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Iron: From Dietary Sources to Utilization in the Body

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Abstract

Iron is a mineral found in every cell of the body. It is considered an essential mineral because it is needed to make the oxygen-carrying proteins haemoglobin and myoglobin, which are the vital part of blood cells. Haemoglobin is found in red blood cells and myoglobin is found in muscles. Iron utilization by the body is dependent on it its bioavailability to the body, which is in turn dependent on several factors, including dietary factors; such that inhibit or enhance its bioavailability. Fortification of iron, which though varies with the type of iron compound, could also determine iron bioavailability. Subject factors, including the iron status of the individual, genetic disorder, nutritional deficiencies and infection/inflammation also contribute to bioavailability. These bioavailability issues, when not resolved would lead to iron-deficiency anaemia, an abnormality that could affect other functioning of the body. In addition, the role of widely consumed food additives such as erythorbic acid on iron bioavailability from mixed diets is important. The iron status of the individual is the overriding factor that determines iron bioavailability, and other host-related factors, such as inflammation, may also play an important role. The current article reviews all these and even more.

Keywords: Iron; Bioavailability; Anaemia; Haemoglobin; Myoglobin; Erythorbic acid

Introduction

Iron is a mineral found in every cell of the body. It is considered an essential mineral because it is needed to make the oxygen-carrying proteins hemoglobin and myoglobin, the vital part of blood cells. Hemoglobin is found in red blood cells and myoglobin is found in muscles [1]. The best sources of iron include: dried beans, dried fruits, eggs (especially egg yolks), iron-fortified cereals, and liver. Others are lean red meat (especially beef), oysters, poultry dark red meat, salmon, tuna, whole grains, etc. Reasonable amounts of iron are also found in lamb, pork, and shellfish. Iron from vegetables, fruits, grains, and supplements are harder for the body to absorb [2].

The human body stores some iron to replace any that is lost. However, low iron levels over a long period of time can lead to iron deficiency anaemia. Symptoms include lack of energy, shortness of breath, headache, irritability, dizziness, or weight loss. However, there could also be too much of iron in the body arising from a genetic disorder called hemochromatosis [3]. Iron metabolism, unlike metabolism of other metals, has no physiologic mechanism for iron excretion, and close to 90% of its daily needs is obtained from endogenous source, namely the breakdown of circulating red cells.

There are iron losses, however, which include obligatory losses in all population groups (skin, intestines, urinary tract, and airways) and menstrual blood losses in women of child-bearing age. To maintain iron balance, the sum of these losses plus the iron required for growth in infants, children, and adolescents, and during pregnancy must be provided by the diet. The only reliable quantitative data for obligatory iron losses, however, are derived from a single study that estimated an average iron loss of 0.9–1.0mg/d (14μg/kg body weight) in men with normal iron status [4].

On the basis of the sum of obligatory and menstrual iron losses and iron needed for growth, the World Health Organization/Food and Agriculture Organization of the United Nations (WHO/FAO), the Institute of Medicine (IOM), and other national organizations have calculated iron requirements for different population groups. To translate these requirements into recommendations for daily dietary iron intakes requires an estimate of iron bioavailability, defined as the extent to which iron is absorbed from the diet and used for normal body functions [5].

Factors That Influence Iron Bioavailability

There are two types of dietary iron: non-heme iron, which is present in both plant foods and animal tissues, and heme iron, which comes from hemoglobin and myoglobin in animal source foods. Heme iron is estimated to contribute 10-15% of total iron intake in meat-eating populations, but, because of its higher and more uniform absorption (estimated at 15-35%), it could contribute ≥40% of total absorbed iron [5]. Non-heme iron is

usually much less well absorbed than heme iron. All non-heme food iron that enters the common iron pool in the digestive tract is absorbed to the same extent, which depends on the balance between the absorption inhibitors and enhancers and the iron status of the individual. It is important, however, to note that not all fortification iron enters the common pool.

