Risk factors for poor iron status in British toddlers: further analysis of data from the National Diet and Nutrition Survey of children aged 1.5–4.5 years

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Submitted 24 February 1999: Accepted 23 May 2000

Abstract

Objective: To examine risk factors for poor iron status in British toddlers.

Design: National Diet and Nutrition Survey (NDNS) of children aged 1.5–4.5 years. *Setting:* Mainland Britain, 1992/93.

Subjects: Of the 1859 children whose parents or guardians were interviewed, a weighed dietary intake was provided for 1675, and a blood sample obtained from 1003

Results: Mean haemoglobin (Hb) and ferritin levels were significantly lower in younger (1.5–2.5 years) than in older (3.5–4.5 years) children, with boys having significantly lower ferritin levels than girls. Poor iron status (Hb < $110\,\mathrm{g\,l^{-1}}$, ferritin < $10\,\mu\mathrm{g\,l^{-1}}$, or low values for both indices) was associated with lower socioeconomic and employment status. Iron status was directly associated with meat and fruit consumption and inversely with that of milk and milk products, after adjustment for age and gender. The latter association remained significant after further adjustment for sociodemographic variables, energy intake and body weight. Children consuming >400 g day $^{-1}$ of milk and cream were less likely to consume foods in other groups, with those also consuming little meat, fish, fruit and nuts at greatest risk of poor iron status. Few associations were observed between poor iron status and individual nutrient intakes, and iron status was not associated with either iron intake or with consumption of a vegetarian diet.

Conclusions: Overdependence on milk, where it displaces iron-rich or iron-enhancing foods, may put toddlers at increased risk of poor iron status. However, this becomes non-significant when moderate-to-high amounts of foods known to enhance iron status (e.g. meat and/or fruit) are also consumed. Milk consumption in this age group should ideally be part of a mixed and balanced diet including all food groups, and particularly lean meat (or other iron-rich or fortified foods) and fruit. This is particularly relevant for households of lower socioeconomic and employment status.

Keywords
Toddlers
Iron status
Risk factors
Milk consumption

Iron deficiency (ID) is the most commonly reported nutritional disorder during early childhood in the UK¹ and other countries^{2–4}. Young children are at increased risk due to their high physiological demands during this period of rapid growth and development. ID is associated with a number of manifestations, including anaemia, loss of appetite, pallor, general lassitude and apathy, increased susceptibility to infection, and delayed psychomotor and cognitive development^{5–11}. Despite iron therapy, deficits in the latter symptoms in early childhood may result in long-lasting detriment^{9,11,12}.

Anaemia, 85% of which has been attributed to ID, has been estimated to occur in over a half of preschool

children in developing countries (which contain 80% of the global population) compared with 10–11% in developed countries 2,3,13 . The NDNS of British children aged 1.5–4.5 years 14 found that overall around one in 12 children, and among the youngest group (1.5–2.5 years) one in eight, had an Hb concentration below 110 g l $^{-1}$ (the World Health Organization (WHO) level defining anaemia 15). A recent national study 16 found that up to one-third of toddlers from three different Asian groups living in the UK had Hb < 110 g l $^{-1}$.

The prevalence of ID and iron deficiency anaemia (IDA) varies with age, socioeconomic status, ethnicity and according to criteria used for diagnosis^{4,8,17}. Iron status

can be assessed by several laboratory assays, with no single test acceptable for accurately diagnosing ${\rm ID}^4$. The observed overlap in iron status values between anaemic and non-anaemic individuals implies that Hb alone may overestimate the prevalence of ${\rm IDA}^{18}$. Conversely, others have found that low Hb values identified only 3–20% of subjects with other evidence of ${\rm ID}^{19}$. Likewise, the use of a single indicator of storage iron or tissue iron supply, such as ferritin or transferrin saturation, respectively, may overestimate the number of individuals without iron stores¹⁸.

Iron status depends not only on adequate intakes but also on the form of ingested iron (haem/non-haem), the presence of enhancers and inhibitors of dietary iron absorption and the extent of iron losses from the body. The vast majority (typically >90%) of dietary iron occurs in non-haem form (from vegetables and cereals), which is more poorly absorbed than haem iron (from meat and fish), and whose bioavailability is greatly affected by enhancers (e.g. meat, fish, vitamins C and A) or inhibitors (e.g. dietary fibre, tea, coffee, calcium) also present in the diet^{20–22}. Iron status may also be impaired through excessive loss of body iron due to infection, inflammation or parasitic infestation, all of which form a greater cause of ID and anaemia in developing countries with poor sanitation and other factors related to poverty^{23,24}.

ID is more common in children with an overdependence on cows' milk (i.e. excessive consumption with only small quantities of solid food)^{25,26}. Although one study found that no single factor accounted for more than a small proportion of the variance in Hb and ferritin values16, factors exerting the most significant adverse effects on iron status in Asian children were the amount of cows' milk consumed, prolonged use of a feeding bottle, and the mother's place of birth being outside the UK. Two-year-old Asian children who consumed more than 600-700 ml of cows' milk daily were at high risk of ID^{16,25}. Asian weaning diets, in particular, may be overdependent on milk^{16,27}, although in the survey used for the present study, only 2% of participants were of north Indian (Asian) origin. Vegetarian and vegan children are also at greater risk of ID, although this can be avoided and adequate iron status is achievable with vegetarian diets²⁸.

