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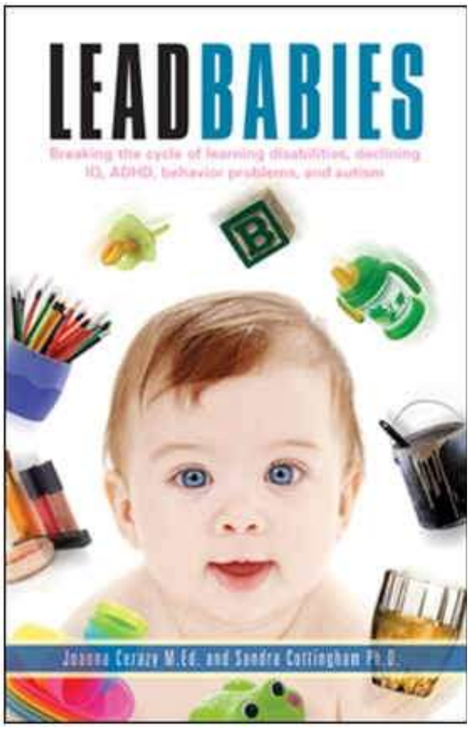
Iron Nutrition and Lead Toxicity

Contents



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Book Review "Lead Babies"

by Joanna Cerazy and Sandra Cottingham, published by Kunati Inc, (USA and Canada), May 2009. Available online at www.nomoreleadbabies.com and Amazon etc.

Review by Anne Roberts, The LEAD Group

I am not a lead sceptic, but before reading this book was inclined to think of lead campaigners as seeing - to slightly misquote the Duke in Shakespeare's *As You Like It* - "Books in the running brooks, sermons in stones, *and lead in everything*."

Well, apparently lead *is* in everything, just about.

In Chapter 2 and Appendix 2, the authors list possible sources of lead – such extensive lists as to make exposure to lead seem virtually unavoidable.

However, the message of the book is that lead *damage is preventable*:

"Nowadays, no one needs to be exposed to lead and to suffer its harmful effects. Our children can be protected from the cycle of damage caused by lead. What you have learned in this book will empower you to take action." (p135)

Cerazy and Cottingham use various "scenarios" to bring to life the effects of lead on the various stages of a child's development, from pre-natal to teenager, beginning with the story of "Sandy and Craig Miller," who have been renovating a 1925-built house, and are now expecting their first child. Almost everything the couple does unwittingly exposes them, and the unborn child, to lead.

There follows an outline of the first, second and third trimesters of foetal development; and, in more detail, conception, and weeks three, four, five and twelve, outlining the critical changes that are taking place.

“We know that toxic exposure during the first trimester of pregnancy interferes with the migration and organization of brain cells, and that any insult at this stage of development affects future brain development stages. Where it was previously supposed that with so little of a human formed, little damage could be done, the reverse has been discovered to be true. A foundation with damage will not support what is built on top of it.” (p20)

Under the heading “Present Versus Past Exposure,” the authors explain that “In pregnancy, the fetus gathers materials it needs to build bone structure from the mother’s bones. Lead from the maternal skeleton is transferred across the placenta to the fetus. Later, additional lead exposure may occur during breast-feeding. A critical factor in how much lead reaches the unborn baby is the amount of lead that has accumulated in the mother’s bones over her lifetime.” (p26)

Chapter 3 continues the story of early childhood lead exposure, this time in toddlers, telling how “Bill and Jennifer Richardson’s” 2-year old daughter “Molly” has changed from a delightful child into one who is hyperactive and difficult to manage. One pair of dotty grandparents has even brought along an heirloom leaded pewter cup as a birthday present for Molly, and gives her a drink out of it.

There follow classroom scenarios, with children who attend “Jackson Elementary School”: “Marcus,” 11 years’ old and autistic, “Bill” and other children, who have learning difficulties.

Finally, there is the story of “Scott”, who has a very low IQ and is skipping school and engaging in petty theft. He is also continuing to be exposed to lead in the one thing he is good at and enjoys: classes which in the US are called “shop” – in this case, learning how to maintain and repair motor vehicles.

The scenarios may strike Australian readers as a little, umm, smaltzy, but they do give a human context to the medical/scientific facts of the very serious and potentially lifetime consequences of prenatal and early childhood exposure to lead. The stories indicate how easy it is for a child to become exposed to lead, even in the best-intentioned or materially well-off families.

In the section on toddlers (“Molly”) we are reminded that “Children are not small versions of adults,” and how this magnifies the effect of what may seem to be very slight exposure to lead.

Not only do children have a larger skin surface in proportion to their body volume, they also, in comparison with adults, “drink more fluids, eat more food, breathe more air relative to their body volume.” (p66) They also absorb lead via the gastrointestinal tract more efficiently than adults (50% relative absorption, compared with adults’ 15% absorption.) (p66) The diagram on page 59 illustrates that “children and adults do not experience the effects of lead equally. Children are impacted earlier and more severely than adults.”

With older children and teenagers (”Scott”), “Mounting evidence has been amassed over recent decades from the fields of behaviour, neuropsychology and biology that confirms that the brain dysfunction caused by early, low-level exposure to lead results in the specific brain dysfunction that is associated with the behaviour we find with ADHD [Attention Deficit Hyperactivity Disorder], delinquency and violence. The change in understanding between what was previously accepted and what is now known is in the area of exposure threshold for damage. It was previously assumed that only high levels of exposure caused damage, and regulations were set accordingly. The more recent realization that neurotoxic effects occur at even the lowest levels of exposure, and that they can occur before a baby is born, has offered new, if not shocking insight into a long list of trends related to behaviour, including school drop-out, delinquency, drug use and violent crime.” (p76)

Use of Magnetic Resonance Imaging (MRI) has revealed that “the reduction in the amount of pre-frontal [lobe] gray matter – a loss of brain neurons observable in the brains of lead-exposed children – is the same as in adults with antisocial personality disorder such as is common in prison populations” [where prisoners are in jail for reasons other than politics, that is].

“The tendency to be “deceitful, reckless, impulsive, irresponsible and lacking in remorse and empathy” is symptomatic of a toxic brain injury, despite the fact that many are heavily invested in the theory that poverty, poor parenting, or negative social influences are to blame.” (p77)

The authors challenge standard preconceptions about the cause of antisocial and criminal behaviour (“Scott is not the result of bad parenting, a low socio-economic status or bad traits genetically inherited”) (p87). They ask why there is “a disproportionate representation of high school drop-outs, prisoners and the poor amongst African Americans?” (p78). Could there be a connection with the lactose intolerance which is very common in African Americans and is the main cause for them avoiding milk products.”

“In the US, African Americans get less than half the daily recommended amount of calcium. Calcium deficiency is a “critical factor in lead susceptibility.” (p78)

A possible connection between lead and addiction to drugs and alcohol is being uncovered which warrants further, long-term research.

"Early and in utero lead exposure, even at very low doses, is known to cause damage to the prefrontal lobes. Research confirms that frontal lobe dysfunction is a risk factor and important predictor for alcohol abuse. Even slight impairment to cognitive ability and language skills has been shown to increase [the] risk…" (p81) Some adult cocaine users have reported "first starting using cocaine as a way of self-medicating symptoms of ADHD." (p81)

The effects of lead on the brain don't end with childhood: "Ongoing lead exposure throughout one's life has the unfortunate effect of diminishing one's intelligence." (p90)

Chapter 6 suggests the possibility that autism may be the outcome of lead "potentiating" with mercury. ("Potentiation" is when the synergistic action of two drugs is greater than the sum of the effects of each when taken on its own.) The authors ask "Would it be outrageous to suppose that when lead potentiates with mercury in the system of a child, the effect is autism?" (p96)

(Which is not to say that without mercury there would be no autism.) The authors suggest the increasing levels of mercury "making its way into the developing brains of babies, both born and in utero, in a way that parallels the saga of lead," may explain the increase in autism in every continent in the world since the early 1980s. (p94) The remainder of Chapter 6 traces the history of use of mercury in some vaccines and in dental fillings.

The symptoms of mercury poisoning are virtually the same as the list of symptoms of autism. "The factor of potentiation makes it at least plausible that mercury, when combined with lead, causes autism; that autism is the brain's response to being hit not with a single toxic metal, but with the additional cumulative insult of a second and possibly others in a window of vulnerability and susceptibility that is not repeated in the human life cycle." (p109)

Chapter 7 discusses the effect of lead on fertility, Chapter 8, Lead and other toxins – "partners in harm", Chapter 9 gives detailed information on how to reduce or prevent exposure to lead: what foods are protective, how to prepare food to avoid lead contamination, what sort of eating utensils to avoid, foods to avoid, foods to minimise… what to avoid in products designed for use by children…personal hygiene, housekeeping, avoiding lead contamination due to occupation or hobbies, how to make outdoor areas around the house safe from lead, and what is the most dangerous thing about renovations, from the point of view of lead.

