Original Article

Iron status among Australian adults: findings of a population based study in Queensland, Australia

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Objectives: To describe the concentrations of serum ferritin among Australian adults by age and sex. Further, the relationships of various social, lifestyle and health factors with serum ferritin concentrations were explored. Design: A total of 1634 adults aged ≥25 years from six randomly selected urban centres in Queensland, Australia participated in the study that was conducted between October and December 2000. Results: Prevalence of depleted iron stores, based on low serum ferritin concentration, was 10.6% among females aged <50 years, 2.8% among females aged ≥50 years and virtually nil among males. In contrast, 16% of the males and 20% of the females aged ≥50 years had elevated serum ferritin concentrations. Significantly higher serum ferritin concentrations were found among females of both age groups who consumed alcohol at a rate of >60 drinks/month, and females aged <50 years who were obese. Lower serum ferritin concentrations were found only among females aged <50 years, with higher education attainment. In multivariable analysis, only the association between higher serum ferritin and obesity was consistent across age-sex groups and statistically significant. Conclusion: Iron deficiency may be a problem among Australian females of reproductive age. Further research is needed to identify the determinants of low iron concentrations in younger females and elevated concentrations of serum ferritin in males and older females in order to develop preventive measures.

Key Words: Serum ferritin, iron status, iron deficiency, elevated serum ferritin, anaemia, prevalence, cross-sectional survey

INTRODUCTION
Although iron deficiency is a major public health problem in developing countries, it can also be a significant problem in industrialized countries.[1] The consequences of iron deficiency in adults include poor haematopoiesis that result in anaemia, decreased work capacity and impaired immune function.[2-4] Further, maternal iron deficiency anaemia is associated with a higher incidence of low birth weight, premature delivery and increased maternal mortality.[5-6] On the other hand, elevated iron stores, as assessed by serum ferritin, are associated with increased risk of diabetes mellitus, atherosclerosis and cancer.[7-9]

Only a few studies in the late 1980s have reported the iron status of the Australian population. For example, the National Heart Foundation of Australia conducted a risk factor prevalence survey in 1989 and reported that the prevalence of iron deficiency among Australian women aged 25-69 years was 8%.[10] Another study published in 1990, reported the serum ferritin concentrations of employees of two large Australian organizations.[11] The prevalence of iron deficiency was found to be 8.9%. This workplace study also examined the association between serum ferritin and some lifestyle factors, such as alcohol intake and exercise.[11] The Australian Longitudinal Study on Women's Health provided more recent data on iron deficiency, but this was based on self-reported informa-

tion, and was thus considered to be less reliable.[12] Therefore considering the role of iron in human health, it is important to determine the current prevalence of iron deficiency and as well as the prevalence of elevated iron stores for the Australian population.

The purpose of this study was to describe the population distribution of serum ferritin (SF) concentrations among adults in Queensland, Australia. We also explored the relationships of various social, lifestyle and health-related factors with SF levels.

SUBJECTS AND METHODS
Subjects
The study was conducted in Queensland, Australia between October and December 2000 as part of a national study, the Australian Diabetes, Obesity and Lifestyle Study (AusDiab), to determine the prevalence of diabetes and associated cardiovascular risk factors among adults

aged 25 years and over.[13] Six urban sites were randomly selected from census collector districts (CDs) in Queensland. The CDs were selected without replacement and with probability proportional to size.Non-institutionalised adults aged $\geq$25 years who were residing in private dwellings were included in the survey if they had lived at that address for a minimum of 6 months prior to the survey. Persons with physical or intellectual disabilities that precluded participation in the study were not included.

Trained interviewers conducted house-to-house interviews, and eligible participants were invited to participate in the study. Details of the sampling framework and overall study design have been published elsewhere.[14] A total of 1634 adults (688 males and 946 females) in Queensland completed the physical examination. Although the overall response rate in the study was low (approximately 50% of those invited and 30% of those estimated to be eligible), internal validity and quality control of the data collection were of high quality.[14]

All respondents gave informed consent to participate in the survey upon arrival at the testing site. The study was approved by the International Diabetes Institute and The University of Queensland ethics committees.

Methods
Study participants arrived for the study examination having fasted for at least 12 hours. After the fasting blood sample was drawn, participants consumed a 75 gram glucose drink. Two hours after consuming the glucose load, blood was drawn to determine glucose tolerance and additional blood was collected using a vacutainer system to measure serum ferritin levels.