It is unclear whether drinking large quantities of cows' milk during early childhood predisposes to ID due to milk (with its low iron content) being consumed to the exclusion of more iron-rich foods, or whether (alternatively or in addition) specific inhibitory factors in milk (e.g. calcium) are responsible^{20,25}. A review of the effect of dairy foods on iron availability found conflicting evidence due to differences in the nature of diets, the form of dietary iron, differing iron status of subjects, and differences in methods used to assess iron availability²⁹. It was concluded that generally dairy foods have little effect on iron availability when added to complex meals, and

that factors known to enhance iron absorption may override any inhibitory effects of milk and other dairy foods

The aim of the present study was to examine the iron status of British toddlers who participated in the recent NDNS, to determine whether they exhibited convincing evidence of suboptimal iron status and whether this was attributable mainly to diet. We also aimed to identify risk factors related to IDA, which may be of public health concern for toddlers in the UK.

Subjects and methods

The study used data from the cross-sectional NDNS of children aged 1.5–4.5 years in mainland Britain, to examine risk factors for poor iron status. Full details of the NDNS are provided elsewhere¹⁴ and only a brief account is given here.

This NDNS was carried out between July 1992 and June 1993, on behalf of the UK government (Department of Health and the Ministry of Agriculture, Fisheries and Food (MAFF)), by the Social Survey Division of the Office of Population Censuses and Surveys (OPCS) (now known as the Office for National Statistics (ONS)) and the Micronutrient Status Group of the Medical Research Council's Dunn Nutrition Unit (now part of MRC Human Nutrition Research). A postal sift of addresses selected from the Postcode Address File was used to identify a nationally representative sample of 2101 children aged 1.5–4.5 years. Only those children who were living in private households were eligible for inclusion and only one child per household was selected.

A structured interview, carried out by trained fieldworkers, was completed for 1859 children (88% of the identified sample) and was considered to be representative of the population in terms of sociodemographic characteristics, according to the 1992 General Household Survey³⁰ and mid-year population estimates*. The survey design included: a face-to-face interview, usually with the child's mother, to provide information about sociodemographic characteristics of the child's household, medicine usage and eating and drinking habits; a weighed dietary record of all food and drink (including supplements) consumed over four consecutive days (including Saturday and Sunday), which was checked during subsequent visits by the interviewer; a record of bowel movements for the same period; anthropometric measurements; a request to take a blood sample; and a dental examination. Ethical permission for the survey was obtained from the National Health Service Local Research Ethics Committee

*Population Estimates Unit, OPCS, Crown Copyright (unpublished data). In mid-1992 the number of children born in 1988, 1989, 1990 and 1991 and living in Great Britain was estimated to be 3 019 644. Of these children 25% were born in each of the 4 years showing an equal distribution in the population from which the survey sample was drawn.

for each location, and from the MRC Dunn Nutrition Unit's Ethics Committee.

A 4-day weighed dietary record was obtained for 1675 children (i.e. 90% of those completing the interview). A feasibility study before the survey confirmed that a 4-day weighed intake was a suitable method for providing reliable data on the diets of young children³¹. The dietary records were coded and intakes of energy and nutrients calculated from the consumption data, by OPCS, using a nutrient databank compiled by MAFF.

The extent and possible impact of underreporting in the dietary record was also investigated. The method of Schofield et al.³² was employed, based on estimated basal metabolic rate, with minimum cut-off limits applied for acceptable/valid energy intakes³³. Underreporting was estimated to occur in 45% of the 1675 toddlers; a similar proportion to that from a recent national diet and nutrition survey of British adults^{34,35}. To assess possible selective underreporting (e.g. whether toddlers with higher milk consumption reported proportionately lower consumption of meat, fruit, etc.), mean consumption values of each food group were compared (using unpaired Student's t-test) between the two groups (i.e. underreporters and not underreporters). Consistently lower values among underreporters would negate likely selectivity in underreporting.

Only 51% of toddlers provided both weighed dietary records and blood samples. In order to assess the representativeness of the results relating food and nutrient intakes to blood iron status indices, we also investigated whether those who provided both samples (n = 956) possessed different characteristics from those who provided a dietary record but no blood sample (n = 719).

Consent to the request for a blood sample was given for 1157 children (62% of the interview sample) and blood was obtained from 1003 children (54%). Initial blood processing was performed at a local laboratory on the same day, while a portion (1 ml) of the sample was posted, on the same day, to Great Ormond Street Hospital, London, UK for haematological analyses. Analyses for other blood status indices were performed at the MRC Dunn Nutrition Unit, Cambridge, UK and at the University of Ulster, Coleraine, Northern Ireland. Details of analytical methods and quality control are described elsewhere 14 and only those for Hb and ferritin measurements are summarized here. Blood counts, including Hb, were performed using a Coulter S Plus Junior, a Coulter Max M or Coulter T660 electronic cell counter. Hb was measured at 540 nm using a cyanmethaemoglobin colorimetric technique. Plasma ferritin was measured with Becton-Dickinson monoclonal antibody-coated tubes, using an immunoradiometric (IRMA) assay ('sandwich technique').

