhea may ensue with the passage of fatty acids in the stool.

Our data have shown that the presence of disaccharide malabsorption by biochemical parameters was present in only 7.6% of cases (n = 7) on admission. The presence of malabsorption without clinical findings of disaccharide intolerance may reflect functional abnormalities of the gut after acute enteritis. With impaired digestion of lactose, bacterial fermentation of unabsorbed carbohydrates results in the release of smaller molecules with consequent increase in the osmolality and in the elaboration of large quantities of lactic acid and hydrogen. This would explain the findings of acidic stool (pH  $<6.0$ ) with  $\geq 0.5$  mg % of reducing substances as measured by Clinitest, suggestive of disaccharide malabsorption but not necessarily intolerance. Evidence of disaccharide intolerance is always provided by clinical observations.

Five out of the seven patients with disaccharide malabsorption were still on breastfeeding and two of the patients were exclusively breastfed. Signifi-

cant fecal reducing substances have been demonstrated among normal breastfed infants (Davidson and Mullinger, 1970; Hoyle *et al.*, 1980). The carbohydrate content of breastmilk is approximately 7 to 7.5 g/l. Lactose at this concentration during acute enteritis may not be totally digested and absorbed. However, despite the findings of disaccharide malabsorption, none of our patients exhibited disaccharide intolerance. The customary advantages of breastfeeding in the prevention and case management of diarrhea should be reiterated: antiinfective properties, decrease in further exposure to pathogens, acceptability and psychosocial benefits.

In our study, there was a mean percentage weight gain of  $4.56 \pm 3.44$  from admission to discharge. There was only one patient in the study who had a negative weight gain at the end of observation. This was a 1 year old whose duration of diarrhea after the start of intervention lasted for 90 hours. This patient did not show evidence of disaccharide malabsorption. Hospital based investigations of dietary intake by children with diarrhea have consistently shown a reduction in food intake, and hence, caloric intake during the early stage of the illness (Clemens *et al.*, 1988; Sarker *et al.*, 1982). A clinical study done in Bangladesh has noted a 30-50% decrease in caloric intake during the acute phase of the disease. In a growing child, these losses are significant and could be manifested by a slowing of growth or by persistent weight loss even after rehydration. The weight gain among our patients from admission to discharge clearly demonstrates that disaccharide absorption is substantial and intake of lactose containing milk is beneficial even during the early stage of acute diarrhea.

The severity of diarrhea, in terms of stool output and duration of illness was reported with a significant decrease in the mean stool output (Mahalanabis *et al.*, 1983; Patra *et al.*, 1984), from the first to the second day of intervention. Thirty-seven patients were discharged improved after 24 hours of observation with only a third of the patient remaining after 72 hours. Our observation reinforces the promotion of continued feeding during diarrhea, as disaccharidase levels in the intestinal mucosa have been reported to be dramatically reduced during fasting. The gradual reintroduction of lactose containing formula was shown to be well tolerated and did not aggravate the diarrhea.

There was no patient who had reappearance of dehydration.

The mean duration of illness of our patients after intervention was from  $2.25 \pm 1.44$  days. Taking into account the duration of diarrhea prior to admission of  $2.50 \pm 1.40$  days, total duration was well within the usual course of five to seven days for acute watery diarrhea. Earlier reports by Dagan *et al.* (1984) have reported the duration of diarrhea in the hospital to be  $6.28 \pm 0.67$  days among patients less than one year of age who were likewise started with a cows' milk-based lactose containing formula in increasing concentrations and volumes. In the same study, children who were fed with a soy-based, disaccharide free formula had a mean duration of diarrhea of  $3.48 \pm 0.23$  days. The differences in the ages of the patients, the degree of dehydration, the nutritional status prior to the illness and the stool pathogens may explain the differences in terms of duration of illness.

Rotavirus was the most common enteropathogen in our study, accounting for 63% ( $n = 35/56$ ), but was observed in only three of the seven patients with disaccharide malabsorption on admission. This is in contrast to other studies done elsewhere. Davidson *et al.* (1984) have reported that among 33 Australian children with acute diarrhea, lactose malabsorption, as diagnosed by breath hydrogen test, was present in 88% of patients with rotavirus enteritis, sixty percent of these patients being clinically lactose intolerant. The increased frequency of lactose intolerance among patients with rotavirus enteritis (Davidson *et al.*, 1975) have been postulated to be related to a specific affinity of the pathogen for lactase, which may serve as a receptor site during the infection (Hjelt *et al.*, 1985; Rodriguez *et al.*, 1977). The duration of rotavirus infection therefore is limited by the replacement of desquamated, mature, lactase containing villus cells by more immature lactase deficient cells. On the other hand, a recent study done in Bangladesh among children with acute diarrhea has shown enterotoxigenic *E. coli* to be the most common organism isolated in patients with lactose malabsorption (35.5%) (Brown *et al.*, 1979). None of our patients in the study with enterotoxigenic *E. coli* as a stool isolate exhibited disaccharide malabsorption.

