# Iron status among obese Egyptian adolescents

Hayam K. Nazifa, Azza A. El Shaheedb, Karima A.I. El-Shamyc, Manal A. Mohsenb, Nevein N. Fadlc, Rehab S.I. Moustafab

<sup>a</sup>Department of Medical Studies, Institute of Postgraduate Childhood Studies, <sup>b</sup>Department of Child Health, <sup>c</sup>Department of Medical Physiology, Medical Division, National Research Centre, Cairo, Egypt

Correspondence to Manal A. Mohsen, PhD, Department of Child Health, Medical Division, National Research Centre, El-Bohoth St., Dokki, PO Box 12311, Cairo, Egypt Tel: +20 100 410 0332; e-mail: manalmohsen@hotmail.co.uk

Received 26 January 2015 Accepted 20 September 2015

Journal of the Arab Society for Medical Research 2015, 10:76–81

#### Background/aim

Obesity and iron deficiency are the most common nutritional disorders in the world. The aim of this work was to assess the iron status, including serum total iron binding capacity (TIBC), transferrin saturation, serum ferritin and soluble serum transferrin receptor (sTfR), among obese Egyptian adolescents.

#### Patients and methods

A cross-sectional case—control study was conducted on 80 adolescents aged 12–14 years who attended the Nutritional Clinic of the National Research Centre. They were divided into two equal groups of 40 adolescents each: the obese group and the nonobese group. Thorough clinical examination including anthropometric measurements was performed. Haemoglobin level was assessed using the cyanmethaemoglobin method. Serum iron, TIBC, transferrin saturation, serum ferritin and sTfR were analysed using ELISA.

#### Results

Obese adolescents revealed significantly lower levels of haemoglobin, serum iron, ferritin and transferrin saturation, and significantly higher diastolic blood pressure and higher TIBC and sTfR, compared with the nonobese group. Obese adolescents had higher frequency of iron deficiency anaemia compared with nonobese adolescents (80 vs. 17.5%).

#### Conclusion

Weight loss programmes for obese adolescents are essential for correction of iron status. Iron fortification for obese adolescents is recommended.

#### Keywords:

children, iron deficiency, obesity

J Arab Soc Med Res 10:76–81 © 2015 The Arab Society for Medical Research 1687-4293

# Introduction

Anaemia is one of the most important nutritional deficiencies affecting various social and socioeconomic strata. It is more common in developing countries, with children and adolescents being at a significantly higher risk for the condition. Recent studies have revealed a prevalence of iron deficiency anaemia (IDA) of around 20% in adolescents [1].

Nutrition during childhood has a significant impact on lifelong health. Obesity and iron deficiency (ID) are two of the most common nutritional disorders worldwide [2]. The prevalence of childhood overweight and obesity is increasing, with the worldwide prevalence having doubled or tripled in industrialized countries over the past few decades [3]. ID continues to be the most prevalent single micronutrient deficiency-related disease in the world, affecting billions of people [4]. Data from NHANES III were examined for an association between ID and weight [5]. The prevalence of ID increased as BMI increased from normal weight to more than the 85th percentile for age and sex to more than the 95th percentile for age and sex (2.1, 5.3 and 5.5%, respectively). Obesity was a risk factor for IDA in both boys and girls, but rates were approximately three times higher in girls [5]. However, whether being overweight or obese is associated with increased risk for anaemia or IDA is still being debated [6].

Adolescence is a time of increased iron needs because of the expansion of blood volume and increases in muscle mass. The incidence of ID among adolescents appears to be rising [7]. The aetiology of anaemia in obese individuals is uncertain but may be related to low-quality diets or increased needs relative to body weight [5,8]. A number of factors have been typically proposed to explain the association between ID and obesity. Eating unbalanced meals – for example, low-cost fast food that is particularly rich in carbohydrates and fat but contains a low quantity of essential nutrients such as iron - has been claimed as the most important cause for this association [7]. Radically changing this sound point of view, a recent study investigating the intake of heme and nonheme iron in a convenience sample of more than 200 obese patients showed that

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

differences in iron intake, or of dietary factors known to affect iron absorption, were not associated with the lower serum iron concentrations found in obese patients. Additional studies investigating other factors that may be associated with the hypoferremia of obesity are warranted [9].

