

Body iron stores in middle-aged North Indian patients with type 2 diabetes and obesity

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ABSTRACT

Introduction: Evidence from various epidemiological and clinical studies suggests that iron overload is proinflammatory and proatherosclerotic. Excess body iron has been positively associated with insulin resistance, type 2 diabetes and obesity.

Aim of the Study: To study the relationship of body iron stores with type 2 diabetes and obesity in middle aged North Indian population.

Materials and Methods: The participant population consisted of four groups of randomly selected participants (between 40 and 65 years of age and postmenopausal women); Group A: Normal individuals (controls), Group B: Obese nondiabetic individuals, Group C: Lean diabetic patients, Group D: Obese diabetic patients. Blood was examined for hematological, biochemical estimations, C-reactive protein, and serum ferritin (SF).

Observation and Results: A total of 197 participants were enrolled. The mean SF levels (ng/ml) among males were: Group A ($n = 18$) 148.56 ± 119.90 ; Group B ($n = 25$) 129.11 ± 94.77 ; Group C ($n = 27$) 127.96 ± 109.65 and Group D ($n = 22$) 148.36 ± 104.94 . The mean SF levels (ng/ml) among females were: Group A ($n = 23$) 67.44 ± 37.59 ; Group B ($n = 25$) 59.62 ± 43.56 ; Group C ($n = 24$) 77.97 ± 91.46 and Group D ($n = 33$) 66.46 ± 86.05 . No statistical difference was found among the groups in both the sexes.

Conclusions: Our observation is in sharp contrast to the earlier studies published from the West stressing that iron stores are increased in obesity and diabetes. We conclude that SF may not be a strong risk factor in the pathogenesis of obesity and diabetes in middle aged North Indians.

Key Words: Diabetes, obesity, serum ferritin

INTRODUCTION

Type 2 diabetes mellitus (T2DM) and obesity are increasingly assuming importance as public health problems in our country, especially among the North Indians. Both conditions have a complex etiopathogenesis and epidemiology. It is well-known that across the globe Indians are genetically and environmentally predisposed to develop metabolic syndrome (MS), diabetes and cardiovascular disease (CVD) at younger age and lower indices of body fat. The traditional risk factors are not totally able to explain this increased propensity.

Worldwide increased body iron store (BIS) has been emerging as putative risk factor for development of insulin resistance and CVD. Basic, clinical, and epidemiological studies have generated enough scientific evidence to

suggest that iron overload is proinflammatory and proatherosclerotic.

Measurement of BIS involves use of many variables, of which serum ferritin (SF) has been found to be a reliable tool, providing that confounding effects by inflammatory, hepatic, or neoplastic diseases are excluded.^[1]

Body circumference measures, such as waist circumference (WC) represent adiposity and these have been undisputedly associated with, insulin resistance syndrome and T2DM, thus WC may be significantly associated with BIS as reflected by SF concentrations.

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This study was carried out to study the relationship of BIS with T2DM and obesity in North Indian population. The basic idea was to evaluate the utility of SF as an additional marker of MS; and to identify the subgroup of individuals at risk for iron-related tissue damage, diabetes and CVD.

MATERIALS AND METHODS

This was a cross-sectional observational study carried out on a randomized sample of patients presenting to our medical outpatient department for a period of 10 months (February-November 2009). The protocol was approved by the Institutional Ethics committee and informed consent was obtained. The study population was categorized into four groups consisting of: Group A: normal healthy individuals (controls); Group B: Obese nondiabetic individuals; Group C: Lean diabetic patients; Group D: Obese diabetic patients.

This study was restricted to men and postmenopausal women to eliminate the influence of pregnancy, hormones and reduce possible confounding by iron deficiency. The study was also restricted to persons between 40 and 65 years of age to eliminate age related bias in SF values. Individuals having recent acute or chronic infectious, inflammatory or neoplastic condition; or with laboratory evidence of inflammation (C-reactive protein >0.5 mg/dl or white blood cell count >11,000/uL, platelet count >400,000/uL) were excluded. Patients with evidence of hepatic/renal disease were also eliminated. Similarly, other exclusion criteria were patients on aspirin, iron, or hormonal therapy. Smokers and individuals with alcohol consumption of >20 g/day were also excluded. Individuals who had anemia (hemoglobin <12 g/dl, mean red cell volume >100 fL, or with history of overt blood loss were also refused enrollment. Blood donors or recipients were also excluded from the study population.