Inhibitors of iron absorption

Phytate: In plant-based diets, phytate (myo-inositol hexakisphosphate) is the main inhibitor of iron absorption. The negative effect of phytate on iron absorption has been shown to be dose dependent and starts at very low concentrations of 2-10 mg/meal [6]. The molar ratio of phytate to iron can be used to estimate the effect on absorption. The ratio should be <1:1 or preferably <0.4:1 to significantly improve iron absorption in plain cereal or legume-based meals that do not contain any enhancers of iron absorption, or <6:1 in composite meals with certain vegetables that contain ascorbic acid and meat as enhancers [6,7]. Food processing and preparation methods, which include milling, heat treatment, soaking, germination, and fermentation, can be used to remove or degrade phytate to a varying extent [6,7]. The addition of exogenous phytase or its activation during food processing, or the addition to a meal just before human consumption, has been shown to improve iron absorption significantly [8,9].

Polyphenols: Polyphenols occur in various amounts in plant foods and beverages, such as vegetables, fruit, some cereals and legumes, tea, coffee, and wine. The inhibiting effect of polyphenols on iron absorption has been shown with black tea and herb teas [10]. At comparable amounts, the polyphenols from black tea were shown to be more inhibiting than the polyphenols from herb teas and wine [11]. The fact that polyphenol quantity, as well as type, influences iron absorption was also shown in a study with spices. Chili, but not turmeric, inhibited iron absorption in Thai women, although turmeric contained more polyphenols than chilli [12]. In cereals and legumes, polyphenols add to the inhibitory effect of phytate, as was shown in a study that compared high and low polyphenol sorghum. After complete phytate degradation, iron absorption from low-polyphenol sorghum increased significantly, whereas iron absorption from high-polyphenol sorghum was not improved [13].

Calcium: Calcium has been shown to have negative effects on non-heme and heme iron absorption, which makes it different from other inhibitors that affect non-heme iron absorption only [14]. Initially, the inhibitory effect was suggested as occurring during the transport of iron across the basolateral membrane from the enterocyte to the plasma because absorption of both forms of iron is equally inhibited, but more recently, it was suggested that the inhibition takes place during the initial uptake into the enterocytes [15]. Dose-dependent inhibitory effects were shown at doses of 75-300mg when calcium was added to bread rolls and at doses of 165mg calcium from milk products. In a recent study the addition of 200mg calcium to a maize-based test meal had no significant effect on iron absorption from NaFeEDTA. It is proposed that single-meal studies show a negative effect of calcium on iron absorption, whereas multiple-meal studies, with a wide variety of foods and various concentrations of other inhibitors and enhancers, indicate that calcium has only a limited effect on iron absorption [16].

Proteins: Whereas animal tissues have an enhancing effect on non-heme iron absorption, animal proteins, such as milk proteins, egg proteins, and albumin, have been shown to inhibit iron absorption. The two major bovine milk protein fractions, casein and whey, and egg white were shown to inhibit iron absorption in humans. Proteins from soybean also decrease iron absorption. Phytate was shown to be the major inhibitor in soy protein isolates, but even after complete phytate degradation iron absorption from soy protein isolates was only half that of the egg-white control (which allows inter-study comparison), which suggests that soy protein itself is inhibiting [17].

Enhancers of iron absorption

Ascorbic acid: Many single-meal radioisotope studies in human volunteers have shown convincingly the dose-dependent enhancing effect of native or added ascorbic acid on iron absorption. The enhancing effect is largely due to its ability to reduce ferric to ferrous iron but is also due to its potential to chelate iron. Ascorbic acid will overcome the negative effect on iron absorption of all inhibitors, which include phytate, polyphenols and the calcium and proteins in milk products and will increase the absorption of both native and fortification iron. In fruit and vegetables the enhancing effect of ascorbic acid is often cancelled out by the inhibiting effect of polyphenols [18]. Ascorbic acid is the only main absorption enhancer in vegetarian diets, and iron absorption from vegetarian and vegan meals can be best optimized by the inclusion of ascorbic acid–containing vegetables [19].