The present work aimed to determine the iron status, including determination of serum levels of total iron binding capacity (TIBC), transferrin saturation, serum ferritin and soluble serum transferrin receptor (sTfR), in obese Egyptian adolescents and explore the link between iron status and obesity, as both have significant public health implications.

### Patients and methods

A cross-sectional case-control study was conducted on 80 adolescents aged 12-14 years. They were divided into two equal groups of 40 each. Forty obese adolescents (21 girls and 19 boys), selected randomly from obese cases who attended the nutritional clinic at the National Research Centre, constituted the first group. The second group comprised sex-matched and age-matched lean adolescents who consulted the Paediatric Clinic in the National Research Centre for minor complaints. They were considered as the control group. None of the adolescents in the study population were taking any medication or had clinical evidence of endocrine or metabolic disease. Adolescents with chronic conditions or with organic causes of obesity, who were on iron supplements, or were taking medications that could affect body weight, and girls with menorrhagia were excluded from this study. Their socioeconomic level was above average. This study was conducted over a period of 12 months starting from January 2011 to December 2011.

Written informed consent was obtained after explanation of the aim of the study. The study was approved by the Medical Ethics Committee of the National Research Centre.

Children were subjected to thorough clinical examination, including anthropometric measurements. Height was measured to the nearest 0.1 cm using a Tanita Stadiometer (USA), and weight was determined to the nearest 0.01 kg using a Tanita Digital Balancer, with the patient dressed in minimum clothes and wearing no shoes. BMI was calculated as weight (in kg) divided by height squared (in m) (kg/m²). BMI exceeding the 95th percentile was used for obesity diagnosis. Control adolescents had BMI below the 85th percentile, based on the Egyptian Growth Charts [10]. The waist and hip circumferences were measured, and the waist/hip ratio was calculated. The landmarks, instruments and techniques used followed the WHO standardized anthropometric methods [11].

After resting for 5 min, blood pressure (BP) was measured in the sitting position using an appropriately sized cuff on both arms [12]. The systolic blood pressure (SBP) and diastolic blood pressure (DBP) used in the analysis were derived from the mean BP measured on both arms.

Blood samples were obtained aseptically using vein puncture. A volume of 5 ml venous blood was drawn from each adolescent after an overnight fast (8 h) and was divided into two parts. The first part was whole blood collected in plastic tubes with, which served as anticoagulant for haemoglobin assay. Samples for haemoglobin evaluation were subjected to immediate assay. The second part was collected in plain, clean plastic tubes without additives for serum iron, TIBC, serum ferritin and sTfR assessment. It was kept at room temperature for 2 h, allowed to clot, and then centrifuged; the serum was separated and kept in plastic Eppendorf tubes and frozen at -20°C until the time of assay.

Haemoglobin level was assessed cyanmethaemoglobin method for the in-vitro determination of haemoglobin in whole blood with heparin or ethylene-diamine-tetra-acetic acid. It was applied according to the International Committee for Standardization in Haematology [13]. Serum iron and TIBC were assessed using the quantitative colorimetric determination of iron, and the unsaturated iron binding capacity in serum was assessed using Stanbio Laboratory Kits (Texas, USA) according to Burtis and Edward [14]. The reference value for serum iron for boys in this age group is 60–120 µg/dl and that for girls is 50–120  $\mu g/dl,$  whereas the reference value for TIBC for this age group is 250-400 µg/dl according to Behrman et al. [15]. Circulating ferritin concentration in serum was quantitatively determined by means of a microplate immunoenzymometric assay (ELISA) using Monobind Inc. kits (Lake Forest, CA, USA), as done by Burtis [16]. The reference value for this age group of children is less than 10 ng/ml, as per Fairbanks and Klee [17]. sTfR is an indicator of iron status that is unaffected by the acute-phase response [18]. It was assessed using the quantitative measurement of human sTfR in serum using ELISA kits of BioVendor Research and Diagnostic products. The reference value for this age group of children is 0.9–3.3 µg/dl [18,19].

