European Journal of Clinical Nutrition (2007) 61, 865–869
© 2007 Nature Publishing Group All rights reserved 0954-3007/07 $30.00
www.nature.com/ejcn

ORIGINAL ARTICLE

Low anemia prevalence in school-aged children in Bangalore, South India: possible effect of school health initiatives

S Muthayya[1], P Thankachan[1], MB Zimmermann[2], M Andersson[2], A Eilander[3], D Misquith[4], RF Hurrell[2] and AV Kurpad[1]

[1]*Division of Nutrition, St John's Research Institute, St John's National Academy of Health Sciences, Bangalore, India;* [2]*Human Nutrition Laboratory, Institute of Food Science and Nutrition, Swiss Federal Institute of Technology Zürich, Zürich, Switzerland;* [3]*Unilever Food and Health Research Institute, Vlaardingen, The Netherlands and* [4]*Department of Community Health, St John's Medical College, St John's National Academy of Health Sciences, Bangalore, India*

Objective: Anemia is a serious public health problem in Indian school children. Since 2003, simple health intervention programs such as antihelminthic treatment and vitamin A supplementation have been implemented in primary schools in the Bangalore region, Karnataka, India. This study examines the prevalence of anemia in school children who are beneficiaries of this program.
Design: Cross-sectional survey.
Setting: Bangalore district, South India.
Subjects: A total of 2030 boys and girls, aged 5–15 years, attending schools in the Bangalore district.
Interventions: School-based, twice yearly intervention: deworming (albendazole 400 mg, single oral dose) and vitamin A supplementation (200 000 IU, single oral dose).
Main outcome measures: Anemia prevalence based on measure of blood hemoglobin (Hb).
Results: Mean age and blood Hb concentration of all children were 9.5 ± 2.6 years and 12.6 ± 1.1 g/dl (range 5.6–16.7), respectively. The overall anemia prevalence in this group was 13.6%. Anemia prevalence was lower in boys than girls (12.0%; $n = 1037$ vs 15.3%; $n = 993$ respectively, $P < 0.05$). There was no significant difference in anemia prevalence between children in urban and rural locations (14.6 and 12.3%, respectively).
Conclusions: The current low anemia prevalence in Bangalore could be due to the impact of school-based intervention programs that have been in place since 2003. The beneficial interactions of deworming and vitamin A supplementation could have widespread implications for current preventive public health initiatives. There is now need for the development of clear policy guidelines based on these simple and integrated interventions.
Sponsorship: This research was supported by the Micronutrient Initiative, Ottawa, Canada, and Unilever Food and Health Research Institute, Vlaardingen, The Netherlands.

European Journal of Clinical Nutrition (2007) **61,** 865–869; doi:10.1038/sj.ejcn.1602613; published online 24 January 2007

Keywords: anemia; hemoglobin; albendazole; vitamin A; school children; Bangalore

Introduction

Anemia is a serious public health problem in India. A national survey has reported high anemia prevalence rates of 74% in children below 5 years of age and 52% in young women (NFHS-2, 1998–99). Anemia figures for Karnataka State from the same survey are close to the national average, at 71 and 42% for preschool children and women, respectively. An estimated 50-95% of the anemia in India is iron (Fe) deficiency anemia (Seshadri, 1996).

Correspondence: Dr S Muthayya, Division of Nutrition, St John's Research Institute, St John's National Academy of Health Sciences, Bangalore 560 034, India.
E-mail: sumithra@iphcr.res.in

Guarantor: S Muthayya.

Contributors: SM, PT, MBZ and MA were involved in study design and implementation, data interpretation and writing of the manuscript. DM and AE were involved in study design, implementation and revising of the manuscript. AVK and RFH were involved in study design, data interpretation and writing of the manuscript. The authors have no conflict of interest.

Received 3 July 2006; revised 14 November 2006; accepted 14 November 2006; published online 24 January 2007

There are few data sources on the Fe or anemia status of school going children. It is unclear if children of school age have the same high levels of anemia seen in preschool children. Further, they are a neglected group in terms of micronutrient interventions, not reached by the intervention strategies aimed at preschool children or pregnant women. In the last 3 years, several studies in India have reported a high prevalence of anemia, between 42 and 63% in this age (Gomber *et al*., 2003; Kumar *et al*., 2003; Rao, 2003; Sethi *et al*., 2003). The etiology of anemia is multifactorial and, therefore, there is an urgent need to determine the causes, and to find efficacious methods of intervening to reduce the burden of anemia in children, particularly in young children.

