

Original Article

Haematocrit levels and anaemia in Australian children aged 1-4 years

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The aim of this study was to describe the prevalence of anaemia, mean haematocrit levels, and the risk factors influencing haematocrit in participants of the 1995 National Survey of Lead in Children. A nationally-representative cross-sectional survey of children aged 1-4 years inclusive was done. Mean haematocrit and the proportion with anaemia using both the US and WHO haematocrit-based criteria were calculated. Multivariate regression was used to identify factors associated with haematocrit. Mean haematocrit level was 38.8% (95% CI: 38.6 - 39.1%) and varied with age of child, state/territory of residence and whether the child was taking supplements. It did not vary by sex, Aboriginal identification, maternal birthplace, whether the child ate meat or any other selected characteristic. The factors identified explained only 4% of the variation in haematocrit levels. The prevalence of anaemia was 3.3% (95% CI: 2.4 - 4.5%) based on the US criteria and 2.0% (95% CI: 1.3 - 3.1%) based on the WHO criteria. The prevalence of anaemia in this national survey was lower than the prevalence of iron deficiency anaemia reported in several more localised studies.

Key words: anaemia, haematocrit, national survey, children, Australia

Introduction

World-wide, iron deficiency anaemia is the most common nutritional deficiency. Generally, the highest prevalence is found in pre-school aged children, adolescents and women of reproductive age.¹ Several localised studies have been conducted in Australia in recent years. Karr *et al.*, found a prevalence of 1.1% (95% CI: 0.1-2.1%) in a representative sample of children aged 9-62 months living in the central and southern Sydney areas, after excluding 0.8% with thalassaemia.² This was highest (3%) in 2-year old children, but fell to 0% in children aged 3 years and older. A subsequent study of children from the same area who had mothers born in Arabic countries, found a prevalence of 6% after excluding 5.5% with haemoglobinopathies.³ By contrast, a study from Adelaide reported that the prevalence of iron deficiency anaemia was 6% in Caucasians aged 6-24 months. Although this study also reported a higher prevalence in Asian than Caucasian children, the children were a non-representative sample and, in particular, the Asian children were recruited if the "workers were concerned that they may have a high risk of iron deficiency anaemia".⁴ A small number of studies have found a higher prevalence of anaemia in Aboriginal children in rural areas.⁵⁻⁷ As each of the above surveys was conducted in a single location, their generalisability to the wider Australian pre-school-aged population is uncertain.

None of these studies examined risk factors for iron deficiency anaemia owing to its low prevalence. Instead, the risk factors for iron depletion, which has a higher prevalence, were examined. In the general Sydney sample,

there were significant associations with age, non-use of supplements and eating meat less than four times per week.² An earlier case-control study from the same area found associations with low intake of haeme iron and high intake of cow milk.⁸ In the Arabic sample, the risk factors were preterm delivery, mother having migrated to Australia within the previous eight years and high intake of cow's milk.³ In the Adelaide group, the risk factors were short duration of breastfeeding, early introduction of cow's milk and high intake of cow's milk.⁴ Thus there is some consistency, but also disagreement regarding the risk factors for iron depletion in these studies. Some of the inconsistency may be related to the differing definitions of iron depletion that were used.

To date there has been no national survey of anaemia or iron status in young Australian children. In 1995, the Australian Institute of Health and Welfare conducted a nationally representative survey of Australian children aged 1-4 years inclusive to examine lead exposure.⁹ Haematocrit levels were measured as part of this survey so that statistical analysis could be carried out on both corrected and uncorrected lead levels to maximise opportunities for comparison with other studies.⁹ Anaemia can be defined

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Accepted 23 July 2004

Table 1. Demographic characteristics of the survey population with usable samples compared to the 1996 census population aged 1-4 years (unweighted results).

