THE LANCET

Vol 338 Saturday 6 July 1991 No 8758

ORIGINAL ARTICLES

Nutritional supplementation, psychosocial stimulation, and mental development of stunted children: the Jamaican Study

S. M. GRANTHAM-MCGREGOR C. A. POWELL S. P. WALKER J. H. HIMES

There is little unequivocal evidence that nutritional supplementation of undernourished children has a beneficial effect on their mental development. The effects of nutritional supplementation, with or without psychosocial stimulation, of growth-retarded (stunted) children aged 9–24 months were assessed in a study in Kingston, Jamaica. 129 children from poor neighbourhoods were randomly assigned to four groups—control, supplemented only, stimulated only, and supplemented plus stimulated. A group of matched non-stunted children (n = 32) was also included. The supplement comprised 1 kg milk-based formula per week for 2 years, and the stimulation weekly play sessions at home with a community health aide. The children's development (DQ) was assessed on the Griffiths mental development scales. Initially the stunted groups' DQs were lower than those of the non-stunted group, and those of the control group declined during the study, increasing their deficit. Stimulation and supplementation had significant independent beneficial effects on the children's development. Estimates of the supplementation effect ranged from 2·2 (95% confidence limits −1·4, 5·7) for the hand and eye subscale to 12·4 (5·4, 19·5) for the locomotor subscale and those for the stimulation effect from 6·4 (2·8, 10·0) for hand and eye to 10·3 (3·3, 17·3) for locomotor. The treatment effects were additive, and combined interventions were significantly more effective than either alone. These findings suggest that poor mental development in stunted children is at least partly attributable to undernutrition.

Lancet 1991; **338:** 1–5.

Introduction

It is estimated that 40% of children under 5 years of age in developing countries have low heights for age (stunting).[1] There has been much debate about whether stunting is a harmless adaptation to poor dietary intakes or has functional consequences.[2] Several cross-sectional observational studies have shown that stunting is associated with poor developmental attainment in young children[3,4] and poor school achievement or intelligence levels in older children.[5-7] However, stunted children usually suffer from many disadvantages[8] which themselves may detrimentally affect development. Control for all these factors in observational studies is unlikely to be possible.[9] Furthermore, the quality of stimulation and undernutrition may interact in their effect on development.[10]

Preventive intervention studies in which pregnant women and their offspring were given nutritional supplementation[11-14] show design faults,[10] such as lack of random assignment to treatment groups[13,14] and lack of control for the extra attention the children may receive.[12-14] Nevertheless, there is some suggestion that nutritional supplementation has small beneficial effects on children's development.

There has been only one well-documented study of supplementation of already undernourished children, which included developmental measures.[15] Supplementation alone did not improve the children's development. Unfortunately, neither the children's dietary intakes nor growth were reported, so it is difficult to be certain that their dietary intakes did increase. Children receiving supplementation combined with stimulation showed pronounced benefits.

ADDRESSES: **Tropical Metabolism Research Unit, University of the West Indies, Jamaica** (S M Grantham-McGregor, MD, C. A. Powell, PhD, S. P. Walker, PhD), **and School of Public Health, University of Minnesota, USA** (J H. Himes, PhD). Correspondence to Dr S. M Grantham-McGregor, Tropical Metabolism Research Unit, University of the West Indies, Mona, St Andrews, Jamaica.