Appropriate aliquots of blood were drawn into plain tubes for SF. The serum tubes were then centrifuged at 3000 rpm for 10 minutes. Following this, an aliquot of the serum was placed into an ACS tube for ferritin testing, and then frozen and packed in dry ice and shipped to the Queensland Health Pathology Services laboratory at Princess Alexandra Hospital in Brisbane. SF was measured using the Bayer Advia Centuar automated immunoassay system (Bayer, Melbourne, Australia). Chemiluminescent labels are used in this immunoassay system.

Demographic and lifestyle data were collected using standardised questionnaires. Dietary intake of iron was calculated from an eighty-item food frequency questionnaire.[15]

Statistical analysis
The frequency distributions of selected variables were examined. Because SF concentration was positively skewed, the natural logarithmic transformation was used for examining the difference between groups. For presentation, the SF results were back transformed and presented as geometric means and exponentiated values of the 95% confidence intervals of the transformed values. The distributions of SF by age group and sex are shown using box plots which indicate the median, quartiles and more extreme values.

The prevalence of various degrees of iron deficiency was determined by the following criteria: for both males and females, SF <12 μg/L was indicated as depleted iron stores and SF between 12.0 μg/L and <20 μg/L was con-

sidered as marginally iron deficiency.[16] Elevated SF was defined for males as SF >400 μg/L; for females aged <50 years, SF >200 μg/L; and for females aged >50 years, SF >300 μg/L.[16] The differences in prevalence between groups were compared using chi-squared tests.

Individuals with a genetic predisposition, such as hereditary haemochromatosis, accumulate more iron than normal and this leads to high iron storage or iron overload.[17] Elevated SF may also be the result of some chronic condition, infection or inflammation.[18-21] Thus subjects with elevated SF (as described above) were excluded from all statistical analyses except for determination of prevalence of iron deficiency for which the total sample was included.

To examine the relationship between various demographic and lifestyle factors and SF concentrations, each variable was grouped on the basis of a priori logical categories as described elsewhere.[22] The diagnostic criteria for the presence of diabetes, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) were based on values for venous plasma glucose concentration (fasting and 2hour measurements) outlined in the World Health Organization (WHO) report on the Diagnosis and Classification of Diabetes Mellitus.[23] The SF concentrations were compared separately for males, and females aged <50 years (considered to be of reproductive age) and females aged $\geq$50 years (considered post-menopause). The differences in mean values between groups were compared using analysis of variance (ANOVA). The effects on SF levels of all the demographic, lifestyle and health variables considered together were analysed using multiple linear regression. Data were analysed using the Stata statistical software (version 8; Stata Corp, College Stations, TX).[24]

RESULTS
Of the 1634 participants, there were slightly more females (58%) than males (42%). Approximately half (47%) of the females were below 50 years of age. Details of the characteristics of males, females aged <50 years and females aged $\geq$50 years are presented in Table 1.
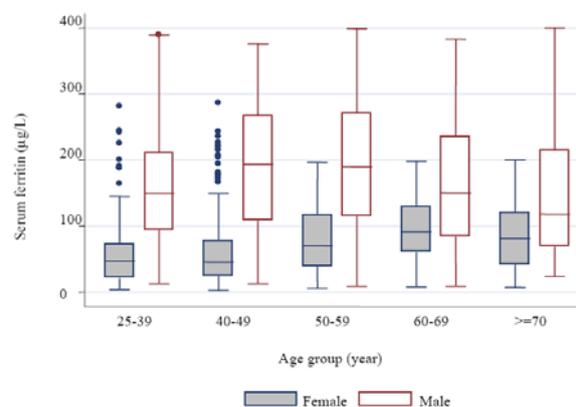

Figure 1. Box plot for serum ferritin (SF) by sex and age for the Queensland AusDiab study, 2000. Participants with elevated SF (for males as SF >400 μg/L; for females aged <50 years as SF >200 μg/L and for females aged $\geq$50 years as SF >300 μg/L.) were excluded. Medians are shown as horizontal lines; boxes show inter-quartile ranges; dots show outlying values.

Table 1. Characteristics of the study participants in the Queensland AusDiab study, 2000: numbers and column percentages, except for the first row.