Characteristic	1996 Census		1995 National Lead Survey in Children			
		%	Boys		Girls	
			N	%	N	%
Age (years)	1	24.9	160	22.9	126	18.8
	2	25.0	172	24.6	204	30.4
	3	25.1	178	25.5	197	29.3
	4	25.0	189	27.0	145	21.6
	Total	100	702	100	672	100
Residence	New South Wales	33.8	196	28.0	181	26.9
	Victoria	24.4	161	23.0	152	22.6
	Queensland	18.7	122	17.5	117	17.4
	South Australia	7.5	53	7.6	67	10.0
	Western Australia	9.7	65	9.3	71	10.6
	Tasmania	2.6	60	8.6	47	7.0
	Northern Territory	1.4	12	1.7	8	1.2
	Australian Capital Territory	1.7	30	4.3	29	4.3
Identification [@]	Non-Indigenous	97.8	630	90.1	628	93.5
	Indigenous	2.2	34	4.9	12	1.8
	Missing	0	35	5.0	32	4.8

[@] based on children aged 0-4 in the 1996 Census

using haematocrit levels.^{1,10} Therefore, we took advantage of these data to describe the prevalence of anaemia, the average haematocrit level and the factors which influence haematocrit in a representative sample of Australian children.

Materials and methods

The 1995 National Survey of Lead in Children (NSLIC) is described elsewhere.⁹ Briefly, sampling frames were constructed by the Australian Bureau of Statistics for non-remote and remote areas separately based on the census collector districts so that all children in Australia aged 1-4 years would have an equal probability of being sampled. In February and March 1995, 175 trained interviewers visited households in the selected areas. The initial interview included an extensive questionnaire about factors which might affect lead levels. At a second visit, a blood sample was drawn from all children in the target age range (1-4 years inclusive) for whom parental consent was given. Haematocrit was measured by inductively coupled plasma mass spectrometry at Royal North Shore Hospital, Sydney. In the context of the current analysis, it is worth noting that the children were not selected because they might have high lead levels.

The NSLIC dataset contains many variables relating to demographic, environmental, occupational, residential history and household characteristics collected by questionnaires available in English and seven other languages. To avoid finding spurious associations resulting from performing multiple tests, a subset of characteristics was selected on *a priori* grounds. The socio-demographic variables were: child age, sex, jurisdiction of residence, Indigenous identification, highest levels of education and income in the household, mother's country of birth, presence of a smoker in the household and lead

level in the child. Maternal country of birth was classified as Australia, an English-speaking country (New Zealand, North America and the British Isles) or a non-English-speaking country.

The only nutrition-related questions asked were whether the child ate "beef, lamb and/or pork", whether the child ate "chicken and/or fish", whether the child took vitamin or mineral supplements and whether the house had a vegetable garden.

We excluded data for all blood samples that had any degree of haemolysis or contained clots on arrival at the laboratory. Anaemia was defined in two ways: the World Health Organisation (WHO) definition is haematocrit <33% for children aged 6-59 months¹; the United States (US) definition is haematocrit \leq 32.9% for children aged <2 years and \leq 33% for children aged 2-<5 years.¹⁰

Haematocrit was normally distributed and so its predictors were examined using multiple linear regression. All variables, including age, were entered as indicator variables, overall 2-sided F-tests were performed and the distribution of the residuals from the final model was checked. However the prevalence of anaemia was so low that only classification within demographic categories was done. As indicated below, the age and jurisdiction of residence distribution did not quite match the 1996 Census,¹¹ and so *post-hoc* sampling weights were calculated to correct this. Analyses were done using Stata 7 (StataCorp, College Station, TX) and allowed for sample weights and clustering within households. Ethical clearance for the NSLIC was given by the Australian Institute of Health and Welfare Ethics Committee and for the current analysis by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research.