TABLE I—CHARACTERISTICS AT ENROLMENT OF CHILDREN, PARENTS, AND SOCIAL BACKGROUND: SUBJECTS WHO COMPLETED THE STUDY

	Control	Supplemented	Stimulated	Supplemented + stimulated	Non-stunted	Differences*
Child						
M/F	19/14	18/14	16/14	18/14	18/14	..
Age (mo)	18·5 (4·5)	18·8 (3·7)	18·8 (4·4)	18·7 (3·8)	18 8 (4·4)	0·1 (6·0)
Weight for age†	−2·4 (0·5)	−2·4 (0·6)	−2·6 (0·5)	−2·7 (0·6)	0 (0·7)	2·5 (0·9)
Weight for length†	−0·8 (0·7)	−1·0 (0·8)	−1·1 (0·6)	−1·2 (0·7)	0 (0·8)	1·0 (1·1)
Length for age†	−2·9 (0·6)	−2·9 (0·6)	−3·0 (0·7)	−3·1 (0·7)	0·1 (0·6)	2·9 (0·9)
Birthweight <2·3 kg	7	0	4	9	0	..
Guardian						
Age (yr)	21·8 (4·3)	26·0 (6·1)	23·9 (6·1)	26·8 (8·4)	26·4 (7·1)	1·9 (9·7)
PPVT	82·1 (17·6)	87·1 (18·9)	85·8 (19·7)	86·6 (22·9)	96·7 (20·0)	11·6 (28·0)
No employed	2	8	7	11	10	..
Social background						
Housing rating	7·7 (1·7)	7·4 (1·8)	7·6 (1·0)	6·8 (1·5)	8·5 (1·4)	1·1 (2·1)
HOME	17·5 (5·0)	15·7 (4·4)	16·3 (4·2)	16·0 (3·9)	17·4 (3·4)	1·0 (5·6)

Data given as mean (SD) or number of subjects
*Mean difference between non-stunted and combined stunted groups
†Values in Z scores of NCHS reference data

However, the effects of supplementation and stimulation could not be separated because there was no group which received stimulation only.

Although a large amount of money is spent world wide on food for undernourished children, there is little unequivocal evidence that their development benefits. In particular, no study has focused on stunted children.

We report here a 2-year intervention study of nutritional supplementation and psychosocial stimulation of stunted children. The study design resembled a clinical trial as far as possible. We also sought an interaction between supplementation and stimulation.

Methods

A house-to-house survey was carried out in most of the poor neighbourhoods in Kingston, Jamaica. It identified all children aged 9–24 months whose lengths were below −2 SD of the NCHS reference data for age and sex.[16] The children were enrolled in the study if they met other criteria—singleton pregnancy, birthweight over 1·8 kg, standard of housing and maternal education below defined levels, and no obvious mental or physical handicap. Further selection details have been reported previously.[17] The sample (n = 129) was stratified by sex and age (16 months and under, over 16 months), then children were assigned systematically to one of four groups—control (n = 33), supplemented only (n = 32), stimulated only (n = 32), and both supplemented and stimulated (n = 32). The initial order of group assignment was determined randomly. For example, every fourth child was assigned to the supplemented and stimulated group. A fifth group of children, non-stunted (n = 32), with lengths over −1 SD of the NCHS reference data, was selected by matching of each control child with the child of the same sex who lived nearest and was within 3 months of age.

Free medical care was available to all children. The children in the four stunted groups were visited every week by a community health aide, and a history of the previous week's illnesses was recorded. Visits to the control group were intended to allow for any extra attention that the supplemented children might receive. Visiting was used instead of a placebo because it was considered unethical for health service personnel to give a true placebo (ie, a low-calorie supplement) to undernourished children.

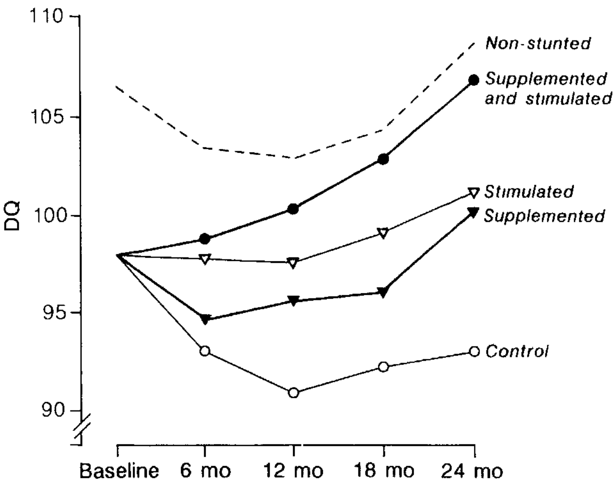
The stimulated groups took part in a programme of psychosocial stimulation similar to those we have used previously with success.[18,19] The community health aides visited the homes for 1 h per week and taught the mothers how to play with their children in such a way as to promote their development. Homemade toys were left in the home at each visit, and the mothers were encouraged to play with their children daily. A supervisor monitored 10% of all visits.