Iron status has been assessed by measuring serum iron [14], transferrin and ferritin concentrations [16]. Transferrin saturation was calculated by finding the molar ratio of serum iron and twice the serum transferrin (because each transferrin molecule can bind two atoms of iron) using the following formula: transferrin saturation = [serum iron ( $\mu$ g/dl)/transferrin (mg/dl)] ×71.2 [20]. Anaemia was considered according to WHO standards as haemoglobin less than 11.5 g/dl and severe anaemia as less than 7.0 g/dl [21].

IDA was defined as concurrent ID and anaemia. The laboratory criteria used in this work to diagnose ID were as follows: Serum iron < 60  $\mu$ g/dl in boys and < 50  $\mu$ g/dl in girls were assigned as ID [15]. Serum ferritin <10 ng/ml [17] and serum transferrin saturation less than or equal to 15 (Transferrin Saturation (TS) = serum iron/TIBC ×100) [22] were also diagnosed as ID. ID was defined as the presence of at least two abnormal age-corrected iron parameters.

Data analysis was performed using SPSS (version 16; SPSS Inc., Chicago, Illinois, USA). Simple statistics and bivariate analysis were used. For comparing two means, Student's t-test of significance was utilized. The  $\chi^2$ -test of significance was used to compare the frequency between two categorical variables. P-values less than 0.05 were considered statistically significant.

### Results

The results of obese adolescents showed highly significant increases (P<0.01) in weight, BMI, waist circumference (WC), hip circumference and waist to hip ratio when compared with the control group. DBP was significantly higher (P<0.01) in obese adolescents than in the control group, whereas nonsignificant changes were reported in SBP between the two groups, as shown in Table 1.

The present results showed that symptoms and signs of anaemia were prominent in obese adolescents: there were increases in the frequency of tachypnea, easy fatigability and lack of concentration in obese adolescents compared with the control group, as well as increases in the frequency of rate of pallor and nail changes (Table 2).

The present results found that the studied obese adolescents had highly significantly reduced (P < 0.01) haemoglobin content, serum iron, ferritin and transferrin saturation levels, and highly significant increases in TIBC and sTfR levels, compared with the control group (Table 3).

Results also showed that the frequency of IDA (serum iron for boys <60  $\mu$ g/dl, for girls <50  $\mu$ g/dl, transferrin saturation  $\leq$ 15% and haemoglobin concentration

Table 1 Anthropometric and blood pressure measurements of the obese Egyptian adolescents under study

Variable	Obese adolescents	Control	P
	(n = 40)	(n = 40)	value
Weight (kg)	81.3 ± 5.4	54.3 ± 7.0	0.001**
Height (cm)	$154.1 \pm 9.3$	$153.1 \pm 3.8$	0.503
BMI (kg/m²)	$33.0 \pm 3.1$	$21.3 \pm 1.0$	0.001**
Waist circumference (cm)	$99.4 \pm 10.6$	58.9 ± 1.5	0.001**
Hip circumference (cm)	$60.1 \pm 5.8$	44.7 ± 1.2	0.001**
Waist to hip ratio (cm)	$1.7 \pm 0.2$	$1.3 \pm 0.0$	0.001**
Systolic BP (mmHg)	$108.1 \pm 9.0$	$104.5 \pm 8.5$	0.068
Diastolic BP (mmHg)	$68.9 \pm 5.6$	65.8 ± 4.9	0.009

All data are expressed as mean  $\pm$  SD; BP, blood pressure; \*\*P < 0.01, highly significant.