Demographic data, detailed medical history, and behavioral information were collected. A meticulous physical examination was carried out. The height was measured to the nearest 0.1 cm, weight to the nearest 0.01 kg, and WC and hip circumference (HC) to the nearest 0.1 cm according to the standard guidelines and equipments. WC was measured in a horizontal plane at the level of the mid-point of the iliac crest and lower rib margin using nonstretchable flexible tape at the end of normal expiration, in the fasting state, with the subject standing erect and looking straight forward and observer sitting in front of the subject. HC was measured in a horizontal plane at the maximum extension of the buttocks. The variables were computed as: Waist-to-hip circumference ratio (WHR), body mass index (BMI = weight/[height²]).

Biochemical analysis was performed in the hospital laboratory using standard procedures. Since SF is an acute-phase reactant and does not accurately reflect the body's iron stores in the presence of inflammation, serum C-reactive protein estimation was done to exclude patients with underlying inflammation. Patients were considered to have diabetes if they met at least one of the following definitions: fasting plasma glucose >126 mg/dl on two or more occasions, postprandial glucose >200 mg/dl, or a random glucose >200 mg/dl with symptoms, or patient currently on insulin or oral hypoglycemic agents.

Patients were considered to be obese if their BMI was 25 kg/m² or more; high WC was defined as larger than 90 cm in men and 80 cm in women. Individuals were classified into the groups on the basis of their BMI (25 or more classified as obesity as recommended in the Consensus Statement for Diagnosis of Obesity, Abdominal Obesity and the MS for Asian Indians and Recommendations for Physical Activity, Medical and Surgical Management) and not on the basis of the WC since we found that even among the nonobese individuals very few individuals had WCs as per the norms established for Asian Indians, especially among the North Indian women.^[2]

These lower cut-offs for obesity were important in our study population as several investigators have shown that Asian Indians are more predisposed to develop insulin resistance and cardiovascular risk factors at lower levels of BMI when compared to other ethnic groups. Asian Indians have different body composition, elevated percentage body fat, and abdominal adiposity at lower or similar BMI levels when compared to white Caucasians. In this study, we needed distinctly dissimilar groups to compare the iron stores, thus we decided on lower cut off of BMI as recommended by the consensus statement above.

For the estimation of SF, overnight fasting blood samples were obtained, centrifuged at 1500 G for 30 min and frozen at -70° C. Quantitative estimation of SF was carried out by using the enzyme immunoassay, ORG 5FE SF kits procured from the ORGENTEC Diagnostika GmbH, Carl-Zeiss-Straße 49 55129 Mainz-Germany were used. We used the following available reference range of SF (as on the kits) for the age and sex matched population: Male (20-50 years): 34-310 ng/ml; female (20-50 years): 22-112 ng/ml; male (51-90 years): 4-665 ng/ml; female (51-90 years): 13-651 ng/ml.

Data analysis

Comparison of the mean SF values of each group was drawn against the control group using the *t*-test by means of computerized software available (Statistical Package for the Social Sciences, SPSS Inc).

OBSERVATION AND RESULTS

Of the total 197 participants, 92 were males and 105 females. Group A-D comprised of 41 healthy normal controls, 50 obese individuals, 51 nonobese diabetic individuals, and 55 obese diabetic individuals, respectively. Mean age of all the groups were comparable. As can be observed from Tables 1 and 2, the groups are quite distinct as per the mean body mass indices. However as is obvious from the tables, the WC of the females even in the nonobese category is on the higher side.

A wide variation was observed in the SF values among the individuals in each group. Mean values of SF in all our groups were on the lower side of the reference range available. Two individuals each (men) in the control group and the obese Group (A and B) and three individual each from the diabetic Groups (C and D) had SF in the upper normal range. No single individual had abnormally high SF levels.