The supplemented groups received 1 kg milk-based formula per week.[20] Sufficient formula was provided for the child to receive 3·15 MJ (750 kcal) and 20 g protein daily, if our instructions were followed. We expected that sharing and substitution would take place[21] and therefore gave this large amount (about 66% of the RDA for energy). In addition, 0·9 kg cornmeal and skimmed milk powder were given to the family, in an attempt to minimise sharing of the supplement.

24 h dietary recalls for each child were done for 2 days at enrolment and after 6 months and 15 months. Each child was weighed and length, head and arm circumferences, and skinfolds were measured on enrolment, then every 6 months. Development was assessed by means of the Griffiths Mental Development

TABLE II—UNADJUSTED MEAN (SD) DEVELOPMENTAL QUOTIENTS (DQ) ON GRIFFITHS TEST

—	Control (n = 33)	Supple-mented (n = 32)	Stimul-ated (n = 30)	Supple-mented and stimul-ated (n = 32)	Non-stunted (n = 32)
DQ					
Baseline	97 (10)	98 (9)	100 (10)	98 (7)	106 (10)
6 mo	92 (7)	94 (8)	100 (7)	99 (11)	104 (10)
12 mo	90 (6)	95 (12)	99 (8)	100 (12)	103 (9)
18 mo	91 (7)	96 (11)	101 (8)	103 (12)	104 (9)
24 mo	92 (8)	100 (13)	103 (9)	107 (12)	109 (10)
Locomotor					
Baseline	102 (13)	104 (12)	103 (11)	98 (11)	114 (12)
6 mo	95 (11)	97 (13)	101 (11)	98 (14)	111 (15)
12 mo	93 (9)	101 (21)	100 (9)	103 (16)	115 (16)
18 mo	102 (20)	108 (21)	111 (19)	112 (17)	123 (20)
24 mo	108 (21)	124 (27)	122 (21)	126 (26)	136 (21)
Hearing and speech					
Baseline	95 (14)	94 (12)	99 (15)	97 (10)	104 (13)
6 mo	92 (15)	96 (13)	98 (11)	99 (15)	99 (15)
12 mo	92 (17)	97 (23)	102 (17)	101 (20)	102 (12)
18 mo	92 (17)	98 (20)	103 (15)	109 (22)	106 (17)
24 mo	97 (17)	102 (20)	108 (18)	110 (21)	111 (16)
Hand and eye					
Baseline	101 (12)	102 (12)	105 (11)	106 (10)	109 (13)
6 mo	98 (10)	98 (9)	107 (10)	104 (12)	107 (13)
12 mo	94 (10)	97 (12)	104 (10)	105 (14)	102 (12)
18 mo	90 (8)	93 (10)	97 (10)	97 (13)	99 (9)
24 mo	87 (8)	89 (10)	94 (12)	97 (13)	96 (11)
Performance					
Baseline	89 (12)	91 (12)	94 (13)	89 (9)	98 (13)
6 mo	83 (10)	86 (10)	92 (11)	92 (15)	93 (17)
12 mo	81 (10)	86 (11)	91 (16)	91 (14)	89 (12)
18 mo	80 (12)	84 (11)	92 (14)	92 (16)	89 (16)
24 mo	77 (13)	84 (15)	86 (14)	93 (14)	91 (13)



Mean DQs of stunted groups adjusted for initial age and score, compared with non-stunted group adjusted for age only.

Scales,[22] which have been modified for Jamaica. Four subscales—locomotor (large muscle activities such as running and jumping), hand and eye coordination, hearing and speech, and performance (shape recognition, block construction, and block patterns)—were used. All tests were done by one of two testers, who were trained by the same person, and who did not know the children's group assignment. In addition the testers assessed equal numbers of children from each group at each test session. Differences between testers for the development quotient (DQ) of 15 children assessed on the same occasion were small (mean 0·87 [SD 1·88]) with an intraclass coefficient of reliability of 0·96. The testers were observed in about 5% of all tests. These observations indicated a high level of standardisation throughout the study (intraclass $R = 0·97$).

