Nutritional stunting in Egypt: which nutrient is responsible?

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التقزُّم التغذوي في مصر: ما المغذيات المسؤولة عنه؟

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الخلاصة: تمت دراسة العلاقة بين التخلف الخطي في النمو وبين عوز مغذيات معينة لدى الأطفال في مصر. وتم انتقاء 107 من الأطفال المتقزمين يتراوح عمرهم من 10 إلى 18 عاماً، وتمت دراسة تاريخهم الصحي، وأُجريت لهم اختبارات مختبرية وفحوص جسمية. وتمت إحالة حالات منتقاة منهم إلى وحدة الأشعة لقياس العمر العظمي. وتم تعيين 39 طفلاً منهم كشواهد للدراسة. ودلت الاختبارات على وجود انخفاض ملموس في مستويات الهيموغلوبين المصلي، والألفاتوكوفيرول، والفيتامين "أ"، والمغنزيوم بين الأطفال المتقزمين بالمقارنة مع الشواهد. كما بيَّنت الاختبارات انخفاض مستويات الزنك في مجموعة الأطفال المتقزمين ولكنه انخفاض لا يُعتد به. وتستنتج الدراسة وجود عوز لعدة مغذيات في وقت واحد في الأطفال المتقزمين، وقد تكون جميعها مسؤولة عن التقزم.

ABSTRACT We looked at the relationship between linear growth retardation and deficiencies of certain nutrients in Egyptian children. A group of 107 stunted children aged between 10 and 18 years were subjected to history-taking, physical examination and laboratory investigations. Selected cases were referred to radiology for assessment of bone age. Thirty-nine children were enrolled as controls. Serum haemoglobin, α-tocopherol, retinol and magnesium levels were significantly decreased in stunted children compared with the controls. Serum zinc levels were also lower in the stunted group but not significantly so. We conclude that several nutrient deficiencies occur simultaneously in stunted children and all of them may be responsible for stunting.

Le retard de croissance d'origine nutritionnelle en Egypte : quel nutriment est en cause ?

RESUME Nous avons examiné la relation entre le retard de croissance statural et les carences en certains nutriments chez des enfants égyptiens. Un groupe de 107 enfants âgés de 10 à 18 ans présentant un retard de croissance a fait l'objet d'une anamnèse, d'un examen physique et d'examens de laboratoire. Certains cas ont été orientés vers un radiologue pour évaluation de l'âge osseux. Trente-neuf enfants appariés ont été inclus comme témoins. Les taux d'hémoglobine, d'alpha-tocophérol, de rétinol et de magnésium sériques étaient significativement réduits chez les enfants présentant un retard de croissance par rapport aux témoins. Les niveaux de zinc sérique étaient également plus faibles dans le groupe des enfants présentant un retard de croissance mais de manière moins significative. Nous concluons que plusieurs carences en nutriments surviennent simultanément chez les enfants présentant un retard de croissance et que toutes ces carences peuvent être responsables du retard de croissance.

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Introduction

Linear growth retardation (stunting) is common among children in Egypt [1]. It has been shown repeatedly that extreme short stature of preschool children in developing countries is not genetically determined but is a consequence of poor socioeconomic conditions [2]. Stunting in children is related to concurrent, and possibly later, delayed mental and motor development [3]. The negative effects in adulthood include limited work capacity owing to reduced muscle mass [4] and increased obstetric risks in women because of short stature [5]. Studies have shown that deficiencies of single or multiple micronutrients, such as zinc, iron, vitamin A or iodine [6–8], may cause linear growth retardation. The effects of infection on linear growth are also well known [9]; for example, an association between intestinal parasitosis and reduced height in children has been described [10]. Inadequate dietary energy intake may also be a factor in the process of linear growth retardation [11].

This study looked at the relationship between deficiencies of certain nutrients and stunting among Egyptian children.

Methods

The study included 107 stunted children attending the outpatient clinic of the Nutrition Institute in Cairo from September 2000 to January 2001. Stunting was defined as height-for-age of 2 or more standard deviations below the expected mean of the reference standard [12]. Patients with known causes of short stature were excluded (e.g. hormonal, chromosomal, bone disease and coeliac disease). Children of short mothers (maternal height less than 145 cm) [13] were also excluded from the study. The stunted children were 69 males and 38 fe-

males, aged between 10 and 18 years. All children were clinically in good health with no complaint apart from short stature. The control group included 39 children (25 males and 14 females); they were recruited from the relatives of patients attending the outpatient clinic for medical complaints. The control group was chosen to have a similar distribution of age, sex and socioeconomic status as the study children. Those with acute infections or chronic complaints were excluded. None of the children had received multivitamins or mineral preparations before the study.