Table 2. Proportion of children who are anaemic according to two criteria by demographic characteristics, weighted to the age- and jurisdiction population distribution in the 1996 Census and corrected for clustering within households

Characteristic	WHO criteria		US criteria*		
	% anaemic	95% CI	% anaemic	95% CI	
Total	2.0	1.3 – 3.1	3.3	2.4 – 4.5	
Sex	Boys	2.5	1.4 – 4.5	4.0	2.6 – 6.0
	Girls	1.6	0.8 – 2.9	2.6	1.6 – 4.1
Age (years)	1	1.8	0.7 – 4.3	1.8	0.7 – 4.3
	2	2.5	1.3 – 4.7	4.9	3.1 – 7.6
	3	1.1	0.4 – 2.7	3.1	1.7 – 5.4
	4	2.8	1.4 – 5.4	3.5	1.9 – 6.2

* US criterion is the 5th centile of the haematocrit distribution in children with no hemoglobinopathies or iron deficiency

Results

It is estimated that 4112 children aged 1-4 years lived in the selected areas and 3542 were located.⁹ Blood samples were taken from 1575 children but only 1371 had useable results. These children came from 1106 households; 947, 193, 10 and two households had one, two, three and four children respectively participating in the survey. Of the non-usable samples, 52 contained clots, 133 were partly or fully haemolysed and 19 samples did not have haematocrit determined. Overall, the age of the children with useable results was similar to the 1996 Census distribution¹¹ but with a small deficit of 1-year old children (Table 1). The regional distribution was also similar, though with a small deficit of children from New South Wales.¹¹ Probably owing to the small numbers, the distribution of the 46 Aboriginal children did not reflect the national distribution because only 17% lived in NSW, 10% in Qld, 32% in WA and 15% in the NT compared to the national proportions of 30%, 28%, 14% and 13% respectively.¹²

The prevalence of anaemia using the WHO criteria was 2.0% (95% CI: 1.3-3.1%) overall (Table 2). Using the US criteria, the classification of 18 children changed the overall prevalence of anaemia was 3.3% (95% CI: 2.4-4.5%). The mean haematocrit level was 38.8% and this varied among the jurisdictions (Table 3). In addition, age, child's lead level and use of supplements and presence of a vegetable garden, had *P* values near or below 0.05 in the univariate analysis. In the multivariate model, only jurisdiction of residence, age and use of supplements were significant predictors of haematocrit but together they explained only 4% of the variation in haematocrit levels in the population (Table 4). This result did not change if the two territories with small populations were excluded from the analysis.

Discussion

This appears to be the first description of the prevalence of anaemia from a national Australian survey of children aged 1-4 years. The prevalence is very low. Including all the haemolysed and clotted samples in the analysis made little difference to the mean haematocrit (for all 783 boys, mean haematocrit was 38.8% and for all 772 girls, the

mean was 38.9%). Therefore we do not believe that excluding these samples has biased our findings. Our results cannot be compared directly to previous Australian studies because the definition of anaemia is based on haematocrit rather than haemoglobin and all anaemias are included in the current analysis. Although haematocrit is used for anaemia screening in other countries, documents that publish cutoff values for both characteristics do not describe how the haematocrit-based prevalence of anaemia relates to the haemoglobin-based prevalence of anaemia.^{1,10} Graciter *et al.*,¹³ used lower cutoffs of 31% haematocrit and 10g/dL haemoglobin to define anaemia to examine this question for children aged 12-23 months seen in a number of surveys. They found that the haematocrit-based definition generally yielded a lower prevalence of anaemia than the haemoglobin-based definition. However the difference was small when the prevalence was low, e.g 3.3% anaemic by haemoglobin and 3.0% anaemic by haematocrit.¹³ Therefore our haematocrit-based prevalence of 2.0-3.3% may be a close estimate of the unknown prevalence of anaemia based on haemoglobin levels.