On enrolment, the children's homes were visited, and details of the social background and the children's medical histories were recorded (table I). The mother's verbal IQ was assessed on the Peabody Picture Vocabulary Test (PPVT),[23] and a modified version of Bettye Caldwell index of stimulation in the home[24] (HOME) was done. The standard of housing was rated on a 12-point scale in which equal weight was given to water supply, toilet, crowding, and household possessions.

2 children were lost from the stimulated group, and were omitted from the analyses. All other children had all five developmental assessments.

The intervention effects were evaluated by multiple regression analyses of the final developmental measures, in the stunted groups only. The basic approach was to include as independent variables the initial developmental measure and age, and the child's supplementation and stimulation status each coded as dichotomous variables. This is a conservative assessment of intervention effects that takes into account both regression to the mean and correlated errors of measurements. The age of the child was included as a quadratic term if it significantly improved the model fit. In addition, in each analysis, several possible covariates which we thought were most likely to affect development or treatment responses were offered. These included sex, birthweight, initial length for age and weight for length of the child; the housing rating and HOME; and mother's age, PPVT, and employment status. Interaction terms of supplementation × stimulation, initial length × interventions, initial weight for length × interventions, and initial age × interventions were also offered. The interaction terms were rescaled by centring to avoid colinearity with the treatment and other covariates. In each analysis, the tester was an additional covariate.

To compare the stunted groups with each other and with the non-stunted group, the multiple regression analyses were repeated with the non-stunted group included. Each of the five group memberships were coded independently (0, 1), and entered into the regression models instead of the two treatment variables. The interaction terms were omitted for these analyses.

Results

At enrolment, each of the stunted groups had significantly lower mean DQs than the non-stunted group (table II) and by chance, the control group had a lower mean DQ than the stimulated group (ANOVA $p<0·001$: post analysis of variance comparisons, non-stunted and each stunted group $p<0·01$; control and stimulated $p<0·05$).

The DQs of the stunted groups adjusted for the children's initial scores and ages are shown in the figure. The non-stunted group's scores adjusted for age only are shown for comparison. DQ in the control group declined steeply during the first year then levelled off, whereas that in the supplemented and stimulated group increased gradually throughout. The scores for the stimulated only and supplemented only groups lay between the control and supplemented plus stimulated group throughout. The partial regression coefficients from the multiple regression analyses of the final developmental measures are shown in table III. The stimulation and supplementation coefficients

TABLE III—REGRESSION COEFFICIENTS (95% CONFIDENCE LIMITS) OF TREATMENT VARIABLES AND OTHER SIGNIFICANT COVARIATES IN MULTIPLE REGRESSION ANALYSES OF DEVELOPMENTAL AND SUBSCALE QUOTIENTS