Table 2 Symptoms and signs of anaemia in the obese Egyptian adolescents under study

Symptoms of	Obese adolescents	Control	$\chi^2$	P value
anaemia	(n = 40)	(n = 40)		
Palpitation				
Yes	4 (10)	3 (7.5)	3.74	0.1
No	36 (90)	37 (92.5)		
Tachypnea				
Yes	23 (57.5)	0 (0)	32.2	<0.0001**
No	17 (42.5)	40 (100)		
Easy fatigabili	ty			
Yes	24 (60)	12 (30)	10.91	0.001**
No	16 (40)	28 (70)		
Lack of conce	entration			
Yes	25 (62.5)	10 (25)	10.80	0.001**
No	15 (37.5)	30 (75)		
Signs of anae	emia			
Pallor				
Yes	21 (52.5)	6 (15)	18.15	<0.0001**
No	19 (47.5)	34 (85)		
Glossitis or	stomatitis			
Yes	0 (0)	0 (0)	_	_
No	40 (100)	40 (100)		
Nail change	S			
Yes	21 (52.5)	7 (17.5)	16.34	0.0001**
No	19 (47.5)	33 (82.5)		
Hyperdynam	nic circulation			
Yes	0 (0)	0 (0)	_	_
No	40 (100)	40 (100)		

All data are expressed as n (%); \*\*P < 0.01, highly significant.

Table 3 Haemoglobin and iron status results of the obese Egyptian adolescents under study

Variable	Obese adolescents (n = 40)	Control $(n = 40)$	P value
Hb (g/dl)	10.1 ± 1.3	12.4 ± 1.3	0.001**
Iron (mg/dl)	$48.5 \pm 10.8$	69.8 ± 11.8	0.001**
TIBC (mg/dl)	$343.4 \pm 52.0$	$292.6 \pm 37.1$	0.001**
Transferrin saturation (%)	$14.8 \pm 5.7$	$24.5 \pm 5.7$	0.001**
Ferritin (ng/ml)	$21.7 \pm 1.4$	$23.4 \pm 1.7$	0.001**
sTfR (mg/dl)	$5.6 \pm 2.2$	$2.7 \pm 1.9$	0.001**

All data are expressed as mean  $\pm$  SD; Hb, haemoglobin; sTfR, soluble serum transferrin receptor; TIBC, total iron binding capacity; \*\*P < 0.01, highly significant.

<11.5 g/dl) was significantly greater (P < 0.01) in obese adolescents than in the control group, as shown in Fig. 1.

### Discussion

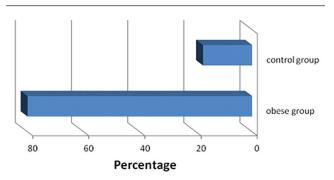
IDA is the main micronutrient deficiency that may affect adolescents. It is one of the most universally prevalent diseases in the world today particularly in developing countries, and it is a major public health problem. ID has been considered an important risk factor for ill health and is estimated to affect two billion people worldwide [23]. The consequences of IDA include diminished cognitive function and exercise performance [24].

ID is the most common nutritional deficiency in the USA and has been linked to obesity in adults and children from both industrialized and transition countries [25]. Speculation of the aetiology of obesity-associated ID has included inadequate dietary iron intake, increased iron requirements due to increased body size and blood volume, and menstrual irregularities [26].

ID and abnormal BMI are two nutritional disorders worldwide, particularly in developing countries [27]. During adolescence, 20% of final adult height and 50% of adult weight are achieved. Because of this rapid growth, adolescents are especially vulnerable to anaemia [28]. The maximum physical, psychological and behavioural changes take place during these years of life, and it has been found that less than 2% of teenagers eat sufficient essential foods and nearly 20% of girls and 7% of boys do not consume adequate amounts of even one of the food groups [28]. Frequently dieting or constrained eating, missing meals, vegetarian eating styles, high carbohydrate meals, and fast foods are all risk factors for anaemia in adolescents. Despite bigger iron needs, many adolescents, especially girls, do not take enough iron from their diets. Seventy five percent of teenage girls do not consume an adequate diet, especially those containing iron, and do not meet their dietary requirements for compensation of iron loss from menstruation, compared with only 17% of teenage boys. Therefore, teenagers are prone to ID and IDA. ID during adolescence causes mental and cognition disturbances, fatigue and lower work capacity [29,30]. It is expected that about 11% of female adolescents are iron deficient in the USA [31].