We could not exclude hemoglobinopathies or non-iron deficiency anaemia and so, theoretically, our study should have yielded a higher prevalence than previous Australian studies^{2,4} but we found a lower prevalence. The US haematocrit cut-offs are based on the 5th centile of the haematocrit distribution from their Third National Health and Nutrition Examination Survey calculated after excluding "persons who had a high likelihood of iron deficiency".¹⁰ Hence the expected prevalence using the US definition is 5% provided that persons who are likely to be iron deficient are excluded from the analysis. We conclude that the prevalence of anaemia is lower in Australia than the US because, firstly, the confidence interval around the prevalence calculated using the US definition excluded the value of 5% and secondly, because we were unable to exclude children with a high likelihood of iron deficiency. The WHO haematocrit cut-off is based on a conversion factor from haemoglobin levels, but the basis of the haemoglobin level chosen is unclear¹ and so the expected prevalence is unclear.

Table 3. Mean haematocrit levels according to demographic and lifestyle characteristics, weighted to the age- and jurisdiction population distribution in the 1996 Census and corrected for clustering within households

Characteristic		<i>N</i>	Mean	Haematocrit 95% CI	<i>P</i>
Total		1371	38.8	38.6-39.1	-
Sex	Boys	699	38.7	38.4-39.1	0.4
	Girls	672	38.9	38.6-39.2	
Age (years)	1	286	38.5	38.0-39.0	0.02
	2	376	38.8	38.4-39.2	
	3	375	38.7	38.3-39.0	
	4	334	39.4	38.9-39.9	
Residence	New South Wales	377	38.6	38.1-39.1	<0.0001
	Victoria	313	39.3	38.9-39.7	
	Queensland	239	38.2	37.6-38.8	
	South Australia	120	38.3	37.6-38.9	
	Western Australia	136	39.6	38.6-40.6	
	Tasmania	107	39.8	38.2-41.4	
	Northern Territory	20	38.1	36.8-39.3	
	ACT	59	41.0	39.8-42.2	
Identification	Non-Indigenous	1260	38.8	38.6-39.1	0.8
	Aboriginal	46	39.1	37.6-40.6	
	Missing	68	38.6	37.8-39.3	
Income	<\$20,000	345	38.7	38.2-39.2	0.7
	\$20-30,000	305	38.9	38.4-39.4	
	\$30-40,000	308	39.0	38.6-39.4	
	>\$40,000	338	38.9	38.5-39.2	
	Missing	75	38.4	37.4-39.5	
Education	Bachelor or higher	310	38.8	38.4-39.2	0.8
	Trade	371	38.9	38.4-39.3	
	Other	290	39.0	38.5-39.5	
	None/missing	400	38.7	38.3-39.1	
Marital status	Married/de facto	1138	38.9	38.6-39.1	0.8
	Single parent	145	38.8	38.0-39.6	
	Other	88	38.6	37.8-39.4	
Maternal birth	Australian born	1045	38.8	38.5-39.1	0.9
	English-speaking countries	132	38.8	38.0-39.6	
	Non-English-speaking	147	38.9	38.4-39.4	
	Missing/not in household	47	39.0	37.9-40.0	
Smoker present in household	no	948	38.9	38.6-39.2	0.5
	yes	422	38.7	38.3-39.1	
Child's lead level	≤0.49 µmol/L	1275	38.9	38.6-39.2	0.09
	>0.49 µmol/L	96	38.3	37.5-39.0	
Takes vitamin/mineral supplements	no	1179	38.7	38.5-39.0	0.02
	yes	188	39.4	38.8-39.9	
Eats beef, lamb, pork	no	88	38.5	37.6-39.4	0.4
	yes	1282	38.9	38.6-39.1	
Eats fish, chicken	no	36	39.1	37.9-40.4	0.6
	yes	1335	38.8	38.6-39.1	
Vegetable garden	no	910	38.7	38.4-39.0	0.06
	yes	461	39.1	38.8-39.5	

Intra-person variation affects biochemical parameters and this has not been corrected for either in the references, our work or previous Australian reports. This means that a number of children who had anaemia or iron depletion on one occasion would not have this on a second occasion, even in the absence of any treatment, and is due to a phenomenon called regression to the mean.¹⁴ Looker *et al.*,

report that an initial 10% prevalence of impaired iron status (based on mean corpuscular volume, transferrin saturation and erythrocyte proto-porphyrin) dropped to 4% when corrected for within-person variability.¹⁵ Therefore population-based surveys using a single measurement occasion for anaemia, iron depletion or deficiency overestimate the true prevalence of these conditions.