As part of an integrated health program over the last 2–3 years, simple nutrition intervention programs have been implemented in primary schools in the Bangalore urban and rural region in the state of Karnataka, India. These Government programs include antihelminthic treatment and vitamin A supplementation distributed at the school once or twice yearly with the aid of teachers. Previous studies of similar interventions, distributed separately or in combination, have reported varying degrees of efficacy against anemia in different countries (Mejia and Chew, 1988; Gilgen and Mascie-Taylor, 2001; Bhargava *et al*., 2003; Stoltzfus *et al*., 2004; Tanumihardjo *et al*., 2004). In addition, significant relationships have been established between worm load, low vitamin A status and anemia prevalence in children (Wolde-Gebriel *et al*., 1993; Dreyfuss *et al*., 2000). Although there are no previous data available on anemia in school children in Bangalore city, the implementation of these interventions offers an opportunity to examine the anemia prevalence in school children in this area, and to compare it to prevalence reported from another city in Karnataka where such data were available without these interventions.

This paper describes the prevalence and severity of anemia in school-going children in the study areas in both urban (but low socioeconomic) and rural Bangalore region of Karnataka, South India. The data are compared to previously reported urban anemia prevalence data from the same state in school going children.

Subjects and methods

During the baseline assessment of several recent nutritional intervention studies carried out from St John's Research Institute, Bangalore, hemoglobin (Hb) was measured in 2030 children, aged 5–15 years. The study sites were three urban Government-aided schools, namely, Franciscan Primary School ($n = 529$; 5–11 years), Maria Niketan Primary School ($n = 386$; 7–10 years) and St. Charles Primary School ($n = 229$; 7–10 years) and six rural Government schools, Kugur Primary School ($n = 152$; 5–15 years), Kugur High School ($n = 284$; 12–15 years), Thindlu Primary School ($n = 172$; 5–15 years), Mugalur Primary School ($n = 163$; 5–14 years), Kuthaganahalli Primary School ($n = 72$; 5–12 years) and Doddathimmasandra Primary School ($n = 43$; 5–10 years). Children attending the urban schools lived in poor neighborhoods, with high population density and poor sanitation and limited access to water. Rural school children lived in villages at least 40 km from Bangalore, with no sanitation or access to running water in their homes. Informed, written consent was obtained from the parents of the children and oral consent was obtained from the children. The protocol of the study was reviewed and approved by the ethical committee at St John's National Academy of Health Sciences, Bangalore.

In the studies that were conducted in these schools, children in grades between 1 and 10 were screened, although this was not uniform across schools because of organizational differences in sites, for example, children in urban schools were in grades 2–5 and children in rural schools were in 1–10. All schools in this study reported that they had implemented deworming (Albendazole 400 mg, single oral dose) and vitamin A supplementation (200 000 IU, single oral dose, with the exception of one school) at least before 12 months of the baseline screening. Data were collected between August 2004 and January 2006.

Height and weight were measured and transformed into *z*-scores of weight-for-age (WAZ) and height-for-age (HAZ). Five ml of blood were collected by venipuncture into EDTA-containing vacutainers and transported on cold packs to the laboratory at St John's. Hb was measured on an automated Coulter AcT Diff^2 hematology analyzer (Beckman Coulter, Krefeld, Germany) using three level controls (Liquicheck, Bio-Rad Laboratories, Irvine, CA, USA). Whole blood was analyzed within 12 hours of blood sampling and anemia was defined as Hb <11.5 g/dl in children aged 5–11 years and <12.0 g/dl in children aged $\geqslant 12$ years (WHO, 2001). Anemia was classified by severity as follows: (*i*) Mild – 10.0 –11.5/12.0 g/dl; (*ii*) Moderate – 8.0–9.9 g/dl; and (*iii*) Severe - <8.0 g/dl.