For the analyses presented here, data reduction was performed using Excel (Microsoft Corp., USA), while data analysis was carried out using DataDesk (Data Descriptions

Inc., USA). Analyses excluded all participants who were unwell with eating affected (16% of those with diary data, n = 266) and those with an α_1 -antichymotrypsin (ACT) level $> 0.65 \text{ g l}^{-1}$ (n = 81 or 11% of those with an ACTmeasurement) in order to exclude those with infection or inflammation. Much of the anaemia, and raised ferritin levels, observed in paediatric practice has been attributed to mild acute infection^{36,37}. The food group analyses also excluded those receiving iron supplements (2%, n = 30). Age and gender adjustments were also included. Logistic and multiple regressions, analyses of variance and unpaired Student's t-tests were performed, with $P \le$ 0.05 deemed significant. Where necessary, variables were transformed to normality using either logarithmic or square root transformations. Food groups were divided into fifths, or where more than 20% of children had zero consumption for a particular food group, binary variables were used. Linear and quadratic trends in associations between dependent and independent variables are described.

Results

Children who provided a food record, but not a blood sample, did not differ significantly with respect to age and gender profiles, anthropometry or geographical region of domicile, compared with those who provided both. Of 50 calculated nutrient intakes, spanning all major macro- and micronutrients, the only nutrients which differed ($P \le 0.05$) between these two groups were retinol, α - and β -carotenes and total sugars, for which the intakes were higher in the group that provided a blood sample. The difference in sugars became non-significant after adjustment for energy intake. Therefore, although the group that provided blood may have differed in some subtle respects from that which did not, these differences were not of major concern for the present study.

Eight per cent of children had an Hb concentration $<110 \text{ g l}^{-1}$, while 41% had Hb $<120 \text{ g l}^{-1}$. Twenty per cent of children had ferritin concentrations $<10 \text{ µg l}^{-1}$, while 31% were $<12 \text{ µg l}^{-1}$. Twenty-four children (3.4%) had both low Hb ($<110 \text{ g l}^{-1}$) and low ferritin ($<10 \text{ µg l}^{-1}$). The proportions of toddlers with low Hb did not vary significantly between those underreporting and not underreporting (7% vs 8%, respectively, P = 0.66), although the difference between those with ferritin $<10 \text{ µg l}^{-1}$ in the two groups was of borderline significance (17% vs 23%, respectively; P = 0.051).

Table 1 shows the mean concentrations of Hb and ferritin by age group and gender. Older children had significantly higher mean concentrations of Hb and ferritin than younger children, with females having significantly higher concentrations of ferritin than males.

Table 2 shows sociodemographic variables associated with poor iron status. After adjustment for age and gender, the proportion of children with low Hb was

Table 1 Mean concentration of haemoglobin and ferritin by age group and gender^a

	Blood	haemoglobin (g	g l ⁻¹)	Serum ferritin (μg I ⁻¹)			
Subject characteristic	Mean	SD	n	Mean†	95%CI‡	n	
Age group (years)							
1.5–2.5	120	9	233	14.9	3.2-71.1	226	
2.5-3.5	122	9	261	17.9*	4.4-72.1	257	
3.5-4.5	123**	9	233	18.4**	4.6-71.8	228	
Gender							
Male	122	10	358	15.6	3.6-70.0	351	
Female	121	8	369	18.5**	4.7-74.1	360	

 $^{^{}a}$ Excludes those with α_{1} -antichymotrypsin values >0.65 g l $^{-1}$ and those unwell with eating affected.

Within age or gender groups, the mean is significantly different from age group 1.5–2.5 years, or from males, $*P \le 0.05$; $**P \le 0.01$.

significantly greater in those whose head of household was unemployed or economically inactive, those receiving state benefits, those whose mothers had fewer qualifications than O levels or equivalent, those with

low household incomes (particularly if ≤£6000 year⁻¹), those who had never been breast-fed (particularly compared with those breast-fed for >6 months), and those more difficult to feed for their age. The proportion