Comparison of the mean SF values of each group was drawn against the control group, that is, Group A. When the Groups B-D were compared to their sex matched control Group A to examine the difference in their SF concentrations, no statistically significant difference was established. The groups were then mutually compared and again no significant statistical difference was found. Thus, our result showed that as supposed the SF was neither increased in the nondiabetic obese nor in the lean or the obese diabetic individuals. The data analysis is shown for the males and females in Tables 1 and 2, respectively.

DISCUSSION

Iron has been linked to the pathophysiology of various diseases primarily because of its chemical property of getting reversibly oxidized and reduced. This unique property makes iron potentially hazardous because it leads to generation of powerful reactive oxidant free radicals. Emerging scientific evidence suggests that iron overload is proinflammatory and proatherosclerotic. Excess body iron can impose oxidative injury that is associated with several cardiovascular risk factors including dyslipidemia, insulin resistance, and inflammation.^[3] Intake of dietary iron, especially highly bioavailable heme iron, was recently associated with greater risk of T2DM and coronary heart disease.^[4] A low-iron diet improves cardiovascular risk profiles.^[5]

Diabetes is associated with increased SF levels. Many elegant studies have concluded that BIS are positively associated with the development of glucose intolerance, T2DM, and gestational diabetes.^[6,7] A study from Finland supports the theory that increased iron stores, even in the range not considered to be associated with hemochromatosis, contribute to the development of noninsulin dependent diabetes.^[8] In a Turkish study within the diabetic subgroup SF was increased in those participants having poor glycemic control when compared to participants with good control.^[9] These observations have been confirmed and reiterated in many other ethnic groups worldwide.^[10-13] Raj and Rajan from India have published their data on 86 diabetic patients and found a positive correlation between SF, fasting blood sugar and HbA1c.^[14] Smotra and Kudyar in a similar study concluded that increased SF levels are associated with

Table 1: Summary of the anthropometric and SF data of males

Group	No. of participants	Mean age (years)	Mean BMI (kg/m ²)	Mean WC (cm)	Mean waist/hip ratio	Mean SF (ng/ml)	P value of mean SF values Patients vs controls
A	18	50.61	21.55	84.64	0.92	148.56 ± 119.90	
B	25	49.20	30.16	102.20	0.99	129.11 ± 94.77	NS
C	27	51.04	21.91	84.89	0.94	127.96 ± 109.65	NS
D	22	52.41	30.10	107.55	1.02	148.36 ± 104.94	NS
Total	92	50.78	26.04	94.96	0.97	137.18	

NS: Not significant, BMI: Body mass index, SF: Serum ferritin, WC: Waist circumference

Table 2: Summary of the anthropometric and SF data of females

Group	No. of participants	Mean age (years)	Mean BMI (kg/m ²)	Mean WC (cm)	Mean waist/hip ratio	Mean SF (ng/ml)	P value of mean SF values Patients versus controls
A	23	49.57	22.29	84.52	0.93	67.44 ± 37.59	
B	25	48.28	31.5	104.52	0.99	59.62 ± 43.56	NS
C	24	52.50	23.33	84.90	0.94	77.97 ± 91.46	NS
D	33	52.06	29.73	102.68	0.99	66.46 ± 86.05	NS
Total	105	50.71	27.07	95.90	0.98	67.68	

NS: Not significant, BMI: Body mass index, SF: Serum ferritin, WC: Waist circumference

increased serum insulin reflecting insulin resistance, poor glycemic control and complications of T2DM.^[15]

In recent years, increased iron stores have been found to predict the development of T2DM while iron depletion was protective. Most of these hypothesis were derived from the observations that DM is a common complication of hemochromatosis^[11] and other iron overload states;^[16] 53-80% of patients with hemochromatosis develop diabetes.^[8,11] Frequent blood donations, leading to decreased iron stores, have been demonstrated to reduce postprandial hyperinsulinemia^[17] in healthy volunteers and to constitute a protective factor for the development of T2DM.^[18]