Statistical analyses were performed with the SPSS program (version 13.0, SPSS, Chicago, IL,USA). Epinfo was used for anthropometry calculations (Epinfo version 3.3.2, CDC, Atlanta). A χ^2 test was carried out to compare anemia prevalence between gender and geographical location (urban/rural). Two-sided *P*-values <0.05 were considered statistically significant.

Results

Data are reported for 2030 children between the ages 5 and 15 years. Mean age and blood Hb concentration of all children were 9.5 ± 2.6 years and 12.6 ± 1.1 g/dl (range 5.6–16.7), respectively. Nearly 50% of the children were underweight (<-2 WAZ), and 25% were stunted (<-2 HAZ).

The overall anemia prevalence in this group was 13.6% (Table 1). In children who had anemia ($n = 285$), 11.2%,

Table 1 Summary of anemia prevalence in all children (urban and rural)

Group	*All children*			*Boys*			*Girls*		
	Total number	*Number anemic*	*Prevalence (%)*	*Total number*	*Number anemic*	*Prevalence (%)*	*Total number*	*Number anemic*	*Prevalence (%)*
All children	2030	276	13.6	1037	124	**12.0**[a]	993	152	**15.3**
Urban children	1144	167	**14.6**	581	79	**13.6**[b]	563	88	**15.6**
Rural children	886	109	**12.3**	456	45	**9.9**	430	64	**14.9**

[a] Significantly different from girls using a χ^2test ($P<0.05$).
[b] Significantly different from rural boys using a χ^2test ($P<0.05$).

2.1% and 0.3% had mild, moderate and severe anemia, respectively. Boys ($n=1037$) had a significantly lower anemia prevalence, 12.0% compared to girls, 15.3% ($n=993$, $P<0.05$). The distribution of anemia prevalence in all children across ages is presented in Figure 1. There is a sharp decline in anemia prevalence from nearly 50% at 5 years of age to close to 10% between 7 and 11 years. This trend was seen in both genders. Thereafter, anemia prevalence increased in both boys and girls.

The agewise distribution of anemia prevalence separately in urban and rural children, categorized by gender, is presented in Figure 1. There was no significant difference in anemia prevalence between children in urban and rural locations (14.6 and 12.3, % respectively). However, there was a significant difference in anemia prevalence between urban and rural boys (Table 1). The prevalence of anemia was comparable between genders in urban children; however, rural girls had a significantly higher anemia prevalence than rural boys ($P<0.05$). The reversal trend from a low to high prevalence for anemia began earlier (at about age 10 years) in urban children when compared to rural children (at about age 12 years). However, the lack of data in urban children above 11 years weakens this observation.