Variable	Male n[*]	Male %	Female <50 y n	Female <50 y %	Female ≥50 y n	Female ≥50 y %
Demography						
Totals (row %)	688	42.1	444	27.2	502	30.7
Age group						
25-39	144	21.3	196	45.0	-	-
40-49	159	23.6	239	55.0	-	-
50-59	152	22.6	-	-	197	40.4
60-69	132	19.6	-	-	152	31.1
≥70	87	12.9	-	-	139	28.5
Total	674	100.0	435	100.0	488	100.0
Socio-economic						
Educational level						
Secondary school or less	184	27.0	167	37.8	288	57.6
Trade certificate, bachelor's degree	436	64.0	236	53.8	192	38.4
Post graduate	61	9.0	37	8.4	20	4.0
Total	681	100.0	440	100.0	500	100.0
Anthropometry						
Body Mass Index (kg/m^2)						
Normal (<25)	219	32.1	219	51.3	197	39.5
Overweight (25-30)	315	46.1	101	23.6	170	34.1
Obese (>30)	149	21.8	107	25.1	132	26.4
Total	683	100.0	427	100.0	499	100.0
Lifestyle-related behaviors						
Smoking status						
Never	337	50.1	254	58.5	332	67.1
Former	235	34.9	107	24.7	117	23.6
Current	101	15.0	73	16.8	46	9.3
Total	673	100.0	434	100.0	495	100.0
Alcohol intake						
None	119	17.4	102	23.1	144	28.8
≤ 60 drinks/month	432	63.2	315	71.4	332	66.4
> 60 drinks /month	133	19.4	24	5.5	24	4.8
Total	684	100.0	441	100.0	500	100.0
Physical activity						
Sedentary	118	17.4	101	23.0	101	20.3
Insufficient	243	35.7	145	33.0	158	31.8
Sufficient	319	46.9	194	44.0	238	47.9
Total	680	100.0	440	100.0	497	100.0
Health-related						
Serum Cholesterol						
<5.5 mmol/L	318	46.3	194	43.7	232	46.4
5.5-<6.5 mmol/L	225	33.8	167	37.6	160	32.0
≥ 6.5 mmol/L	144	21.0	83	18.7	108	21.6
Total	687	100.0	444	100.0	500	100.0
Diabetes						
Normal	478	70.8	320	73.5	345	70.7
IFG/IGT**	148	21.9	76	17.5	96	19.7
Diabetes	49	7.3	39	9.0	47	9.6
Total	675	100.0	435	100.0	488	100.0
Diet related						
Dietary iron intake						
<12 mg/day	276	43.1	172	41.6	207	44.4
12-16 mg/day	174	27.1	127	30.8	118	25.3
>16 mg/day	191	29.8	114	27.6	141	30.3
Total	641	100.0	413	100.0	466	100.0
Use of Vitamins						
Yes	161	25.4	130	31.5	195	41.8
No	474	74.6	282	68.5	272	58.2
Total	635	100.0	412	100.0	467	100.0

[*] Due to missing values, total n is not the same for all variables. ** Impaired fasting glucose/Impaired glucose tolerance

Table 2. Haemoglobin (g/L) and serum ferritin concentration (μg/L) of Australian adults aged 25 and over in the Queensland AusDiab study, 2000.

Subgroup	N	Mean ± SD	Median	25-75 percentile	p-value*
Haemoglobin					
Males	688	154 ± 9.5			
Females <50 years	443	136 ± 9.9			<0.001
Females ≥50 years	502	140 ± 10.0			
Serum Ferritin§					
Males	576		162	93.5 - 248	
Females <50 years	431		46.0	22.0 - 75.0	<0.001**
Females ≥50 years	400		80.5	44.5 - 120	

§Subjects with elevated serum ferritin levels were excluded from the analysis. These were defined males with SF>400 μg/L, and females aged <50 year with SF>200 μg/L and females aged >50 year with SF >300 μg/L.
*Based on ANOVA. **Using logarithmically transformed data for serum ferritin.

Table 3. Prevalence of iron deficiency and elevated serum ferritin concentrations among adults aged 25 years and over, in the Queensland AusDiab study, 2000.

