HHS Public Access

Author manuscript

Ann Hematol. Author manuscript; available in PMC 2019 May 19.

Published in final edited form as:

Ann Hematol. 2009 November ; 88(11): 1131–1136. doi:10.1007/s00277-009-0718-6.

Serum ferritin concentrations in Africans with low dietary iron

Victor M. Moyo,
Clinical Affairs, Oncology/Hematology, Ortho Biotech LLC, Bridgewater, NJ, USA

Elisha Mvundura,
Department of Obstetrics and Gynecology, Georgetown Hospital, Washington, DC, USA

Hlosukwazi Khumalo,
Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa

Innocent T. Gangaidzo,
Department of Medicine, University of Zimbabwe Medical School, Harare, Zimbabwe

Thokozile Saungweme,
Department of Medicine, University of Zimbabwe Medical School, Harare, Zimbabwe

Mehdi Nouraie,
The Department of Medicine and Center for Sickle Cell Disease, Howard University

Tracey A. Rouault,
Cell Biology and Metabolism Branch, National Institute of Child Health and Human Development, Bethesda, MD, USA

Zvenyika A. R. Gomo, and
Chemical Pathology, University of Zimbabwe Medical School, Harare, Zimbabwe

Victor R. Gordeuk
The Department of Medicine and Center for Sickle Cell Disease, Howard University

Abstract

In the setting of high dietary, several studies have provided evidence for a strong effect of both high dietary iron and an unidentified genetic locus on iron stores in Africans. To investigate whether these effects are discernible in the setting of low dietary iron, serum ferritin concentrations were measured in 194 Zimbabwean men >30 years of age and 299 postmenopausal women who consumed a non-iron-fortified diet and who did not drink iron-rich traditional beer or other alcoholic beverages. Comparisons were made with non-alcohol drinking African-Americans studied in the third National Health and Nutritional Examination Survey (NHANES III) who consume an iron-fortified diet. As stratified by age and sex, serum ferritin concentrations were significantly lower in the 493 Zimbabweans studied than in 1,380 comparable African-Americans ($P < 0.0005$). Nevertheless, nine Zimbabwean subjects (1.8% of all cases) had modestly elevated serum ferritin concentrations not associated with evidence of inflammation or hepatic dysfunction. These data suggest that mild serum ferritin concentration elevations may occur among

V. R. Gordeuk, The Department of Medicine and Center for Sickle Cell Disease, Howard University, 2041 Georgia Ave. NW, Washington, DC 20060, USA, vgordeuk@howard.edu.

Zimbabweans not exposed to high dietary iron and that iron fortification of the diet may have substantial effects on serum ferritin concentration.

Keywords

African iron overload; Iron status; Ferritin; Dietary iron

Introduction

The prevalence of iron overload in sub-Saharan Africa is the highest in the world, up to 10% in adults in some rural communities [1]. The principal environmental etiologic factor for iron overload in Africans is the consumption of a traditional fermented beverage that is brewed in non-galvanized iron drums and provides increased dietary iron in an ionized and highly bioavailable form [2, 3]. The fact that not all of the individuals who drink the iron-laden traditional beverage develop iron overload has raised the possibility that factors other than the ingestion of excess iron may contribute to the etiology of this condition.

Primary iron overload in European populations is a hereditary condition, and homozygosity for C282Y in the *HFE* gene linked to the HLA locus on chromosome 6 is usually responsible [4]. While African iron overload was formerly regarded as a condition that is exclusively related to ingestion of high dietary iron in the form of traditional home-brewed beer [5], studies of pedigrees from Zimbabwe, Zambia, South Africa, and Swaziland suggest that a gene not related to *HFE* may be implicated in the pathogenesis [6–9]. In these studies, it appears that heterozygotes develop iron overload only in the presence of high dietary iron intake. In this study, we postulated that if homozygosity for an iron-loading locus exists, then affected homozygous individuals would have some degree of elevation in serum ferritin concentrion in the absence of high dietary iron.

