Dietary Vitamin A Intake and Nondietary Factors Are Associated with Reversal of Stunting in Children

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ABSTRACT We examined prospectively the associations between dietary vitamin A intake, nondietary factors and growth in 8174 Sudanese children ages 6–72 mo who were stunted at the start of follow-up. All subjects were weighed and measured at baseline and at 6-mo intervals for 18 mo of follow-up. Dietary vitamin A intake during the prior 24 h was assessed using recall of vitamin A–containing foods at baseline and 6-mo intervals. We examined the association of dietary vitamin A intake with growth and the incidence of recovery of stunting after controlling for age, sex, breast-feeding status and socioeconomic variables. We found that carotenoid intake was associated with a greater incidence of reversal of stunting. Children in the highest quintile grew 13 mm more during the study period than children in the lowest quintile [95% confidence interval (CI): 0–25 mm] in multivariate analyses. The relative risk (RR) of recovery associated with vitamin A intake was greater in infants up to 1 y old (RR = 3.3, CI: 0.9–11.7) than in children ≥3 y of age (RR = 1.0, CI: 0.8–1.3) (P-value for interaction = 0.08). Diets rich in carotenoids may increase the rate of recovery from stunting in children. Dietary effects on growth might be strongest among very young children and those who have been most malnourished. Age, sex, breast-feeding status, socioeconomic status and severity of baseline stunting also were associated with reversal of stunting in this population. J. Nutr. 130: 2520–2526, 2000.

KEY WORDS: vitamin A, stunting, child growth, developing countries, Sudan

Malnutrition is a major public health problem in many parts of the world. One of the clearest manifestations of prolonged malnutrition in childhood is compromised stature; it has been estimated that 43% of children worldwide are stunted (De Onis et al. 1993). The extent to which stunting in early childhood is reversible and the factors that determine the likelihood of reversal of stunting have not been fully elucidated.

Laboratory studies have shown that vitamin A deficiency is associated with poor growth patterns (Underwood 1984). Large cross-sectional studies similarly have observed an association between xerophthalmia, a clinical sign of vitamin A deficiency, and low height-for-age (Brink et al. 1979, Cohen et al. 1993, Santos et al. 1983). Observational studies have found associations between the rate of linear growth and the level of dietary intake of vitamin A (Fawzi et al. 1997b, Ramakrishnan and Martorell 1998). In intervention studies in developing countries, however, the effects of supplementary feeding programs on linear growth have been small (Beaton et al. 1993, Ramakrishnan and Martorell 1998). It has been suggested that the variability in the observed effect of vitamin A supplementation on growth is attributable in part to differences in other characteristics of the respective study populations, such as age, the level of baseline nutritional deficiencies and growth deficits, and the burden of infection (Allen 1994, Bahl et al. 1997).

A number of studies have examined the roles of nondietary factors such as age, sex, socioeconomic status and health status in linear growth and the incidence of stunting. Little research has directly examined predictors of the incidence of recovery from stunting. One study that examined predictors of both stunting and reversal of stunting in young children in a rural setting found that significant predictors of stunting were not necessarily predictors of reversal of this condition (Vella et al. 1994). For example, the presence of family income from non-agricultural sources protected children from the risk of stunting but was not associated with the incidence of recovery.

Additional research is warranted to identify the determinants of recovery from stunting. The objectives of this study were to examine the association between dietary vitamin A intake, nondietary factors and the reversal of stunting in Sudanese children who participated in a longitudinal study of vitamin A and growth.

SUBJECTS AND METHODS

The data used in these analyses are taken from the Sudan Vitamin A Study which was begun in June 1988 to examine the relation between supplementary vitamin A intake and the survival, health and growth of preschool children. The original study population...
consisted of 28,753 children who were between 6 and 72 mo of age at baseline, free of clinical signs of vitamin A deficiency and living in any of five primarily rural regions in northern Sudan. Field personnel were divided into teams of five (2 interviewers, 2 anthropometrists and a supervisor). Interviewers enrolled all eligible children in alternate households and randomly assigned them to receive either 60,000 μg (200,000 IU) vitamin A with 40,000 μg (40 IU) vitamin E or 40 mg vitamin E alone. Follow-up took place at 6, 12 and 18 mo after baseline data collection (rounds 2–4). Children who displayed any signs of xerophthalmia at any round were given vitamin A and excluded from further follow-up. Living children not present at the time of a visit were not followed up further.