After taking verbal consent from the parents, the stunted children and the controls were assessed through the following.

- Educational and clinical history. Parents were asked about: school grade and examination marks, previous diseases or operations, history of drug therapy including inhaled or topical preparations, parental heights and family history.

- Medical history and clinical examination. The clinical assessments included: pubertal state; head and neck for signs of vitamin deficiency; teeth for dental caries or tooth decay; heart, chest and abdomen for chronic diseases; skin for café au lait patches, vitiligo, etc.; other clinical signs, such as white nails and pityriasis alba.

- Anthropometric measurements. These were performed according to standard procedures [12]. Height was measured to the nearest 0.1 cm using a stadiometer. ANTHRO software (version 1.01, 1990) was used to calculate Z-scores for height-for-age, weight-for-height and body mass index (BMI) for age [14].

- Laboratory investigations. Complete analysis of blood, urine and stool samples was carried out on the whole sam-

ple. Analysis of serum levels of retinol, α-tocopherol, zinc and magnesium was carried out on a sample of the stunted children and controls. Blood samples were collected from fasting individuals between 09:00 and 10:00. Sampling was done in a room free from direct light. Serum was rapidly separated by centrifugation at 3000 rpm for 10 minutes. Separated serum aliquots were removed and stored frozen at –70 °C until analysis. Serum retinol and α-tocopherol levels were estimated by high pressure liquid chromatography (HPLC) according to the method of Bieri et al. [15]. Serum zinc and magnesium levels were determined by flame atomic absorption spectrophotometry (Unican 929), according to the method of Falchuk and colleagues [16].

* Radiological examination. Assessment of bone age on the left hand and wrist was made on a sample of cases.

The data were analysed using *SPSS* version 5.0.1.

Results

Clinical histories suggestive of rickets during infancy were found in 21.8% of stunted children. None of the control children had a history of rickets. Moreover clinical examination showed that 27.3% had skeletal manifestations of rickets (frontal bossing, rachitic chest or knock knees). Goitre was visible in 32 (19.3%) of the stunted children (7.8% males and 11.5% females) and none of the control children.

A BMI below the 5th percentile for age indicating malnutrition was found in 43 (40.2%) of the stunted children: 18 (47.4%) of the females and 25 (36.2%) of the males. A BMI between the 85th and 95th percentiles indicating risk of obesity was found in 1 female (2.6%) and none of the males. None of the stunted children had a BMI greater than the 95th percentile (indicating obesity). None of the control group was underweight or obese; risk of obesity was found in 2 females and 3 males.

The mean height-for-age among the stunted children was –2.70 ± 0.99 standard deviations from the reference. The majority of stunted children (68.2%) had moderate stunting (height-for-age between –2 and –3 standard deviations from the reference) (Table 1).

The laboratory results showed stunted children had significantly lower mean serum levels of haemoglobin ($P < 0.01$), magnesium ($P < 0.01$), α-tocopherol ($P < 0.001$) and retinol ($P < 0.05$) compared with the controls. Mean serum zinc levels were lower in the stunted children than in the controls but this was not statistically significant (Table 2). None of the control children showed deficiencies in any of the measured micronutrients except for α-tocopherol (25.6% were deficient compared with 78.2% of stunted children).

Although more females than males had nutrient deficiencies, the differences were not significant (Table 3). Correlation studies (Table 4) showed no significant correlation between the severity of stunting and the serum levels of the studied nutrients. However, a highly significant correlation was found between serum zinc and serum retinol and between serum α-tochopherol and serum retinol levels ($P < 0.01$). There was no significant difference in the prevalence of deficiencies of the studied nutrients between males and females.

With the exception of 2 cases, bone age was delayed by an average of 2 years. One case had 5 years' delay in bone age and another case had bone age corresponding to chronological age.

Table 1 **Severity of growth retardation in stunted children**

Degree of stunted growth relative to reference standard	Males		Females		Total	
	No.	%	No.	%	No.	%
< −2 s	45	65.2	28	73.7	73	68.2
< −3 s	21	30.4	7	18.4	28	26.2
< −4 s	2	2.8	2	5.3	4	3.7
< −5 s	1	1.4	1	2.6	2	1.9
Total	69	100.0	38	100.0	107	100.0

$\chi^2 = 2.03$, $P > 0.05$ comparing males and females.
s = standard deviation.

The prevalence of intestinal parasitosis was similar among the stunted and the control group. The most common recovered organisms were *Entamoeba histolytica* followed by *Oxyuris* species (pinworms) and *Giardia* species.