Chapter 10 gives a three-step inventory of making one's personal environment (house and surrounds) safe, and discusses tests for the presence of lead. Most of the tests mentioned are only available, for people outside the USA, by online purchase although The LEAD Group's DIY-sampling kit can be used to send any type of sample to a lab for lead analysis and includes an interpretation sheet. See www.lead.org.au/clp/products/Do_It_Yourself_Lead_Safe_Test_Kit.pdf

Chapter 11 is about getting the lead out of one's body. The authors say medical supervision is imperative, but this seems to apply only to removal that is "too invasive or aggressive." (p 99) "Just as there are risks in leaving lead in the body, there are risks with removing it. These are concerns that you will need to discuss with your doctor." (p159)

It does appear, on my reading, that the herbal, as opposed to "mainstream" methods are a lot more agreeable and have other health benefits. (However, take a look at Appendix 3, which lists the lead content of traditional remedies and cosmetics reported to contain lead!)

"Herbal" versus "mainstream" treatment is a subject which I think requires more detailed discussion than to be obtained here.

Appendix 1 gives a chronology of the use of lead in the world.

Appendix 2 lists possible sources of lead (in addition to the discussion in Chapter 2). Another comprehensive list can be found at www.lead.org.au/lasn/lasn006.html but the two lists are complementary and a useful comparison can be made between them.

Summing up: an interesting, easy-to-read, informative book, with 146 references to research papers, and a good, straightforward index. The cover – an alert infant making eye-contact with the potential reader- is, considering the subject, perhaps misleading. On the other hand, depicting anything else would be distasteful and off-putting. The cover *does* accord with the optimism of the authors that lead poisoning *can* be prevented.

Editorial

By Professor Brian Gulson, CSIRO, Macquarie University Graduate School of the Environment, and Head of The LEAD Group's Technical Advisory Board

The major article in this edition of LEAD Action News has been nine months in the making so it's more or less Robert Taylor's first "child". When he first came to do volunteer work at The LEAD Group, Robert clearly expressed his deep interest in nutrition and thus this newsletter was conceived. I believe he has created the longest reference list of any document yet written by The LEAD Group. You can find the concise version of Robert's major article (below) in the Factsheets section of our website. At the end of his incredible job of collating, Robert, paraphrasing a line from The West Wing, remarked "noting that lack of iron reduces IQ and too much iron exposes you to acute attacks of diseases endemic in poor countries, God must have decided that poor people can be intelligent or alive, but not both at the same time."

Thanks to Catherine Sweeny who provided us with all the excellent photos of food in this newsletter and to David Ratcliffe who has the humungous job of web-publishing it.

Sadly, this edition of LEAD Action News also contains an obituary, that of Kathryn R. Mahaffey who died on 2nd June 2009, just one week after she kindly offered to review Robert's newsletter article and factsheet. Kate Mahaffey is world-renowned for her decades of work in nutrition specifically relating to lead and mercury poisoning, and the world of lead has suffered a great loss. From a personal point of view, I and my former team at CSIRO owe a tremendous debt to Kate. She was our project officer for the Biokinetics of Lead in Human Pregnancy study run through the US National Institute of Environmental Health Sciences. Our collaboration both scientifically and personally is something I will forever value.

Please use the Contact Us form on our website – www.lead.org.au/cu.html - to send your questions and thoughts on this or any earlier editions of LEAD Action News.

Obituary: Kathryn R. ("Kate") Mahaffey

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Kathryn R. Mahaffey passed away peacefully in her sleep June 2, 2009 after decades of work that advanced the nation's health and environment. She is remembered as a beloved wife, mother, scientist and community member who served as a source of inspiration with her principled and tireless intellect. She was the rare scientist who knew how to apply the lessons from academic research to protect the public heath. Her work changed the face of epidemic heavy metal poisoning, endocrine disruptors and many other environmental pollutants that afflict children, pregnant women and at-risk populations. Literally millions of children have avoided the tragedy of lead and mercury poisoning as a consequence of her work. Dr. Mahaffey was the first to ensure that the number of lead poisoned children in the US was determined accurately through the National Health and Nutrition Examination Survey in the 1970s, an action that enabled the nation to track a more than 90% reduction in children's blood lead levels.

Dr. Mahaffey conducted path-breaking scholarship on mercury poisoning, helping to disentangle the web of bioaccumulation that had stymied previous efforts to seriously address the problem. She was a principal author of the eight-volume Mercury Study Report to Congress that broke new scientific ground while focusing national attention on mercury exposure in the U.S. Most recently, she helped organize an international conference in Japan on reducing exposure to mercury from eating contaminated fish, while balancing key nutrients such as omega-3-fatty acids. As a public health activist, her work won cheers from children's health scientists and attacks from those who considered the facts to be injurious to their interests.

Dr. Mahaffey joined the public service in 1972, working first at the Food and Drug Administration, followed by the National Institute for Occupational Safety and Health, the National Institute of Environmental Health Sciences and numerous positions at the Environmental Protection Agency. Most recently she was a distinguished professorial lecturer at George Washington University, where she taught toxicology. She was also engaged in helping to design new studies, such as the National Children's Study.

The recipient of numerous awards from government and academe, she received the prestigious Arthur Lehman Award for regulatory toxicology from the Society of Toxicology and the Bronze Medal for Commendable Service from EPA for her work on mercury. She was also appointed to many panels by the National Academy of Sciences. She most recently filed a scientific critique of a government report on risks and benefits of fish consumption; in her comments she demonstrated that an attempt to abandon fish advisories, which have helped reduce mercury exposure, was without scientific foundation.

A prolific writer, Dr. Mahaffey published over a hundred manuscripts in the peer-reviewed scientific literature, eight reports to Congress, fifteen book chapters, and seven books.

Her personal life was brimming with the same intensity she brought to science, with achievements in music, sewing, knitting, furniture and interior design. A love of cooking and people made her parties special and memorable. She was a loving friend and family member who endeared people with her unique blend of intellect and tenderness.

She founded and led the Green Group at the Westmoreland Congregational United Church of Christ, Bethesda, Maryland where she also served as a trustee. As a gifted volunteer math tutor and leader of the math club at the Marie Reed Elementary School (one of the poorest elementary schools in Washington DC) she invested many hours with underachieving students.

A native of Mahaffey, Pennsylvania, she graduated from Penn State University and held a doctorate in nutrition, physiology and biochemistry from Rutgers University. Her upbringing in rural Pennsylvania significantly shaped her beliefs that people and the earth are part of an interconnected system requiring essential protection.

Kate Mahaffey is survived by her husband, David Jacobs, her daughter, Harriet Meehan, her son, Bert Kramer, her mother, Harriet Mahaffey, her two sisters, Rebecca Latimer and Deborah Westover, her two step-children, Paul and Robin Jacobs, and her two grandchildren, Lillian Meehan and Evalyn Meehan.

A memorial service was held on Tuesday evening, June 9, 2009, 7:00 PM at Westmoreland Congregational United Church of Christ, 1 Westmoreland Circle, Bethesda, Maryland. In lieu of flowers, contributions can be made to the Kathryn R. Mahaffey Memorial Scholarship Fund, which will enable students to pursue careers in science and public service. Checks can be made out to the Westmoreland Congregational United Church of Christ, with a notation on the check stating "Kathryn Mahaffey Foundation Fund."

Iron Nutrition and Lead Toxicity: Interactions and Impacts

Please note citations are listed below, and also contain a brief guide to the better articles.