Materials and methods

Selection and study of Zimbabwean participants

This study was conducted at seven locations in the highveld region of Zimbabwe ranging from 1,200 to 1,450 m of elevation. Subjects were recruited among members of the Seventh Day Adventist Church. This church teaches abstinence from all alcoholic beverages, including traditional iron-rich beer, and also promotes a primarily vegetarian diet. The study subjects included subjects from both major ethnic groups in Zimbabwe, the Shona and the Ndebele, as well as Chewa descendents from Malawi. The study was approved by the Research Council of Zimbabwe’s Ministry of Health and all participants gave written informed consent.

Men above 30 years of age and postmenopausal women were enrolled if they met the criteria of never having drunk traditional home-brewed beer or other alcoholic beverages and of having no acute illness. Iron overload becomes more apparent after menopause in women because of the protective effect of menstruation and child bearing and after the age of 30 years in men [5]. The participants were questioned whether or not they were vegetarians. They were also questioned regarding symptoms and complications of iron overload and

conditions that may lead to secondary iron overload such as multiple blood transfusions or dyserythropoeitic anemia [10], disorders associated with inflammation, and the use of drugs [5]. Women were questioned about their gynecological and obstetrical history. Clinical examinations were conducted on each participant.

Collection and analysis of blood samples for Zimbabwean subjects

Venous blood samples were collected in the morning using the vacutainer system (Terumo Medical, Elkton, USA) into EDTA tubes and non-anticoagulated tubes. A Coulter Counter model MD8 autoanalyser (Coulter Electronics, Hialeah, FL, USA) was used to measure full blood counts. Westergren erythrocyte sedimentation rates were determined by the method of the International Council for Standardization in Haematology [11]. Serum ferritin levels were measured using a radioimmunoassay (Diagnostic Products Corporation, LA, CA, USA). Liver function tests were measured on a Cobas Mira Plus autoanalyzer (Roche Diagnostics Systems, Montclair, France) using reagents and calibrants from Roche Diagnostic Systems (Johannesburg, South Africa). Markers for hepatitis B and C viral infections were analyzed using Bioelisa enzyme immunoassay techniques (Biokit, Barcelona, Spain). For selected subjects with elevated serum ferritin concentrations, the serum iron concentration and total iron binding capacity were determined with methods modified from those of the International Council for Standardization in Haematology [12, 13], and the transferrin saturation was calculated as the serum iron divided by total iron binding capacity expressed as a percentage.

NHANES III data

Data from The Third National Health and Nutrition Examination Survey were used for comparative analysis. In this survey, 31,311 persons who were a representative sample of the non-institutionalized US population were examined from 89 primary sampling units (county or small group of contiguous counties) between 1988 and 1994. The lower age limit was 2 months, and there was no upper age limit. Data were extracted from the NHANES CD-ROM. Of 8,756 non-Hispanic African-Americans, 2,903 were selected who did not drink alcohol. From this subset, we selected 1,380 individuals consisting of men over 30 years of age and women over 50 years of age. For the NHANES III US dataset, total iron binding capacity and serum iron were determined colorimetrically using an Alpkem RFA analyzer (Alpkem Clackamas, OR, USA) as detailed in methodological procedure for NHANES III [14]. The transferrin saturation was calculated from the ratio of the serum iron to total iron binding capacity expressed as a percentage. Serum ferritin concentration was measured by using the Quantimmune IRMA kit (Biorad Laboratories, Hercules, CA, USA)

Definition of elevated serum ferritin

Because a population-based reference range for serum ferritin concentration has not been determined in Zimbabwe, we compared serum ferritin concentrations obtained in this study with those for non-alcohol drinking African-Americans studied in the third National Health and Nutritional Examination Survey in the United States [15, 16]. We used criteria developed from the second National Health and Nutrition Examination Survey [16] to define elevated serum ferritin concentrations: greater than 200 μg/L in men 30–44 years of age and

in women between the ages of 50 and 64; greater than 300μg/L in men 45–64 years of age and in women over the age of 64; and greater than 400 μg/L in men over the age of 64 years.