The study was approved by the Committee on the Use of Human Subjects in Research at the Harvard School of Public Health, the director general of primary health care at the Ministry of Health in the Sudan and the directors of health for the Khartoum and Central regions.

**Anthropometric measurements.** Anthropometricians measured the height and weight of all children at a central location after each round of household visits was completed. Children were weighed with a Salter scale to the nearest 100 g. Height was measured to the nearest 1 mm with a locally made anthropometer. Recumbent length measurements were taken on children < 85 cm in height. All team members were trained to use carefully standardized methods of anthropometric measurement. We used the Centers for Disease Control Anthropometric Software Package, which is based on National Center for Health Statistics (NCHS)2 growth curves (Hamill et al. 1977), to calculate anthropometric indicators. We considered children with height-for-age measures >2 SD below the NCHS median to be stunted. Children with weight-for-height measures >2 SD below the median were considered wasted. We examined the relation of intake from each of six food groups with growth. We evaluated the roles of the individual foods that accounted for most of the variability between subjects in dietary vitamin A intake as well as their height at the end of follow-up. Adjusting for baseline anthropometry can be expected to attenuate the diet-growth association to the extent that baseline height is on the causal pathway between diet and ultimate size. Therefore, we analyzed the diet-growth association both with and without adjustment for baseline height. Similarly, severity of baseline stunting may be on the causal pathway between vitamin A intake and the incidence of reversal of stunting. Therefore, we examined the role of vitamin A in the reversal of stunting both with and without adjustment for severity of baseline stunting. We used simple and multivariate linear regression models to assess the association between vitamin A intake and the incidence of reversal of stunting, we examined the association to the extent that baseline height is on the causal pathway between diet and ultimate size. Therefore, we analyzed the diet-growth association both with and without adjustment for baseline height. Similarly, severity of baseline stunting may be on the causal pathway between vitamin A intake and the incidence of reversal of stunting. Therefore, we examined the role of vitamin A in the reversal of stunting both with and without adjustment for severity of baseline stunting. We used simple and multivariate linear regression models to assess the association between vitamin A intake and the incidence of reversal of stunting, we examined the association to the extent that baseline height is on the causal pathway between diet and ultimate size. 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the highest quintile of vitamin A intake were 34 mm taller at round 4 than children in the lowest quintile [95% confidence interval (CI): 20–47 mm]. In multivariate analyses, the effect of total vitamin A was substantially attenuated such that intake in the highest quintile was no longer significantly associated with greater attained height, although children in the fourth quintile of intake were 12 mm taller than children in the lowest quintile (95% CI: 0–25 mm).

We also studied the association between dietary vitamin A intake at rounds 1–3 and height at round 4, controlling for height at baseline. That is, we examined the association of dietary intake with change in height during the study period. Total vitamin A intake was not a significant predictor of change in height in multivariate analysis.

Similar analyses were conducted to examine the role of carotenoid and preformed vitamin A intake in growth. Children in the highest quintile of carotenoid intake were on average 40 mm taller at round 4 than children in the lowest quintile (95% CI: 27–52 mm) in univariate analyses. In multivariate analysis, children in the top quintile of carotenoid intake were 16 mm taller than children in the lowest quintile of intake (95% CI: 3–29 mm). Carotenoid intake continued to demonstrate an association with growth after controlling for height at baseline, and children in the highest quintile of intake grew 13 mm more during the study period than children in the lowest quintile (95% CI: 0–25 mm). Intake of preformed vitamin A intake was not associated with net attained height or change in height during the study period in either univariate or multivariate analyses.