Glossary of technical terms

ID – Iron deficiency: low stores of iron in the body
IDA –Iron Deficiency Anaemia: Deficiency and malformation of red blood cells caused by the lack of iron to produce haemoglobin. Not the only cause of anemia. Anemia may be hypochromic (pale blood cells due to lack of iron) or microcytic (small blood cells with fragile membranes and shorter life spans).
Haemoglobin – The iron compound that carries oxygen in red blood cells.
Serum – Material within the blood or bloodstream.
Myoglobin – Iron compound that stores oxygen in the muscles for rapid exertion
Ferritin - Standard iron storage molecule. Can store 4500 iron atoms in tight bonds that are dissolved when the atoms are passed to tranferrin.
Transferrin - Standard iron transporter within the blood. Loosely binds two iron atoms for transport and transfer.
DMT1 (divalent metal transporter 1) – Transports 8 different metals (Fe, Zn, Mn, Co, Cd, Cu, Ni and Pb) at a cellular level. It is the primary iron transporter.
Reticulo-Endothelial Cells – Cells associated with the immune system scattered in relatively fixed positions (such as the spleen or connective tissue)
Macrophages – White blood cells contained within tissue. They engulf aging blood cell, dissolve them and allow the recycling of their contents (specifically iron).
Erythropoiesis – The creation of new red blood cells.
Apoptosis – Cellular suicide (programmed cell death) carried out at the body's direction.
Ellastin and collegin – Connective tissues that provide form and flexibility to a number of body structures including the skin.
Zinc protoporphyrin – A zinc compound which replaces haemoglobin preventing normal oxygen transportation. It can be caused by iron deficiency or the presence of lead during the formation of red blood cells (erythrocytes).
Sickle cell disease – When a specific form of haemoglobin causes the blood cells to deform into a sickle shape. Must be distinguished from the sickle cell trait where a mixture of normal and sickle cells co-exist.
Helobactor Pylori (H Pylori) – Bacteria that inhabits the stomach and are the primary cause of stomach ulcers. It most easily establishes itself in low acidic stomachs and once established further reduces acidity.
ADD/ADHD [Attention Deposit Hyperactivity Disorder] – A neurobehavioral condition involving inattention, impulsiveness and sometimes hyperactivity
Hypertension – Consistently high blood
Gestational Diabetes (or gestational diabetes mellitus, GDM) – a temporary diabetic state that can be produced by pregnancy.
Hepcidin – The hormone that regulates the body's iron metabolism. For a comprehensive outline of it's function see citations 123 and 124.

Haem iron [Haeme or Heme (US)] – Iron that is found in a porphyrin ring that allows it to more easily engage in certain organic interactions.
Phytates – Phosphorus based compounds that bind with calcium, magnesium, iron and zinc in the stomach and gut to produce insoluble (and indigestible) compounds. They have anti-carcinogenic impacts on the gut.
Polyphenols – A large family of organic compounds with antioxidant properties. Major subgroups include tannins, lignins and flavanoids. Like phytates they can bind with other nutrients in the stomach and gut but many appear to have significant health benefits.
Carotenoids – Plant compounds that provide pigments that both absorb and protect plant cells from light. They provide most bright colours in vegetables other than light green.
Sideroblastic anemia – A form of anemia where iron stores are available but cannot be incorporated in blood cells. Lead toxicity, excessive zinc consumption and copper or pyridoxine (B6) deficiency can be contributing causes. It can also be hereditary.
Ischemic stroke – Stroke caused by blockage of a blood vessel
VLDL cholesterol – A form of cholesterol high in triglycerides. It is usually estimated as a percentage of your triglyceride value.

Iron Nutrition and Lead Toxicity: Interactions and Impacts

Iron and lead levels

Low levels of serum (blood) iron are associated with higher blood lead levels in men (1,2), pregnant women (3,4) and particularly in children (5,6,7,8,9), though the evidence for women in general is curiously inconclusive (10,11). The relationship between serum iron and blood lead has genetic elements (12,13).

Iron metabolism: Overview

Iron is an essential micronutrient (14,15,16). The total amounts involved are small; an adult female will have 2-4 grams of iron (around 38mg per kilo) in her body, an adult male up to six (around 50mg per kilo) (17). Males tend to have more due to being larger, not suffering blood loss due to menstruation and some innate differences that begin at puberty (18 p249-251,19). Adult males normally have three times the stored iron of premenopausal females (1000 mg to 300mg seems a widely quoted figure but present author have not sighted the original source), a fact true for vegetarians as well as omnivores (480 mg to 160 mg; the same source problem applies) (20).

The majority of iron in the body is bound in haemoglobin [or hemoglobin (US spelling)] (found in red blood cells [erythrocytes]) where it is used in transportation and processing of oxygen within the body (21,22,23). Up to 10% is used in myoglobin that stores oxygen in the muscles (17,25,24,23). Over 4% is used in lung metabolism (26) playing a vital role in respiration (27). Most of the remainder is stored in the compound ferritin, over two thirds of which is deposited in the liver, the bulk of the remainder being split between bone marrow and reticulo-endothelial cells (17,23,25). Transport of iron within the body is handled by the serum molecule transferrin and at a cellular level by DMT1 [Divalent Metal Transporter 1] (16,17,25). The entire complex system is designed to ensure there is minimal free iron since free iron damages body organs through oxidation due to its highly reactive nature (16, 25).

Smaller trace amounts fulfill key roles within the body with functions such as immune defense (23,28), neural function (29,30,23), DNA synthesis (14,16), cellular energy production (31), liver function (32), apoptosis (33), elastin production (34) and collagen production (35). Iron levels are associated with bone strength and density (36); iron deficiency is linked to stress fractures in female athletes (37).

Iron cannot be systematically excreted from the body and is recycled within the body (23 Fig3,38) predominantly by macrophages of the reticulo-endothelial system. Macrophages of the spleen and liver generally recycle red blood cells before they reach the end of their natural life (120 days) eliminating 1% per day (124). The total iron absorbed from food each day is about 0.06% of total adult body iron (17) although for infants this figure can be multiplied by up to six (25). The main cause of iron loss from the body is blood loss (including significant losses inside the gut (39), particularly for athletes (40 p113,41)). This is the primary determinant of iron status (42,43) though some iron is lost through sweat (peaking within half an hour of heavy sweating) and skin loss (40 p112-114,22). Losses from urine are minimal (about 0.1 mg) (17). For most women menstruation will double to triple iron loss, with losses being slightly higher for adolescents, but it can be even higher (18 p249-251). Diet cannot outweigh heavy blood loss (42,43). Women with heavy menstrual flow should see their doctors as some medication (including the contraceptive pill) can reduce menstrual bleeding.

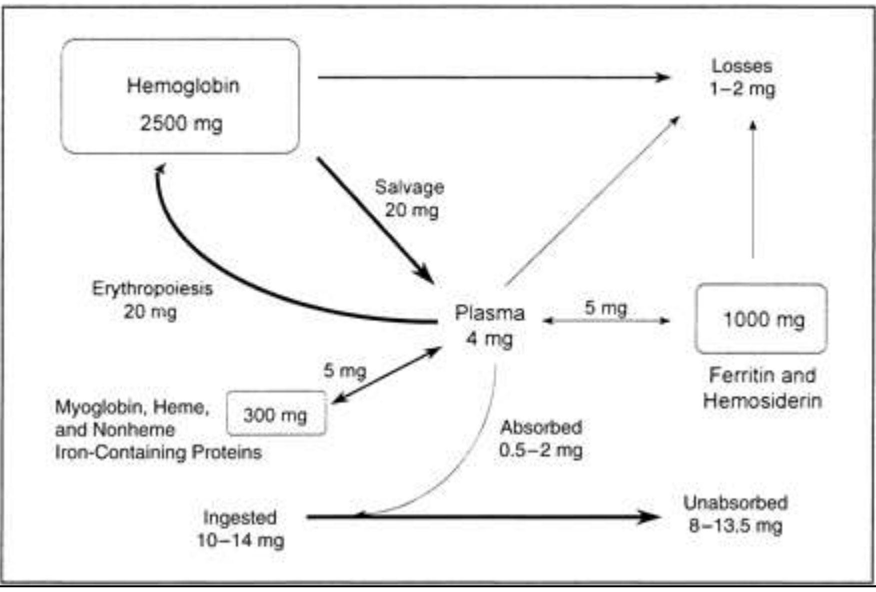


Figure 1 Diagram of iron movement in adult humans with estimates of iron trafficking derived from Bothwell et al. (1979). The above chart represents iron transfers within the body on a typical day. Note the chart reads from the bottom up. The plasma figure represents the extremely small amount of iron not bound in organic molecules. Hemosiderin is a less useful iron storage molecule than ferritin whose primary purpose seems to be to protect the body from the presence of unbound iron and which accumulates in body organs.
From Beard, John L. **Iron Requirements in Adolescent Females** J. Nutr. 2000

Iron lead interactions

Iron and lead occupy similar niches within the human body and so compete for likely binding sites particularly during absorption (44,45). While the primary toxicity of lead in the body is due to its ability to mimic calcium it also interferes with the iron metabolism in ways that are fairly well understood (45,46). The displacement of iron by zinc in the haemoglobin, producing zinc protoporphyrin (also a result of iron deficiency)(22,45), is one of the primary consequences of lead toxicity (46 p6). This leads to reduced oxygen supply as iron is the element responsible for most of haemoglobin's oxygen carrying capacity (21) producing hypochromic anemia. Lead also reduces the production of red blood cells (erythropoiesis) (45,46 p6), their size (microcytic anemia) (47) and their longevity (47). It prevents the normal increase of erythopoiesis in response to anemia (45).