Table 4. Final model for predictors of haematocrit levels, weighted to the age- and jurisdiction population distribution in the 1996 Census and corrected for clustering within households

Variable	Group	Haematocrit (%)	95% CI	P
Constant	-	38.2	37.7; 38.8	<0.0001
Age#	2 years old	0.3	-0.3; 0.8	0.02
	3 years old	0.1	-0.4; 0.7	
	4 years old	0.8	0.2; 1.5	
Jurisdiction#	Victoria	0.6	-0.04; 1.3	<0.0001
	Queensland	-0.5	-1.3; 0.3	
	South Australia	-0.4	-1.1; 0.4	
	Western Australia	1.0	-0.1; 2.1	
	Tasmania	1.2	-0.5; 2.9	
	Northern Territory	-0.5	-2.0; 1.0	
	Australian Capital Territory	2.4	1.2; 3.6	
Vitamin & mineral supplements	Takes v does not take	0.6	0.07; 1.1	0.03

referent group for age is 1 year old, for jurisdiction is New South Wales; overall r^2 for model: 0.04

The single Torres Strait Islander child in the survey did not have a usable sample. There was no difference in haematocrit levels between Aboriginal and non-Indigenous children. Earlier studies reporting high levels of anaemia in Aboriginal children have been done in rural and remote areas.⁵⁻⁷ However most Aboriginal people live in urban areas and this was reflected in the NSLIC (J Donovan, personal communication). Although the sample size is small, our findings results suggest that results from remote areas should not be extrapolated to the urban Aboriginal population.

We found that haematocrit levels were the same regardless of maternal birthplace although the numbers were too small to allow examination in great detail. Unlike some other surveys^{2,8} we did not find that red meat intake was related to anaemia. This may be due to the crudeness of the questions that were asked. However, trials of dietary intervention have had inconsistent results^{16,17} and so this finding may also indicate that a single food item is not pre-eminent for preventing anaemia. It was not possible to examine a number of associations described previously. The National Health Survey and the National Nutrition Survey were also in the field in 1995 and ascertained duration of breastfeeding, age of introduction of cow milk, years since immigration, and collected a 24-hour dietary recall from those aged 2 years and older.¹⁸ It is unfortunate that, although all three surveys collected data in the same year, there was no relationship between them that would have permitted better use of the biochemical data. One interesting relationship would have been a comparison of iron status and dietary intakes. Population intakes should be compared to the estimated average requirement (EAR), not the recommended dietary intake.^{19,20} As the Australian dietary reference data do not describe the EAR, we used the US value of 3.4mg/day for this age group.¹⁹ After correction for within-person variation in dietary intake, the 10th centile of iron intake in Australian children aged

2-3 years was 5.3 mg/day,²¹ indicating that less than 10% of this age group have inadequate iron intakes. This is consistent with the low prevalence of anaemia in the NSLIC.

In conclusion, the prevalence of anaemia is very low, 2-3.3%, in children aged 1-4 years in Australia. Although mean haematocrit levels increased with age and supplement use and varied among the jurisdictions, these factors explain little of the variation in haematocrit within the population. To examine risks associated with dietary intake and other factors, future national surveys would need to examine iron status, rather than anaemia, to exclude haemoglobinopathies and inflammatory diseases such as respiratory infections²² and to over-sample subgroups where the prevalence is thought to be higher. Our results justify the extra expense involved in a more detailed assessment of iron status rather than collecting only haemoglobin or haematocrit. In addition, to determine the true prevalence, agreement is needed on the definition of iron deficiency and depletion and, ideally, a sub-sample should be re-tested to reduce the effects of intra-individual variability on the prevalence results.