We considered the associations of the nondietary factors in the highest quintile of vitamin A intake were 34 mm taller at round 4 than children in the lowest quintile [95% confidence interval (CI): 20–47 mm]. In multivariate analyses, the effect of total vitamin A was substantially attenuated such that intake in the highest quintile was no longer significantly associated with greater attained height, although children in the fourth quintile of intake were 12 mm taller than children in the lowest quintile (95% CI: 0–25 mm).

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In univariate analysis, total vitamin A intake was significantly associated with a greater incidence of recovery from stunting (Table 2). Children in the highest quintile of total vitamin A intake experienced a 54% greater incidence of recovery from stunting than children in the lowest quintile (95% CI: 1.32–1.80). This association was not present after controlling for other predictors of reversal of stunting.

Carotenoid intake was significantly associated with a greater incidence of reversal of stunting in crude analysis, with children in the highest quintile experiencing a 66% higher rate of recovery than children in the lowest quintile relative risk (RR) = 1.66, 95% CI: 1.43–1.92]. Carotenoid intake continued to show an association with the probability of overcoming stunting in multivariate analyses, with a risk ratio of 1.19 comparing children in the highest and lowest quintiles (95% CI: 1.00–1.41). A significant association was not found after controlling for severity of baseline stunting.

In univariate logistic regression analysis, the relative risk of reversal of stunting comparing children in the highest and lowest quintiles of preformed vitamin A intake was 1.28 (95% CI: 1.10–1.50). There was no association between preformed vitamin A intake and reversal of stunting in multivariate analyses.

We examined the relative risk of recovery from stunting associated with each additional serving of specific foods and food groups over the course of three nonconsecutive days using multivariate linear regression models. Each additional serving of green leafy vegetables was associated with an 8% increase in the incidence of recovery (multivariate RR: 1.08, 95% CI: 1.01–1.16). Each additional serving of yellow/orange vegetables was associated with a 6% increase in the incidence of recovery (RR = 1.06, 95% CI: 1.01–1.11). Of the six foods that accounted for most of the between-person variability in vitamin A consumption, garden rocket (a green leafy vegetable) was the only food significantly associated with growth in these analyses. Each additional serving of garden rocket was associated with an 11% increase in the incidence of reversal of stunting (multivariate RR = 1.11, 95% CI: 1.00–1.23).

We considered the associations of the nondietary factors in
our models with the incidence of reversal of stunting using multivariate analysis (Table 3). Age and severity of stunting at baseline were the two strongest predictors of the incidence of reversal of stunting by round 4. Relative to infants 6–12 mo old, children who were 1–2 y old at baseline were more likely to recover from stunting (RR = 1.62, CI: 1.15–2.29). Children > 2 y old at baseline were significantly less likely to recover from stunting than infants. The risk ratio for 2- to 3-y-olds was 0.41 (95% CI: 0.28–0.60); for children ≥ 3 y old at the start of follow-up, the risk ratio was 0.58 (95% CI: 0.40–0.84).

As expected, severity of baseline stunting was the strongest predictor of the incidence of recovery. Compared with children who were 2–3 Z-scores below the median height-for-age, those who were 3–4 Z-scores below the median experienced an 88% lower likelihood of recovery (RR = 0.12, 95% CI: 0.10–0.14), and those who were ≥4 Z-scores below the median were 96% less likely to recover (RR = 0.04, 95% CI: 0.03–0.05).

Socioeconomic status, measured by both household water supply and maternal literacy, also was a significant predictor of the incidence of reversal of stunting. Children in homes that lacked an inside water supply had a 20% lower incidence of reversal of stunting (RR = 0.80, 95% CI: 0.69–0.92). The incidence of reversal among children whose mothers were illiterate was 27% lower than among those whose mothers were literate (RR = 0.73, 95% CI: 0.63–0.84). Among subjects who were 18 mo or younger at baseline, those who were breast-fed at baseline had a 31% lower incidence of reversal than those not breast-feeding (RR = 0.69, 95% CI: 0.56–0.86). Boys also had a lower incidence of recovery from stunting (RR = 0.85, 95% CI: 0.75–0.95).

Supplementation status was not associated with multivariate attained height, change in height or the incidence of recovery from stunting in any of our analyses.