These are far from the only effects. Lead's effect on the iron metabolism impacts on functions as diverse as cardiovascular response, neurotransmitter behavior, nerve transmissions, liver detoxification and bone development (46 p7). Lead is neurotoxic killing brain calls directly through apoptosis and interfering with brain function in a variety of ways (45,48,49). Individuals with sickle cell disease may be particularly vulnerable (45). Iron deficiency increases the rate of lead transfer to the brain , at least in rats, since these metals share a common transporter (DMT1) (50). Rat studies also indicate iron may be able to reduce lead induced apoptosis in the brain (51) and reduce lead related disruption during brain development (52).

Iron status: iron deficiency and iron deficiency anemia

Diet, digestion and blood loss (53) are the main factors that reduce iron levels but other factors such as H. Pylori infections (54,38) and genetics (38) also have impacts. Serum ferritin is the traditional way of measuring iron status but more recent tests for such things as total iron binding capacity, serum tranferrin saturation, free erythrocyte protoporphyrin, and serum tranferrin receptors in conjunction with haemoglobin measurements can more accurately establish the status of iron within the body (40 p100-104,26 p10-18).

Low iron levels (iron deficiency[ID]) affect over 20% of OECD populations (55). If iron levels become low enough iron deficiency anemia [IDA] occurs as the body lacks the iron to form enough new blood cells (17,25,56). While IDA affects less than 3% of the general population (57) it is much higher among some subgroups: pre-menopausal women (55,57,58,59,60), adolescent women (61,62), women who exercise (63,64,65,66), pregnant women (60,67,68), children (55,58,69,70), obese children (71,72) and some ethnic groups (57,59,67,68,69,70,72). Vegetarians (or vegans) in first world countries tend to have lower iron stores but not significantly higher rates of IDA (73,74,75,20). Vegetarian women should note this may be of particular concern during pregnancy (see below). In many third world populations the iron deficiency/anaemia figures can rise much higher (76,77,78,79,80,183). It would be difficult to measure the typical iron level differences between vegetarians and non vegetarians in third world conditions given the high proportion of individuals in these populations with IDA (effectively having no iron stores). It should also be noted that the World Heath Organization has questioned the use of the term iron deficiency anemia in at least one of its publications, since "presence of anaemia in a subject is a statistical rather than a functional concept" and that for an individual "has no immediate physiological meaning" (18 p258-260). Iron deficiency can be easily misdiagnosed because the early symptoms resemble the symptoms of ADD/ADHD (55), a disorder it may be linked to (81,82). In addition high lead levels in and of itself interferes with iron's effectiveness within the body, leading to increased iron deficiency (45,46 p6,83).

Iron deficiency and pregnancy

Among pregnant women iron deficiency can occur even when pre-pregnancy iron levels were adequate (58,84) since iron requirements increase as the pregnancy proceeds (84). Third trimester iron requirements of 5-7.5mg/day (84) cannot be met even from high bioavailability diets (from which under 5mg may be absorbed (84,85)) and must be met from the body's iron stores or supplementation. The average first world woman has approximately 300mg of iron stored; the estimated net requirements of pregnancy are 580mg (84). Danish studies indicate less than 20% of women enter pregnancy with the minimal iron stores required (86) and this figure can only be higher in the third world. However supplementation levels should be set with an awareness that iron absorption increases during pregnancy (84,87,88) and that high iron levels may have detrimental health effects (explained later in this article).

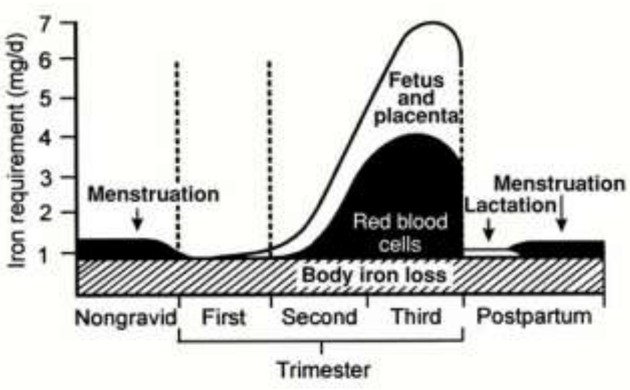


Figure 2: Estimated daily iron requirements during pregnancy in a 55-kg woman. The above chart graphically illustrates the strain of pregnancy on the iron metabolism. It does not take into account possible blood loss during birth which Bothwell estimates as costing 150mg of iron. *From:* Bothwell, Thomas H **Iron requirements in pregnancy and strategies to meet them** Am J Clin Nutr 2000 72: 257S-264

Borderline to moderate maternal IDA has limited impact on fetal iron levels (88,89,90) since the foetus receives most iron via the placenta rather than from maternal iron stores (91). However maternal iron levels may impact on the risk of ID or IDA developing in infancy (92,93,94,95) for reasons that are not clear given that breast milk iron is not directly related to maternal iron levels (96,97,98), except though significant IDA (97,99). Some studies do show a link between ID and iron stores (as opposed to other iron parameters) (100,101,102,95) which may explain this (as iron depletes as the infant grows (103,104)) but the reasons why these results are not universal is not clear (89,90). The fetus's iron status is significantly impacted by maternal hypertension (103), gestational diabetes (103), smoking (103,102), consuming alcohol (105), severe IDA (103) and premature delivery (103) and low birth weight (103,84). Provided a neonate (newborn) is neither underweight, premature or otherwise iron deficient it should have sufficient iron stores (around 75 mg per kilo) to maintain iron levels until six months of age with breastfeeding but after that point iron depletes rapidly (103,104,18 p247). Note that with infants early introduction of complementary foods (before 6 months) or prolonging heavy breast feeding (>6 times per day beyond 6 months) are negatively associated with iron status (106,107,104).

Females planning pregnancy should seek to maintain robust iron levels since supplementation after pregnancy is discovered largely misses the first trimester when ID has significant impact, effecting fetal weight and risk of problems in later trimesters (94,108,59), possibly due to the impact on the development of placenta (109). Iron stores should be increased prior to pregnancy as iron absorption may fall in the first trimester (84) and significant stores reduce dependence on supplementation (which some find difficult to maintain due to side effects (110,111)). Significant iron depletion can occur during birth (19 Fig3) though the actual depletion from breast feeding is less than from menstruation (84). Mothers are frequently iron depleted after giving birth (108,95).

Iron deficiency and lead levels

There seems little doubt that rectifying severe iron deficiency significantly impacts blood lead levels (7,45,112,113,114,3). In pregnant women dietary iron intake has more of an impact on newborn blood lead than that of any other micronutrient; the impact is double that of calcium (3). It also worth noting that research indicates that low maternal iron levels increase the risk of schizophrenia in offspring by up to four times (115) while high lead levels can roughly double the same risk (116). How these two risk factors interact with each other is not yet known. With children correcting iron (or zinc) deficiency may, though not necessarily will, lead to the cessation of pica (the compulsive consumption of non-food items such as paint and clay) which can be a source of lead contamination (117).

On the other hand the evidence for supplementation where iron intake is adequate is poor (118,119); for example, a recent large double blind study indicates no impact on blood lead levels from iron (or zinc) supplementation (120). The same may be true of low lead levels with or without iron deficiency (121,122). There might still be advantages to iron supplementation for individuals whose environments remain lead contaminated and whose primary exposure is through ingestion (45,119). A recent study of rats' brains found that low dose iron supplementation had more effect on lead damage than high dose iron supplementation (51).

Iron regulation within the body

The body regulates the intake of iron, so iron absorption falls as iron levels rise (17), due to the influence of the hormone hepcidin (25,123,124). The more hepcidin is produced by liver cells the less iron will be absorbed (25,123,124,38).

An individual who is suffering IDA may absorb up to fifteen times more iron than an individual with high iron levels (149) partially due to increases in DMT1 inside the duodenum (large intestine) (45). Unfortunately if iron is not present in sufficient quantities the DMT 1 molecules will instead transport other metals such as lead, increasing lead absorption up to 7 fold (45).

Hepcidin production may be stimulated by disease related inflammation (123) or exercise that impacts the joints (125,126) though research in the field is in its early stages (the hormone was only discovered in 2000 (38) and it's regulation of iron metabolism was widely recognized in 2004).

The inability to regulate iron intake (Hemochromatosis) is a generally heritable defect that is found in European and particularly Slavic populations which tends to lead to iron toxicity (and consequent organ failure) in later life (127,128,129,38). Similar defects can be present in people of African (AIO or siderosis (127)) or Solomon Islander descent (53), and there may be others yet to be discovered (128).