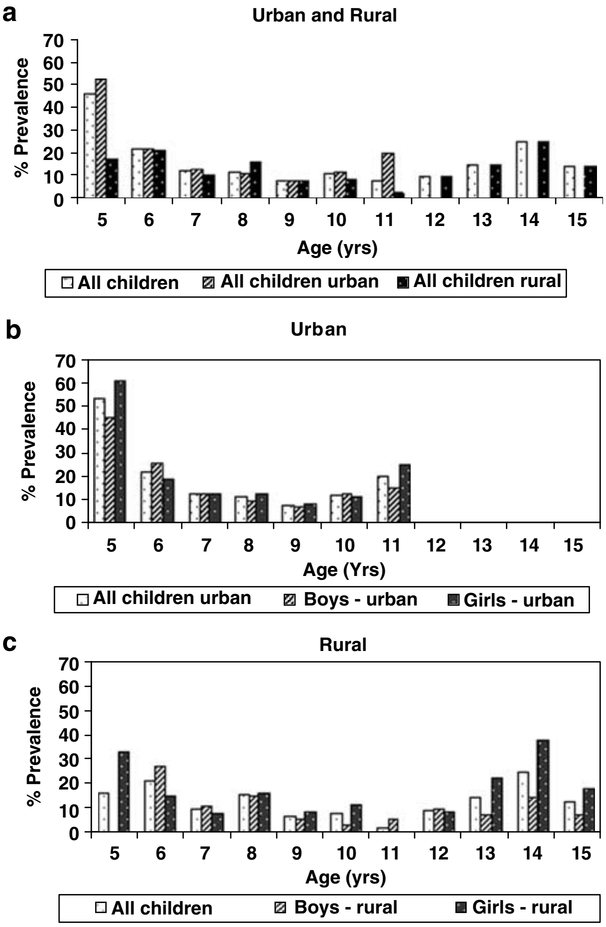

Figure 1 Percentage anemia prevalence stratified by location, age and gender in school age children. Panels **a**, **b**, **c** represent all children (urban and rural, $n=2030$), urban children ($n=1144$) and rural children ($n=886$) respectively. No data were available for the following age/gender/location groups: (**a**), ages 12–15 (urban children); (**b**), ages 12–15 (both genders); and (**c**), age 5 (boys) and age 11 (girls).

Discussion

Our findings indicate that the mean prevalence of anemia is only 13.9% in school-age children in the Bangalore region, Karnataka, South India. It is also worth noting that the prevalence was consistently low between the ages of 7 and 11 years in all sites studied. These data contrast sharply with reports of 3–4 times higher anemia prevalence in school age children from other parts of India (Gomber *et al.*, 2003; Kumar *et al.*, 2003; Sethi *et al.*, 2003). For example, a survey in the city of Gulbarga in northern Karnataka reported an anemia prevalence of 61% in this age group among girls (Kumar *et al.*, 2003). This study was conducted before state government health interventions such as antihelminthic treatment and vitamin A supplementation, targeting school children (personal communication from authors of Kumar *et al.* (2003).

Therefore, a possible explanation for the current low anemia prevalence in Bangalore could be the impact of these integrated child health programs that have been in place since 2003 (Akshara Dasoha, 2003). Specifically, these include the regular oral administration of albendazole (400 mg) and vitamin A of 200 000 IU twice yearly, as well as a free daily lunch. Indeed, the Akshara Dasoha scheme as proposed (2003) was more comprehensive, as it also envisioned the supplementation of Fe and folate. However, these were not implemented in the schools in this study, probably because of a lack of supply.

Both the antihelminth treatment and vitamin A intervention can improve Fe availability for erythropoesis in Fe-deficient children (see below). It is likely that most childhood anemia in India is owing to Fe deficiency (Seshadri, 1996). This is supported by data from a subgroup of anemic children in the present study ($n = 54$), in whom, Fe deficiency (defined by a low serum ferritin and elevated transferrin receptor concentration) accounted for about 93% of the anemia prevalence (S Muthayya, unpublished data). Indian children residing in poor neighborhoods have been shown to have a high prevalence of helminth infections (Sur *et al.*, 2005). The intensity of helminthic infection and faecal egg count are strongly and inversely associated with Hb concentrations (Stoltzfus *et al.*, 1997; Dreyfuss *et al.*, 2000). This might partly explain, why anemia prevalence is much lower than expected in this study group following the deworming intervention. Vitamin A was also administered to the study children and may have had an additional positive impact on their Hb and Fe status. Vitamin A status has been shown to be poor in school-aged children in other Indian studies (Dwivedi *et al.*, 1992; Kapil *et al.*, 1996; Khandait *et al.*, 1999). Nutritional surveys have shown a close association between vitamin A deficiency and anemia (Mejia and Chew, 1988; Bloem *et al.*, 1989). Vitamin A repletion may reduce anemia by improving utilization of stored Fe for erythropoiesis (Zimmermann *et al.*, 2006), and by enhancing immunity and reducing the anemia of infection (Semba and Bloem, 2002). Intervention studies in preschool and school-going children also confirm that vitamin A supplementation improves Hb concentration and other measures of Fe status (Mohanram *et al.*, 1977; Mejia and Chew, 1988; Mwanri *et al.*, 2000).

The beneficial interactions of deworming and vitamin A supplementation could have widespread implications for current preventive public health interventions. In combination with these interventions, the consumption of a simple, rice-based, lunch supplying roughly 300–400 kcal/day may not only improve the overall nutritional status of school-age children, but also contribute a small amount of additional Fe each day. Although the amount of Fe in the rice-based lunch is low, the Fe may be well-absorbed, given the relatively low amount of inhibitors such as phytates in rice.