Iron status		Prevalence (%)		p-values*
	Male	Female <50 years	Female ≥ 50 years	
Depleted iron store[†]	0.3	10.6	2.8	<0.001
Marginal iron deficiency[‡]	1.2	9.7	2.2	<0.001
Elevated serum ferritin[§]	16.3	2.9	20.3	<0.001

[†] Serum ferritin (SF) <12 μg/L was considered to indicate depleted iron stores for males and females.
[‡] SF between 12.0 μg/L and <20.0 μg/L was considered to indicate marginal iron deficiency for males and females.
[§] Elevated serum ferritin concentration was defined for males as SF >400 μg/L; for females aged <50 years as SF >200 μg/L and for females aged ≥50 years as SF >300 μg/L.[17]
* p-values from chi-squared tests.

Mean haemoglobin and SF concentrations were significantly lower among females than males ($p< 0.001$, Table 2). Further, females aged <50 years had significantly lower haemoglobin and SF than females aged ≥50 years ($p<0.001$).

Concentrations of SF in males were significantly higher than females for all age groups ($p<0.001$, Figure 1). For males, SF levels increased with age and reached maximum levels in the 40-59 year age groups, after which they declined. For females between 25 and 49 years of age, there was no appreciable difference in SF concentration; however SF levels increased after the age of 50 years.

Among females aged <50 years, 10.6% were found to have depleted iron stores and another 10% appeared to have marginal iron deficiency (Table 3). The prevalence of depleted iron stores or marginal iron deficiency was uncommon among males and females aged ≥50 years. About 16% of the males and 20% of the females aged ≥50 years had elevated SF levels. The prevalence of anaemia, determined by haemoglobin (Hb), was low in all subgroups: 0.6% in males (Hb <130 g/L), 3.8% in females aged <50 years and 2.0% in females aged ≥50 years (Hb<120 g/L).

Table 4 shows the bivariate relationships of selected socio-economic, lifestyle, health and diet related factors with SF concentrations; stratified by sex, and in females, by age. Among females aged <50 years, those with post graduate education had significantly lower SF concentrations than other education groups. In females aged <50 years, those who were obese had statistically significantly

higher SF levels than who were overweight or of normal weight. Males and females aged <50 years who had alcohol intakes of >60 drinks/month had significantly higher SF concentrations than those who consumed less or no (none) alcohol, and in females aged ≥ 50 years, those who consumed no alcohol (none) had significantly lower SF compare to the other groups. Never smokers had lower SF concentrations than current or former smokers but the differences were not statistically significant. No consistent and statistically significant associations were found between SF and physical activity, serum cholesterol, diabetes, dietary intake of iron and use of vitamin supplements in any of the sub-groups. When all the variables were considered simultaneously using multiple regression, the only consistent finding across all age-sex groups was that significantly higher SF was associated with obesity.

DISCUSSION
The present study reports, for the first time in many years, the distributions of SF concentrations on a large community sample of Australian adults. As would be expected, there were marked differences in concentrations of SF between males and females. SF in males reached maximum levels in the 40-59 year age groups and then declined. This decline with age could be due to either a decline in iron intake or a decrease in iron absorption. While SF concentrations were higher in females in the post-menopausal age groups (50 years and over) than in pre-menopausal age groups, the difference was considerably less than that between males and females. Leggett et al

Table 4. Serum ferritin concentration (μg/L) (geometric mean and 95% confidence interval) by selected variables for adults 25 years and over in the Queensland AusDiab study, 2000.