An acute diarrheal illness can indeed have a



profound effect on the nutritional status of the child. And the more marginal the child's nutritional status at the onset, the more important this consideration becomes. Continued breastfeeding and/or gradual reintroduction of a cow's milk-based lactose containing formula has been shown to be beneficial.

In conclusion, this one year prospective study was undertaken to determine the incidence of disaccharide malabsorption and intolerance among hospitalized patients with acute watery diarrhea who were initially admitted with mild to moderate dehydration. The effect of breastfeeding and/or a cow's milk-based lactose containing formula on the severity of the diarrhea and extent of weight gain was investigated. Pertinent findings revealed the following:

1. Seven out of ninety two patients (7.6%) had disaccharide malabsorption by biochemical parameters (stool pH < 6.0 and reducing sugar  $\geq$  0.5%) on admission. However, none of these patients exhibited clinically significant disaccharide intolerance. The repeat biochemical parameters of these patients and the rest of the study population on the 24th and the 48th hour of observation were no longer suggestive of malabsorption.

2. Determination of the stool pH alone was shown to be an unreliable screening test for the diagnosis of sugar malabsorption. A majority of the stool specimens with acidic pH (pH < 6) contained nonsignificant amounts of reducing substances (less than 0.5mg%).

3. The study has shown that breastfeeding and/or the intake of a cow's milk-based lactose containing formula did not increase the severity of the diarrhea. A significant decrease in the stool output ( $145.85 \pm 130.26$  vs  $115.43 \pm 95.65$  g/kg admission weight) was noted on the second day as compared to the first day of observation. The mean duration of illness of our patients after intervention was from  $2.25 \pm 1.44$  days. Taking into account the duration of diarrhea prior to admission of  $2.50 \pm 1.40$  days, total duration was well within the usual course of five to seven days for acute watery diarrhea.

4. There was a mean percentage weight gain of  $4.56 \pm 3.44$  from admission to discharge. The extent of weight gain of our patients clearly demonstrates that disaccharide absorption is substantial

and intake of lactose containing formula is beneficial even during the early stage of acute diarrhea.

5. Rotavirus was the most common enteropathogen in our study, accounting for 63% (n = 35/56), but in only three patients with rotavirus enteritis was malabsorption of disaccharide seen. An increased frequency of disaccharide intolerance among patients with rotavirus infection was not demonstrated in this study.

On the basis of these findings the following **recommendations** are proposed:

1. We support the present recommendation of the World Health Organization of continued feeding during acute diarrhea. Lactose at a concentration equivalent to that of half strength cow's milk formula gradually increasing to full strength after 24 hours can be well tolerated by children with acute enteritis.

2. Transient disaccharide malabsorption may occur in a patient with gastroenteritis, however this does not always signify intolerance. Therefore the potential for carbohydrate intolerance should be carefully addressed in clinical protocols prepared for medical personnel. Misconceptions about transient intolerance to carbohydrates all too often lead to the use of a lactose-free formula or prolonged withholding of food.

3. Breastmilk even with a higher carbohydrate content as compared to artificial infant formula has been shown to be well tolerated even among patients with acute enteritis. The promotion of continued breastfeeding therefore during diarrhea should be emphasized in the nutritional component of diarrhea treatment programs.

4. Further investigations are needed to compare the use of different formulas and feeding regimens in acute diarrhea and to determine which is the most effective in promoting weight gain and decreasing the severity of the illness.

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APPENDIX 1

Assessment of degree of hydration.

A. Mild dehydration

Presence of at least one of the following in a child with diarrhea:

1. sunken eyes
2. sunken fontanel
3. dry mucus membranes

with absence of:

1. loss of skin elasticity, i.e., skin retracts in less than two seconds after pinching
2. poor urine output for the last 6-8 hours.

B. Moderate dehydration

Loss of skin elasticity, i.e., skin retracts slowly more than two seconds after pinching in a child with diarrhea plus at least one of the followings:

1. sunken eyes
2. sunken fontanel
3. dry mucus membranes
4. absent urine output for the last 6-8 hours

C. Severe dehydration

Signs of moderate dehydration in a child with diarrhea plus signs of shock:

1. weak rapid pulse
2. cold extremities
3. sensorial change (lethargy to coma)

APPENDIX 2

Composition of infant formula.

Ingredients	
Protein (g/l)	15
Source	cow's milk
Whey:Casein	60:40
Fat (g/l)	37
Coconut oil (%)	55
Soybean oil (%)	45
Carbohydrate (g/l)	7
Lactose (%)	100
Minerals (g/l)	2.9
Na (mg/l)	145
K (mg/l)	690
Cl (mg/l)	450
Ca (mg/l)	450
P (mg/l)	300
Fe (mg/l)	12
Vitamins	
A (IU)	2000
D (IU)	400
E (IU)	10
C (mg)	55
Folic acid (µg)	50
B1 (mg)	0.5
B2 (mg)	0.6
Niacin (mg)	8.0
B6 (mg)	0.4
B12 (µg)	1.5
Biotin (µg)	150
Panhotenic acid (mg)	3.0
K1 (µg)	150
Ca/liter	670
% Protein	9
% Fats	49
% CHO	42
Renal solute load (per 1)	100
Osmolality (mOsm/l)	278