In this study, confirming the link between adiposity and serum iron levels, obesity was associated with poor iron status. Recent studies have ruled out

Figure 1



Comparison between the obese adolescents group and the control group as regards the frequency of iron deficiency anaemia.

inadequate dietary iron intake as the source of this problem [9]. Latest research has focused on the impact of obesity-associated low-grade inflammation on systemic iron metabolism [32]. Results of the present study showed a highly statistically significant increased weight, BMI, WC, hip circumference and waist to hip ratio in obese adolescents when compared with the control group. This is in agreement with Salem et al. [33], who reported a highly significant difference between obese and control children as regards BMI, WC, hip circumference and waist to hip ratio in both boys and girls.

Among the factors that can interfere with BP, the literature shows that White children have a greater chance of having higher BP compared with non-White children, as they tend to have a higher fat percentage. Excess fat plays a role in the blood hypertension pathogenesis, either directly associated with the metabolic characteristics of adipocytes located mainly in the abdominal region or indirectly by means of hyperinsulinaemia resulting from the insulin resistance and consequent disorder in glucose metabolism [34].

The variation in body fat distribution is an important morphological indicator related to endocrine and metabolic complications, which predispose to the development of cardiovascular diseases. Individuals with centripetal disposition of body fat tend to have a higher incidence of blood hypertension. Increased WC has been related to higher BP levels. A cross-sectional study with obese and eutrophic children aged 7-12 years found that individuals with increased WC were 2.8 times more likely to have high BP compared with those with adequate WC [35].

In this study, the prevalence of hypertension increases progressively with increasing BMI, and studies have detected hypertension in over 30% of obese children [36].

Blood hypertension, although treatable and easily measurable and assessable clinically, is a silent disease, whose degenerative and cumulative effect is greater among younger individuals because of their longer exposure. Excess weight causes abnormalities in BP and therefore predisposes to cardiovascular diseases. Many of the abnormalities that occur in this age group are reversible if there is early detection and intervention to combat the risk factors for cardiovascular diseases, as the process of atherosclerosis and left ventricular hypertrophy may be reversed or minimized by reducing weight [35].

The adverse impact of excess weight on the multiple cardiovascular risk factors, such as high BP, requires primary prevention at early ages, as studies indicate that excess weight in childhood and adolescence tends to persist into adulthood. Thus, the prevalence of high BP identified in the study population, especially among adolescents with severe obesity, strengthens the importance of preventing obesity and its comorbidities [35].

These results showed that the frequency of IDA(serum iron in boys <60  $\mu$ g/dl, that in girls <50  $\mu$ /dl, transferrin saturation ≤15% and haemoglobin concentration <11.5 g/dl) was greater in obese adolescents than in the control group [(32 (80%) vs. 7 (17.5%)]. These results go hand in hand with those of Manios *et al.* [37], who reported a high prevalence of ID and IDA in obese children and adolescents of both sexes.

Previous reports have proposed different mechanisms to explain the high rate of ID among obese children, including consumption of high calorie, iron-poor diets, sedentary lifestyle resulting in decreased myoglobin breakdown, and increased iron requirements for increased red cell mass. A potential mechanism explaining the impact of obesity on iron status may involve the increased production of the protein hepcidin. Hepcidin is a key regulator of iron homeostasis, decreasing intestinal iron absorption and promoting iron sequestration in macrophages, effectively lowering serum iron and the bioavailability of iron [35].

# **Conclusion and recommendations**

In this study a high frequency of IDA was observed among the studied obese adolescents in Egypt, the magnitude of which warrants the need for multiple, large studies in different Egyptian districts.