Definition of unexplained serum ferritin elevation

While the serum ferritin concentration reflects body iron stores [17, 18], inflammation or hepatocellular damage from various causes may also influence serum ferritin concentration [19]. In addition, certain anemias may lead to secondary iron-loading. Because we wished to identify subjects with elevated serum ferritin concentration due to a possible primary iron-loading process, we excluded patients whose increased serum ferritin concentrations were associated with anemia (hemoglobin <13 g/dL in men or <12 g/dL in women) [20, 21], with ongoing hepatocellular damage as shown by elevated values for aspartate amino transaminase or alanine amino transaminase (>50 IU/L) or with an ongoing inflammatory process as suggested by an elevated erythrocyte sedimentation rate (>9 mm/h) [22]. For African-Americans, we used the same hemoglobin and transaminase criteria mentioned above, and since we did not have data on erythrocyte sedimentation rate, a C-reactive protein value within the normal range (0–1 mg/dL) was used as criteria for inclusion into the analysis. The normal range of values for the C-reactive protein in African-Americans was based on the normal ranges given in the laboratory manual for NHANES III [14].

Statistical analysis

Student's *T* test was used to compare groups for normally distributed continuous variables and the Kruskal–Wallis test for skewed variables. The Fisher's exact test or Pearson chi-square test was used to compare proportions. Analysis of variance (ANOVA) was used to compare laboratory tests according to hepatitis status. Transferrin saturations were compared between Zimbabweans with unexplained serum ferritin elevations and comparable African-Americans after stratifying for age and sex. The level of statistical significance was taken as two-sided $P < 0.05$.

Results

The clinical characteristics of the Zimbabwean study population are summarized in Table 1. More women than men were studied because recruitment was usually done during the week when most men were at work and because generally more women than men attend the Seventh Day Adventist Church in the age categories that were selected. Median serum ferritin concentrations were less than 100 μg/L in both men and women. Among the men, 23% were positive for hepatitis B surface antigen and 6% were positive for antibody to hepatitis C. Among the women, 10% were positive for hepatitis B surface antigen and 11% were positive for antibody to hepatitis C.

Because of the substantial prevalence of positivity for hepatitis B surface antigen and/or antibody to hepatitis C, we examined clinical variables according to hepatitis virus status as shown in Table 2. Complete blood counts, erythrocyte sedimentation rates, and serum ferritin concentrations did not differ significantly according to the presence of hepatitis C antibody or hepatitis B surface antigen. Alanine amino transaminase and aspartate amino

transaminase were significantly elevated in the participants with hepatitis C but not in those positive for hepatitis B surface antigen.

Four hundred fifty Zimbabwean men over the age of 30 years and women over the age of 50 years were compared to 1,380 African-Americans of the same age range and sex (Table 3). (Forty-three postmenopausal women <50 years of age were excluded from this analysis.) As expected for a population living at higher altitude, non-alcohol drinking Zimbabwean participants had significantly higher hemoglobin concentrations than comparable African-Americans. Zimbabwean men over the age of 30 and postmenopausal women had significantly lower serum ferritin concentrations when compared to their abstinent African-American counterparts. Based on a serum ferritin concentration of less than 12 μg/L, the proportion of subjects with iron deficiency was significantly higher in Zimbabwean subjects (3.3%) when compared to their American counterparts (1.0%; $P = 0.002$).

Twenty-four of the Zimbabwean subjects had an elevated serum ferritin concentration using criteria developed for the United States population (see “Materials and methods”), but in 15 of these, there was concomitant anemia, inflammation, or elevated liver function tests. Nine subjects (1.8%), seven men and two women, had unexplained elevated serum ferritin concentrations. The clinical characteristics of these subjects are summarized in Table 4. Their ages fell within the fourth and sixth decades. The mean (±SE) transferrin saturation of 34 ± 3% in these nine Zimbabweans was significantly higher than the transferrin saturation of 26 ± 0.7% in 220 non-alcohol-drinking African-Americans who fell within the same age range and met similar selection criteria for an elevated serum ferritin concentration ($P = 0.046$).