Impacts of iron deficiency

Low iron levels by themselves produce cognitive decline (130) especially among young children (131,132,133), exacerbating lead's impact. For example iron deficiency impacts on depression level following pregnancy (134) and interferes with the ability of the mother to interact with the child (135,136). At the same time ID in the child impairs walking which weakens the maternal bond and reduces cognitive stimulus at a key stage of development (137,138,139).The fact that a child with IDA has a muted facial (140) and auditory recognition (141) (including the mother's face and voice) combined with poorer object recall (142) and a more uneven temperament (133,139,142) is unlikely to be helpful to this crucial bond. All this may have significance since it has been suggested that an enriched environment may mitigate the impact of lead on cognitive development (143). In rectifying iron deficiency in children, verbal and motor skills are likely to improve independently of any lead impacts (118,140,144) though correction in latter childhood cannot totally repair early iron deficiency (145,133,30).

Iron and diet

Iron levels can be modified by diet (146,147,148) though the role of individual nutrients should not be overstated (149) and removal of iron inhibitors may be more important than supplementation aimed at increasing iron absorption. For example phosphorus and phytates may have twice the inhibitory effect than Vitamin C has at enhancing iron absorption (150). It would not be wise to begin vitamin C enhancement without first looking at phosphorus and phytate levels in the diet if your aim is to increase iron levels.

It is worth noting phosphorus is a micronutrient that is as important as iron, though one that is oversupplied in most western diets (151). It is important to remember that many of the compounds that inhibit iron absorption are themselves either essential or helpful nutrients, so the emphasis should be on balancing and offsetting impacts or separating consumption by at least two hours rather than on eliminating items.

Also keep in mind that since only a minute fraction of the body's total iron is absorbed per day (approximately 0.2 (212) -5 mg (84,85) of 2-5g in the normal adult body counterbalanced by losses of 0.8-3mg (18 p249-251)) rapid changes in iron status should not be expected (147,148,75). Individuals who are iron deficient should be aware that as iron levels rise measurable storage levels (serum ferritin) may even initially fall as iron storage is not the body's highest priority (152).

Iron absorption enhancers: Meat consumption & vegetarian diets

In terms of iron enhancement one of the easiest methods is increasing meat in the diet. A significant quantity of the iron in meat is haeme [or heme (US spelling)]. Between 15-40% of haeme iron in the diet is absorbed compared with 1-15% of non-haeme iron (75). Haeme iron is absorbed in a completely different manner than non-haeme iron and is not susceptible to most factors that inhibit or enhance non-heme iron absorption (153). The mechanism of haeme iron absorption is still poorly understood (153). Not only is haeme more easily absorbed than non-haeme iron but meat proteins (more accurately the amino acids that make them up (154,155,156)) enhance iron (and zinc) absorption even if the iron is non-haeme (157,158). While of secondary significance to adults (159) this can be especially useful for weaning infants (160) given the tight balance between iron absorption and loss (104,18 p247). Meat's importance is best demonstrated by the fact that premenopausal female omnivores can absorb six times as much iron as similar vegetarians (75). Cooked beef contains more haeme iron (65% of iron content) than cooked pork (39%) and poultry or fish (26%) (161). Haeme iron absorption seems little influenced by rising or falling iron stores (162,153) though there appears to be a limit on how much can be absorbed at a given time (158) and overall dietary haeme absorption still seems linked to iron status (163).

For vegetarians or vegans a good supplementation technique is through cooking acidic vegetables (such as tomatoes or cabbage) in non enameled cast iron pots which has been consistently shown to significantly increase dietary iron (165 p60,166,167); a technique that works equally well for non-vegetarians and which may be preferable to iron supplementation in pill form. For this purpose it should be noted that materials do not have to be naturally high in iron to improve iron status (168). Should there be difficulty in finding non-enameled cast iron cookware Lodge Cast Iron Cookware of Tennessee proves a range that is widely distributed. Note that the iron in many vegetables is more bioavailable (capable of being absorbed) when cooked rather than raw (341).

Iron cooking vessels: The following items have their iron content more than doubled when cooked in iron container without a protective surface. *Rear Row:* red cabbage, tomato, rice, corn meal *Front Row:* tomatoes, capsicum (bell or banana peppers in USA), pureed vegetables, wild rice, apple sauce, scrambled egg, corn meal, *Foreground:* scrambled egg *Not pictured:* milk

Vegetarians should note non haeme iron absorption can be compromised if stomach acidity is impaired (for instance by the use of antacids), since absorption of non-haeme iron in the gut requires the transformation and maintenance of iron in ferrous (Fe^{2+}) form (169 p154,170). If the stomach is insufficiently acidic the iron will not convert from ferric (Fe^{3+}) to ferrous form (Fe^{2+}) inside the duodenum (large intestine) and is unlikely to be absorbed (171). Furthermore the primary molecular iron transporter, DMT1, which is critical to this process operates effectively only at low (acidic) pH (172 p524,38). It is worth noting stomach acidity reduces with age (173) and that reduced stomach acidity can be a consequence of Helicobacter Pylori infection (174).

Iron absorption enhancers: Vitamin C and other food acids

Vitamin C (ascorbic acid) clearly enhances non-haeme iron absorption (165 p11,175,176), though its impact should not be overstated (149). The extreme increases shown in single meal experiments (some higher than 200%) (177,178,179,165 p11) are far more modest in whole diet studies (150,159,175,180) and not supported by studies of the iron stores of individuals who consume vitamin C supplements (150,176,180). As with most other enhancers and inhibitors it will only impact if consumed with food it can mix with in the stomach; Vitamin C taken four hours before a meal has no impact (177). There is no increase in effect once 100mg of ascorbic acid has been ingested (165 p12). The primary impact of vitamin C is to accentuate the creation and maintenance of soluble, absorbable iron compounds in the gut (171); the primary determinant of whether this available iron is absorbed is still iron status and hepcidin levels (149,179,38). The increased iron availability created by vitamin C is still dependant, to a lesser extent (171), on stomach acidity (181). Vitamin C may also enhance iron's capacity to displace lead during food absorption (182). Ascorbic acid is found in a wide range of vegetables as well as fruit but cooking destroys up to 75% (183,342). While oranges are a good source of vitamin C many fruits are far richer including guava, kiwi and black currant (342,343).

Vitamin C: 240 g of the foods (pictured above) should provide sufficient Vitamin C to optimize iron absorption (up to 960 g if cooked, for juice equivalent check labels). Top row: watercress, kohlrabi [kohl rabi, german turnip] (leaves), silver beet (spinach in Australia), popcorn. *Middle Row:* kohlrabi (bulbs), grapefruit, orange, lemon, cauliflower *Bottom row:* papaya [paw paw in Australia], strawberries, lime, dill, kaffir lime [K-lime, makrud lime]. *Not pictured:* Lychee

The fact that non-citric fruit (184) or vitamin C supplementation alone (150,180) does not necessarily enhance iron status indicates that vitamin C does not operate in isolation. It is worth noting citric acid can enhance iron absorption and may have a complementary role (175). However apple juice is almost as effective at enhancing iron absorption as orange juice possibly due to it's malic acid content (185). Food acids that have shown iron absorption enhancement include citric (found in oranges, grapefruit, lemon and limes), malic (apples ,grapes and wines), tartaric (grapes, bananas, tamarinds and wines) and lactic (yoghurt, sauerkraut and fermented pickled] vegetables), but studies contain inconsistencies that make predictions on their overall effects unreliable (175,186).

Vitamin C: 120 g of the foods (pictured above) should provide sufficient Vitamin C to optimize iron absorption (up to 480g if cooked, for juice equivalent check labels). Top row: parsley, guava (juice pictured), blackcurrant (juice pictured), kale *Middle Row:* radish, capsicum (bell pepper in US), kiwi fruits, broccoli *Bottom row:* feijoa, baby capsicums, brussel sprouts, guava, horse radish *Not pictured:* Mustard greens, red peppers, thyme

Iron absorption enhancers: Vitamin A, carotenoids and oily fish

Food Acids: These come in a variety of forms and many foods contain more than one type. *Lactic acid:* One of the more consistent iron enhancers. Yogurt (far left) and pickled vegetables (including sauerkraut with pictured can) are the most common sources. *Tartaric Acid:* Grapes, bananas, wine pictured. *Malic acid:* Grapes, apples, wine pictured. *Citric acid:* Pictured in our Vitamin C photos on the previous page are oranges, grapefruit, lemon and lime all of which are rich in citric acid

Until recently it was believed that Vitamin A enhanced iron absorption (165 p28) but new research indicates that vitamin A enhances the body's ability to transfer iron out of storage and its ability to construct haemoglobin from iron (187,188). It may limit the impact of iron inhibitors by preventing them binding to iron (189) though more recent research implicates the related carotenoids, including beta-caratone, which provide the colour compounds in most vegetables that are not light green (190,191,192). Fish oil (and/or carbohydrates) enhances iron absorption where certain significant inhibitors are present (193,194); whether it does so when such inhibitors are not present in significant quantities is another issue (195).