Cooking, industrial processing, and storage degrade ascorbic acid and remove its enhancing effect on iron absorption [20]. Several derivatives of ascorbic acid are less sensitive to heat and oxygen. Erythorbic acid, an ascorbic acid derivative, is widely used as an antioxidant in processed foods in industrialized countries. In developed nations, its intake from processed foods may reach 200mg/d [21] and erythorbic acid intake could be as high, if not higher, than ascorbic acid intake. Although it has little vitamin C activity, its enhancing effect on iron absorption appears to be almost double that of ascorbic acid [22]. The abundance of such compounds in the American diet might help explain why it has not been possible to demonstrate clearly the enhancing effect of vitamin C on iron absorption in multiple-meal studies of self-selected diets [23].

Muscle tissue: Single-meal radioisotope studies have consistently shown an enhancing effect of meat, fish, or poultry on iron absorption from vegetarian meals and 30g muscle tissue is considered equivalent to 25mg ascorbic acid. As with ascorbic acid, it has been somewhat more difficult to demonstrate the enhancing effect of meat in multiple meals and complete diet

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studies. Reddy et al. 2006 reported only a marginal improvement in iron absorption (35%) in self-selected diets over 5d when daily muscle tissue intake was increased to ≈300g/d, although, in a similar 5-d study, 60g pork meat added to a vegetarian diet increased iron absorption by 50% [24].

The nature of the "meat factor" has proved elusive. Most evidence indicates that it is within the protein fraction of muscle tissue; however, it is also possible that other muscle tissue components are involved [5]. There is good evidence to support the enhancing effect of cysteine-containing peptides, which are rich in digests of myofibrillar proteins and which, like ascorbic acid, could both reduce and chelate iron. Unlike other proteins, myofibrillar proteins are digested extensively by pepsin in the stomach and thus could bind iron and prevent its precipitation at the higher pH of the duodenum.

Studies with Caco-2 cells have indicated that glycosaminoglycans and l-α-glycerophosphocholine might also contribute to the enhancement of nonheme iron absorption by meat. It is difficult, however, to extrapolate from Caco-2 cells to humans and purified sulfated and unsulfated glycosaminoglycans did not increase iron absorption from a liquid formula meal in young women, although it is possible that other glycosaminoglycans that occur naturally may be enhancing [25-28]. Armah et al. [26] reported that purified l-α-glycerophosphocholine increased iron absorption in women who consumed a vegetable lasagna low in inhibitors, although to a lower extent than ascorbic acid. The enhancing effect of l-α-glycerophosphocholine was not confirmed in women who consumed a high-phytate maize meal, although iron absorption from this meal was increased by ascorbic acid (and EDTA).

Fortification of iron

Bioavailability of fortification iron varies widely with the iron compound used [29] and foods sensitive to color and flavor changes are usually fortified with water-insoluble iron compounds of low bioavailability. Iron compounds recommended for food fortification by the World Health Organization (WHO) include ferrous sulfate, ferrous fumarate, ferric pyrophosphate, and electrolytic iron powder. Many cereal foods, however, are fortified with low-cost elemental iron powders, which are not recommended by WHO and these have even lower bioavailability.

Subject factors: Subject factors may include iron status of the individual, genetic disorder, nutritional deficiencies and infection/inflammation.

Iron status: The iron status of individuals mainly influences the absorption of non-heme iron, whereas heme iron absorption is generally less affected [30]. There is an inverse correlation between iron status and iron absorption, and with the use of ferritin as an indicator of iron status the relation can be described mathematically [31]. A study in young women showed that the regulation of iron absorption by ferritin was less pronounced when iron was added as a water-insoluble compound (micronized dispersible ferric pyrophosphate), compared with ferrous sulfate [32]. These findings are important for fortification practices because they show that the different compounds are more or less suitable for repletion of iron-deficient subjects.

Further studies should be performed in iron-deficient and iron-replete individuals and with different fortification compounds. A study in Indian women investigated the effect of enhancers (ascorbic acid) and inhibitors (tea polyphenols) of iron absorption in an iron-deficient anemic group, compared with a nonanemic iron-replete control group. The difference in iron absorption between the groups was defined by the iron status, but the magnitude of the enhancing and inhibiting effect was shown to be independent of iron status [33].