—	DQ	Locomotor	Hearing and speech	Hand and eye	Performance
Enrolment to 12 mo					
Stimulated*	5·7 (2·8, 8·7)	5·8 (1·5, 10·0)	5·1 (−1·1, 11·3)	6·9 (3·1, 10·6)	7·0 (2·8, 11·1)
Supplemented*	3·4 (0·5, 6·3)	6·6 (2·3, 10·9)	2·6 (−3·5, 8·7)	1·2 (−2·5, 4·9)	3·1 (−1·0, 7·3)
Initial age	1·5 (1·0, 2·0)	1·7 (1·0, 2·4)	1·4 (0·6, 2·3)	0·2 (−0·3, 0·7)	1·4 (0·7, 2·1)
Initial score	0·7 (0·5, 1·0)	0·7 (0·5, 1·0)	0·5 (0·2, 0·7)	0·3 (0·2, 0·5)	0·5 (0·3, 0·7)
Covariates†	B	A	J, I	K, G, I	..
Adjusted R^2	0·38	0·35	0·21	0·28	0·22
12 mo to 24 mo					
Stimulated*	2·8 (0·2, 5·5)	3·5 (−2·0, 9·0)	5·5 (−0·1, 11·0)	3·8 (0·3, 7·2)	5·5 (1·2, 9·7)
Supplemented*	2·7 (0·1, 5·2)	3·0 (−2·7, 8·6)	2·6 (−2·8, 8·1)	1·5 (−1·7, 4·7)	5·8 (1·7, 9·9)
Initial age	0·3 (−0·4, 0·6)	0·8 (0·1, 1·5)	−0·4 (−1·1, 0·3)	−0·1 (−0·5, 0·3)	1·0 (0·5, 1·5)
12 month score	0·8 (0·7, 0·9)	1·1 (0·9, 1·3)	0·6 (0·4, 0·7)	0·5 (0·3, 0·6)	0·4 (0·3, 0·6)
Covariates†	B, H, F	E, I, B, F, C	..	J, A	I, H
Adjusted R^2	0·67	0·62	0·37	0·39	0·42
Enrolment to 24 mo					
Stimulated*	7·3 (4·0, 10·6)	10·3 (3·3, 17·3)	10·0 (3·5, 16·6)	6·4 (2·8, 10·0)	8·2 (3·8, 12·6)
Supplemented*	6·1 (2·9, 9·4)	12·4 (5·4, 19·5)	5·7 (−0·7, 12·2)	2·2 (−1·4, 5·7)	7·1 (2·7, 11·5)
Initial age	1·7 (1·2, 2·3)	3·7 (2·7, 4·8)	0·5 (−0·4, 1·4)	0·2 (−0·2, 0·7)	1·9 (1·2, 2·6)
Initial score	0·7 (0·5, 1·0)	1·1 (0·7, 1·5)	0·3 (0·04, 0·6)	0·3 (0·2, 0·5)	0·4 (0·2, 0·7)
Covariates†	H, C	I, E	D, L	G, J	H
Adjusted R^2	0·42	0·36	0·15	0·25	0·32

*No = 1, yes = 2

†A = length for age; B = length for age × supplementation; C = length for age × stimulation; D = weight for height; E = weight for height × stimulation, F = age²; G = age × stimulation, H = PPVT; I = tester; J = sex; K = guardian employed, L = HOME

TABLE IV—REGRESSION COEFFICIENTS (95% CONFIDENCE LIMITS) OF CONTRASTS BETWEEN GROUPS OVER 24 MONTHS

—	DQ	Locomotor	Hearing and speech	Hand and eye	Performance
Differences from non-stunted					
Control	−8·4 (−13·6, −3·2)	−12·9 (−22·9, −2·8)	−6·7 (−16·2, 2·7)	−7 7 (−13·0, −2·5)	−12·0 (−18·7, −5·2)
Stimulated	−0·5 (−5·4, 4·4)	−1·1 (−11·2, 8·9)	4·8 (−5·0, 14 7)	−0 9 (−6·2, 4·4)	−3·3 (−9·9, 3·3)
Supplemented	−1·9 (−6·9, 3·2)	−0·6 (−10·5, 9·3)	0·8 (−9·1, 10·6)	−5·4 (−10·7, −0·2)	−4·2 (−10·9, 2·4)
Supplemented + stimulated	5·0 (−0·1, 10·1)	8·4 (−2·3, 19·1)	8·4 (−1·5, 18·4)	1·1 (−4·1, 6·3)	5·0 (−1·7, 11·8)
Differences from supplemented + stimulated					
Control	−13·4 (−17·9, −8·8)	−21·3 (−30·7, −11·9)	−15·2 (−23·8, −6·5)	−8·8 (−14·0, −3·7)	−17·0 (−23·4, −10·6)
Stimulated	−5·5 (−10·2, −0·8)	−9·6 (−19·3, 0·1)	−3·6 (−12·2, 5 1)	−2·0 (−7·2, 3·2)	−8·3 (−14·9, −1·7)
Supplemented	−6·9 (−11·4, −2·3)	−9·0 (−18·5, 0·4)	−7·6 (−16·2, 0·9)	−6·6 (−11·7, −1·4)	−9·2 (−15·7, −2·8)
Differences from supplemented only					
Control	−6·5 (−11·1, −1·9)	−12·3 (−21·6, −2·9)	−7·5 (−16·1, 1·0)	−2·3 (−7·4, 2·8)	−7·8 (−14·2, −1·3)
Stimulated	1·4 (−3·3, 6·1)	−0·5 (−10·1, 9·0)	4·1 (−4·7, 12·8)	4·6 (−0·7, 9·8)	0·9 (−5·6, 7·5)
Differences from stimulated only					
Control	−7·9 (−12·6, −3·1)	−11·7 (−21·1, −2·3)	−11·6 (−20·3, −2·8)	−6·8 (−12·1, −1·6)	−8·7 (−15·3, −2·0)

are estimates of the difference in scale points between treatment and no treatment, with control for initial age and status and all other significant covariates.