Alcohol: the ultimate special case

Alcohol reverses the effect of genes governing the hormone hepcidin (decreasing hepcidin production even as iron consumption and stores rise) leading to much higher absorption(196,197,323) but loss of the ability to regulate iron is a high price to pay for reasons outlined later. Two standard drinks a day (the current Australian recommendation for safe alcohol) is sufficient to impact iron levels reducing the risk of iron deficiency; more than this increases the risk of high iron levels (198). The polyphenols in red wine may have a slight negative impact on this (199). In Africa significant consumption of low alcohol, high iron beer has been a major factor in the prevalence of high iron levels, though it has also protected some groups of women from iron deficiency (200,201). It is important to note that in spite of high iron levels individuals who consume significant amounts of alcohol are up to five times more likely to have severely elevated blood lead (11,10,1,202) and, in the case of pregnant women, are more likely to transfer lead to the fetus (203). Significant alcohol consumption during pregnancy increases the infant's chance of developing ID or IDA (105).

General iron absorption inhibitors: Calcium

Calcium can reduce iron absorption by 50-60% (204,165 p15) but the experimental data contains inconsistencies and its impact on a whole diet is difficult to assess (205,159,164). It is the only inhibitor that affects both haem and non haem iron (206,164). Its impact is dose dependant (18 p254); a single slice of cheese (128 mg) has no impact on iron bioavailability in a hamburger (206). Maximal impact requires 300-600mg with higher amounts having no significant additional impact (18 p254). One of the highest iron absorption rates is from human milk though the often quoted 49% being absorbed by the child is optimistic; it is more likely to be under half that (compared to under 10% for cows milk) (207). Cow's milk can compromise a child's iron status in part by accentuating intestinal bleeding (208) and by reducing iron consumption (107). Significant quantities of cow's milk should not be given to children under 12 months of age (208,107).

Iron absorption inhibitors: Non-haem iron

The following comments on inhibitors apply only to non-haem iron.

Calcium & soy: The pictured quantity of milk or cheese would minimize iron absorption, half that would have little impact. For milk products (like yogurt) check label (impacts at >300ml with impact rapidly accelerating). Unfermented soy products (beans, milk and meat substitutes) inhibit iron absorption but are high in iron.

Soy proteins inhibit iron absorption (158,209,210) but it should be noted that this may be counterbalanced by the fact that soy is very high in iron (211). Fermented soy products can actually enhance iron absorption (212,213) but may not do so in all cases (195). Products containing calcium or soy also tend to be high in phosphorus (151) or its compounds (as are chicken, nuts, legumes [beans, lentils etc], soft drinks, meat and fish) (151) which may inhibit absorption (150).

Of these phosphorus compounds phytic acid [phytate in salt form] (found abundantly in whole grains, bran, nuts and seeds (165 p61,214 p42)) is the most significant reducing iron absorption by as much as 90% (215,216) due to the formation of insoluble (and therefore indigestible) iron compounds in the gut (214 p41). Even small quantities significantly inhibit iron absorption (217). However this inhibitory effect is significantly reduced by the presence of ascorbic acid, with vitamin C's impact being proportional to the phytate content (165 p11). Baking involving yeast (most bread making) greatly reduces the presence of phytates (217,218).

Phytates: The most powerful iron inhibitors (pictured above). Least inhibitory when baked with yeast (*right rear:* wholegrain bread) and should always be consumed with vitamin C (*left rear:* apple & blackcurrant juice). *Middle row:* baked beans, beans (black turtle, black eye, lima, white, barlotti), bran, peanuts *Front row:* sunflower & sesame seeds, peas, beans, nuts (almond, brazil, cashew), muesli

Tannins (polyphenols found in tea) can reduce absorption by up to 90% (219,186) (generally closer to two thirds(220,219)) but dissipate rapidly while other polyphenols found in coffee have roughly half the effect but are longer lasting (220).The impact of polyphenols (including tannin) can be considerable (221,222,176) but should not be overstated (223,224). They do not seem to have significant effects on individuals not otherwise iron deficient (223,224) possibly due to the presence of carotenoids and vitamin C in most diets which can negate the impact of the polyphenols (189,190,191,225) though rat studies also suggest the composition of saliva may be modified by regular tannin consumption (226). Some nuts, sorghum, chocolate, red wine and legumes contain significant polyphenols (227). The author has yet to see clear evidence that caffeine has any significant effect on iron status in humans in spite of this rumour having wide currency on the web.

Polyphenols: The items pictured at left contain polyphenols that may inhibit iron absorption. Note the considerable overlap with phytates. *Left to Right* Nuts (almond, brazil, cashew) beans (black turtle, black eye, lima, white, barlotti), coffee, tea, wine, beans, peas, chocolate, nuts (peanuts) lentils, peanut butter, baked beans.

Carotenoids & Vitamin C: The items (pictured at left) are high in both of these nutrients and should optimize iron absorption when polyphenols are present: *Left to right:* kale (in pot), thyme, banana capsicum (banana pepper in USA), capsicum (bell peppers in USA), red pepper, guava, broccoli, feijoa, kiwi fruit.

General Note: Not all polyphenols inhibit iron absorption and there insufficient evidence to know exactly how effectively individual food items that contain carotenoids can offset those that do.

Carotenoids – Some of the carotenoids found in the items pictured above may be able to counteract the inhibitory impact of polyphenols in coffee & tea. *Top row* Silver beet (spinach in Australia), yellow Indian corn, endives, lettuce, ruby red grapefruit juice, basil. *Middle row* Squash, red cabbage, broccoli, watermelon, pink grapefruit, cabbage, pumpkin. *Lower row* bananas, asparagus, carrots, tomatoes, red onions, red peppers, feijoa, guavas, apples, red peppers, beans, peas, banana capsicum (banana peppers in USA), avocado. *Not pictured* Pimentos, pepper grass, parsley, kiwi fruit.

Oxalic acid is one of the more widely mentioned inhibitors, though available research is very limited. It is found in variable quantities in dark green leafy vegetables (notably spinach), cocoa, chocolate, nuts, berries and beans (228,229). It can be found in large quantities in some non-western diets (230). While several studies have shown a variable degree of inhibition (186,231) a recent study showed none (232). From what little research on humans is readily available the overall impact on a western diet is unlikely to be significant. However, spinach, while containing significant iron (and vitamin C), remains a poor source of bioavailable iron as not only does it contain oxalates but also calcium, polyphenols and phytic acid (186,231). Some studies have placed iron absorption from spinach as low as 1.4% (186).

Eggs: Products that contain egg whites (such as pavlova centre rear) severely inhibit iron absorption and should be replaced with products such as papaya and egg yolk pudding (as vitamin C will enhance iron absorption from the egg). Simple ceramic egg separators are readily available. Traditional Italian gelato (not all gelato) may use egg yolks (right bowl) or less frequently egg whites (left bowl).

Egg whites (egg albumin) can inhibit iron absorption by almost 80% (233 table 2). The overall effect is about -27% per egg (234) and has been used as a standard experimental control due to to the predictable consistency of the result (209,210). Regular consumption can have a noticeable effect on iron levels (183). Egg yokes do not significantly inhibit iron absorption in humans and are a good complementary food for infants provided potential egg allergies are taken into account (231,232).