Nutritional deficiencies: Vitamin A and riboflavin deficiencies have been shown to influence iron metabolism and absorption. Human studies showed that the correction of riboflavin deficiency improved the response to iron supplements [34]. An absorption study in Gambian men indicated that the efficiency of iron use is impaired in riboflavin deficiency but that iron absorption is unaffected.

Infection/inflammation: The peptide hepcidin, produced in the liver and adipose tissue, has been identified as a key regulator of iron homeostasis [35]. Hepcidin expression is increased in chronic inflammation and obesity and may contribute to the increased prevalence of iron deficiency observed in overweight populations [36-38]. A cross-sectional study in Thai women showed that obesity is associated with decreased iron absorption and increased inflammation, independent of iron status [39]. A study in school-aged children showed that overweight children had higher hepcidin concentrations and lower iron status compared with normal-weight children. The iron intake and bioavailability of the 2 groups were not significantly different, which suggests hepcidin-mediated decreased iron absorption or increased iron sequestration in overweight children [40]. Two recent small-scale studies have shown an inverse correlation of hepcidin concentration and iron absorption in iron-replete healthy women and men [41,42]. Further studies in populations with a broad range of iron status are needed to investigate fully the role of hepcidin on iron absorption.

Genetic disorders: Hemochromatosis is a disorder of excessive iron accumulation that affects up to 1 in 150 people in populations of Northern European origin. The effect of the disorder on iron absorption has been studied in control subjects and in homozygous and heterozygous subjects [43]. Homozygous subjects showed increased heme and non-heme iron absorption, whereas the non-heme iron absorption of heterozygous subjects from meals with moderate iron content was not shown to be different from control subjects. However, increased absorption was seen in heterozygous subjects from meals highly fortified with iron. These results were not confirmed in later studies in male C282Y heterozygotes and were suggested to be related to improved methods of genotyping and feeding of test meals (ie, single compared with multiple meals) [44].

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The other important group of genetic disorders that leads to iron overload is thalassemias and related hemoglobinopathies, which occur mainly in South and Southeast Asia, the Middle East, and the Mediterranean [45]. Thalassemia homozygotes have ineffective erythropoiesis that stimulates iron absorption even if iron stores are adequate, which leads to a risk of iron excess when regular transfusions are given to correct anemia [46]. Heterozygotes for α-thalassemia 1, β-thalassemia, and hemoglobin E are usually asymptomatic and have mild anemia but they may be at risk of iron overload if they have some degree of impaired erythropoiesis. To investigate this potential risk, a study was carried out in Thai women heterozygous for α-thalassemia 1, β-thalassemia, hemoglobin E, and compound HbE/β-thalassemia and control subjects, which measured iron absorption and use from rice meals with the use of stable isotope techniques [47]. In subjects with α-thalassemia 1 and β–thalassemia, but not hemoglobin E, iron use was lower and absorption was significantly higher than in control subjects, and absorption was not adequately down-regulated with increased iron stores. In countries with mandatory iron fortification of commonly consumed food products and concurrent high prevalence of thalassemia, the occasional monitoring of iron stores may be useful for early identification of potential iron overload.

Unresolved Iron Bioavailability Issues

Vitamin A and carotenoids

Vitamin A deficiency, such as iron deficiency, leads to anaemia. Vitamin A can affect several stages of iron metabolism, which include erythropoiesis and the release of iron from ferritin stores [48]. Isotopic studies that investigated the influence of vitamin A on iron absorption have, however, reported contradictory findings. A series of radio-iron studies from Venezuela has consistently shown that vitamin A and β-carotene enhance iron absorption from iron-fortified maize bread, wheat bread, and rice meals [49]. In the belief that the different findings may be related to the vitamin A status of the subjects, Davidson et al. added vitamin A to iron-fortified maize gruels fed to vitamin A-deficient Ivorian children. In this study, the additional vitamin A significantly decreased iron absorption, although the inhibition disappeared three weeks after provision of high-dose vitamin A supplements to the children. The Ivorian children in this study were also iron deficient, which might have influenced vitamin A metabolism [50]. The interaction of iron and vitamin A metabolism is clearly complex and subject factors or methodologic issues could explain the contradictory findings. The possible influence of carotenoids on iron absorption is important because carotenoids are widely present in fruit and vegetables.