Regular screening for IDA in obese adolescents is fundamental.

Weight loss programmes may be essential for correction of iron status. Iron fortification for obese adolescents is recommendable.

# Financial support and sponsorship

Ni1

### **Conflicts of interest**

There are no conflicts of interest.

## References

- 1 Castro de Andrade Cairo R, Rodrigues Silva L, Carneiro Bustani N, Ferreira Marques CD. Iron deficiency anemia in adolescents; a literature review. Nutr Hosp 2014; 29:1240–1249.
- 2 Harris RJ. Nutrition in the 21st century: what is going wrong Arch Dis Child 2004; 89:154–158.
- 3 Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. Int J Pediatr Obes 2006; 1:11–25.
- 4 Stoltzfus R. Defining iron-deficiency anemia in public health terms: a time for reflection. J Nutr 2001; 131:565S–567S.
- 5 Nead KG, Halterman JS, Kaczorowski JM, Auinger P, Weitzman M. Overweight children and adolescents: a risk group for iron deficiency. Pediatrics 2004; 114:104-108.
- 6 Gartner A, El Ati J, Traissac P, Bour A, Berger J, Landais E, et al. A double burden of overall or central adiposity and anemia or iron deficiency is prevalent but with little socioeconomic patterning among Moroccan and Tunisian urban women. J Nutr 2014; 144:87–97.
- 7 Seltzer CC, Mayer J. Serum iron and iron-binding capacity in adolescents. Comparison of obese and non obese subjects. Am J Clin Nutr 1963; 13:354–361.
- 8 Pinhas-Hamiel O, Newfield RS, Koren I, Agmon A, Lilos P, Phillip M. Greater prevalence of iron deficiency in overweight and obese children and adolescents. Int J Obes Relat Metab Disord 2003; 27:416-418.
- 9 Menzie CM, Yanoff LB, Denkinger BI, McHugh T, Sebring NG, Calis KA, Yanovski JA. Obesity-related hypoferremia is not explained by differences in reported intake of heme and nonheme iron or intake of dietary factors that can affect iron absorption. J Am Diet Assoc 2008; 108:145–148.
- 10 Ghali I, Salah N, Hussien F, Erfan M, El-Ruby M, Mazen I, et al. (2002). Egyptian growth curves for infants, children and adolescents. Published in: Crecere nel mondo. Satorio A, Buckler JMH and Marazzi N. Ferring Publisher, Italy, 2008.
- 11 World Health Organization. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee (WHO Technical Report Series No. 854). Geneva, Switzerland: World Health Organization; 1995
- 12 National High Blood Pressure Education Program (NHBPEP) Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004; 114:555–576.
- 13 [No authors listed]. International committee for standardization in haematology. Recommendations for haemoglobinometry in human blood. Br J Haematol 1967; 13:71–75.
- 14 Burtis CA, Edward AR. (Ed), Tietz Textbook of Clinical Chemistry, Second Edition, W.B. Saunders Company, Philadelphia, London, Toronto, Montreal, Sydney, Tokyo, 1994; p. 2195.
- 15 Behrman R, Kliegman R, Jenson H. Nelson textbook of pediatrics. 16th ed. Collingwood, Ontario: WB Saunders Company; 2000. 32–50.
- 16 Burtis, CA, Ashwood ER, ed., Tietz Textbook of Clinical Chemistry 3rd Edition, W.B. Saunders, Philadelphia, PA,1999.
- 17 Fairbanks VF, Klee GG. Biochemical aspects of hematology. In CA Burtis, ER Ashwood, editors *Tietz textbook of clinical chemistry*. 3rd ed. Philadelphia: W.B. Saunders Company; 1999. 1642–1644.
- 18 Baillie FJ, Morrison AE, Fergus I. Soluble transferrin receptor: a discriminating assay for iron deficiency. Clin Lab Haematol 2003; 25:353–357.
- 19 Fernandez-Real JM, Moreno JM, López-Bermejo A, Chico B. Vendrell J. Ricart W. Circulating soluble transferring receptor according to glucose tolerance status and insulin sensitivity. Diabetes Care 2007; 30:604–608.