Discussion

Serum ferritin concentrations of non-alcohol drinking black Africans were significantly lower than serum ferritin concentrations of non-alcohol drinking African-Americans of similar age range and of the same sex. The Zimbabweans had a diet not fortified with iron in comparison the African-Americans who consume a diet fortified with iron. Despite the low serum ferritin concentrations among the Zimbabweans as a whole, a small number had modest unexplained elevations in serum ferritin concentration by a definition developed for the American population.

The non-alcohol-drinking Zimbabweans had significantly higher hemoglobin concentrations but similar mean corpuscular volumes compared to non-alcohol-drinking African-Americans. The higher hemoglobin concentrations are probably explained by the relatively high altitude in areas where the Zimbabwean subjects live (1,200 to 1,400 m above sea level). We cannot exclude the possibility that the different assays used to measure serum ferritin concentration in the two populations may account for some of the observed differences. In the area of Zimbabwe where the study was undertaken, hookworm infestation is rare and tends to be subclinical and not a significant cause of iron deficiency anemia [23, 24]. Thus, hookworm-related intestinal blood loss would not explain the lower serum ferritin concentrations in this population. Living at an altitude of 1,200 to 1,400 m would not be expected to influence serum ferritin concentration [25, 26].

It is conceivable that the lower serum ferritin concentrations in Zimbabweans may reflect truly lower iron stores because of dietary differences between Zimbabwe and the United States. An important difference between the present Zimbabwe study population and the United States population is that flour is not fortified with iron in Zimbabwe. Flour has been fortified with iron in the USA since 1941, with bread, rolls, and buns having an iron content of 17.5 to 27.5 mg iron/kg [27]. There is evidence that iron fortification increases the iron status of populations. In Venezuela, fortification of precooked yellow and white maize with 20 mg\kg iron led to a reduction in the prevalence of anemia from 37% in 1992 to 15% in 1994 [28]. In Sweden, an increase in the iron status of the population appeared to be due to both iron fortification and an increase in the sale of medicinal iron preparations for iron supplementation [29].

In addition, the consumption of meat may be relatively low in this Zimbabwe study population. The Seventh Day Adventist Church promotes a vegetarian diet amongst its members, and in this study, 10% of the Zimbabwean participants were vegetarian. Food iron of animal origin (i.e., hem iron) is generally better absorbed than iron from vegetables, fruit, and grain. The percentage of iron absorbed from vegetables reaches the highest point of 10% in severely iron-deficient subjects, whereas 10% to 20% of the iron present in beef and pork is absorbed by subjects with normal iron status [30, 31]. Epidemiological studies in Western populations have shown an inverse relationship between meat consumption and the prevalence of iron deficiency [32].

Although the iron stores in the Zimbabwe population that we studied were lower than the African-American population on the basis of serum ferritin concentrations and although we used criteria for elevated serum ferritin developed for the United States population [16], we still found nine subjects (1.8%) with modestly raised serum ferritin concentrations unexplained by traditional beer consumption, alcohol, anemia, inflammation, or hepatocellular dysfunction. We have not proved increased iron stores in these individuals, which would require liver biopsy or quantitative phlebotomy, and it is possible that these are normal subjects whose ferritins lie in the upper tail of normal values. Nevertheless, as mentioned above, the elevated ferritin criteria were developed for the United States population with higher meat consumption, iron fortification, and higher serum ferritin concentrations. Furthermore, the relative but significant elevation in transferrin saturations also goes with increased iron stores. Interestingly, our mixture modeling studies of transferrin saturation in African-Americans points to the possibility of an iron-loading tendency in a similar proportion (0.9–2.1%) of the population [33, 34].

In conclusion, our findings suggest a need for further studies to better understand the roles of dietary iron content and iron-loading alleles in determining iron stores of both Africans and African-Americans. Studies to document the safety of various levels of iron fortification may also be in order.