Reports of school performance showed that 10.3% of the stunted children had dropped out of school: most of them had failed at the third primary level. Nearly half the children (46.3%) reported repeating one or more school years, having to take summer examinations to catch up for the school year or passing their examinations with low grades. A further 39.2% of stunted children showed good to average

Table 2 **Comparison of serum levels of haemoglobin and micronutrients in stunted and control children**

Variable	Mean levels ± s	P-value
Haemoglobin (g/dL)		
Stunted (n = 107)	11.66 ± 1.13	
Control (n = 39)	12.30 ± 0.68	< 0.01
Retinol (µg/dL)		
Stunted (n = 77)	43.87 ± 17.45	
Control (n = 39)	52.66 ± 12.01	< 0.05
α-tocopherol (µg/dL)		
Stunted (n = 78)	329.83 ± 155.28	
Control (n = 39)	606.86 ± 125.68	< 0.001
Zinc (µg/dL)		
Stunted (n = 57)	93.46 ± 16.78	
Control (n = 39)	97.38 ± 12.33	> 0.05
Magnesium (mg/dL)		
Stunted (n = 54)	2.09 ± 0.37	
Control (n = 39)	2.39 ± 0.27	< 0.01

s = standard deviation.

Table 3 **Prevalence of deficiency of the micronutrients in stunted children**

Micronutrient	Children with deficient levels[a]		Children with normal levels		P-value
	No.	%	No.	%	
α-tocopherol (µg/dL)					
Males (n = 44)	33	75.0	11	25.0	
Females (n = 34)	28	82.4	6	17.6	
Total (n = 78)	61	78.2	17	21.8	> 0.05
Retinol (µg/dL)					
Males (n = 43)	4	9.3	39	90.7	
Females (n = 34)	5	14.7	29	85.3	
Total (n = 77)	9	11.7	68	88.3	> 0.05
Zinc (µg/dL)					
Males (n = 33)	8	24.2	25	75.8	
Females (n = 24)	5	20.5	19	79.2	
Total (n = 57)	13	22.8	44	77.2	> 0.05
Magnesium (mg/dL)					
Males (n = 31)	4	12.9	27	87.1	
Females (n = 23)	7	30.4	16	69.6	
Total (n = 54)	11	20.4	43	79.6	> 0.05

[a]Deficiency levels defined as follows: retinol < 20 µg/dL, α-tocopherol < 500 µg/dL, zinc < 70 µg/dL [25], magnesium < 1.6 mg/dL [31].

achievement while a minority (4.2%) was very good. All the control group children showed good to average school achievement; none reported repeating a school year.

Discussion

Height reflects the genetic composition of a population as well as its socioeconomic, health and nutritional status [17]. The role

Table 4 **Correlation between the degree of growth retardation and serum micronutrient levels and between different micronutrient levels in stunted children**

Variable	R	P-value
Degree of stunting versus retinol levels	0.0488	> 0.05
Degree of stunting versus α-tocopherol levels	−0.0694	> 0.05
Degree of stunting versus zinc levels	0.1851	> 0.05
Degree of stunting versus magnesium levels	0.2025	> 0.05
Serum retinol levels versus zinc levels	0.4044	< 0.01
Serum retinol levels versus α-tocopherol levels	0.3046	< 0.01

of environment is illustrated by the improvements in average height shown by populations over the last century in both European and non-European countries [18]. In developing countries, poor nutrition plays a major role in the etiology of stunting, although there is no clear consensus as to which nutrients are important. Deficiencies of energy, protein, zinc and iron have all been implicated [6]. Since most stunted children in this study were of normal weight-for-height, the quality of their diet may be a more important factor than the quantity.

In the present study 27.3% of the stunted children had skeletal manifestations of old rickets (frontal bossing, rachitic chest or knock knees), and 21.8% of them had a history highly suggestive of rickets during the first 2 years of life. The influence of vitamin D on bone mineralization is particularly important in children during the development of long bones, as bone mineralization is an integral part of the process of bone development [19]. Overt vitamin D deficiency in children leads to rickets. Sub-clinical vitamin D deficiency may be associated with deficits in bone mineralization in both adults and children but the exact definition of this condition remains controversial [20]. In our study the children who had a history of rickets had probably suffered vitamin D deficiency during their early and late childhood. Stunting was reported during the nineteenth century among children involved in coal mining, probably owing to the suppressive effect of sunlight deprivation on skeletal development [21].

Stunting seemed to have a relationship to school performance. All the control group children showed good to average school achievement; none reported repeating a school year. However, 10% of the stunted children reported failing at school.