This relationship of diabetes and excess iron is bi-directional. Iron affects glucose metabolism, which in turn impinges on several iron metabolic pathways. The clinical impact of these intimate interactions depends on the genetic predisposition and the time frame in which this network of closely related signals acts.^[19] The mechanisms behind hyperferritinemia in MS may be ferritin gene over expression as a part of adaptive adipocyte response to iron-induced oxidative stress. Adipocytes modulate metabolism through adiponectin in response to iron stores. Gabrielsen *et al.* observed that iron negatively regulates adiponectin transcription via FOXO1-mediated repression. SF was increased and adiponectin was decreased in T2DM and in obese diabetic subjects. These findings demonstrate a causal role for iron as a risk factor for insulin resistance.^[20] In a population study by Martinelli *et al.*, hepcidin (key iron regulatory hormone) levels increased significantly and linearly with increasing number of MS, paralleling the trend of SF. The authors apprehended that due to the pleiotropic effects of hepcidin, insulin resistance may worsen and contribute to the cardiovascular complications of MS.^[21] A novel adipokine visfatin predominantly secreted by visceral adipose tissue has also been found to be significantly associated with parameters of iron metabolism, especially in participants with altered glucose tolerance.^[22] It has been postulated that elevated iron stores may induce diabetes by several mechanisms including oxidative injury to pancreatic beta cells, impaired hepatic and muscle insulin extraction, and impaired peripheral glucose disposal.^[23] Systemic iron overload is not only linked in the causation and induction of diabetes, but also associated with the progression of the disease. Iron induced damage has also been shown to modulate the development of chronic diabetes complications^[9,19] such as microangiopathy and atherosclerosis.^[23]

Serum ferritin levels are often elevated in MS (dysmetabolic hyperferritinemia) or associated with a true hepatic iron overload (dysmetabolic iron overload syndrome). Elevated iron stores have been positively associated with the

prevalence of the MS.^[24] However, only a few studies have reported the relationship between the SF concentrations and various indices of adiposity, including the visceral and subcutaneous fat area.^[25] In Indians, central (abdominal) adiposity is a characteristic feature, which correlates directly with the metabolically active visceral adipose tissue.^[2] Gillum reported that the SF concentrations were correlated with the WHR.^[12] Increased SF levels have been found in overweight and obese women with polycystic ovary syndrome and has been shown to improve with metformin therapy.^[26] Elevated SF concentrations early in gestation are associated with an increased risk gestational diabetes. The association, at least in part, is mediated by the maternal fat mass and obesity.^[7]

Recently, the European Prospective Investigation into Cancer and Nutrition-Potsdam study examined the association between BISs and risk of type 2 diabetes through a case – cohort study. They concluded that high ferritin levels are associated with higher risk of type 2 diabetes independently of established diabetes risk factors.^[27] Zhao *et al.* have recently conducted a systematic meta-analysis where they analyzed the association of BISs or dietary heme-iron intake with T2DM risk and have suggested that increased ferritin levels and heme-iron intake are both associated with higher risk of T2DM.^[28]

Indians in general have been found to have low BIS when compared to other Asians, partly probably due to the predominant vegetarian diet.^[29] It has been also shown in many studies that the mean SF concentration is significantly lower in Asian men when compared with the Native-Americans.^[11,30] In India, few if any studies have examined the relationship of SF and insulin resistance syndrome or diabetes.^[14,15,31,32] The data from these small case-control studies have been conflicting.

Our study highlights that there is no evidence of iron overload in the nondiabetic obese, lean diabetic and obese diabetic North Indians. Since our study does not favor iron overload in this population, we may assume that excess iron may not contribute to the pathogenesis of these diseases in Indians. It has been observed that mean SFs increased across heme-iron intake quartiles but decreased across nonheme iron intake quartiles. Thus, nonheme iron intake should not be positively associated with diabetes risk. Luan *et al.* found no significant association between nonheme-iron intake and diabetes^[10] and Lee *et al.* demonstrated that nonheme iron intake was negatively associated with diabetes.^[33]

The most important reason for low SF levels even in the obese and diabetic Indians appears to be the predominant vegetarian diet and nonheme iron consumption in our

patients.^[34] It has been established by various cross-country surveys that nonheme iron contributes about 90-95% of total daily iron in Indian diets, in contrast to the predominant highly bio-available heme iron in the western diets. Further Indian diet is plagued by low iron content (largely cereal based) and poor absorption (due to dietary lack of ascorbic acid and alpha-tocopherol). The data also suggest that the average iron density of an Indian diet is not more than 8.5 mg/1000 Kcal.^[34] Iron and other micronutrient deficient diet coupled with occult gastrointestinal blood loss, dietary restrictions in obesity and diabetes, certain medications and unrecognized renal involvement all may contribute to poor iron stores in Indians.