Table 2 Sociodemographic variables associated with poor iron status*

	% of children with poor iron status in each sociodemographic group						
Sociodemographic group†	1	2	3	4	5	n	Linear trend (P value)
Haemoglobin <110 g l⁻¹							
Employment status of HoH (1 working;	6	13	11	-	_	727	0.02
2 unemployed; 3 economically inactive)							
State benefits received (1 not receiving; 2 receiving)	6	11	_	_	_	727	0.02
Mothers' qualifications (1 above A level; 2 A level; 3 O level or equivalent; 4 CSE or equivalent; 5 none)	5	5	4	11	14	725	0.001
Household income (1 £25 000; 2 £18 000-25 000; 3 £10 000-18 000; 4 £6000-10 000; 5 £0-6000)	7	5	5	6	13	703	0.02
Feeding (1 easy to feed for age; 2 average; 3 difficult)	5	8	10	_	-	726	0.04
Breast-feeding (1 breast-fed; 2 never breast-fed)	6	10	_	_	-	716	0.02
Breast-feeding duration (1 >6 months; 2 3-6 months; 3 <3 months; 4 never)	3	6	7	10	-	715	0.007
Ferritin $<$ 10 $\mu g \Gamma^{-1}$							
Social class (1 non-manual; 2 manual; 3 armed forces; 4 never worked)	17	23	33	42	-	707	0.01
Eating behaviour (1 eats most things; 2 eats reasonable variety; 3 fussy or faddy eater)	18	20	28	-	-	710	0.03
Haemoglobin <110 g I^{-1} and ferritin <10 μ g I^{-1}							
State benefits received (1 not receiving; 2 receiving)	2	5	-	-	-	711	0.04
Mothers' qualifications (1 above A level; 2 A level; 3 O level or equivalent; 4 CSE or equivalent; 5 none)	1	4	2	5	6	709	0.04
Appetite (1 good; 2 average; 3 poor)	1	5	4	-	-	711	0.03

HoH, Head of household.

[†] Geometric mean.

^{‡ 2.5-97.5} percentile.

^{*} Adjusted for age group and gender. Excludes those with α_1 -antichymotrypsin values >0.65 g l⁻¹ and those unwell with eating affected.

[†] The following sociodemographic variables were not associated (P > 0.05) with iron status: family type; supplements of vitamins/minerals; mother smoking; father smoking; region; allergies affecting eating; other food avoidance; number of children in household; prematurity of birth; low birth weight; birth order; ethnic group. Measures of socioeconomic status are based on social class of HoH (based on their occupation), employment status of HoH, whether the HoH was receiving family credit and/or income support, and the mothers' highest educational qualification. Educational qualifications (England and Wales): CSE (Certificate of Secondary Education) and O level (Ordinary level or General Certificate of Education) examinations, usually attained at 14–15 years. CSE (a lesser qualification than O level) and O levels have now been replaced by the GCSE (General Certificate of Secondary Education) examination. A (Advanced) level examinations are usually taken at age 17–18 years.

Table 3 Consumption of food groups associated with poor iron status*

	% of children with poor iron status in each food group (fifths)						<i>P</i> value		
Iron status category and food group†	1	2	3	4	5	n	Linear trend	Quadratic trend	
Haemoglobin <110 g l⁻¹									
Meat and fish	13	6	3	8	7	717	NS	0.01	
Fruit and nuts	10	11	5	5	5	717	0.04	NS	
Ferritin $<$ 10 μ g Γ^{-1}									
Milk and milk products	15	14	22	25	26	701	0.002	NS	
Subgroup: milk and cream	15	17	18	25	26	701	0.009	NS	
Meat and meat products	28	21	13	19	21	701	NS	0.02	
Meat and fish	29	21	15	18	19	701	0.03	NS	
Hb < 110 g Γ^1 and ferritin < 10 μ g Γ^1									
Milk and milk products	1	2	3	4	7	701	0.007	NS	
Subgroup: milk and cream	2	1	3	4	7	701	0.02	NS	
Fat spreads	7	1	2	2	5	701	NS	0.004	
Fruit and nuts	5	5	3	1	1	701	0.02	NS	
Soft drinks (excluding fruit juice)	6	4	5	1	1	701	0.01	NS	

NS, not significant if P > 0.05.

Milk and cream includes condensed, evaporated and dried milks, goat's milk, sheep's milk, soya milk, milk shakes, infant formula and all creams.

of children with low ferritin was significantly greater in those whose head of household was of manual status or in armed forces or who had never worked, and in those who were fussy or faddy eaters. The proportion of children with both low Hb and low ferritin was greater in households receiving benefits, in those whose mothers had few or no qualifications at O level or equivalent, and in those without a good appetite.

Food group consumption was divided into fifths or treated as binary variables to examine the associations with poor iron status (Table 3). After adjustment for age and gender, the highest proportions of children with low Hb or low ferritin were those with consumption of meat and meat products, and its subgroup meat and fish, in the lowest fifth of their respective distributions. High consumption of fruit and nuts was associated with a low proportion of children having low Hb, or low Hb with low ferritin. The proportion of children with low ferritin, or low Hb with low ferritin, was higher in those consuming large quantities of milk and milk products, or the subgroup milk and cream.

The inverse relationship between milk and milk product consumption and iron status remained significant after further adjustment for age, gender, energy intake and body weight (P=0.03), and even after adjustment for age, gender and all 10 sociodemographic variables associated with iron status (see Table 2) (P=0.02). The proportion of children with both low Hb and low ferritin was greater in those consuming >400 g day⁻¹ (two glasses) of milk and cream compared with those consuming <400 g day⁻¹ (6% vs 2%; P=0.02). The

association remained significant after adjustment for age, gender and the 10 sociodemographic variables associated with iron status (Table 2) (P = 0.04), and after adjustment for age, gender, energy intake and weight of child (P = 0.05). Twenty-six per cent of children in the survey were receiving $>400 \text{ g day}^{-1}$ of milk and cream, with liquid milks comprising the vast majority.