All of this group repeated the third primary year but some of them passed the fourth with difficulty and then failed to pass the fifth grade. This may indicate that the age of onset of stunting in those children is early, occurring before the age of maximum brain maturation. Many of the other stunted children showed average to below average school achievement, usually passing their examinations with low grades. The negative effect of factors causing stunting on the brain functions cannot be ignored in these children and it is likely that they may be failing to reach their full potential in brain development. Mendez and Adair demonstrated that moderate to severe stunting in the first 2 years of life is associated with poor performance on cognitive tests in late childhood [3]. Associations between height-for-age and school achievement have been found in older children [17]. Nearly 40% of stunted children showed good to average achievement while a small percentage were very good, which suggests that brain functions can proceed unabated even while somatic growth is affected.

Goitre was visible in nearly one-fifth of stunted children (7.8% males and 11.5% females) compared with none of the controls: figures that are higher than those reported recently among schoolchildren in Cairo (3.2% males and 7.9% females) [1]. The high prevalence of goitre among the stunted children reaffirms the need to continuously monitor the iodine fortification programme that was implemented in Egypt in 1996. Marginal iodine deficiency is associated with short stature and its effects are probably underestimated [22].

Mean retinol (vitamin A) levels were significantly lower in the stunted children; serum retinol below 20 µg/dL was present in 11.7% of stunted children and none of the controls. None of the children had ocu-

lar manifestations of vitamin A deficiency. Abd El Maksoud et al. found that the prevalence of retinol deficiency among stunted children was more than double than in normal children [23].

The most striking result in this study was the higher prevalence of α-tocopherol (vitamin E) deficiency among the stunted children (78.2%) compared with the controls (25.6%). This difference was highly significant. Rosado reported a high proportion of α-tocopherol deficiency (68%) among stunted preschool Mexican children [24]. Vitamin E not only contributes to the stabilization of membrane structure, but also stabilizes active agents such as vitamin A, hormones and enzymes [25]. Without vitamin E, pituitary and adrenal functions may be impaired as these glands may suffer the cumulative effects of oxidation [26]. Although there was no significant difference in mean serum zinc levels between the stunted and the control children, 22.3% of stunted children had levels below 70 μg/dL compared with none of the controls. Our results are similar to those of Rosado [24] who found that 25% of stunted children had low serum zinc. Zinc deficiency presents as growth diminution, possibly as a way of reducing the demand for zinc by the most metabolically active tissues, although a reduction of zinc concentrations in tissues and fluids is not usually seen [27].

In the present study magnesium levels were significantly lower in stunted children than in the controls. Magnesium is a cofactor in nearly all enzyme reactions requiring ATP in the body. It also serves the important functions of protein formation and DNA production. Magnesium deficiency affects the production of the biologically active form of vitamin D and thereby promotes osteoporosis [28].

A highly significant positive correlation was found between zinc and serum retinol levels. Zinc participates in the absorption, mobilization, transport and metabolism of vitamin A, most likely through its involvement in protein synthesis and cellular enzyme functions. There is also evidence that vitamin A affects zinc absorption and utilization. Thus, fluctuation in the status of one or both micronutrients may be expected to alter the metabolism of the other, with functional consequences for the health of the individual [29]. Correlations between serum levels of α-tochopherol and retinol were highly positive. Both these vitamins are fat soluble and α-tochopherol maintains the biological activity of vitamin A [25].

Among the stunted children in this study, there was a delay in bone age by an average of 2 years. Martorell et al. stated that the potential for catch-up growth increases as maturation is delayed and the growth period is prolonged [30]. However, maturational delays in developing countries are less than 2 years, only enough to compensate for a small fraction of the growth retardation in early childhood [30].

The prevalence of intestinal parasitosis was similar among the stunted and the control group, hence a relationship with linear growth retardation could not be demonstrated.

Conclusion

Decreased levels of several micronutrients were associated with linear growth retardation and it is highly likely that several nutrient deficiencies occur simultaneously in stunted children. Deficiencies of nutrients such as vitamin D, α-tocopherol, magnesium and iodine may be underestimated at these ages and there may be many others that should be considered.

References

1. *Food consumption pattern and nutrition intake among different population groups in Egypt. Final report supported by WHO/EMRO.* Cairo, Nutrition Institute, 2000.

2. Martorell R et al. Poverty and stature in children. *Nestlé nutrition workshop series*, 1988, 14:5774.

3. Mendez MA, Adair LS. Severity and timing of stunting in the first two years of life affect performance on cognitive tests in late childhood. *Journal of nutrition*, 1999, 129(8):1555–62.

