

*The Hemochromatosis Natural Helper's Guide*

# **The HEMOCHROMATOSIS Natural Helper's Guide**

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## **What is Hemochromatosis?**

Hemochromatosis is an inherited genetic condition that causes the body to absorb and store too much iron in the tissues. The condition is also called "iron overload" or "iron storage overload" disease. It's dangerous because while iron is an essential component of the hemoglobin in our red blood cells and lets these cells transport oxygen, in its unbound form it also has a destructive nature that accelerates the oxidation or "rusting" of body tissues.

Of all the genetic disorders, hemochromatosis is the most common, and routinely affects over one million people in just the United States alone. This means that approximately one million Americans per year are overloading with excess iron – twice as many as those who suffer from iron deficiency anemia. Additionally, hereditary hemochromatosis is also the most commonly inherited liver disease in Europe.

Hereditary hemochromatosis (HH), also known as genetic hemochromatosis (GH), is triggered by a defective gene that causes one amino acid to be substituted for another in a protein. About 1 in 127 to 1 in 270 patients inherit the gene from both parents to get the disease; the gene is prevalent in Hispanics and in about 20% of people of northern European stock.

In terms of total figures, approximately 33 million Americans (10% of the population) are carriers for hemochromatosis but don't know it, and only a minority of these carriers actually manifests the disease.

Usually, most people have inherited the gene from an ancestor of centuries ago ... in particular, ancestors of Irish, German, English, Scottish, Welsh origin. A person who inherits two identical genes ("homozygous") will always pass on the disease to their offspring.

The physical basis of hemochromatosis is simple: the intestines absorb twice as much iron from food as normal, and thus excess iron slowly builds up in the body tissues. Many cases of hemochromatosis go undiagnosed because doctors and patients are unaware of the condition and don't know what to look for. The early symptoms include fatigue, sore joints and frequent infections, so they are easy to mistake for other conditions.

Nevertheless, as the excess iron builds up in the organs – especially in the liver, heart, spleen, and pancreas – it tends to destroy cells. Some people have no outward symptoms whatsoever until the condition matures in mid-age, at which time they may have 200 times the normal levels of iron!

That's when it can really cause problems.

Joint pain and sore joints are the most common early complaints of people who have hemochromatosis. Other common symptoms include fatigue, weakness and lack of energy. There can also be a loss of libido/sex drive, abdominal pains and swelling, and various heart problems, such as heart flutters. There can also be a history of frequent infections, skin bronzing or hair loss.

The symptoms tend to occur in men between the ages of 30 and 50 and in women over age 50 after they stop menstruating. Women are less at risk for iron buildup than men because of the blood, and thus iron they lose during their monthly menstruation. However, many people have no symptoms at all when they are first diagnosed.

If the condition persists without being diagnosed, by the time someone is fifty or sixty with iron build-up, the organs may have literally “rusted inside” and will show damage of some sort. Iron is nature’s rusting (“oxidation”) agent, and the buildup of excess iron levels within our bodies is one of the ways by which our body becomes oxidized, and therefore prematurely aged.

The excess iron is harmful because it is a catalyst for the generation of free radical activity, and free radicals have been identified as an underlying cause of cancer, atherosclerosis, liver cirrhosis, neurological disease, and other aging-related disorders.

Liver cirrhosis, liver cancer, heart failure, diabetes, arthritis are all possibilities for hemochromatosis sufferers if the excess iron builds up to cause tissue damage. The damage to one’s liver and pancreas is especially dangerous because the harmful results can be permanent. One of the side effects of hemochromatosis is a yellowish skin complexion and diabetes like symptoms that give HH the name “bronze diabetes.” Other possibilities from hemochromatosis include an enlarged liver (hepatomegaly), cirrhosis (liver scarring), and spleen enlargement (splenomegaly).

The following are some of the symptoms of hereditary hemochromatosis (HH):

- Chronic fatigue and weakness
- Sore or aching joints, especially in the knuckle and first joint of the first and second fingers
- Frequent infections (colds, flu and other signs of weakened immune system)
- Abdominal pain/swelling
- Red palms
- Impotence – low libido (males) – sterility - infertility
- Cirrhosis of the liver (with or without history of alcohol use)
- Liver cancer (with or without history of alcohol use)
- An enlarged liver or other liver disease
- Arthritis or joint pain (or joint replacement)
- Slightly elevated liver enzymes
- “Bronze diabetes” – abnormal gray or bronze discoloration of the skin

- Early menopause/irregular menses
- Darkening of the skin without exposure to the sun
- Always feeling "cold"
- Hair loss, loss of body hair
- Weight loss
- Cancer
- Headaches
- Hypothyroidism (thyroid deficiency)
- Adrenal gland damage
- Heart irregularities/heart failure/heart attack (particularly in younger men)

The screening to check for hemochromatosis involves multiple tests, not just one blood test. Doctors should measure a number of different factors to determine if you suffer from hemochromatosis. A skilled hematologist, who is an expert at blood conditions, is often the best professional to consult concerning your blood iron levels.

For instance, "low iron" on one blood test does NOT rule out hemochromatosis. Hemochromatosis often goes undiagnosed because it can show up on a blood test as low hemoglobin, just as does iron depletion. A person can actually be anemic and still be suffering from iron overload hemochromatosis!

Therefore, because the symptoms can be diverse and vague and can mimic the symptoms of many other conditions, and because blood tests can be misinterpreted, hemochromatosis often goes undiagnosed and untreated. Frankly, many doctors just don't think to test for it and doctors most often focus on the conditions caused by hemochromatosis—arthritis, liver disease, fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome, heart disease, or diabetes—rather than search for the underlying cause.