Acknowledgments

This study was supported in part by the NIH research grant no. UH1-HL03679–07 from the National Heart, Lung and Blood Institute and the Office of Research on Minority Health and by Howard University General Clinical Research Center grant no. MO1-RR10284, by National Institute of Child Health and Human Development contract

no. 1-HD-3–3196, by a grant from the J.F. Kapnek Charitable Trust (Philadelphia, PA), and by a grant allocation from the Research Board of the University of Zimbabwe (Harare, Zimbabwe).

References

1. Gordeuk VR, Boyd RD, Brittenham GM (1986) Dietary iron overload persists in rural sub-Saharan Africa. Lancet 1 (8493):1310–1313 [PubMed: 2872439]
2. Bothwell TH, Seftel H, Jacobs P, Torrance JD, Baumslag N (1964) Iron overload in bantu subjects; studies on the availability of iron in bantu beer. Am J Clin Nutr 14:47–51 [PubMed: 14106870]
3. Saungweme T, Khumalo H, Mvundura E et al. (1999) Iron and alcohol content of traditional beers in rural Zimbabwe. Cent Afr J Med 45(6):136–140 [PubMed: 10695182]
4. Feder JN, Gnirke A, Thomas W et al. (1996) A novel MHC class I-like gene is mutated in patients with hereditary haemochromatosis. Nat Genet 13(4):399–408 [PubMed: 8696333]
5. Bothwell TH, Charlton RW, Cook JD, Finch CA (1979) Alcohol, iron and liver disease: iron metabolism in man Blackwell, Oxford
6. Gordeuk V, Mukiibi J, Hasstedt SJ et al. (1992) Iron overload in Africa. Interaction between a gene and dietary iron content. N Engl J Med 326(2):95–100 [PubMed: 1727237]
7. McNamara L, MacPhail AP, Gordeuk VR, Hasstedt SJ, Rouault T (1998) Is there a link between African iron overload and the described mutations of the hereditary haemochromatosis gene? Br J Haematol 102(5):1176–1178 [PubMed: 9753041]
8. Moyo VM, Gangaidzo IT, Gomo ZA et al. (1997) Traditional beer consumption and the iron status of spouse pairs from a rural community in Zimbabwe. Blood 89(6):2159–2166 [PubMed: 9058740]
9. Moyo VM, Mandishona E, Hasstedt SJ et al. (1998) Evidence of genetic transmission in African iron overload. Blood 91(3):1076–1082 [PubMed: 9446671]
10. Gordeuk VR (1992) Hereditary and nutritional iron overload. Baillieres Clin Haematol 5(1):169–86 [PubMed: 1596591]
11. ICSH recommendations for measurement of erythrocyte sedimentation rate (1993) International Council for Standardization in Haematology (Expert Panel on Blood Rheology). J Clin Pathol 46 (3):198–203 [PubMed: 8463411]
12. (1978) Recommendations for measurement of serum iron in human blood. Br J Haematol 38(2): 291–4 [PubMed: 638077]
13. (1978) The measurement of total and unsaturated iron-binding capacity in serum. Br J Haematol 38(2):281–7 [PubMed: 638076]
14. Gunter EW, Lewis BG, Koncikowski SM (1996) (Center for Disease control, Department of Health and Human Services). Laboratory methods for the Third National Health and Nutritional Examination Survey (NHANES III), 1988–1994
15. Center for Food Safety Applied Nutrition, Food and Drug Administration (1984) Expert Scientific Working Group. Assessment of the IronNutritional Status of the U.S. Population Based on Data Collected in the Second National Health and Nutritional Examination Survey, 1976–1980
16. (1985) Summary of a report on assessment of the iron nutritional status of the United States population. Expert Scientific Working Group. Am J Clin Nutr 42(6):1318–1330 [PubMed: 4072962]
17. Cavill I, Jacobs A, Worwood M (1986) Diagnostic methods for iron status. Ann Clin Biochem 23(Pt 2):168–171 [PubMed: 3767262]
18. Jacobs A, Miller F, Worwood M, Beamish MR, Wardrop CA (1972) Ferritin in the serum of normal subjects and patients with iron deficiency and iron overload. Br Med J 4(5834):206–208 [PubMed: 5082548]
19. Custer EM, Finch CA, Sobel RE, Zettner A (1995) Population norms for serum ferritin. J Lab Clin Med 126(1):88–94 [PubMed: 7602240]
20. World Health Organization (1968) WHO Scientific Group, Nutritional Anaemias World Health Organization, Geneva
21. World Health Organization (1972) WHO Scientific Group, Nutritional Anaemias World Health Organization, Geneva