Variable	Male Mean	Male 95% CI[*]	Female <50 y Mean	Female <50 y 95% CI[*]	Female ≥50 y Mean	Female ≥50 y 95% CI[*]
Socio-economic						
Educational level		p=0.28		p=0.004		p=0.89
Secondary school or less	151	134-170	47.0	40.9-54.0	68.7	62.0-75.7
Trade certificate, bachelor's degree	140	130-150	40.0	35.8-45.1	70.8	63.6-79.1
Post graduate	128	101-161	27.4	20.0-37.4	71.5	50.5-101
Anthropometry						
Body Mass Index (kg/m^2)		p=0.15		p=0.03		p=0.18
Normal (<25)	144	118-144	38.1	33.8-43.1	64.7	57.8-73.3
Overweight (25-30)	147	134-160	42.1	35.8-49.7	75.9	68.0-85.0
Obese (>30)	150	130-172	50.4	42.1-60.3	69.4	59.7-78.8
Lifestyle-related behaviours						
Smoking status		p=0.44		p=0.06		p=0.83
Never	137	126-149	37.7	33.5-42.5	68.7	62.7-75.1
Former	146	131-163	45.6	38.8-53.4	70.8	61.4-81.9
Current	151	131-173	47.9	39.3-58.3	73.7	58.3-92.6
Alcohol intake		p=0.06		p=0.026		p=0.023
None	128	110-147	43.6	36.8-51.7	59.7	51.9-68.5
≤ 60 drinks/month	141	130-152	39.2	35.4-43.4	74.3	68.3-80.7
> 60 drinks /month	160	143-180	65.4	43.4-96.5	74.0	43.3-112.8
Physical activity		p=0.58		p=0.54		p=0.31
Sedentary	133	113-155	42.5	35.3-51.1	73.7	63.9-85.0
Insufficient	141	128-157	38.5	33.0-45.2	64.1	56.5-72.6
Sufficient	144	133-157	42.9	37.8-48.7	71.5	64.0-79.8
Health-related						
Serum Cholesterol		p=0.79		p=0.48		p=0.06
<5.5 mmol/L	138	127-152	38.9	33.3-44.2	63.4	57.3-68.4
5.5-<6.5 mmol/L	146	132-160	43.8	38.1-49.9	76.7	67.4-86.4
≥ 6.5 mmol/L	143	126-162	42.5	34.1-53.1	73.7	62.6-87.0
Diabetes		p=0.57		p=0.51		p=0.70
Normal	143	132-153	40.9	37.1-45.1	69.4	63.4-75.4
IFG/IGT**	136	120-155	38.9	30.8-48.7	69.4	58.9-81.0
Diabetes	156	125-195	47.9	34.9-65.4	76.7	59.7-99.1
Diet related						
Intake of Iron		p=0.53		p=0.12		p=0.46
<12.0 mg/day	137	124-150	45.9	39.7-53.0	65.8	58.1-74.3
12-16 mg/day	1487	132-167	38.9	33.1-45.5	74.1	64.0-85.9
≥16 mg/day	1447	129-161	37.3	31.6-44.0	68.1	59.9-77.5
Use of vitamins		p=0.08		p=0.57		p=0.33
Yes	129	113-147	39.2	33.2-46.2	67.5	60.3-75.5
No	146	137-157	41.4	37.3-46.1	72.5	65.9-79.8

[*] Confidence Interval.
ANOVA was performed based on log transformed values for each categorical variable and for each age-sex group separately.
** Impaired fasting glucose/Impaired glucose tolerance

also showed a very similar pattern of variation for SF concentrations between Australian males and females in an earlier workplace study, although median values for males and post-menopausal females were much higher than those observed in our community based study.[11] This difference may be explained by the fact that we excluded subjects with elevated SF levels while they were included in the other study. It is essential to exclude subjects with elevated levels to describe the "normal range" of a population, as elevated SF may not reflect true iron storage but could be due to inflammation, chronic conditions or haemochromatosis.[17-21] It is important to note that there are no single ideal biological measures for iron status; nevertheless plasma iron, SF, transferrin and transferrin saturation levels or a combination of some of these or all of these are frequently used as indices of biological iron

status.[25] In recent years SF has been widely used as a marker of body iron stores or iron status in nutritional surveys and clinical assessment.[26] However, many studies showed a marked elevation of SF during the acute and chronic phases of infection or haemochromatosis.[17,21] More recently the transferrin receptor (TfR) assay has been used because it was hoped that this would be more stable than ferritin.[27] Yet several studies showed an effect of infection such as malaria on plasma TfR levels, although changes occur considerably less than changes in ferritin.[28] Moreover, the test is expensive and was not routinely available for use at the time of the study. Therefore, we used SF to assess iron status.

The findings of the present study indicate that iron deficiency may be a problem among Australian females as 1 in every 5 females aged <50 years has some form iron

deficiency (either iron depleted stores or marginal iron deficiency). Earlier studies used a cut-off <10 µg/L for SF to define iron deficiency.[10-11] One study reported an overall prevalence of 8.9% among Australian females aged 17-65 years working in a bank and/or insurance corporation.[11] Another study reported an iron deficiency prevalence of approximately 8% among women 20-69 years of age.[10] Using the same cut-off of <10 µg/L for SF, we found the prevalence of iron deficiency among women <50 years of age to be 7.1%, indicating a slight decrease in the prevalence of iron deficiency among Australian females over the past decade. However, it is worth mentioning that the previous studies included a wider age range than the present study, hence it may be difficult to draw a conclusion.