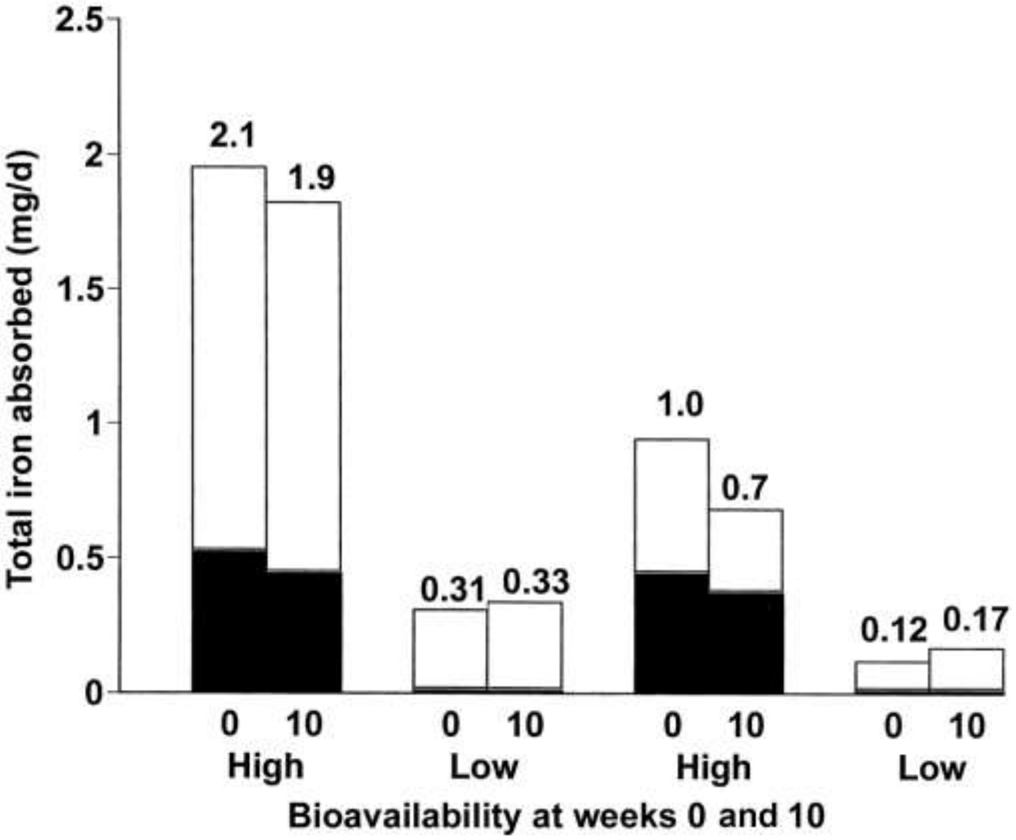
Metallic micronutrients: complex interactions

The issue of metallic micronutrients is complex. Copper is a classical example. Copper deficiency inhibits iron absorption in rats (237) and may prevent iron supplementation from being effective since copper plays a key role in iron absorption and transfer within the body (238,239,38,14). The problem is iron itself can interfere with copper absorption (240,165 p6). Indeed significant copper deficiency is extremely rare with healthy individuals on western diets except through zinc and, to a lesser extent, iron and/or vitamin C supplementation (239,241,242,243) and simply increasing copper intake may not counteract the effect of supplementation (242,243). High iron intake can even have different effects on copper levels in the foetus and mother (244). Copper levels (and selenium levels) also fall if an individual is iron deficient (245).

Zinc can inhibit iron absorption (and, to a lesser extent, visa-versa) (240,246,247,248,249,159 p5) but appears to be dose and ratio dependant (249). With rats and humans there is little effect if the iron/zinc ratio is around 2/1(249,250) and significant doses are required for there to be impact (251). Iron supplementation should have minimal impact on zinc levels (252,246,253) though an impact is possible (254,274). Zinc and iron have an even more complicated relationship where their effects on physiological outcomes (e.g. anaemia) are concerned (253,246,247,280). Iron and zinc compete primarily for absorption in the gut (247,255); separating the consumption of iron or zinc supplements by several hours (256,257,252) or consuming iron supplements on other than a daily basis (274) should minimize problems. Zinc's consistently demonstrated impact on copper levels is probably more significant (239,240,241,243,165 p7). Manganese severely impacts iron absorption (258) and its absorption is hindered by high iron stores (259).

Iron absorption and the dominant role of inhibitors

It is worth noting that the total impact of inhibitors may negate efforts at reasonable iron supplementation or fortification (260); several countries have populations whose iron absorption rates are between 2-4% (216,261). High iron bioavailability diets that minimize iron inhibitors and maximize iron enhancers significantly increase iron absorption (by a factor of 6 or more, see chart opposite) in individuals with low iron stores, though it should be noted that studies where individuals select their own food consistently return poorer outcomes than those where meals were selected by experimenters (85). Note from the accompanying chart that male omnivores (normally having three times the iron stores of women omnivores) absorb half as much as females in spite of having higher iron intakes and probably do not consistently meet their daily iron requirements even on a high bioavailability diets. For individuals on high bioavailability diets absorption decreases over time while on low bioavailability diets (which provide under 40% of daily requirements) absorption increases. The body's ability to regulate iron absorption is clearly demonstrated but so is the massive impact of inhibitors.



From: **Hunt, Janet R High-, but not low-bioavailability diets enable substantial control of women's iron absorption in relation to body iron stores, with minimal adaptation within several weeks Am J Clin Nutr 2003 78: 1168-1177**

Women [left 4 columns] in the present study who were treated with the same maintenance (12 wk) and test (2 d) diets tended to have adaptations in a similar but much less pronounced pattern as was seen in the men [right 4 columns] in a companion study (11). The men, with their higher iron stores, absorbed considerably less nonheme iron (clear column) than did the women, and similar amounts of heme iron (black column), even though the men's greater energy requirements resulted in proportionately greater iron intake (13 mg total iron for women, 15–16 mg for men). The figures above each bar designate total iron absorbed (mg/d). For these data, 9 women and 14 men were tested with the high-bioavailability diet, and 7 women and 17 men were tested with the low-bioavailability diet

Iron supplementation: cautions and limitations

Iron supplementation either directly by pill or multi-vitamin, or the taking of iron enhances (such as Vitamin C) should be handled with extreme care; particularly with children (19,262). Infants are unable to down regulate their iron absorption in the same manner as adults (263). Supplementation of infants (with iron drops or fortified formula; normal adult supplements must never be used) should only be undertaken where iron deficiency has been clearly established since supplementing iron sufficient infants can have severe short term and long term consequences (262,264,265,266,106). For infants gains may be fleeting (267) though this must be set against the crucial nature of this period for brain development (29,30,142).

When individuals are not iron deficient higher iron intake may not translate into higher iron stores (268,269).Where iron stores do rise the range of iron indicators affected may not be as wide with iron supplements as with food supplementation (270). Where supplements are taken low dosages may be almost as effective as high doses (51,88,133,271,272,273,274) and may even produce superior long term results with less risk (25 p529S,264, 265,266,273). Nor should it be assumed that providing a wider range of vitamin supplements will automatically improve outcomes (275,94). Some individuals find daily iron supplements produce significant side effects (mostly gastrointestinal) (110,111,276,277,282). While iron supplements can offer a more rapid improvement in immediate iron levels their effect may not be as long lasting (147) and continuing use has significant negative risks.

Iron supplementation: dangers

As the body does not excrete iron the cumulative build up from supplementation can be dangerous; a continuous load exceeding 1-2 mg/day can results in iron overload (278). In addition the interactions between micronutrients can be complex; iron supplementation can interfere with zinc absorption impacting on the immune system (254). This is of concern given that mild iron deficiency may reduce the risk of acute illness in areas where certain infectious diseases are prevalent (279,280,281). Even a non-toxic iron enhancer such as Vitamin C (for which a UK expert panel declined to set a recommended maximum intake (282)) can have deleterious impacts once other micronutrient impacts are taken into account: at least one case of serious copper deficiency has been reported in association with vitamin C (283). With Vitamin C it is instructive to compare Roth's list of impacts (283) (which include possible indirect effects) with those from the fact sheets of the Pauling institute (284) and the Feinberg School of Medicine(285) (which do not). To quote from a review of US military observations "Single-nutrient supplementation … should be implemented only after nutritional counseling and diet modification." (276)

Iron supplementation: daily or weekly/bi-weekly

Studies of pregnant women indicate that weekly iron supplementation is preferable to daily supplementation (286,287,288) supporting studies that show the ability to absorb non-haem iron from food may decline after daily supplementation (289,290). Supplementation should work best with individuals who consume significant haem iron since it is unaffected by this decline (289). Daily supplementation may still be preferable for children or individuals who are severely iron depleted since most studies show daily supplementation reduces anaemia at a faster rate (291,292,293,344,84). Many studies show minimally different results between daily and weekly supplementation (294,295) so, unless significant anaemia is an issue, the choice between them is generally determined by other criterion. For instance intermittent provision of iron supplements may address the problem of infectious and parasitic diseases noted above (296) and promote better absorption of elements that compete with iron (276). Equally important to the individual is the radical reduction in the risk of side effects (whose extent, prevalence and threshold dose is widely disputed but do occur) (297,294). For pregnant women who are not anaemic daily supplementation at other than low dosages can increase the risks of premature birth or low weight birth (298).

High iron levels: risk and damage

It is worth remembering that iron is in itself a neurotoxin and that in the USA is the largest cause of fatal accidental poisonings in children under 6 (14). High levels of iron can result from genetic factors, dietary overload, increased dietary absorption, sideroblastic anemia and even iron absorption through the lungs in the case of metal workers (53). High iron levels are not limited to individuals with genetic abnormalities and vary quite widely between ethnic and racial groups (299,129).