In addition to studying the main effects of these factors on the reversal of stunting, we examined the extent to which they modified the association between dietary vitamin A intake and growth by conducting individual logistic regressions within strata of these factors (results not shown). Although the interaction was not significant (P = 0.08), the relationship between dietary vitamin A intake (comparing those in the highest quintile with those in the lowest quintile) and the incidence of reversal of stunting appeared to vary by age. The magnitude of the diet-recovery association was strongest among subjects who were up to 12 mo old at baseline (RR = 3.3, 95% CI: 0.9–11.7). Each subsequent age group experienced a progressively more attenuated association between diet and growth, and children ≥ 3 y old at baseline did not

### Table 2

<table>
<thead>
<tr>
<th>Quintile of intake</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>P value for linear trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total vitamin A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds of reversal²</td>
<td>1.00</td>
<td>1.14 (0.97, 1.33)</td>
<td>1.14 (0.99, 1.33)</td>
<td>1.42 (1.23, 1.64)</td>
<td>1.54 (1.32, 1.80)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Odds of reversal³</td>
<td>1.00</td>
<td>1.01 (0.85, 1.20)</td>
<td>0.99 (0.84, 1.16)</td>
<td>1.11 (0.95, 1.31)</td>
<td>1.15 (0.96, 1.37)</td>
<td>0.0745</td>
</tr>
<tr>
<td>Carotenoids</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds of reversal²</td>
<td>1.00</td>
<td>1.26 (1.08, 1.47)</td>
<td>1.41 (1.20, 1.64)</td>
<td>1.46 (1.21, 1.77)</td>
<td>1.66 (1.43, 1.92)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Odds of reversal³</td>
<td>1.00</td>
<td>1.02 (0.87, 1.21)</td>
<td>1.10 (0.93, 1.33)</td>
<td>1.10 (0.90, 1.36)</td>
<td>1.19 (1.00, 1.41)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Preformed vitamin A⁴</td>
<td>1.00</td>
<td>1.09 (0.96, 1.23)</td>
<td>1.28 (1.10, 1.50)</td>
<td>1.28 (1.10, 1.50)</td>
<td>1.28 (1.10, 1.50)</td>
<td>0.0020</td>
</tr>
<tr>
<td>Odds of reversal²</td>
<td>1.00</td>
<td>1.09 (0.96, 1.23)</td>
<td>1.28 (1.10, 1.50)</td>
<td>1.28 (1.10, 1.50)</td>
<td>1.28 (1.10, 1.50)</td>
<td>0.0020</td>
</tr>
<tr>
<td>Odds of reversal³</td>
<td>1.00</td>
<td>1.08 (0.95, 1.23)</td>
<td>0.97 (0.82, 1.14)</td>
<td>0.97 (0.82, 1.14)</td>
<td>0.97 (0.82, 1.14)</td>
<td>0.7969</td>
</tr>
</tbody>
</table>

1 RR < 1.0 indicates a lower incidence of recovery, relative to the comparison group; RR > 1.0 indicates a greater relative incidence of recovery.
2 From logistic regression model including quintiles of dietary vitamin A intake (4 dummy variables) during the 12 mo of follow-up as predictors of height after 18 mo.
3 From logistic regression model including age, wealth, availability of water in the house, maternal literacy, region of residence and breast-feeding status at baseline, gender, capsule and quintiles of dietary vitamin A intake (4 dummy variables).
4 Quartiles 2, 3 and 4 are merged into one variable because of limited variation in intake.