Over the first 12 months, there was a significant benefit with stimulation in the DQ and all subscales (all $p < 0·01$) except hearing and speech ($p < 0·1$). With supplementation there were benefits in the locomotor subscale and DQ (both $p < 0·01$).

In the second year the effect of stimulation was significant in the performance and hand and eye subscales and the DQ, and the effect on hearing and speech approached significance ($p < 0·06$). The effects of supplementation were significant in the performance subscale and DQ.

Over the whole 2 years, stimulation improved all the subscales and the DQ (all $p < 0·01$). Supplementation had a significant effect on the locomotor and performance subscales and the DQ (all $p < 0·01$). The effect on hearing and speech was not significant ($p < 0·08$). The stimulation × supplementation interaction term was not significant in any of the regressions. Initial nutritional status did not make a consistent contribution to the variance over the 2 years.

The multiple regression analyses of the final DQ scores were repeated with the five groups entered as independent variables instead of the two interventions. Over the 2 years the three treated groups and the non-stunted group improved significantly more than the control group in DQ (all $p < 0·01$). The supplemented plus stimulated group improved significantly more than the supplemented only ($p < 0·01$) or the stimulated only ($p < 0·05$) group. The difference between the supplemented plus stimulated and non-stunted groups approached significance ($p < 0·052$). Other differences are shown in table IV.

Discussion

This is the first convincing demonstration that nutritional supplementation has a beneficial effect on stunted children's mental development. Not only was the development of the supplemented children better than that of the controls, but also the children who received both interventions did better than those who received only stimulation. Thus, the finding was replicated within the study. Experimental studies of this kind are difficult to carry out for both ethical and logistical reasons. However, most of the requirements of a clinical trial were satisfied. The children were randomly assigned to groups, and the testers were unaware of the assignment. We controlled for any extra attention the supplemented children may have received by visiting all children the same number of times. However, there was no true placebo. Unlike many longitudinal field studies, missing data were not a source of bias.

Preliminary analysis of the growth and dietary intake data has been reported.[17,20] The supplemented groups had higher dietary intakes at 6 months than at baseline and the children showed a growth spurt, albeit small. Nevertheless, there was an inevitable lack of control over the actual increase in the children's total dietary intakes. The lack of precision in the measurement of the supplement consumed would have minimised the observed effects of supplementation.[25]

Another difficulty is that the Griffith's test was neither designed nor standardised for Jamaica. However, it has been extensively used in Jamaica;[26] it gives stable results over several years and accords well with the Stanford Binet[18] and school achievement levels (unpublished).

The control group showed a fall in DQ points in the first year and then the score levelled off. A decline is expected in poor Jamaican children at this age.[26] The benefits of the intervention were therefore mainly in reducing the decline in the first year. However, in the second year, the supplemented only and stimulated only groups showed small improvements, and the supplemented plus stimulated group showed substantial improvements. All groups scored lower in the performance subscale than in the locomotor subscale, as has been previously described.[26] Supplementation mainly benefited locomotor development in the first year. However, the beneficial effect spread to the performance subscale in the second year and was beginning to affect hearing and speech. This pattern is similar to findings from preventive studies. In one study supplementation was given to mothers in pregnancy and lactation, and the offspring showed motor benefits only.[11] In two other studies supplement was also given to the offspring,[27,28] and the first developmental improvements were found in the motor and manipulative activities. Improvements appeared later in other areas of development.