22. Gillum RF, Mussolino ME, Makuc DM (1995) Erythrocyte sedimentation rate and coronary heart disease: the NHANES I Epidemiologic Follow-up Study. J Clin Epidemiol 48(3):353–361 [PubMed: 7897457]
23. Goldsmid JM (1968) Studies on intestinal helminths in African patients at Harari Central Hospital, Rhodesia. Trans R Soc Trop Med Hyg 62(5):619–629 [PubMed: 5707916]
24. Goldsmid JM (1972) Ternidens deminutus (Railliet & Henry, 1909) and hookworm in Rhodesia and a review of the treatment of human infections with T. deminutus. Cent Afr J Med 18(11):1–14 [PubMed: 5016569]
25. Cook JD, Boy E, Flowers C, Daroca Mdel C (2005) The influence of high-altitude living on body iron. Blood 106(4):1441–1446 [PubMed: 15870179]
26. Khraisha S, Awidi A (1987) Comparative study of serum ferritin and its correlation with different blood parameters of students at Amman and Dead Sea levels. Aviat Space Environ Med 58 (10): 987–988 [PubMed: 3675470]
27. Waddell J (1974) The bioavailability of iron sources and their utilization in food enrichment. Fed Proc 33(7):1779–1783 [PubMed: 4365303]
28. Layrisse M, Chaves JF, Mendez C et al. (1996) Early response to the effect of iron fortification in the Venezuelan population. Am J Clin Nutr 64(6):903–907 [PubMed: 8942415]
29. Hallberg L (1982) Iron nutrition and food iron fortification. Semin Hematol 19(1):31–41 [PubMed: 6763337]
30. Layrisse M, Martinez-Torres C (1971) Iron absorption from food and iron supplementation of foods. In: Brown BW, Moore CV (eds) Progress in Haematology. Grune & Strratton, New York, pp 137–160
31. Martinez-Torres C, Layrisse M (1973) Nutritional factors in Iraon deficiency: food iron absorption. In: Callender ST (ed) Clinics in Haematology Saunders, London, pp 339–352
32. Takkunen H, Seppanen R (1975) Iron deficiency and dietary factors in Finland. Am J Clin Nutr 28(10):1141–1147 [PubMed: 1242290]
33. Gordeuk VR, McLaren CE, Looker AC, Hasselblad V, Brittenham GM (1998) Distribution of transferrin saturations in the African-American population. Blood 91(6):2175–2179 [PubMed: 9490706]
34. McLaren CE, Li KT, Gordeuk VR, Hasselblad V, McLaren GD (2001) Relationship between transferrin saturation and iron stores in the African American and US Caucasian populations: analysis of data from the third National Health and Nutrition Examination Survey. Blood 98(8): 2345–2351 [PubMed: 11588029]

Table 1

Clinical characteristics of 493 Zimbabwean subjects grouped according to sex

	Men	Women	*P*
N	194	299	
Age (years)	44 (35–58)	57 (51–−62)	<0.001
Vegetarians (no. and %)	21 (10.8%)	27 (9.0%)	0.51
Number of pregnancies	–	7 (5–8)[a]	–
Number of spontaneous abortions	–	1 (0–1)[b]	–
Ferritin (μg/L)	84 (58–126)	70 (39–108)	<0.001
Hemoglobin (g/dL)	15.0 (14.1–15.8)	13.5 (12.8–14.3)	<0.001
Erythrocyte sedimentation rate (mm/h)	10 (4–24)	32 (17–49)	<0.001
Aspartate amino transaminase (IU/L)	26 (21–33)	25 (20–30)	0.291
Alanine amino transaminase (IU/L)	16 (10–24)	14 (10–19)	0.007
Alkaline phosphatase (IU/L)	90 (72–111)	104 (86–124)	0.043
Gamma glutamyl transferase (IU/L)	17 (10–23)	15 (9–24)	0.253
Hepatitis B surface antigen positive (no. and %)	45 (23.2%)	31 (10.4%)	<0.001
Antibody to hepatitis C positive (no. and %)	11 (5.7%)	34 (11.4%)	0.032