Other important reason is a very low prevalence of the C282Y or H63D gene mutations responsible for iron overload in Indians as compared to the Caucasians. In a study on nonalcoholic liver disease from India, it was shown that in Indians there was no significant association of iron overload with this component of MS.^[32] In fact in a small case-control study on Indian coronary heart disease patients, the BIS were found to be lower as compared to controls.^[31] Having said so, however it strongly needs to be emphasized that iron even if not in excess is still available as catalytic iron to mediate in free radical reactions and its role in inducing oxidative injury and contribution to metabolic abnormalities cannot be completely ignored.

CONCLUSIONS

Our observation of normoferritinemia in obesity and diabetes is in sharp contrast to the earlier studies published from the West. Further research is needed to elucidate the mechanism and significance of these findings in Indians. It is also important that such similar studies are done at community level with larger sample size to impart good statistical power to the study. Furthermore, large prospective studies may help to establish or negate a causal relationship between iron overload, MS and diabetes. Nevertheless, this data reassures us that public health and individual strategies to supplement iron must continue, without apprehension of precipitating MS or diabetes.

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REFERENCES

- Bozzini C, Girelli D, Olivieri O, Martinelli N, Bassi A, De Matteis G, *et al.* Prevalence of body iron excess in the metabolic syndrome. *Diabetes Care* 2005;28:2061-3.
- Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, *et al.* Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. *J Assoc Physicians India* 2009;57:163-70.
- Williams MJ, Poulton R, Williams S. Relationship of serum ferritin with cardiovascular risk factors and inflammation in young men and women. *Atherosclerosis* 2002;165:179-84.
- van der A DL, Peeters PH, Grobbee DE, Marx JJ, van der Schouw YT. Dietary haem iron and coronary heart disease in women. *Eur Heart J* 2005;26:257-62.
- Rajpathak S, Ma J, Manson J, Willett WC, Hu FB. Iron intake and the risk of type 2 diabetes in women: A prospective cohort study. *Diabetes Care* 2006;29:1370-6.
- Jiang R, Manson JE, Meigs JB, Ma J, Rifai N, Hu FB. Body iron stores in relation to risk of type 2 diabetes in apparently healthy women. *JAMA* 2004;291:711-7.
- Chen X, Scholl TO, Stein TP. Association of elevated serum ferritin levels and the risk of gestational diabetes mellitus in pregnant women: The Camden study. *Diabetes Care* 2006;29:1077-82.
- Witte DL, Crosby WH, Edwards CQ, Fairbanks VF, Mitros FA. Practice guideline development task force of the College of American Pathologists. Hereditary hemochromatosis. *Clin Chim Acta* 1996;245:139-200.
- Canturk Z, Cetinarslan B, Tarkun I, Canturk NZ. Serum ferritin levels in poorly- and well-controlled diabetes mellitus. *Endocr Res* 2003;29:299-306.
- Luan de C, Li H, Li SJ, Zhao Z, Li X, Liu ZM. Body iron stores and dietary iron intake in relation to diabetes in adults in North China. *Diabetes Care* 2008;31:285-6.
- Acton RT, Barton JC, Passmore LV, Adams PC, Speechley MR, Dawkins FW, *et al.* Relationships of serum ferritin, transferrin saturation, and HFE mutations and self-reported diabetes in the Hemochromatosis and Iron Overload Screening (HEIRS) study. *Diabetes Care* 2006;29:2084-9.
- 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;25:639-45.
- Jehn ML, Guallar E, Clark JM, Couper D, Duncan BB, Ballantyne CM, *et al.* A prospective study of plasma ferritin level and incident diabetes: The Atherosclerosis Risk in Communities (ARIC) Study. *Am J Epidemiol* 2007;165:1047-54.
- Raj S, Rajan GV. Correlation between elevated serum ferritin and HbA1c in type 2 diabetes mellitus. *Int J Res Med Sci* 2013;1:12-5.
- Smotra S, Kudyar RP. Relationship between serum ferritin and type-2 diabetes mellitus. *J K Sci* 2008;10:170-4.
- Dubois-Laforgue D, Caillat-Zucman S, Boitard C, Timsit J. Clinical characteristics of type 2 diabetes in patients with mutations of HFE. *Diabetes Metab* 2000;26:65-8.
- Facchini FS. Effect of phlebotomy on plasma glucose and insulin concentrations. *Diabetes Care* 1998;21:2190.
- Ascherio A, Rimm EB, Giovannucci E, Willett WC, Stampfer MJ. Blood donations and risk of coronary heart disease in men. *Circulation* 2001;103:52-7.
- Fernández-Real JM, López-Bermejo A, Ricart W. Cross-talk between iron metabolism and diabetes. *Diabetes* 2002;51:2348-54.
- Gabrielsen JS, Gao Y, Simcox JA, Huang J, Thorup D, Jones D, *et al.* Adipocyte iron regulates adiponectin and insulin sensitivity. *J Clin Invest* 2012;122:3529-40.
- Martinelli N, Traglia M, Campostrini N, Biino G, Corbella M, Sala C, *et al.* Increased serum hepcidin levels in subjects with the metabolic syndrome: A population study. *PLoS One* 2012;7:e48250.
- Swaminathan S, Fonseca VA, Alam MG, Shah SV. The role of iron in diabetes and its complications. *Diabetes Care* 2007;30:1926-33.