We found that, one in every six males (16%) and one in every five females ≥50 years of age (20%) had elevated SF. Fleming et al conducted a study among elderly white Americans aged 69-96 years and found an overall prevalence of high iron stores of 12.9% (13.9% in males and 12.2% in females) using a SF cut-off of >300 µg/L in males and >200 µg/L in females.[29] In the present study the prevalence of elevated SF was higher than in Fleming's study even though we used a higher cut-off to define elevated SF.[29] SF is considered to be a sensitive indicator of body iron stores and thus elevated SF concentrations may reflect possible high body iron stores.[30-31] However, as mentioned earlier, as a positive acute phase protein; SF concentration increases in response to inflammation or concurrent infection.[21] One limitation in the present study was that we do not have information on subclinical infection or other inflammatory conditions. Thus there might be an overestimation of the prevalence of elevated SF concentration and an underestimation of iron deficiency. Fleming et al examined the potential effects of chronic disease on iron measures among elderly subjects of the Framingham Heart Study cohort.[29] They reported that chronic disease had little effect on population prevalence estimates of iron deficiency. Similarly, only 1% of the elevated iron stores were attributable to chronic disease.[29] Thus we consider the issue of acute phase protein to be of small practical significance for overall iron status assessment.

Our analysis found several demographic, lifestyle and health-related factors associated with SF. Females <50 years of age with higher education levels had lower SF levels compared to those with lower education levels. This result supports the findings of the US NHANES studies that showed an inverse relationship between education and SF.[32] This could be due to greater preoccupation with body weight and image by females with higher educational levels. In the present study, females <50 years of age with BMI <25 kg/m^2 had significantly lower SF than females with higher BMI, and only obesity was associated with SF in the multivariate analyses, which supports our assertion. Previous studies have also shown higher SF levels among over-weight or obese adults than those who were of normal weight.[32-33]

In the present study, we observed significantly higher SF levels in females < 50 years of aged who had an alcohol consumption level of >60 drinks/month and in drinkers compared to non-drinkers among females ≥50 years of age. While among males, SF increased with alcohol intake but the effect was not statistically significant. Other studies have reported similar findings but in men only.[11,32] Regular consumption of alcohol is responsible for the disruption of normal iron metabolism in humans, resulting in excess deposition of iron in the liver.[25] We found no significant relationship between the level of physical activity and SF concentrations, as reported in other studies.[11,34-35] Several studies have reported higher SF levels in subjects with diabetes mellitus.[29,35-36] However, it was not clear whether these higher levels were the result of excess body iron stores or a higher BMI or if they reflect inflammation.[32] In the present study, we excluded subjects with very high SF levels in order to reduce the possible effect of acute phase reactants. Although we observed higher SF levels in people in all three sub-groups who had diabetes mellitus compared to people with normal glucose tolerance or IFG/IGT, the differences between groups did not reach statistical significance. Further we found no association between serum total cholesterol and SF levels in any of the three sub-groups. A study of Danish males and females also found no association between SF and serum cholesterol.[34] In contrast, others have shown higher SF levels in subjects with higher serum cholesterol.[33] Further in all three sub-groups, SF levels were somewhat higher in current smokers compared to never-smokers and former smokers, although the differences were not statistically significant. Millman and Kirchhoff also found no association between SF and tobacco smoking in Danish females, but a negative association in Danish males.[34] Some other studies have also reported higher SF levels among smokers.[32]

We found no consistent or significant association between dietary iron intake or use of vitamin supplements and SF levels in any of the sub-groups. This was not surprising as the dietary iron intake was only a crude measure based on a food frequency questionnaire. Also we do not have information on specific vitamin or mineral content of supplements for those who reported supplement usage.

It is important to note that the results of the bivariate analysis may have been confounded by interrelated factors. When we used multiple regression, SF was found to be significantly associated only with obesity and the finding was consistent across all three age-sex groups.

In conclusion, while anaemia is uncommon in this population, iron deficiency may be a problem among females of reproductive age. Further research is needed to clearly identify the determinants of low iron levels in younger females and elevated levels of SF in males and older females and to develop preventive strategies.