Results are presented as median and interquartile range unless otherwise indicated

P = value difference in means or proportions by *t* test and chi-square statistic comparing men and women

[a] *n* = 283

[b] *n* = 282

Table 2

Laboratory tests according to hepatitis C serology and hepatitis B surface antigen status

	Negative (*N*=375)	Hepatitis C positive (*N*=42)	Hepatitis B surface antigen positive (*N*=73)	Both positive (*N*=3)	*P* (for overall ANOVA)
Hemoglobin (g/dL)	14.0 (0.1)	14.0 (0.2)	13.9 (0.1)	14.4 (0.7)	0.7
White blood cells (×10^3/μL)	5.5 (0.1)	5.5 (0.2)	5.2 (0.2)	5.0 (0.8)	0.4
Platelets (×10^3/μL)	234 (3)	230 (10)	220 (7)	182 (36)	0.2
Aspartate amino transaminase (IU/L)	25 (25–25)	34 (32–36)[a]	24 (23–25)	39 (32–48)	<0.0001
Alanine amino transaminase (IU/L)	14 (14–14)	18 (17–20)[a]	14 (14–15)	30 (22–42)	0.004
Erythrocyte sedimentation rate (mm/h)	30 (1)	26 (4)	34 (3)	30 (15)	0.4
Ferritin (μg/L)	68 (66–71)	70 (62–79)	85 (77–93)	83 (54–129)	0.2

Results, given as mean (SE) or geometric mean (SE range), are from analysis of variance models with adjustment for age, sex, and vegetarian status

[a]Signficantly different from negative group in post hoc analysis

Table 3

Comparison of hemoglobin and ferritin concentrations between Zimbabwean blacks and African-Americans by age and sex

	Samples	Hemoglobin (g/dL)	Ferritin (μg/L)
Men 30–49			
Zimbabwean	114	15.2 (14.4–16.1)	86 (56–126)
American	387	14.6 (14.0–15.3)[a]	157 (93–263)
P		<0.0001	<0.0001
Men 50+			
Zimbabwean	80	14.5 (13.5–15.4)	84 (61–127)
American	436	13.9 (13.1–14.8)[b]	173 (94–301)
P		0.002	<0.0001
Women 50+			
Zimbabwean	256	13.5 (12.8–14.3)	73 (39–109)
American	557	12.8 (12.1–13.4)[c]	130 (72–210)
P		<0.0001	<0.0005

Results are presented as median and interquartile range

[a] $n = 380$

[b] $n = 431$

[c] $n = 554$

Table 4

Clinical summary of Zimbabweans with elevated serum ferritin concentrations and values for hemoglobin, erythrocyte sedimentation rate, and liver enzymes within the reference range

Characteristic	Men (*n*=7; values in median and range or no. and %)	Women (*n*=2; individual values given)
Age (years)	36 (30–52)	53, 57
Serum ferritin (μg/L)	338 (237–415)	332, 344
Transferrin saturation (%)	36 (30–46)	33, 34
Hemoglobin (g/dL)	15.8 (15.7–16.6)	15.0, 15.2
γ-Glutamyl transferase (IU/L)	27 (11–43)	26, 57
Aspartate aminotransferase (IU/L)	29 (23–34)	22, 29
Alanine aminotransferase (IU/L)	16 (14–38)	11, 45
HbsAg positive	1 (14.4%)	Yes, yes
Anti-HCV positive	1 (14.3%)	No, no