The proportion of children consuming >400 g day⁻¹ of milk and cream was less in older children aged 3.5–4.5 years (P < 0.0001) and, after adjustment for age and gender, in those whose mothers had attained O levels or equivalent (P = 0.02), in those who were moderately easy to feed (P = 0.03), and in those eating a reasonable variety of foods (P = 0.01). In addition, Caucasian children were less likely to consume >400 g day⁻¹ of milk and cream than children from other ethnic groups (Asian, Black, Chinese, other) (24% vs 44%; P = 0.002). Associations remained significant after adjustment for age, gender, energy intake and body weight.

The direct association between milk and cream consumption and poor iron status, and possible displacement of other foods richer in iron, was investigated further (Table 4). Children who consumed >400 g day⁻¹ of milk and cream were less likely to consume the following food groups: cereals and cereal products, eggs and egg dishes; fat spreads; meat and meat products; meat and fish; vegetables (including potatoes and chips); sugar, preserves and confectionery; fruit juice and soft drinks (excluding fruit juice). The prevalence of poor iron status was greater in children consuming >400 g day⁻¹ of milk and cream plus low consumption of meat and fish, and

^{*} Adjusted for age group and gender. Excludes those with α_1 -antichymotrypsin values >0.65 g l⁻¹, those unwell with eating affected, and those taking iron supplements.

[†] The following food groups were not associated (P > 0.05) with poor iron status: cereals and cereal products (and subgroup: breakfast cereals); eggs and egg dishes (binary variable); fish and fish dishes (binary variable); vegetables (including potatoes); salad/vegetables (excluding potatoes); potatoes (including chips); sugar, preserves and confectionery; fruit juice (binary variable); coffee (binary variable); tea (binary variable); commercial infant foods and drinks (binary variable).

Table 4 Relationship between food group consumption and percentage of children consuming >400 g day⁻¹ of milk and cream*

	$\%$ of children consuming ${>}400~{\rm g~day}^{-1}$ of milk and cream in each food group (ascending fifths‡ of food group consumption)						
Food group†	1	2	3	4	5	Linear trend (P value)	
Cereals and cereal products	29	29	24	24	23	0.04	
Eggs and egg dishes	28	23	_	_	-	0.05	
Fat spreads	32	28	21	22	25	0.02	
Meat and meat products	33	25	24	22	24	0.03	
Subgroup: meat and fish	32	26	23	24	22	0.01	
Vegetables (including potatoes)	32	19	25	27	25	0.03	
Subgroup: potatoes (including chips)	34	26	20	22	26	0.01	
Sugar, preserves and confectionery	33	28	28	18	20	< 0.001	
Soft drinks (excluding fruit juice)	45	38	23	15	11	< 0.0001	
Subgroup: fruit juice	27	22	_	_	_	0.03	
Coffee (made up)	26	16	_	_	_	0.02	
Commercial infant food and drinks	25	36	_	_	_	0.03	

^{*} Adjusted for age group and gender. Excludes those with α_1 -antichymotrypsin values >0.65 g l⁻¹, those unwell with eating affected, and those taking iron supplements.

fruit and nuts, compared with those consuming $<400 \text{ g day}^{-1}$ of milk and cream (9% vs 2%; P=0.02). This relationship remained significant after adjustment for age, gender and 10 sociodemographic variables associated with iron status (P=0.03); and after adjustment for age, gender, energy intake and body weight (P=0.05). Iron status was not associated with milk consumption ($>400 \text{ g day}^{-1}$) if moderate or high amounts of meat and fish and/or fruit and nuts were also consumed.

Underreporting was not found to be selective across the range of food groups. Although difficult to ascertain unequivocally from the data available, those toddlers with a high milk consumption (however defined) did not appear to selectively underreport the consumption of meat and meat products or fruit, nor was consumption of those foods with a high fibre content found be selectively overreported. However, those estimated to be underreporting food consumption did differ in some respects from those without evidence of underreporting. Although not differing significantly in sociodemographic terms, underreporters comprised a smaller proportion consuming $>400~{\rm g~day}^{-1}$ milk and cream, compared with those not underreporting (18% vs 33%; P < 0.0001). This should be borne in mind when interpreting the results.

The relationships between milk and milk product consumption on the one hand, and of selected nutrients on the other, were also analysed (not presented). Milk and milk product consumption was directly associated with intakes of energy, iron, zinc, retinol, protein, fat, calcium and phosphorus. It was also associated directly with the blood status indices retinol, zinc protoporphyrin and red blood cell count, and inversely with mean corpuscular volume and ferritin.

Few associations between poor iron status and intakes of individual nutrients (including supplements) were observed ($P \le 0.05$). Low Hb was related to higher

retinol intakes (P = 0.03), while low ferritin was related to higher calcium (P = 0.02), sugar (P = 0.02) and intrinsic milk sugar (P = 0.02) intakes, and to lower *cis*-polyunsaturated fatty acid intakes (P = 0.03). Low Hb combined with low ferritin was associated with higher intakes of calcium (P = 0.05) and retinol (P = 0.0007).