AUTHOR DISCLOSURES
Faruk Ahmed, Terry Coyne, Annette Dobson and Christine McClintock, no conflicts of interest.

REFERENCES

1. World Health Organization (WHO). Iron deficiency anaemia. Assessment, prevention and control: a guide for programme managers. WHO/NHD/01.3 Geneva: WHO, 2001
2. Edgerton VR, Gardner GW, Ohira Y, Gunawardena KA, Senewiratne B. Iron deficiency anaemia and its effect on work productivity and activity patterns. Br Med J. 1979;2: 1546-1549.
3. Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. Morbidity and Mortality Weekly Report. 1998;47:1-29.
4. Beard JL. Iron biology in immune function, muscle metabolism and neuronal functioning. J Nutr. 2001;131:581S-589S.
5. Scholl TO, Hediger ML, Fisher RL, Shearer JW. Anaemia vs iron deficiency: increased risk of preterm delivery in a prospective study. Am J Clin Nutr. 1992;55:985-988.
6. Brabin BJ, Hakimi M, Pelletier D. An analysis of anaemia and pregnancy related maternal mortality. J Nutr. 2001;131: 604S-614S.
7. Fernandez Real JM, Ricart-Engel W, Arroyo E, Balanca R, Casamitjana-Abella R, Cabrero D, Fernandez-Castaner M, Soler J . Serum ferritin as component of the insulin resistence syndrome. Diabetes Care. 1998;21:62-68.
8. Corti MC, Gaziano M, Hennekens CH. Iron status and risk of cardiovascular disease. Ann Epidemiol. 1997;7:62-68.
9. Gray KF. Prospects for the prevention of free radical disease, regarding cancer and cardiovascular disease. Br Med Bull. 1993;49:679-599.
10. National Heart Foundation (NHF). Risk Factor Prevalence study: Iron Status-Draft Report, Australia. Canberra, 1991.
11. Leggett BA, Brown NN, Bryant SJ, Duplock L, Powell L, Halliday JW. Factors affecting the concentrations of ferritin in serum in a healthy Australian population. Clin Chem. 1990;36:1350-1355.
12. Research Institute for Gender and Health. Australian Longitudinal Study on Women's Health data book. New South Wales, Australia: University of Newcastle, 1996.
13. Dunstan DW, Zimmet PZ, Welborn TA, de Courten M, Cameron A, Sicree R, Dwyer T, Colagiuri S, Jolley D, Knuiman M, Atkins R, Shaw J. The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. Diabetes Care. 2002;25: 829-834.
14. Dunstan DW, Zimmet PZ, Welborn TA. Cameron A, Shaw J, de Courten M, Jolley D, McCarty D. The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)--methods and response rates. Diabetes Res Clin Pract. 2002;57:119-129.
15. Hodge A, Patterson AJ, Brown WJ, Ireland P, Giles G. The Anti Cancer Council of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in young to middle aged women in a study of iron supplementation. Aust N Z J Public Health. 2000;24:576-583.
16. McDonnell SM. Hereditary hemochromatosis-Preventing chronic effects of this under diagnosed disorder. Postgrad Med. 1997;102:83-85.
17. Barton JC. A brief history of hemochromatosis. In: Edward CQ, Barton JC, eds. Hemochromatosis: genetics, pathophysiology, diagnosis and treatment. Cambridge, United Kingdom: Cambridge University Press. 2003; pp. 3-7.
18. Tuomainen TP, Nyyssonen K, Tervahauta A, Korpela H, Lakka T, Kaplan GA , Salonen JT. Body iron stores are associated with serum insulin and blood glucose concentrations. Population study in 1013 eastern Finnish men. Diabetes Care. 1997;20:347-8.
19. Tuomainen TP, Punnonen K, Nyyssonen K, Salonen JT. Association between body iron stores and the risk of acute myocardial infarction in men. Circulation. 1998;97:1461-6.
20. Stevens RG, Jones DY, Micozzi MS, Taylor PR. Body iron stores and the risk of cancer. N Engl J Med. 1988; 319: 1047-52.
21. Witte DL. Can serum ferritin be effectively interpreted in the presence of the acute-phase response? Clin Chem. 1991; 37:484-485.
22. Coyne T, Ibiebele TI, McNaughton S, Rutishauser IHE, O'Dea K, Hodge AM, McClintock C, Findlay MG, Lee A. Evaluation of brief dietary questions to estimate vegetable and fruit consumption using serum carotenoids and red cell folate. Public Health Nutr. 2005;8:298-308.
23. World Health Organization (WHO). Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications; Part1: Diagnosis and Classification of Diabetes Mellitus. Geneva: Department of Non-communicable Disease Surveillance, WHO, 1999.
24. StataCorp. Stata Statistical Software, release 8.0. College Station, TX: Stata Corporation, 2003.
25. Cobiac L, Baghurst K. Iron status and dietary iron intakes of Australians. Food Australia. 1993;45:S1-S24.
26. Cook J. The nutritional assessment of iron status. Arch Latinoam Nutr. 1999; 49: 11S-14S.
27. Cook JD. The measurement of serum transferring receptor. Am J Med Sci. 1999;318:269-276.
28. Verhoef H, West CE, Ndeto P, Burema J, Beguin Y and Kok, F.J. Serum transferrinreceptor concentrations indicate increased erythropoiesis in Kenyan children with asymptomatic malaria. Am J Clin Nutr. 2001;74:767-775.
29. Fleming DJ, Jacques PF, Tucker KL, Massaro JM, D'Agostino RB, Wilson PWF, Wood RJ. Iron status of the free-living, elderly Framingham Heart Study cohort: an iron replete population with a high prevalence of elevated iron stores. Am J Clin Nutr. 2001;73:638-646.
30. Cook JD, Lipschitz DA, Miles LEM, Finch CA. Serum ferritin as a measure of iron stores in normal subjects. Am J Clin Nutr. 1974;27:681-7.
31. Walters GO, Miller FM, Worwood M. Serum ferritin concentration and iron stores in normal subjects. J Clin Path 1973;26:770-772.
32. Ford ES, Cogswell ME. Diabetes and serum ferritin concentration among US adults. Diabetes Care. 1999;22:1978-1983.
33. Gillum RF. Association of serum ferritin and indices of body fat distribution and obesity in Mexican American men: the third National Health and Nutrition Examination survey. Int. J Obes Relat Metab Disord. 2001;25:639-645.
34. Milman N, Kirchhoff M. Relationship between serum ferritin and risk factors for ischaemic heart disease in 2235 Danes aged 30-60 years. J Intern Med. 1999;245:423-433.
35. Sharifi F, Sazandeh SH. Serum ferritin in type 2 diabetes mellitus and its relationship with HbA1c. Acta Medica Iranica, 2004;42:142-145.
36. Kaye TB, Guay AT, Simonson DC. Non-insulin-dependent diabetes mellitus and elevated serum ferritin level. J Diabet Complications. 1993;7:246-249.