Non-digestible carbohydrates

Non-digestible carbohydrates are widely present in plant foods. They resist digestion in the small intestine but are fermented in the colon to short-chain fatty acids with a variety of reported health benefits, which include increased colonic iron absorption [42]. Although most dietary iron is absorbed in the duodenum, the colon mucosa also expresses the iron absorption proteins divalent metal transporter, ferritin, and ferroportin, as shown in pigs [51]. Ohkawara et al. have reported that infused ferrous iron was absorbed by humans from the colon with ≈30% of the efficiency of the total iron absorption (duodenum and colon). Pectin and inulin have been reported to increase hemoglobin repletion in iron-deficient rats and a mixture of inulin and oligo-fructose increased hemoglobin repletion in iron-deficient pigs [52].

Possible mechanisms for increased colonic iron absorption are the generation of a lower pH, formation of soluble iron complexes, reduction of ferric to ferrous iron by gut microflora, a proliferation of the absorptive area in the colon, and an increase in iron-absorption proteins [42]. Human studies have consistently shown that inulin and oligo-fructose increase colonic calcium absorption, but a balance study and a stable isotope study failed to demonstrate an enhancing effect of inulin on iron absorption. The influence of non-digestible carbohydrates on colonic iron absorption merits further investigation [53-55].

Establishment of a 'bioavailability factor' for DRVs of iron

Diet composition and iron status influence iron bioavailability; however, iron status is the overriding determinant. The iron bioavailability factor for DRVs thus needs to be practically relevant and for a well-defined iron status [14]. It has traditionally been estimated for subjects with no iron stores (serum ferritin<15μg/L). The selection of no iron stores as the reference for the bioavailability factor leads to a higher bioavailability factor and lower dietary iron intake recommendation but still ensures that subjects with low or no iron stores will absorb enough iron to meet their demands. Whereas this seems a pragmatic approach, it remains unclear as to how individuals with adequate iron stores, who absorb much less iron, maintain their iron balance.

Discussion and Conclusion

For its function of making haemoglobin and myoglobin that are essential for carrying oxygen in the blood, Iron is considered as a very essential mineral in the body [1]. Its sources of iron include: dried beans, dried fruits, eggs, iron-fortified cereals, and liver. Others are lean red meat (especially beef), oysters, poultry dark red meat, salmon, tuna, whole grains, etc.

It is noteworthy to mention that iron from vegetables, fruits, grains, and supplements are harder for the body to absorb [2]. Iron utilization by the body is dependent on it its bioavailability to the body, which is in turn dependent on several factors, including dietary factors; such that inhibit or enhance its bioavailability. For instance, phytate, polyphenol, calcium and animal protein by various ways inhibit iron bioavailability. Others such as Ascorbic Acid and animal tissues enhance its

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bioavailability. Fortification of iron, which though varies with the type of iron compound, could also determine iron bioavailability. Subject factors, including the iron status of the individual, genetic disorder, nutritional deficiencies and infection/inflammation also contribute to bioavailability.

These bioavailability issues, when not resolves would lead to iron-deficiency anaemia, an abnormality that could affect other functioning of the body. These unresolved bioavailability issues include the mechanism by which calcium inhibits iron absorption, the nature of the meat factor, and the influence of vitamin A, carotenoids and non-digestible carbohydrates on iron bioavailability. In addition, the role of widely consumed food additives such as erythorbic acid on iron bioavailability from mixed diets needs clarification. The iron status of the individual is the overriding factor that determines iron bioavailability, and other host-related factors, such as inflammation, may also play an important role. Obesity is an inflammatory disorder and would be predicted to decrease iron bioavailability. Traditionally, in industrialized countries, a mean iron bioavailability factor has been used to generate DRVs for iron for all population groups, irrespective of diet.