A detailed search of the literature on anemia prevalence in India does not provide any evidence for a secular trend such as a gradual reduction in anemia prevalence in the last 20 years (Seshadri, 1996; Gomber *et al.*, 2003; Kumar *et al.*, 2003; Rao, 2003; Sethi *et al.*, 2003). Hence, the low prevalence of anemia observed in the present study could be attributed to the combined school based interventions that have been operational in the study area in the last 3 years. A limitation of the present report is the lack of baseline anemia prevalence data before the implementation of these interventions, as well as lack of data on helminthic infections, Fe deficiency and vitamin A deficiency in school children in the study areas. Therefore, the implications of this cross-sectional study need to be verified by careful longitudinal studies where pre-intervention baseline values are available on vitamin A status and parasite prevalence. However, we believe this report is valuable because firstly, we have used data from a similar location in the same state for comparison (Gulbarga city, Kumar *et al.*, 2003) and, secondly, the present prevalence of anemia is well below what might be expected from a secular trend. Finally, some of the children in the present study could effectively represent the same cohort of preschool children studied in the NFHS assessment in Bangalore in 1998–1999, in whom the anemia prevalence was reported to be about 70% (NFHS-2, 1998–99). Importantly, from a public health viewpoint, these interventions did not include Fe supplements and are indicative of the impact that simple integrated health initiatives can have on reducing the burden of anemia in the school child in the community. Additionally, as these combined interventions are cost effective and affordable, and as they are school-based programs, it is most likely that they will ensure a greater level of compliance, and, therefore, sustainability. The point has to be made, however, that this report only assesses the prevalence of anemia, and not Fe deficiency, which could still be more widely prevalent and, therefore, interventions such as Fe fortification are needed to overcome this problem.

The high anemia prevalence (43%) in the youngest children (5 years) just entering school, compared to that of their older peers is noteworthy (Figure 1). This may indicate the low coverage of nutrition interventions aimed at vulnerable preschool children. For example, surveys in Karnataka state report only 56% coverage of vitamin A supplementation to preschool children through the routine immunization program (UNICEF, 2001).

The data in this report add to the already existing, and convincing, studies available on the efficacy of school based distribution of antihelminthic treatment and vitamin A supplementation in reducing anemia prevalence in school children. There is now need for the development of clear policy guidelines for the wider implementation and evaluation of these simple and integrated interventions. On the basis of these findings, we would also recommend further assessment of the prevalence of anemia and its potential causes, including helminth infections, Fe deficiency and vitamin A deficiency.

Acknowledgements

The assistance of Dr R Goud, Dr D Moretti, Ms A Vani and Ms L Sebastian is gratefully acknowledged. We thank Mr T M Vijay Bhaskar, Secretary, Department of Education, Government of Karnataka, the Departments of Health and Women and Child Development, Government of Karnataka, for their support and cooperation.

References

Akshara Dasoha (2003). To the children of all Government Primary School in the State, Government Order No. ED 24 MMS 2003. In: office of the Joint Director of Public Instruction (Midday Meal Scheme). Government of Karnataka, India, Karnataka.

Bhargava A, Jukes M, Lambo J, Kihamia CM, Lorri W, Nokes C *et al.* (2003). Anthelmintic treatment improves the hemoglobin and serum ferritin concentrations of Tanzanian schoolchildren. *Food Nutr Bull* **24**, 332–342.

Bloem MW, Wedel M, Egger RJ, Speek AJ, Schrijver J, Saowakontha S *et al.* (1989). Iron metabolism and vitamin A deficiency in children in northeast Thailand. *Am J Clin Nutr* **50**, 332–338.

Dreyfuss ML, Stoltzfus RJ, Shrestha JB, Pradhan EK, LeClerq SC, Khatry SK *et al.* (2000). Hookworms, malaria and vitamin A deficiency contribute to anemia and iron deficiency among pregnant women in the plains of Nepal. *J Nutrit* **130**, 2527–2536.

Dwivedi SN, Banerjee N, Yadav OP (1992). Malnutrition among children in an urban Indian slum and its associations. *Indian J Matern Child Health* **3**, 79–81.

Gilgen D, Mascie-Taylor CG (2001). The effect of anthelminthic treatment on helminth infection and anaemia. *Parasitology* **122** (Pt 1), 105–110.