Original Article

Iron status among Australian adults: findings of a population based study in Queensland, Australia

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澳大利亞成人鐵狀況：澳洲昆士蘭族群研究的發現

目的：描述性別、年齡別澳大利亞成人血清鐵蛋白濃度。進而探討不同社經地位、生活型態和健康因子與血清鐵蛋白濃度的關係。設計：在 2000 年 10 月至 12 月間，隨機選取澳洲昆士蘭 6 個都會中心，共 1634 位年齡大於等於 25 歲的成人參與研究。結果：基於低血清鐵蛋白濃度，50 歲以下女性鐵存量用盡的盛行率為 10.6％；50 歲及以上者 2.8％，男性差不多為零。相較之下，16％50 歲及以上的男性及 20％的女性為高血清鐵蛋白濃度。兩個年齡層女性每月喝酒大於 60 杯，年齡小於 50 歲的肥胖女性，其血清鐵蛋白濃度顯著較高。低血清鐵蛋白濃度只在小於 50 歲且教育程度較高的女性中發現。在多變項分析中，跨年齡性別，只有高血清鐵蛋白和肥胖的關係一致，並達統計顯著。結論：鐵缺乏可能是澳洲育齡婦女的一個問題。為了發展預防措施，未來研究需要找出年輕女性低鐵濃度及男性和年老女性高血清鐵蛋白濃度的決定因素。

關鍵字：血清鐵蛋白、鐵狀況、鐵缺乏、高血清鐵蛋白、貧血、盛行率、橫斷性研究。