No association was observed between iron status and vegetarian diets (those which excluded all meat and meat products n=20; or those excluding all meat and fish n=14), although the sizes of these subgroups were clearly very small (not presented). The effect of tea drinking on iron status was also investigated. Thirty-seven per cent of toddlers were found to consume tea, with an age and gender distribution similar to the sample as a whole. When toddlers were classified into tea drinkers or non-tea drinkers, no significant association was found with iron status (low Hb and/or low ferritin), after adjustment for age and gender.

Discussion

This study has attempted to identify risk factors for poor iron status in British children aged 1.5–4.5 years. Since no single measure can accurately diagnose ID, iron status was assessed by the following indicators: Hb level <110 g l⁻¹ (the WHO definition for anaemia¹⁵), ferritin level <10 μg l⁻¹ (an earlier sign of ID, reflecting depletion of iron stores) and low Hb combined with low ferritin.

Eight per cent of children had an Hb level $<110 \text{ g l}^{-1}$, 20% had a ferritin level $<10 \text{ µg l}^{-1}$ and 3.4% had low Hb with low ferritin, as a composite index of iron status. These proportions were higher than in some developed countries^{38–40} and similar to others^{26,41}, although low relative to many developing countries¹³. In agreement with other findings^{40–42}, poor iron status was associated

[†] The following food groups were not associated with the consumption of >400 g day⁻¹ of milk and cream: breakfast cereals; fish and fish dishes; salad/vegetables (excluding potatoes); fruit and nuts; tea (made up).

[‡] Where more than 20% of children had a zero consumption binary variables are presented.

with lower sociodemographic variables, in terms of employment status, social class, state benefits being received, mother's qualifications, household income, previous breast-feeding, and the eating, feeding and appetite characteristics of the child. To reduce the high prevalence of poor iron status in these vulnerable groups, targeted community-based nutrition and dietary health education programmes may be desirable, such as those implemented successfully in the USA (Women, Infants and Children (WIC) and Food Stamp programmes). In the last few decades, both programmes have led to significant reductions in the prevalence of ID and IDA and to improved nutrient intakes and status overall in socially disadvantaged preschoolers^{43–45}.

As expected, good iron status in the present study was associated directly with meat and fruit consumption. Meat (especially red meat) is rich in haem iron that is relatively well absorbed²⁰, and both meat⁴⁶ and fruit²⁰, with its generally high vitamin C content, are known to enhance the absorption of non-haem iron, which comprised over 90% of total iron intake. Consumption levels of soft drinks (excluding fruit juice) were also independently associated with better iron status (higher Hb and ferritin levels). This may be due to soft drinks replacing milk consumption, though it is also possible that their vitamin C content enhanced iron absorption.

No association was found between iron status and iron intake (total, haem or non-haem iron), probably because the bioavailability of non-haem iron, which forms the vast majority of total iron intake, is dependent on the varying proportions and complex interactions between enhancers and inhibitors in the diet^{20,21}. Paradoxically, a higher retinol intake was associated with lower Hb (possibly due to the high retinol content of milk and milk products) but higher retinol status was associated with higher Hb. The NDNS report¹⁴ noted that there was no association between plasma retinol and its dietary intake. Other studies have reported interactions between vitamin A and iron status^{47,48}, with direct correlations between plasma retinol and iron status in children also observed⁴⁸.

Low ferritin levels were associated with relatively high intakes of sugar, calcium and intrinsic milk sugars. Neither low Hb nor low ferritin was related to high fibre intakes, as might have been expected. However, fibre is associated with fruit and hence also with high vitamin C intake. The food group cereals and cereal products (which includes breads) was strongly related to iron intake but not significantly related to iron status, possibly due to the inhibitory effects of non-starch polysaccharides.

Consumption of milk and milk products was inversely associated with ferritin and with good composite iron status (higher Hb and ferritin levels). Such inverse associations have also been observed by others^{4,16,25,27}. ID is common in children consuming large quantities of cows' milk, particularly if they consume only small amounts of solids²⁵. However, one review²⁹ concluded

that dairy foods may have little effect on iron availability when added to complex meals, since components which enhance iron absorption may override any inhibitory effects of milk.

After adjustment for age and gender, children consuming >400 g day⁻¹ of milk and cream had a significantly greater chance of having poor iron status than those consuming <400 g day⁻¹. This inverse relationship remained significant after further adjustment for sociodemographic factors, energy intake and body weight. Those children who were younger, or more difficult to feed, or whose mothers had attained few or no qualifications at O level standard or higher, or who were of non-Caucasian ethnicity, were more likely to consume >400 g day⁻¹ of milk and cream. Others have reported that Asian weaning diets may be over-dependent on milk^{16,27}, but the number of Asian children in the present study (n = 27, with dietary records) was too small to account for the observed relationships.

Consumption of >400 g day⁻¹ of milk and cream in the present study appeared to displace a wide variety of other food groups from the diet, including iron-rich and iron-enhancing foods such as meat, fish, eggs, cereals and fruit. Iron status in children consuming >400 g day⁻¹ of milk and cream plus moderate or high amounts of meat and fish, and/or fruit and nuts, was no poorer than for all children who consumed <400 g day⁻¹.