The iron bioavailability factors for mixed diets in industrialized countries would appear to range from 14% to 18% for subjects with no iron stores. The iron bioavailability factors for vegetarian diets appear to range from 5% to 12%. A high intake of fortification iron would be expected to lower dietary bioavailability because cereal foods are commonly fortified with low-bioavailability elemental iron powders. Because both the consumption of iron-fortified foods and the bioavailability of iron-fortification compounds vary widely, the contribution of fortification iron to the bioavailability factors is difficult to estimate. In addition, it should be noted that regulation of iron absorption with iron status depends on the solubility of the iron compounds in the gastrointestinal tract.

References

1. IMFNB (2001) Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academy Press, US.
2. Jacobs P, Wood L, Bird AR (2000) Erythrocytes: better tolerance of iron polymaltose complex compared with ferrous sulphate in the treatment of anaemia. Haematology 5(1): 77-83.
3. Monsen ER, Hallberg L, Layrisse M (1978) Estimation of available dietary iron. Am J Clin Nutr 31(1): 134-41.
4. Hunt JR, Zito CA, Johnson LK (2009) Body iron excretion by healthy men and women. Am J Clin Nutr 89(6): 1792-1798.
5. Hunt JR, Zeng H (2004) Iron absorption by heterozygous carriers of the HFE C282Y mutation associated with hemochromatosis. Am J Clin Nutr 80(4): 924-31.
6. Hurrell RF (2004) Phytic acid degradation as a means of improving iron absorption. Int J Vitam Nutr Res 74(6): 445-452.
7. Tuntawiroon M, Sritongkul N, Rossanderhulten L (1990) Rice and iron-absorption in man. Eur J Clin Nutr 44: 489-497.
8. Egli I, Davidsson L, Zeder C, Walczyk T, Hurrell R (2014) Dephytinization of a Complementary food based on wheat and soy increases zinc, but not copper, apparent absorption in adults. J Nutr 134(5): 1077-1080.
9. Troesch B, Egli I, Zeder C, Hurrell R.F, de Pee S, et al. (2009) Optimization of a phytase-containing micronutrient powder with low amounts of highly bioavailable iron for in-home fortification of complementary foods. Am J Clin Nutr 89(2): 539-544.
10. Hurrell R.F, Lynch SR, Trinidad TP, Dassenko SA, et al, (1989) Iron absorption in humans as influenced by bovine milk proteins. Am J Clin Nutr 49: 546-552.
11. Cook JD, Reddy MB, Hurrell RF (1995) The effect of red and white wines on non-heme-iron absorption in humans. Am J Clin Nutr 61(4): 800-804.
12. Tuntipopipat S, Judprasong K, Zeder C (2006) Chili, but not turmeric, inhibits iron absorption in young women from an iron-fortified composite meal. J Nutr 136(12): 2970-2974.
13. Hurrell RF, Reddy MB, Juillerat MA, Cook JD (2003) Degradation of phytic acid in cereal porridges improves iron absorption by human subjects. Am J Clin Nutr 77(5): 1213-1219.
14. Hallberg L, Rossander-Hulthen L, Brune M, Gleerup A (1993) Inhibition of haem-iron absorption in man by calcium. Br J Nutr 69(2): 533-540.
15. Roughead ZK, Zito CA, Hunt JR (2005) Inhibitory effects of dietary calcium on the initial uptake and subsequent retention of heme and nonheme iron in humans: comparisons using an intestinal lavage method. Am J Clin Nutr 82(3): 589-597.
16. Lynch SR (2000) The effect of calcium on iron absorption. Nutr Res Rev 13(2): 141-158.
17. Hurrell RF, Lynch SR, Trinidad TP, Dassenko SA, Cook JD (1988) Iron absorption in humans: bovine serum albumin compared with beef muscle and egg white. Am J Clin Nutr 47(1): 102-107.
18. Ballot D, Baynes RD, Bothwell TH (1987) The effects of fruit juices and fruits on the absorption of iron from a rice meal. Br J Nutr 57(3): 331-343.
19. Hallberg L, Rossander L (1982) Effect of different drinks on the absorption of non-heme iron from composite meals. Hum Nutr Appl Nutr 36(2): 116-23.
20. Teucher B, Olivares M, Cori H (2004) Enhancers of iron absorption: ascorbic acid and other organic acids. Int J Vitam Nutr Res 74(6): 403-419.
21. Sauberlich HE, Tamura T, Craig CB, Freeberg LE, Liu T (1996) Effects of erythorbic acid on vitamin C metabolism in young women. Am J Clin Nutr 64(3): 336-346.
22. Fidler MC, Davidsson L, Zeder C, Hurrell RF (2004) Erythorbic acid is a potent enhancer of nonheme-iron absorption. Am J Clin Nutr 79(1): 99-102.
23. Cook JD, Reddy MB (2001) Effect of ascorbic acid intake on non-heme-iron absorption from a complete diet. Am J Clin Nutr 73(1): 93-98.
24. Bach Kristensen M, Hels O, Morberg C, Marving J, Bugel S, Tetens I (2005) Pork meat increases iron absorption from a 5-day fully controlled diet when compared to a vegetarian diet with similar vitamin C and phytic acid content. Br J Nutr 94(1): 78-83.
25. Huh EC, Hotchkiss A, Brouillette J, Glahn RP (2004) Carbohydrate fractions from cooked fish promote iron uptake by Caco-2 cells. J Nutr 134(7): 1681-1689.
26. Arman CN, Sharp P, Mellon FA (2008) L-alpha-glycerophosphocholine contributes to meat's enhancement of nonheme iron absorption. J Nutr 138(5): 873-877.