In our study, the effect of supplementation on DQ developed gradually, and improvement continued in the last 6 months. The children might have improved further if supplementation had continued. In contrast, stimulation had an early effect which tended to lessen later. We have shown several times that deprived Jamaican children benefit from stimulation.[18,19] The main interest therefore centred on the possible interaction of stimulation with supplementation. Although it is often suggested that nutrition and stimulation interact in their effects on development,[10] there was no indication of a significant or near significant interaction in this study, which was designed to detect such effects.

The size of the benefit from the individual interventions was probably clinically significant, and the effects appeared to be additive. The group receiving both interventions made a substantial improvement, and caught up with the non-stunted group. However, even the non-stunted group's development was well behind that reported for middle class Jamaican children.[26,29]

The findings strongly suggest that at least part of the deficit in development found in stunted children is attributable to undernutition. It is not clear from these data which nutrient or nutrients led to the improved development. However, this study and a report on the benefits of supplementing children of low birthweight[30] indicate that nutrition probably plays a larger part in child development than previously acknowledged. The findings highlight the waste in human potential of millions of stunted third world children.

This study was funded by the Ford Foundation, USA, with assistance from the Population Council. S. G. McG. is partially supported by the Wellcome Trust, UK. The supplement was donated by Cow & Gate, UK, Grace Kennedy & Co Ltd, Jamaica Commodity Trading Co, and Seprod Ltd, Jamaica. We thank S. Chang, D. Simeon, P. Fletcher, K. McDonald, S. Lalljie, L. Dacosta, and J. Meeks Gardner for their assistance.