Conclusions

Although it contains many essential nutrients, an over-dependence on milk may put young children at increased risk of poor iron status, owing to its displacement of iron-rich or iron-enhancing foods from the diet. This risk becomes non-significant when moderate to high amounts of iron-rich or iron-enhancing foods (e.g. meat and fruit) are also consumed. Milk consumption in this age group should ideally be part of a mixed and balanced diet which includes all food groups, particularly meat (or iron-rich alternatives), iron-fortified foods, fruit and vegetables. This advice should be targeted particularly at households of lower socioeconomic and employment status.

Acknowledgements

The National Diet and Nutrition Survey of children aged 1.5–4.5 years was funded jointly by the Ministry of Agriculture, Fisheries and Food and the Department of Health and conducted by the Office of Population Censuses and Surveys, in conjunction with the Medical Research Council's Dunn Nutrition Unit. The dataset was provided by The Data Archive, University of Essex, Colchester CO4 3SQ, UK. We are grateful to Dr Rachel Thompson, University of Southampton, for helpful advice. CWT and CMW were funded by the Ministry of Agriculture, Fisheries and Food.

References

- 1 Department of Health. Weaning and the Weaning Diet. Report on Health and Social Subjects, No. 45. London: HMSO, 1994.
- 2 De Maeyer E, Adiels-Tegman M. The prevalence of anaemia in the world. *World Health Stat. Q.* 1985; **38**: 302–16.
- 3 Viteri FE. Iron: global perspective. In: Ending Hidden Hunger: a Policy Conference on Micronutrient Malnutrition. Atlanta, GA: Task for Child Survival and Development, 1992: 145–84.
- 4 Morbidity and Mortality Weekly Report (MMWR). Recommendations to Prevent and Control Iron Deficiency in the United States. Report No. 47 (RR-3). Atlanta, GA: US Department of Health and Human Sciences, CDC, 1998.
- 5 Weinburg ED. Iron and susceptibility to infectious disease. *Science* 1974; **184**: 952–6.
- 6 Aukett MA, Parks YA, Scott PH, Wharton BA. Treatment with iron increases weight gain and psychomotor development. *Arch. Dis. Child.* 1986; 61: 849–57.
- 7 Lozoff B, Brittenham GM, Wolf AW, et al. Iron deficiency anemia and iron therapy effects on infant developmental test performance. *Pediatrics* 1987; **79**: 981–95.
- 8 Oski FA. Iron deficiency in infancy and childhood. *New Engl. J. Med.* 1993; **329**: 190–3.
- 9 Walter T. Impact of iron deficiency on cognition in infancy and childhood. Eur. J. Clin. Nutr. 1993; 47: 307–16.
- 10 Brock JH. Iron and immunity. J. Nutr. Immunol. 1993; 2: 47– 106.
- 11 Lansdown R, Wharton BA. Iron and mental and motor behaviour in children. In: *Iron: Nutritional and Physiological Significance. The Report of the British Nutrition Foundation's Task Force.* London: Chapman and Hall, 1995: 65–78.
- 12 Hurtado EK, Claussen AH, Scott KG. Early childhood anemia and mild or moderate mental retardation. *Am. J. Clin. Nutr.* 1999; **69**: 115–19.
- 13 Florentino RF, Guirriec R. Prevalence of nutritional anemia in infancy and childhood with emphasis on developing countries. In: Steckel A, ed. *Iron Nutrition in Infancy and Childhood*. New York: Nestlé/Raven Press, 1984: 61–74.
- 14 Gregory JR, Collins DL, Davies PSW, Hughes JM, Clarke PC. National Diet and Nutrition Survey: Children Aged 1.5 to 4.5 years. Vol. 1: Report of the Diet and Nutrition Survey. London: HMSO, 1995.
- 15 WHO/UNICEF/UNU. *Iron Deficiency: Indicators for Assessment and Strategies for Prevention*. Geneva: World Health Organization, 1998.
- 16 Lawson MS, Thomas M, Hardiman A. Iron status of Asian children aged 2 years living in England. Arch. Dis. Child. 1998: 78: 420–6.
- 17 Worwood M. Measurement of iron status. In: *Iron: Nutritional and Physiological Significance. The Report of the British Nutrition Foundation's Task Force.* London: Chapman and Hall, 1995: 23–32.
- 18 Worwood M. The laboratory assessment of iron status an update. *Clin. Chim. Acta* 1997; **259**: 3–23.
- 19 Taylor PG, Martinez-Torres C, Mendez-Castellano H, *et al.* The relationship between iron deficiency and anemia in Venezuelan children. *Am. J. Clin. Nutr.* 1993; **58**: 215–18.
- 20 Fairweather-Tait S, Hurrell RF. Bioavailability of minerals and trace elements. *Nutr. Res. Rev.* 1996; **9**: 295–324.
- 21 Lynch SR. Interaction of iron with other nutrients. *Nutr. Rev.* 1997; **55**: 102–10.
- 22 García-Casal M, Layrisse M, Solano L, et al. Vitamin A and β-carotene can improve non-heme iron absorption from rice, wheat and corn by humans. J. Nutr. 1998; 128: 646–50.
- 23 Fleming AF. Haematological manifestations of malaria and other parasitic diseases. Clin. Haematol. 1981; 10: 983–1011.
- 24 Adish AA, Esrey SA, Gyorkos TW, Johns T. Risk factors for iron deficiency in preschool children in northern Ethiopia. *Public Health Nutr.* 1999; 2: 243–52.