### Table 3

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Subjects, n</th>
<th>RR (95% CI) of reversal of stunting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A supplementation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>3996</td>
<td>1.00</td>
</tr>
<tr>
<td>Yes</td>
<td>4178</td>
<td>0.97 (0.86, 1.09)</td>
</tr>
<tr>
<td>Age at baseline, y =1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–2</td>
<td>230</td>
<td>1.00</td>
</tr>
<tr>
<td>1–2</td>
<td>1719</td>
<td>1.62 (1.15, 2.29)</td>
</tr>
<tr>
<td>2–3</td>
<td>1684</td>
<td>0.41 (0.28, 0.60)</td>
</tr>
<tr>
<td>3+</td>
<td>4541</td>
<td>0.58 (0.40, 0.84)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>4125</td>
<td>1.00</td>
</tr>
<tr>
<td>Male</td>
<td>4049</td>
<td>0.85 (0.75, 0.95)</td>
</tr>
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<td>Water in house</td>
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<tr>
<td>Yes</td>
<td>3347</td>
<td>1.00</td>
</tr>
<tr>
<td>No</td>
<td>4827</td>
<td>0.80 (0.69, 0.92)</td>
</tr>
<tr>
<td>Maternal literacy</td>
<td></td>
<td></td>
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<tr>
<td>Yes</td>
<td>2139</td>
<td>1.00</td>
</tr>
<tr>
<td>No</td>
<td>5962</td>
<td>0.73 (0.63, 0.84)</td>
</tr>
<tr>
<td>Breastfeeding at baseline</td>
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<td></td>
</tr>
<tr>
<td>No</td>
<td>6474</td>
<td>1.00</td>
</tr>
<tr>
<td>Yes</td>
<td>1692</td>
<td>0.69 (0.56, 0.86)</td>
</tr>
<tr>
<td>Severity of stunting at baseline</td>
<td></td>
<td></td>
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<tr>
<td>≤ 2 – 3 SD</td>
<td>4276</td>
<td>1.00</td>
</tr>
<tr>
<td>≤ 3 – 4 SD</td>
<td>2308</td>
<td>0.12 (0.10, 0.14)</td>
</tr>
<tr>
<td>≤ 4 SD</td>
<td>1590</td>
<td>0.04 (0.03, 0.05)</td>
</tr>
</tbody>
</table>

1 From linear regression models that included age, sex, wealth, availability of water in the house, maternal literacy, region of residence, breast-feeding status, supplementation status and quintile of dietary vitamin A intake (4 dummy variables).
2 Relative risk (RR) < 1.0 indicates a lower incidence of recovery relative to the comparison group.
appear to benefit at all from higher levels of vitamin A intake (RR = 1.0, 95% CI: 0.8–1.3).

The relative risk of recovery associated with vitamin A intake was 2.2 (95% CI: 1.0–4.8) among the children who were most severely stunted at baseline (≤4 Z-scores below the median), compared with 0.9 (95% CI: 0.8–1.2) among those least stunted at baseline (2–3 Z-scores below the median). Although this suggests that dietary vitamin A is associated with growth in the children who are most malnourished at baseline, modification of the diet-growth association by severity of baseline stunting was not significant (P for interaction = 0.34).

The association of dietary intake with recovery from stunting did not vary between children who were randomized to vitamin A supplementation and those who received a placebo. The relative risk of recovery associated with the highest quintile of dietary vitamin A intake was 1.1 in supplemented children and 1.2 in unsupplemented children (P for interaction = 0.62).

DISCUSSION

We found that carotenoid intake was associated with attained height among children who were stunted at baseline, as well as with changes in height and the incidence of reversal of stunting during the period of follow-up in this population. Our analyses suggested that the association between total vitamin A intake and growth was strongest among infants. These results are based on multivariate analyses controlling for the possible confounding effects of age, sex, and baseline socioeconomic and breast-feeding status.

The dietary assessment method used in this study is associated with measurement error (Beaton et al. 1983), resulting in random misclassification of exposure status. This error may have resulted in an underestimation of the magnitude of the existing associations between diet and growth.

The possibility of residual confounding by the variables in the model, or confounding by unmeasured dietary and nondietary variables, cannot be excluded. One potential confounder of the diet-growth association is total energy intake. Although data were not collected on total energy intake, energy intake calculated from the limited list of foods in the survey was not associated with growth in earlier analyses (Fawzi et al. 1997b), suggesting that the protective effects observed were not due to effects of overall food consumption.