4. Haas JD et al. Early nutrition and later physical work capacity. *Nutrition review*, 1996, 54(suppl.):41–8.

5. Martorell R et al. Long-term consequences of growth retardation during early childhood. In: Hernandez M, Argente J, eds. *Human growth: basic and clinical aspects.* Amsterdam, Elsevier Science, 1992:143–8

6. Neumann GC, Harrison GG. Onset and evolution of stunting in infants and children. Examples from the Human Nutrition Collaborative Research Support Program. Kenya and Egypt studies. *European journal of clinical nutrition.* 1994, 48(suppl.):90–120.

7. Allen LH. Nutritional influences on linear growth: a general review. *European journal of clinical nutrition*, 1994, 48 (suppl.):75–89.

8. Rivera JA et al. Zinc supplementation improves the growth of stunted Guatemalan infants. *Journal of nutrition*, 1998, 128:556–62.

9. Stephensen CB. Burden of infection on growth failure. *Journal of nutrition*, 1999, 129(suppl.):543–8.

10. Egger RJ et al. Association between intestinal parasitosis and nutritional status in 3–8 year old children. *Tropical geographic medicine*, 1999, 42:312–23.

11. Hautvast LA et al. Food consumption of young stunted and non-stunted children in rural Zambia. *European journal of clinical nutrition*, 1998, 53:50–9.

12. *Measuring changes in nutritional status. Guidelines for assessing the nutritional impact of supplementary feeding programs for vulnerable groups.* Geneva, World Health Organization, 1983.

13. *Second report on the world nutrition situation: a report compiled from information available to the United Nations agencies of the ACC/SCN / prepared in collaboration with the International Food Policy Research Institute (IFPRI), Washington DC.* Geneva, United Nations ACC Sub-Committee on Nutrition, 1992.

14. *Physical status, the use and interpretation of anthropometry.* Geneva, World Health Organization, 1995 (WHO Technical Report Series, No. 854).

15. Bieri JG et al. Simultaneous determination of retinol and á-tocopherol in plasma or red cells by high pressure liquid chromatography. *American journal of clinical nutrition*, 1979, 32:2143–9.

16. Falchuk KH et al. Determination of zinc and selenium in biological samples by atomic absorption spectrophotometry. *Methods of enzymology*, 1988, 158:422–35.

17. Grantham-McGregor SM, Fernald LC, Sethuraman K. Effect of health and nutrition on cognitive and behavioral development in children in the first three years of life. *Food and nutrition bulletin*, 1999, 20(1):76–99.

18. Ulijaszek SJ. Between population variation in pre-adolescent growth. *European journal of clinical nutrition*, 1994, 48 (suppl.):1–14.

19. Davies PS et al. Vitamin D seasonal and regional differences in preschool children in Great Britain. *European journal of clinical nutrition*, 1999, 53:195–8.

20. Jones G et al. Vitamin D level in pre-pubertal children in Southern Tasmania prevalence and determinants. *European journal of clinical nutrition*, 1999, 52: 824–9.

21. Kirby PT. Causes of short stature among coal-mining children, 1823–1850. *Economic history review*, 1995, 48:687–99.

22. Gomo ZA et al. Urinary iodine concentration and thyroid function in adult Zimbabweans during a period of transition in iodine status. *American journal of clinical nutrition*, 1999,70:882–91.

23. Abd El-Maksoud AM et al. Anthropometric measurements and plasma retinol levels of preschool children in Egypt. *Scientific medical journal*, 1997, 9(4):1–10.

24. Rosado LJ. Separate and joint effects of micronutrient deficiencies on linear growth. *Journal of nutrition*, 1999, 192(suppl.):531–3.

25. Grosch WB. Vitamins. In: Belitz HD, Grosch W, eds. *Food chemistry*, 2nd ed. New York, Springer–Verlag, 1999.

26. Meydani M. Vitamin E. *Lancet*, 1995, 345:170–5.

27. Ruz M et al. Single and multiple selenium, zinc, iodine deficiencies affect rat thyroid metabolism and ultrastructure. *Journal of nutrition*, 1999, 129:174–80.

28. Dreosti IE. Magnesium status and health. *Nutrition review*, 1995, 53(9)(suppl.):23–7.

29. Christian P, West K. Interaction between zinc and vitamin A: an update. *American Journal of clinical nutrition*, 1998, 68 (suppl.):435–41.

30. Martorell RL et al. *Reversibility of stunting: epidemiological findings in children from developing countries*. Atlanta, Center of International Health, Emory University School of Public Health, 2000.

31. Wooton ID, Freeman H. *Microanalysis in medical biochemistry*, 6th ed. Edinburgh, Churchill Livingstone, 1982.