Gomber S, Bhawna, Madan N, Lal A, Kela K (2003). Prevalence & etiology of nutritional anaemia among school children of urban slums. *Indian J Med Res* **118**, 167–171.

Kapil U, Saxena N, Srivastava M, Jailkhani L, Nayyar B, Chitkara P (1996). Assessment of vitamin A deficiency indicators in urban slum communities of National Capital Territory of Delhi. *Asia Pacific J Clin Nutr* **5**, 170–172.

Khandait DW, Vasudeo ND, Zodpey SP, Ambadekar NN, Koram MR (1999). Vitamin A intake and xerophthalmia among Indian children. *Public Health* **113**, 69–72.

Kumar CS, Kumar AH, Sunita V, Kapur I (2003). Prevalence of anemia and worm infestation in school going girls at Gulbarga, Karnataka. *Indian Pediatr* **40**, 70–72.

Mejia LA, Chew F (1988). Hematological effect of supplementing anemic children with vitamin A alone and in combination with iron. *Am J Clin Nutr* **48**, 595–600.

Mohanram M, Kulkarni KA, Reddy V (1977). Hematological studies in vitamin A deficient children. *Int J Vitam Nutr Res* **47**, 389–393.

Mwanri L, Worsley A, Ryan P, Masika J (2000). Supplemental vitamin A improves anemia and growth in anemic school children in Tanzania. *J Nutr* **130**, 2691–2696.

NFHS-2 (1998-99). National Family Health Survey II. In: IIPS, Mumbai, India.

Rao B (2003). Anaemia and micronutrient deficiency. *The National Med J India* **16**, 46–50.

Semba RD, Bloem MW (2002). The anemia of vitamin A deficiency: epidemiology and pathogenesis. *Eur J Clin Nutr* **56**, 271–281.

Seshadri S (1996). A data base on iron deficiency anaemia (IDA) in India: Prevalence, aetiology, consequences and strategies for control. Prepared for Task Force on Micronutrient Malnutrition Control. In: Department of Women and Child Development, Ministry of Human Resources Development., New Delhi.

Sethi V, Goindi G, Kapil U (2003). Prevalence of anemia amongst primary school age children (6–11 years) in National Capital Territory of Delhi. *Indian J Pediatr* **70**, 519–520.

Stoltzfus RJ, Chway HM, Montresor A, Tielsch JM, Jape JK, Albonico M *et al.* (2004). Low dose daily iron supplementation improves iron status and appetite but not anemia, whereas quarterly anthelminthic treatment improves growth, appetite and anemia in Zanzibari preschool children. *J Nutr* **134**, 348–356.

Stoltzfus RJ, Dreyfuss ML, Chwaya HM, Albonico M (1997). Hookworm control as a strategy to prevent iron deficiency. *Nutr Rev* **55**, 223–232.

Sur D, Saha DR, Manna B, Rajendran K, Bhattacharya SK (2005). Periodic deworming with albendazole and its impact on growth status and diarrhoeal incidence among children in an urban slum of India. *Trans R Soc Trop Med Hyg* **99**, 261–267.

Tanumihardjo SA, Permaesih D, Muhilal (2004). Vitamin A status and hemoglobin concentrations are improved in Indonesian children with vitamin A and deworming interventions. *Eur J Clin Nutr* **58**, 1223–1230.

UNICEF (2001). National Report-Multiple Indicator Cluster Survey-2. In: Department of Women and Child Development, Government of India and United Nations Children's Fund (UNICEF), New Delhi.

WHO (2001). *Iron deficiency anemia: Assesment prevention and control-A guide for program manager* In: NHD01.3 ed, WHO: Geneva.

Wolde-Gebriel Z, West CE, Gebru H, Tadesse AS, Fisseha T, Gabre P *et al.* (1993). Interrelationship between vitamin A, iodine and iron status in schoolchildren in Shoa Region, central Ethiopia. *Br J Nutr* **70**, 593–607.

Zimmermann M, Biebinger R, Rohner R, Dib A, Zeder C, Hurrell RF *et al.* (2006). Vitamin A supplementation in children with poor vitamin A and iron status increases erythropoietin and hemoglobin concentrations without changing total body iron. *Am J Clin Nutr* **84**, 580–586.