References

1. United Nations Children's Fund, United Nations University, World Health Organisation. Iron deficiency anaemia. Assessment, prevention and control. A guide for programme managers. World Health Organisation, 2001.
2. 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.
3. Karr MA, Mira M, Alperstein G, Labib S, Webster BH, Lammi AT, Beal P. Iron deficiency in Australian-born children of Arabic background in central Sydney. *Med J Aust* 2001; 174:165-68
4. 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.

5. Harris MF, Cameron B, Florin S. Iron deficiency in Bourke children. *Aust Paediatr* 1988;24: 45-7.
6. Hopkins RM, Gracey MS, Hobbs RP, Spargo RM, Yates M, Thompson RCA. The prevalence of hookworm infection, iron deficiency and anaemia in an Aboriginal community in north-west Australia. *Med J Aust* 1997; 66: 41-44
7. Fraser J. Evaluation of a child health program to prevent and treat anaemia in Arnhemland. *Aust J Rural Health* 1996;4:11-7.
8. Mira M, Aperstein G, Karr M, Ranmuthugala G, Causer J, Niec A, Lilburne A. Haem iron intake in 12-36 month old children depleted in iron: case-control study. *Brit Med J* 1996; 312: 881-3.
9. Donovan J. Lead in Australian children: report on the National Survey of Lead in Children. Australian Institute of Health and Welfare, Canberra 1996.
10. Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. *MMWR* 1998; 47 (No RR-3):[inclusive page numbers].
11. ABS. Population by age and sex. Australian states and territories. June 1992 to June 1997. ABS Catalogue No 3201.0, Canberra, 1997
12. ABS. Experimental projections of the Aboriginal and Torres Strait Islander population. 30 June 1996 to 30 June 2006. ABS Catalogue No 3231.0. Canberra, 1998
13. Graciter PL, Goldsby JB, Nichaman MZ. Hemoglobins and hematocrits: are they equally sensitive in detecting anemias? *Am J Clin Nutr* 1981;34: 61-64.
14. Newell D, Simpson J. Regression to the mean. *Med J Aust* 1990; 153: 166-8.
15. Looker AC, Sempos CT, Liu K, Johnson CL, Gunter EW. Within-person variance in biochemical indicators of iron status: effects on prevalence estimates. *Am J Clin Nutr* 1990; 52: 541-7.
16. Engelmann MD, Sandstrom B, Michaelsen KF. Meat intake and iron status in later infancy: an intervention study. *J Pediatr Gastroenterol Nutr* 1998; 26: 26-33.
17. Makrides M, Leeson R, Gibson RA, Simmer K. A randomized controlled clinical trial of increased dietary iron in breast-fed intakes. *J Pediatr* 1998; 133: 559-62.
18. ABS and HEALTH. National Nutrition Survey. Nutrient intakes and physical measurements Australia 1995. ABS Cat No 4805.0, Canberra 1998.
19. Food and Nutrition Board, Institute of Medicine. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington, 2002. [http:// books.nap.edu/books/0309072794/html/index.html](http://books.nap.edu/books/0309072794/html/index.html)
20. Beaton G H. Recommended dietary intakes: individuals and populations. From Shils M, Olson JA, Shike M, Ross AC (eds). *Modern nutrition in health and disease*. 9th Edn. Williams and Wilkins, Baltimore, 1999.
21. Australian Bureau Statistics. National Nutrition Survey Nutrient Intakes and Physical Measurements Australia 1995. Cat No 4805.0. ABS, Canberra 1998.
22. Yip R, Dallman PR. The roles of inflammation and iron deficiency as caused of anemia. *Am J Clin Nutr* 1998; 48: 1295-1300.