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27. Fair-weather-Tait S, Lynch S, Hotz C (2005) The usefulness of in vitro models to predict the bioavailability of iron and zinc: a consensus statement from the HarvestPlus expert consultation. Int J Vitam Nutr Res 75(6): 371-374.

28. Storcksdieck Bonsmann S, Hurrell RF (2007) Iron-binding properties, amino acid composition, and structure of muscle tissue peptides from in vitro digestion of different meat sources. J Food Sci 72(1): S19–S29.

29. World Health Organization (2006) Guidelines on food fortification with micronutrients. In: Lindsay Allen et al. (Eds), WHO Library Cataloguing-in-Publication Data, Geneva, Switzerland, pp. 1-376.

30. Miret S, Simpson RJ, McKie AT (2003) Physiology and molecular biology of dietary iron absorption. Annu Rev Nutr 23: 283-301.

31. Cook JD, Dassenko SA, Lynch SR (1991) Assessment of the role of nonheme iron availability in iron balance. Am J Clin Nutr 54(4): 717-722.

32. Moretti D, Zimmermann MB, Wegmuller R, Walczyk T, Zeder C, et al. (2006) Iron status and food matrix strongly affect the relative bioavailability of ferric pyrophosphate in humans. Am J Clin Nutr 83(3): 632-638.

33. Thankachan P, Walczyk T, Muthayya S, Kurpad AV, Hurrell RF (2008) Iron absorption in young Indian women: the interaction of iron status with the influence of Tea and Ascorbic Acid. Am J Clin Nutr 87(4): 881-886.

34. Powers HJ (2003) Riboflavin (vitamin B-2) and health. Am J Clin Nutr 77(6): 1352-1360.

35. Bekri S, Gual P, Anty R (2006) Increased adipose tissue expression of hepcidin in severe obesity is independent from diabetes and NASH. Gastroenterology 131(3): 788-796.

36. Nemeth E, Valore EV, Territo M, Schiller G, Lichtenstein A, Ganz T (2003) Hepcidin, a putative mediator of anemia of inflammation, is a type II acute-phase protein. Blood 101(7): 2461-2463.