Childhood morbidity is another potential confounder of the diet-growth relationship. However, childhood morbidity is also likely to be on the causal path between dietary intake and growth; thus it may not be appropriate to control for this factor. We attempted to control for baseline health using variables indicating whether the child had recently experienced a fever, lower respiratory infection, diarrhea or measles before baseline measurements were made. These factors did not affect the associations of interest and were removed from the final models. The lack of association in our models may be due either to a weak association between morbidity and growth or to substantial misclassification of health status using these variables.

As noted earlier, children < 85 cm in height were measured supine and those ≥85 cm were measured standing up. Attained height may be underestimated for children who were measured supine at baseline and standing up at round 4. In multivariate analyses controlling for age, however, we do not expect this factor to contribute to the estimated associations between dietary intake and attained height or change in height.

Animal studies have suggested a causal relationship between vitamin A deficiency and decreased growth in rats, through mechanisms that include loss of appetite and poor absorption and metabolism of other nutrients, followed by recovery in growth with the reintroduction of vitamin A in the diet (Underwood 1984). Observational studies have shown that vitamin A intake is inversely associated with the incidence and severity of childhood infections (Barreto et al. 1994, Beaton et al. 1993, Lie et al. 1993, Underwood and Arthur 1996), which in turn can cause growth faltering through mechanisms including reduced intake, malabsorption and elevated nutritional demands of illness (Black et al. 1984, Martorell et al. 1975, Neumann and Harrison 1994, Rahman and Wahed 1983). Vitamin A also is known to play a role in cellular differentiation and organ growth (Zile et al. 1979), and perhaps in the multiplication and differentiation of cells at the growth plate of long bones (Wolbach 1947). There is evidence that intake of this micronutrient is additionally associated with levels of nocturnal growth hormone secretion (Evain-Brion et al. 1994).

Randomized trials, however, have not tended to find any association between vitamin A supplementation and growth. An intervention study in a population of ~1500 children in Ghana, of whom 48% were stunted, did not show an effect of supplementation on linear growth (Kirkwood et al. 1996). A randomized trial of 3377 children in rural Nepal (West et al. 1997) and two trials in India (Bahl et al. 1997, Ramakrishnan et al. 1995) with ~900 and ~590 children, respectively, all failed to show an association between vitamin A supplementation and growth. Vitamin A supplementation did not have an appreciable effect on linear growth in this study population in the Sudan (Fawzi et al. 1997a). The significant association between dietary vitamin A intake and linear growth concurrent with a lack of effect of supplementation was noted in the study population (Fawzi et al. 1997b) as well as in the smaller study in India cited above (Ramakrishnan et al. 1995). It is possible that other nutrients present in foods rich in vitamin A are responsible in part for the associations observed. Dietary vitamin A also may be more bioavailable than large-dose supplementation. Again, we cannot rule out the possibility that the observed association is a function of confounding. A randomized trial of dietary vitamin A would be required to minimize the risk of observing a confounded association.

We found that linear growth was associated with dietary carotenoid intake, but not with intake of preformed vitamin A. This is consistent with the findings of a study conducted in Peru, in which β-carotene intake was correlated with attained height, but preformed vitamin A intake was not (Graham et al. 1981). It is also consistent with the finding that carotenoid intake was more strongly associated with morbidity and mortality than was preformed vitamin A intake in the present study population (Fawzi et al. 1994 and 1995). The lack of an association between preformed vitamin A intake and improved growth or health in multivariate analyses in this population may be due in part to the limited range of intake of this nutrient, which is found primarily in animal sources.

In multivariate analyses predicting attained height, change in height and the incidence of recovery from stunting, the association between carotenoid intake and growth is present only in those children at the highest quintile of intake, suggesting that there is a threshold below which carotenoids may not exert an effect on growth. However the range of intake in this category (314–965 retinol equivalents per day) is modest, and exactly half of the children in this group have intake levels below the FAO/WHO recommended daily intake of 400 retinol equivalents (FAO/WHO 1967).
Our analyses suggested that dietary vitamin A was more strongly associated with recovery from stunting among the youngest children in the study, and that increasing age was associated with a weaker association between diet and growth. This interaction was of borderline significance. An intervention study of the effects of protein-energy supplementation on growth in Guatemala similarly indicated that infants in the first year of life experienced greater anthropometric benefits from supplementation than older children (Schroeder et al. 1995). It has been suggested that nutritional interventions can exert the greatest effects on growth of stunted children in the first year of life because this represents the time of the greatest potential growth velocity, as well as the period in which insults from infection associated with weaning are most frequent (Lutter et al. 1990, Martorell et al. 1994, Neumann and Harrison 1994, Schroeder et al. 1995).