25 Lawson MS. Iron in infancy and childhood. In: Iron: Nutritional and Physiological Significance. The Report of the British Nutrition Foundation's Task Force. London: Chapman and Hall, 1995: 93–105.

- 26 Oti-Boateng P, Seshadri R, Petrick S, Gibson RA, Simmer K. Iron status and dietary iron intake of 6–24-month-old children in Adelaide. *J. Paediatr. Child Health* 1998; **34**: 250–3.
- 27 Duggan MB. Cause and cure for iron deficiency in toddlers. *Health Visitor* 1993; **66**: 250–2.
- 28 Sanders TAB. Vegetarian diets and children. *Pediatr. Clin. North Am.* 1995; **42**: 955–65.
- 29 Jackson LS, Lee K. The effect of dairy products on iron availability. *Crit. Rev. Food Sci. Nutr.* 1992; **31**: 2559–70.
- 30 Thomas M, Goddard E, Hickman M, Hunter P. 1992 General Household Survey. London: HMSO, 1994.
- 31 White A, Davies PSW. Feasibility Study for the National Diet and Nutrition Study of Children Aged 1.5 to 4.5 years. London: OPCS, 1994.
- 32 Schofield WN, Schofield C, James WPT. Basal metabolic rate. *Hum. Nutr. Clin. Nutr.* 1985; **39C** (Suppl. 1): 1–96.
- 33 Goldberg GR, Black AE, Jebb SA.*et al.* Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify underreporting. *Eur. J. Clin. Nutr.* 1991; 45: 569–81.
- 34 Gregory J, Foster K, Tyler H, Wiseman M. *The Dietary and Nutritional Survey of British Adults*. London: HMSO, 1990.
- 35 Macdiarmid J, Blundell J. Assessing dietary intake: who, what and why of under-reporting. *Nutr. Res. Rev.* 1998; **11**: 231–53.
- 36 Stevens D. Epidemiology of hypochromic anaemia in young children. *Arch. Dis. Child.* 1991; **66**: 886–9.
- 37 Stevens D. Screening toddlers for iron deficiency anaemia in general practice. No investigation can accurately separate normal from pathological [letter]. *BMJ* 1998; 316: 145.
- 38 Hagman U, Bruce Å, Persson L-Å, Samuelson G, Sjölin S. Food habits and nutrient intake in childhood in relation to health and socio-economic conditions: a Swedish multicentre study 1980–81. Acta Paediatr. Scand Suppl. 1986; 328: 1–56.
- 39 Karr M, Alperstein G, Causer J, Mira M, Lammi A, Fett MJ. Iron status and anaemia in preschool children in Sydney. *Aust. NZ J. Public Health* 1996; **20**: 618–22.
- 40 Looker AC, Dallman PR, Carroll MD, Gunter EW, Johnson CL. Prevalence of iron deficiency in the United States. J. Am. Med. Assoc. 1997; 277: 973–6.
- 41 Grant GA. Prevalence of iron deficiency in rural pre-school children in Northern Ireland. *Br. J. Gen. Pract.* 1990; **40**: 112–13
- 42 Owen GM, Kram KM, Garry PJ, Lowe JE, Lubin AH. A study of nutritional status of preschool children in the United States 1968–1970. *Pediatrics* 1974; **53** (4) (Suppl. II): 597–646.
- 43 Yip R, Parvanta I, Scanlon K, Borland EW, Russell CM, Trowbridge FL. Pediatric Nutrition Surveillance System – United States 1980–1991. Morb. Mortal. Weekly Rep. 1992; 41 (SS-7): 1–24.
- 44 Sherry B, Bister D, Yip R. Continuation of decline in prevalence of anemia in low-income children: the Vermont experience. Arch. Pediatr. Adolesc. Med. 1997; 151: 928–30.
- 45 Rose D, Habicht J-P, Devaney B. Household participation in the Food Stamp and WIC programs increases the nutrient intakes of preschool children. *J. Nutr.* 1998; **128**: 548–55.
- 46 Engelmann MDM, Davidsson L, Sandstrom B, Walczyk T, Hurrell RF, Michaelsen KF. The influence of meat on nonheme iron absorption in infants. *Pediatr. Res.* 1998; **43**: 768–73.
- 47 Shatrugna V, Raman L, Uma K, Sujatha T. Interaction between vitamin A and iron: effects of supplements in pregnancy. *Int. J. Vitam. Nutr. Res.* 1997; **67**: 145–8.
- 48 Majia LA, Hodges RE, Arroyave G, Viteri F, Torun B. Vitamin A deficiency and anemia in Central American children. *Am. J. Clin. Nutr.* 1977; **30**: 1175–84.