- 20 Dallman PR. Iron deficiency and related nutritional anemias. In: Nathan DG, Orkin SH, editors. Nathan and Oski's hematology of infancy and childhood. Philadelphia: W.B. Saunders Company; 1987. 274-314.
- 21 Stoltzfus RJ, Dreyfuss ML. Guidelines for the use of iron supplements to prevent and treat iron deficiency anaemia. Washington DC, International Nutritional Anaemia Consultative Group/WHO/ UNICEF, 1998.
- 22 English RM, Bennett SA, Iron status of Australian children, Med J Aust 1990; 152:582-586.
- 23 World Health Organization (WHO). Iron deficiency anaemia: assessment, prevention, and control. A guide for programme managers. Geneva: World Health Organization; 2002.
- 24 Murray-Kolb LE, Beard JL. Iron treatment normalizes cognitive functioning in young women. Am J Clin Nutr 2007; 85:778-787.
- 25 Tussing-Humphreys LM, Liang H, Nemeth E, Freels S, Braunschweig CA. Excess adiposity, inflammation, and iron-deficiency in female adolescents. J Am Diet Assoc 2009; 109:297-302.
- 26 Moayeri H, Bidad K, Zadhoush S, Gholami N, Anari S. Increasing prevalence of iron deficiency in overweight and obese children and adolescents (Tehran Adolescent Obesity Study). Eur J Pediatr 2006; 165:813-814.
- 27 Bhatia D, Seshadri S. Growth performance in anemia and following iron supplementation. Indian Pediatr 1993; 30:195-200.
- 28 DiMeglio G. Nutrition in adolescence. Pediatr Rev 2000; 21:32–33.
- 29 Verdon F, Burnand B, Stubi CL, Bonard C, Graff M, Michaud A, et al. Iron supplementation for unexplained fatigue in non-anaemic women: double blind randomised placebo controlled trial. BMJ 2003; 326:1124.

- 30 Haas JD, Brownlie T IV. Iron deficiency and reduced work capacity: a critical review of the research to determine a causal relationship. J Nutr 2001; 131(2S-2): 676S-688S; discussion 688S-690S.
- 31 McCann JC, Ames BN. An overview of evidence for a causal relation between iron deficiency during development and deficits in cognitive or behavioral function. Am J Clin Nutr 2007; 85:931-945.
- 32 Ausk KJ, Ioannou GN. Is obesity associated with anemia of chronic disease? A population-based study. Obesity (Silver Spring) 2008; 16:2356-2361.
- 33 Salem MA, El Alfy MS, El Beblawy NM. Prevalence of obesity among Egyptian primary school children and its effect on haemostasis and thyroid functions. Egypt J Paediatr 2005; 22: 433-452.
- 34 Costanzi CB, Halpern R, Rech RR, Bergmann ML, Alli LR, Mattos AP. Associated factors in high blood pressure among schoolchildren in a middle size city, southern Brazil. J Pediatr (Rio J) 2009; 85:335-340.
- 35 Noronha JAF, Ramos ALC, Ramos AT, Cardoso MAA, De-Carvalho DF, Medeiros CCM. High blood pressure in overweight children and adolescents. J Hum Growth Dev 2012; 22:196-201.
- 36 Sorof JM, Lai D, Turner J, Poffenbarger T, Portman RJ. Overweight, ethnicity, and the prevalence of hypertension in school-aged children. Pediatrics 2004; 113(Pt 1): 475-482.
- 37 Manios Y, Moschonis G, Chrousos GP, Lionis C, Mougios V, Kantilafti M, et al. The double burden of obesity and iron deficiency on children and adolescents in Greece: the Healthy Growth Study. J Hum Nutr Diet 2013; 26:470-478.