37. Nead KG, Halterman JS, Kaczorowski JM, Auinger P, Weitzman M (2004) Overweight children and adolescents: a risk group for iron deficiency. Pediatrics 114(1): 104-108.

38. Yanoff LB, Menzie CM, Denkinger B (2007) Inflammation and iron deficiency in the hypoferremia of obesity. Int J Obes (Lond) 31(9): 1412-1419.

39. Zimmermann MB, Fucharoen S, Winichagoon P (2008) Iron metabolism in heterozygotes for hemoglobin E (HbE), alpha-thalassemia 1, or beta-thalassemia and in compound heterozygotes for HbE/beta-thalassemia. Am J Clin Nutr 88(4): 1026-1031.

40. Aeberli I, Hurrell RF, Zimmermann MB (2009) Overweight children have higher circulating hepcidin concentrations and lower iron status but have dietary iron intakes and bioavailability comparable with normal weight children. Int J Obes Lond 33: 1111-1117.

41. Roe MA, Heath AL, Oyston SL (2005) Iron absorption in male C282Y heterozygotes. Am J Clin Nutr 81(4): 814–821.

42. Young MF, Glahn RP, Ariza-Nieto M (2009) Serum hepcidin is significantly associated with iron absorption from food and supplemental sources in healthy young women. Am J Clin Nutr 89(2): 533-538.

43. Lynch SR, Skikne BS, Cook JD (1989) Food iron-absorption in idiopathic hemochromatosis. Blood 74(6): 2187-293.

44. Hunt JR (2006) Moving toward a plant-based diet: are iron and zinc at risk? Nutr Rev 60(5Pt 1): 127-134.

45. Weatherall DJ, Clegg JB (2001) Inherited haemoglobin disorders: an increasing global health problem. Bull World Health Organ 79(8): 704-712.

46. Pippard MJ, Callender ST, Warner GT, Weatherall DJ (1979) Iron absorption and loading in beta-thalassaemia intermedia. Lancet 2(8147): 819-821.

47. Zimmermann MB, Zeder C, Muthayya S (2008) Adiposity in women and children from transition countries predicts decreased iron absorption, iron deficiency and a reduced response to iron fortification. Int J Obes Lond 32: 1098-1104.

48. Zimmermann MB, Biebinger R, Rohner F (2006) Vitamin A supplementation in children with poor vitamin A and iron status increases erythropoietin and hemoglobin concentrations without changing total body iron. Am J Clin Nut 84: 580-586.

49. Garcia-Casal MN (2006) Carotenoids increase iron absorption from cereal-based food in the human. Nutr Res 26: 340-344.

50. Jang JT, Green JB, Beard JL, Green MH (2000) Kinetic analysis shows that iron deficiency decreases liver vitamin A mobilization in rats. J Nut 130(5): 1291-1296.

51. Blachier F, Vaugelade P, Robert V (2007) Comparative capacities of the pig colon and duodenum for luminal iron absorption. Can J Physiol Pharmacol 85(2): 185-592.

52. Yasuda K, Roneker KR, Miller DD, Welch RM, Lei XG (2006) Supplemental dietary inulin affects the bioavailability of iron in corn and soybean meal to young pigs. J Nutr 136(12): 3033-3038.

53. Abrams SA, Griffin IJ, Hawthorne KM (2005) A combination of prebiotic short- and long-chain inulin-type fructans enhances calcium absorption and bone mineralization in young adolescents. Am J Clin Nutr 82: 471-476.

54. Coudray C, Bellanger J, Castiglia-Delavaud C, Remesy C, Vermorel M, et al. (1997) Effect of soluble or partly soluble dietary fibres supplementation on absorption and balance of calcium, magnesium, iron and zinc in healthy young men. Eur J Clin Nutr 51(6): 375-380.

55. van den Heuvel EG, Schaafsma G, Muys T, van Dokkum W (1998) Non-digestible oligosaccharides do not interfere with calcium and non-heme-iron absorption in young, healthy men. Am J Clin Nutr 67(3): 445-451.

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