Although point estimates suggested that diets rich in vitamin A may be more strongly linked to growth among the children who were the most malnourished at baseline, this interaction did not approach significance. Although the randomized trial in rural Nepal did not show an effect of vitamin A supplementation on growth, an analysis that included xerophthalmic subjects who were treated outside the randomized design found that supplementation was associated with larger growth increments in xerophthalmic children than in nonxerophthalmic children, suggesting that the effect of vitamin A varies according to baseline deficiency status (West et al. 1997). In a review of epidemiologic research on the determinants of recovery from stunting, it was suggested that the benefits of nutritional improvements tend to be greatest when deficits are highest (Martorell et al. 1994). It has been noted that the exclusion of children with clinical signs of xerophthalmia in all of the randomized trials of vitamin A described here may have attenuated the associations between vitamin A and growth (Ramakrishnan et al. 1995, West et al. 1997). A more powerful study would be required for an adequate test of which nutritional interventions are most effective in the more severely malnourished populations.

The large sample size in this study allowed us also to examine the roles of some nondietary factors in the reversal of stunting: this is an area that remains not fully understood. In our analyses of the direct associations of nondietary factors with growth, we observed that boys in our study were less likely to recover from stunting than girls. A longitudinal study in the Philippines found that stunting was more prevalent among boys than girls in the first 2 years of life, but that boys were at lower risk of being stunted in later childhood relative to girls (Ricci and Becker 1996). This would suggest a higher rate of recovery among boys than girls, which contrasts with our results. On the other hand, a longitudinal population-based study in Senegal observed that stunted preschool boys achieved lower strides in growth than similarly stunted girls, when followed to adolescence (Simondon et al. 1998). The findings of sex differentials in the rate of recovery from stunting and the disparities in the direction of this differential suggest that the sex differences in the rate of reversal may not be biological in nature. It may be worthwhile to explore whether sex differences in the probability of recovery can be explained by social factors such as differential access to health care or differences in diet and care received at home.

Children who were breast-fed at the start of follow-up experienced lower rates of recovery than nonbreast-fed children. Other studies have found an association between breast-feeding beyond infancy and compromised growth (Fawzi et al. 1998, Victoria et al. 1991 and 1984). It has been suggested that the inverse association between prolonged breast-feeding and growth is a function of inadequate complementary feeding (Fawzi et al. 1998). The association may also be due to reverse causality; that is, poor strides in growth may induce some mothers to continue breast-feeding (Marquis et al. 1997, Simondon and Simondon 1998).

Although the results suggest that the incidence of recovery from stunting is greatest among children who were 1–2 years old at baseline, it is important to note that this result may be an artifact of the change in reference populations used by the NCHS at 2 years of age (WHO 1995). As a result of the disjunction in the NCHS reference curve, children appear erroneously to improve in length-for-height when they reach 2 years of age. On the contrary, our results indicate that incidence of recovery from stunting tends to decline with age.

Our findings suggest that diets rich in vitamin A, particularly in the form of carotenoids, can improve growth and increase the rate of recovery from stunting among malnourished children. The results also suggest that dietary effects on growth might be strongest among very young children and among those who have been the most chronically malnourished. The inconsistency of the findings of the many studies regarding the association between vitamin A intake and growth suggests that multiple dietary and nondietary factors may modify the influence of micronutrient intake on growth. The potential of other factors to affect growth directly and to modify the associations between micronutrient exposures and growth points to the importance of addressing the multiple causes of growth faltering in any campaign to improve the health of children in developing countries.

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