High iron levels provide ideal conditions for certain infections (300,301,302) notably malaria (303,280,281) and tuberculosis (300,301). High levels of iron doubles the risk of diabetes (304,305,306) as well as increasing the risk of complications from this disease (306).High iron may increase the risk of cardiovascular problems (307) though current evidence indicates a marginal influence (308,309) except when associated with other factors such as alcohol (310). There is an increased risk of ischemic stroke in postmenopausal women (311).

Having both high iron and high Very Low Density Lipoprotein (VLDL) cholesterol levels appears to double your risk of cancer (312) (excluding breast cancer (313,312)) and triples your risk of Alzheimer's disease (314). For males there appears little cancer risk from high iron alone but there may be some for females (315). Other co-factors that may increase the cancer risks of high iron levels include vitamin C (lung cancer (316)) and alcohol (colon (317) and breast cancer (318)) though it must be emphasized research into links between cancer and iron are ongoing (319). Haem and non-haem iron can also have differing impacts but current research contains significant contradictions; two significant lung cancer studies showed conflicting results (320,321,316). Zinc ameliorates some of these impacts (320,317,316) but bear in mind its impact both on iron and copper levels. Haem iron is associated with colon cancer but chlorophyll (from green leafy vegetables) seems to ameliorate the risk (161); Mormons who eat meat do not have higher rates of colorectal cancer than vegetarian 7th day Adventists (322).

High iron levels impact on the liver and, in conjunction with other factors, can lead to liver failure (323). Haem iron is also associated with gallstone disease (324). Vitamin C could potentially enhance oxidative damage caused by high iron levels (325).

Iron overload may be more of a problem with older individuals than iron deficiency (326,327) and its overall prevalence may be increasing in some western countries (328). Even during pregnancy it is possible to absorb too much iron (329). High levels of iron during pregnancy are associated with gestational diabetes mellitus particularly if combined with obesity (the combination may triple the risk) (330). When considering iron supplementation during pregnancy one should be aware the birth weight of infants can be adversely affected by high maternal iron levels (331,332,298,126) and the effect of iron on other micronutrients (333). It must be emphasized however that for most individuals the risks of high iron during pregnancy are considerably less than those of low iron (59,84,108); the argument for considering supplementation is strong (86,94,288,334,335).

High iron: risk or possibility

Also remember that the body is fairly efficient at preventing high iron levels: the Lexington medical center in North Carolina finds that there are five individuals with low iron for every one with high iron (53). The average male stores the equivalent of over one and a half years food intake so significant intakes over prolonged periods are required for problems to occur. Even with haemochromatosis significant organ damage does not normally occur until an individual is in their forty's (25) – without supplementation over forty years food intake is required. In fact only a minority of individuals (28% of men and just 1.2% of women under the age of eighty) with the most common haemochromatosis genetic defect will actually experience significant overload related disease (128).

"**Iron deficiency is not a diagnosis**"

Seek medical or qualified nutritional advice before treating yourself for abnormal iron levels and remember iron levels may be the result of other medical conditions (152) such as H. Pylori infection (174), hookworm infestation (336,337), drug intake (most frequently aspirin (338,339)) or genetic abnormalities (38). The fact that the gut is both the site of absorption and of predominant loss makes its health a primary factor. In the UK 41% of IDA is attributable to six medical conditions rather than diet or non-disease related blood loss (339). For males in particular IDA may be an early sign of cancer (340). To quote from a slide presentation from Saint Vincent's Hospital Sydney **"Iron deficiency is not a diagnosis."** (25) An inadequately balanced diet may be.

Iron Nutrition and Lead Toxicity - Citations

By Robert Taylor, The LEAD Group Incorporated, June 2009

Notes on Sources (For a guide to source content see end of document):

For reasons of both availability and reader access this article draws predominantly on free to view articles or the better abstracts of pay for view articles. It must be emphasized that the author is a layman. With no medical or biochemical background this article is limited by my lack of familiarity with some of the more technical aspects.

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Source Guide: A guide to the source content of the best citations

For an easy introduction to iron within the body sources 17 and 25 are recommended. Source 18 has some good charts, an effective summary of iron requirements from infancy to adulthood and interesting reflections on the meaning of IDA. Innvista's sheet on IDA (source 56) outlines the symptoms of IDA and provides an historical context. Source 40 has a good explanation of what iron test result mean (as does source 17 p9 for iron deficiency) and the progression from ID to IDA plus a discussion of iron enhancement strategies. For good short summaries of reasons why iron levels may be abnormal see sources 53,152 & 339.

Source 44 is a good and simple description of iron absorption though it does seem to miss a step: the iron conversion from FeIII to FeII in the intestine (sources 170 & 171). It is a dated general article that makes an interesting historical contrast to the far more technical 38 showing how quickly our understanding of the iron metabolism has evolved. For an exploration of the vital role of hepcidin the Ganz articles (123 & 124) are recommended. As to why hepcidin levels would increase in some athletes source 126 explains the connection between exercise, hepcidin levels and inflammation. The article on iron and the genetic effects of alcohol (196) is transformational finally explaining the high levels of iron and lead associated with alcohol consumption. The physiological impact is further discussed in source 323.

Women interested in reading about their iron requirements could start with source 19 (on adolescent female iron requirements). For iron requirements during pregnancy Bothwell's article (84) is thoroughly recommended. For a measured consideration of supplementation requirements during pregnancy sources 59,88,287,288,298 & 335 are all useful. Medical advice should be sought as the level of supplementation can be tailored to your iron status. Source 103 provides a clear outline of the maternal risk factors associated with iron deficiency in newborns.

For the risks and advantages to supplementing infants or children 262 provides a good starting point. Lozoff's study in Chile is a good cautionary tale: encouraging initial results (139) that had to be qualified (though not invalidated) by later outcomes (265,266). None the less this is a crucial period; sources 29 & 30 explain the key nature of iron in the first months of life. 142 & 30 give clear, if mildly technical, explanation of our understanding of iron and the developing brain.

For the impact of lead a good overview is provided by source 46 which contains excellent (if slightly dated) charts on the way lead interferes with the iron metabolism and red blood cell production. Source 45 which provides a good overview of research into iron and lead is unfortunately not only pay for view but has different copyright access in different jurisdictions; the address quoted gives access in the USA, if you live elsewhere you may have to search for the article separately. Source 48 (on neurotoxicity in children) is worth reading though some sections are technical while 49 (on metal neurotoxicity), while excellent, is too technical for most general readers.

For the impact of iron nutrition on lead toxicity source 118 from the CDC provides a good overview. Source 3 indicates fairly clearly which maternal nutrients have the biggest impacts on a newborn's lead levels. That reducing iron deficiency can reduce lead levels is demonstrated clearly by source 112. Source 7 demonstrates it is the iron deficiency that is the key while source while source 9 shows the importance of the severity of the iron deficiency. On the other hand Rosado article (120) demonstrates through a large study that no major blood lead change from iron level improvements can be expected where deficiency is not both widespread and deep; supplementing iron sufficient children whose primary exposure is not through ingestion has little effect.

In terms of maintaining your iron levels through diet the best sources are those that examine the whole diet. Source 85 demonstrates how big a difference an active tailoring of your diet can make though Figure 5 indicates how little difference it will make if you are already at or above the normal well nourished male's storage level (c.1000mg). The fact that an iron sufficient individual may need 4x the RDI to raise his iron storage significantly indicates how futile the effort could be even if it did not carry significant risks (268). Most women, however, routinely face low iron levels which can easily tip towards deficiency if stressed by events such as pregnancy, illness or increased blood loss. This tendency may have developed as a result of the vulnerability to acute infection in our African homeland (279) but it does not render the consequences less real. Source 130 demonstrates the cognitive cost of allowing this to occur while source 134 clearly demonstrates the emotional cost.

Sources 147 & 148 make interesting contrast in diet studies. They achieve comparable outcomes but reach different conclusions on the value of food intake versus iron supplements. Source 176 clearly identifies the dietary elements associated with higher long term iron levels (heme iron, supplemental iron, dietary but not supplementary vitamin C, and alcohol) while the finding that coffee but not tea is associated with lower iron levels provides intriguing circumstantial support for a rat study (226) that indicates saliva can be modified by regular tannin consumption.

Source 150 confirms the importance of vitamin C and meat in enhancing iron absorption while clearly establishing these two dietary components combined cannot outweigh the negative impact of phosphorus and phytate inhibition. How low inhibitors can reduce dietary iron absorption is demonstrated by source 216 which looks at the diets of Moroccan children. On the other hand the fact that removing coffee from the diet (224) can have little effect indicates that some inhibitors are already counterbalanced in some diets. The very mixed results on food acids in different studies (175) confirm the complexity of interactions that occur within and between foods.

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