REFERENCES

1. Keller W. The epidemiology of stunting. In: Waterlow JC, ed. Linear growth retardation in less developed countries. Nestle Nutrition Workshop 14. New York: Raven Press, 1988: 17–40.
2. Waterlow JC. What do we mean by adaptation? In: Blaxter K, Waterlow JC. eds. Nutritional adaptation in man. London: John Libbey, 1985: 1–12.
3. Lasky RE, Klein RE, Yarbrough C, Engle PL, Lechtig A, Martorell R. The relationship between physical growth and infant behavioural development in rural Guatemala. *Child Develop* 1981; **52:** 220–26.
4. Powell CA, Grantham-McGregor SM. The ecology of nutritional status and development in young children in Kingston, Jamaica. *Am J Clin Nutr* 1985; **41:** 1322–31.
5. Jamison DT. Child malnutrition and school performance in China. *J Develop Econ* 1977; **20:** 299–309.
6. Moock PR, Leslie J. Childhood malnutrition and schooling in the Teri region of Nepal. *J Develop Econ* 1986; **20:** 33–52.
7. Clarke NW, Grantham-McGregor SM, Powell C. Health and nutrition predictions of school failure in Kingston, Jamaica. *Ecol Food Nutr* (in press).
8. Martorell R, Mendoza F, Castillo R. Poverty and stature in children. In: Waterlow JC, ed. Linear growth retardation in less developed countries. Nestle Nutrition Workshop 14. New York: Raven Press, 1988: 57–74.
9. Richardson SA. The background history of school children severely malnourished in infancy. In: Schulman I, ed. Advances in pediatrics, vol 21. Chicago: Yearbook Medical Publishers Inc, 1974; 167–95.
10. Grantham-McGregor SM. Field studies in early nutrition and later achievement. In: Dobbing JH, ed. Early nutrition and later achievement. London: Academic Press, 1987: 128–74.
11. Joos SK, Pollitt E, Mueller WH, Albright DL. The Bacon Chow Study: maternal nutritional supplementation and infant behavioural development. *Child Develop* 1983; **54:** 669–76.
12. Waber DP, Vuori-Christiansen L, Ortiz N, et al. Nutritional supplementation, maternal education and cognitive development of infants at risk of malnutrition. *Am J Clin Nutr* 1981; **34:** 807–13.
13. Freeman HE, Klein RE, Townsend JW, Leghtig A. Nutrition and cognitive development among rural Guatemalan children. *Am J Publ Health* 1980; **70:** 1277–88.
14. Chavez A, Martinez C. Growing up in a developing community. Guatemala City: INCAP, 1982.
15. McKay H, Sinesterra L, McKay A, Gomez H, Lloreda P. Improving cognitive ability in chronically deprived children. *Science* 1978; **200:** 270–78.
16. Hamill PVV, Drizd TA, Johnson CL, Reed RB, Roche AF, Moore WM. Physical growth: National center for health statistics percentiles. *Am J Clin Nutr* 1979; **32:** 607–29.
17. Walker SP, Powell CA, Grantham-McGregor SM. Dietary intakes and observed activity levels of stunted and non-stunted children in Kingston, Jamaica. Part I Dietary intakes. *Eur J Clin Nutr* 1990; **44:** 527–34.
18. Grantham-McGregor SM, Schofield W, Powell C. Development of severely malnourished children who received psychosocial stimulation: six year follow-up. *Pediatrics* 1987; **79:** 247–54.
19. Powell CA, Grantham-McGregor SM. Home visiting of varying frequency and child development. *Pediatrics* 1989; **84:** 157–65.
20. Walker S, Powell CA, Grantham-McGregor SM, Himes JH, Chang S. Nutritional supplementation, psychosocial stimulation and growth of stunted children: the Jamaican Study. *Am J Clin Nutr* (in press).
21. Beaton GH, Ghassemi H. Supplementary feeding programs for young children in developing countries. *Am J Clin Nutr* 1982; **35** (suppl).
22. Griffiths R. The abilities of babies. London: University of London Press, 1967.
23. Dunn LN. Peabody picture vocabulary test. Nashville Tennessee: American Guidance Service, 1965.
24. Caldwell BM. Descriptive evaluation of child development and of developmental settings. *Pediatrics* 1967; **40:** 46–50.
25. Barrett DE. Methodological requirements for conceptually valid research studies on the behavioural effects of malnutrition. In: Galler JR, ed. Human nutrition 5. Nutrition and behaviour. New York and London: Plenum Press, 1984; 9–36.
26. Walker SP, Grantham-McGregor SM. Growth and development of West Indian children. Part 2: Development. *West Ind Med J* 1990; **39:** 12–19.
27. Klein R. Malnutrition and human behaviour: a backward glance at an ongoing longitudinal study. In: Levitsky DA, ed. Malnutrition environment and behaviour. Ithaca: Cornell University Press, 1979; 219–37.
28. Mora JO, Clement J, Christiansen N, Ortiz N, Vuori L, Wagner M. Nutritional supplementation early stimulation and child development. In: Brozek J, ed. Behavioural effects of energy and protein deficits Washington DC: DHEW Publ No (N1H) 79–1906: 1979; 255–69.
29. Chambers C, Grantham-McGregor SM. Patterns of mental development among young, middle-class Jamaican children. *Child Psychol Psychiatry* 1986; **27:** 117–23.
30. Lucas A, Morley R, Cole TJ, et al. Early diet in preterm babies and developmental status at 18 months. *Lancet* 1990; **335:** 1477–81.

From The Lancet

Mushrooms as food

The alarming symptoms which occasionally follow the use of fungi when taken as food are familiar to most of our readers. The risk in this particular, however, is less than it might be. In actual market custom we recognise but a very few forms of edible fungi, though it must be allowed that even in these we are liable to deception of a somewhat dangerous kind. It is therefore a matter of some importance that the public mind should be informed as far as possible of the qualities which distinguish the edible from the poisonous varieties. To give a precise definition which would also be comprehensive is, however, no simple matter, and as a matter of fact the number of edible fungi, even in this country, is much greater than is commonly understood. It may be said, however, that a high colour, a scaly or spotted surface, and tough or watery flesh are usually associated with poisonous properties, while the edible species are but seldom highly coloured, scaly, or spotted, but usually white or brownish, and brittle on fracture. The former, moreover, grow clustered on wet or shady ground, the latter singly in dry pastures. The common British mushroom is known by its pink hymenium or gills. Fungi which have a bitter or styptic taste, or which burn the fauces, as well as those which yield a pungent milk, those of livid colour, and those which on bruising assume various hues, ought to be avoided. It should be remembered also that all plants of this class readily undergo decomposition, and should therefore be eaten as fresh as possible.

(May 30, 1891)