23. Jehn M, Clark JM, Guallar E. Serum ferritin and risk of the metabolic syndrome in U.S. adults. *Diabetes Care* 2004;27:2422-8.
24. Iwasaki T, Nakajima A, Yoneda M, Yamada Y, Mukasa K, Fujita K, *et al.* Serum ferritin is associated with visceral fat area and subcutaneous fat area. *Diabetes Care* 2005;28:2486-91.
25. Luque-Ramírez M, Alvarez-Blasco F, Botella-Carretero JL, Sanchón R, San Millán JL, Escobar-Morreale HF. Increased body iron stores of obese women with polycystic ovary syndrome are a consequence of insulin resistance and hyperinsulinism and are not a result of reduced menstrual losses. *Diabetes Care* 2007;30:2309-13.
26. Fernández-Real JM, Moreno JM, Chico B, López-Bermejo A, Ricart W. Circulating visfatin is associated with parameters of iron metabolism in subjects with altered glucose tolerance. *Diabetes Care* 2007;30:616-21.
27. Montonen J, Boeing H, Steffen A, Lehmann R, Fritsche A, Joost HG, *et al.* Body iron stores and risk of type 2 diabetes: Results from the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam study. *Diabetologia* 2012;55:2613-21.
28. Zhao Z, Li S, Liu G, Yan F, Ma X, Huang Z, *et al.* Body iron stores and heme-iron intake in relation to risk of type 2 diabetes: A systematic review and meta-analysis. *PLoS One* 2012;7:e41641.
29. Hughes K. Serum ferritin and iron status in the general population of Singapore, 1993 to 1995. *Ann Acad Med Singapore* 1998;27:507-11.
30. Dhillon BK, Das R, Garewal G, Chawla Y, Dhiman RK, Das A, *et al.* Frequency of primary iron overload and HFE gene mutations (C282Y, H63D and S65C) in chronic liver disease patients in north India. *World J Gastroenterol* 2007;13:2956-9.
31. Gupta R, Rastogi S, Nagar R, Kastia S, Kaul V. Dietary and serum iron, body iron stores and coronary heart disease. *J Assoc Physicians India* 2000;48:489-92.
32. Duseja A, Das R, Nanda M, Das A, Garewal G, Chawla Y. Nonalcoholic steatohepatitis in Asian Indians is neither associated with iron overload nor with HFE gene mutations. *World J Gastroenterol* 2005;11:393-5.
33. Lee DH, Folsom AR, Jacobs DR Jr. Dietary iron intake and type 2 diabetes incidence in postmenopausal women: The Iowa Women's Health Study. *Diabetologia* 2004;47:185-94.
34. Nair KM, Iyengar V. Iron content, bioavailability & factors affecting iron status of Indians. *Indian J Med Res* 2009;130:634-45.

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