Here's the good news. There are a number of solutions available for hemochromatosis when it's found. In addition, if the iron overload caused by hemochromatosis is diagnosed and treated before organ damage has occurred, a person can live a normal, healthy life.

In the next chapter, we'll talk about various screening tests available to confirm the condition.

## Screening for Hemochromatosis

The diagnosis of hereditary hemochromatosis (HH) is arrived at – as is the diagnosis of many conditions - based upon:

- (1) Taking a thorough medical history (including questions about any family history of arthritis or unexplained liver disease and family heritage because of a possible genetic component).
- (2) A physical examination.
- (3) Various blood tests that measure the presence of iron overload.
- (4) DNA genetic testing that searches for specific hemochromatosis mutations.

If your doctor suspects hereditary hemochromatosis, they will order a genetic blood test to look for the HFE mutation that is responsible for the disorder.

Let's talk about the standard blood tests first before we talk about the genetic testing...

### Iron Blood Tests

Hemochromatosis often goes undiagnosed because the condition – even though of high iron – sometimes shows up on a blood tests as low hemoglobin, which you also find in cases of iron depletion anemia. In addition, doctors aren't trained to look for the condition but normally treat the symptoms of hemochromatosis as it affects various organs and body symptoms.<sup>1</sup>

Blood tests for **serum iron**, **total iron binding capacity (TIBC)** and **serum transferrin** are good ways to begin the initial screening for hemochromatosis. Any blood testing lab can do these tests for you, and most doctors will order them for you without any problems.

Your doctor must specifically request an **iron series profile** on the lab requisition form otherwise all three of these specific tests might not be done. I cannot tell you the number of times I've advised people to get blood tests for specific markers only to find out later that only half of the requested measures were done.

You can also call Healthcheck USA ([www.healthcheckusa.com](http://www.healthcheckusa.com)) at 1-800-929-2044 to find a blood testing lab near you. Blood testing for a complete iron profile, which includes, serum iron, TIBC and serum ferritin and genetic test kits that you use at home are available from this company. Remember when asking your doctor for iron testing to mention all three tests by name, and also ask for copies of the reports.

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<sup>1</sup> Acton RT, Barton JC, Casebeer L, et. Al. Survey of physician knowledge about hemochromatosis. Genet Med. 2002 May-Jun;4(3):136-41.

Also remember to take these blood tests in a fasting state, preferably in the morning, because that's when you'll get the most accurate results. Over 50% of individuals have elevated serum iron levels after eating, so if the tests are taken on a full stomach, the iron levels that are measured can be elevated even if there are no increased iron stores

Another good test is the **serum transferrin receptor test**, but it is not yet available at most laboratories. If you suspect hemochromatosis, ask for these four tests which help screen for the condition:

- **Total Iron Binding Capacity (TIBC)** - measures how well your blood can transport iron
- **Serum Iron Test**
- **Serum Ferritin** - shows the level of iron in the liver
- **Serum transferrin receptor** (not yet available at most laboratories)

Once your doctor has these tests he can start making some calculations, and then some preliminary conclusions.

The first calculation is to determine the “percent of saturation,” also known as the “transferrin saturation” level. The transferrin saturation measure is calculated by dividing the serum iron results by the TIBC results and multiplying the fraction by 100 to arrive at a percentage.

$$\text{Transferrin Saturation} = 100 * \frac{\text{Serum Iron Concentration}}{\text{Total Iron Binding Capacity}}$$

Now there are various diagnostic cutoff levels for transferrin saturation and serum ferritin, which have varied across studies, that suggest hemochromatosis. In other words, doctors use different decision thresholds for transferrin saturation, serum ferritin level, and their combined results to predict hemochromatosis.

Here's how to interpret the figures.

A normal transferrin saturation measure is between 20% and 50%. Therefore, a transferrin saturation greater than 50% is a warning flag identifying people who may have iron loading. Some doctors have proposed that the screening cutoff point should be 60% for males and 50% for women. Some doctors recommend a lower screening figure of 40%, too, which is a figure we'll focus on.

A percent of saturation figure greater than 40% and/or a serum ferritin greater than 150 ng/ml (>150 ng/mL) is a general set of looser measures that also suggests the condition,

and we have to say “suggests” the condition because not all individuals with these figures have hemochromatosis. That’s why we call it a “warning flag.”

The initial screening levels for hemochromatosis, that suggest further investigation, are definitely still being argued about. I would rather err on the side of caution, so the lower cutoff values seem more logical than the higher. The higher the levels, however, the higher the probability of true hemochromatosis and the lower the cutoff levels, the lower the probability of the condition.

In some studies, higher cutoff levels (transferrin saturation  $\geq 62\%$  and serum ferritin levels  $\geq 500 \mu\text{g/L}$ ) identified a subgroup of individuals in which *all of them* had hereditary hemochromatosis. A set of less stringent criteria (transferrin saturation  $\geq 45\%$  and serum ferritin levels  $> 200 \mu\text{g/L}$ ) identified a group of individuals in which only 11.5% had hereditary hemochromatosis.<sup>2,3,4</sup>

Basically, the combination of an elevated transferrin saturation and an elevated serum ferritin level are together extremely accurate for predicting hemochromatosis.<sup>5</sup>

Therefore, the screening criteria are not conclusive determinants of hemochromatosis, but only prompt one to perform DNA tests that confirm the presence of the gene defects. After all, other factors can raise various blood iron figures, including the measurement of transferrin saturation.

The reason that transferrin saturation is preferred over serum ferritin in the first place is because it’s a more sensitive and specific test than serum ferritin, which can become elevated for a large variety of reasons. Serum ferritin, in a number of cases, can also be normal in some cases of hemochromatosis, or greatly under suggest the amount of iron accumulated in the body.

Since we’re looking for to help zero in on the easily missed condition of hemochromatosis, and since serum ferritin can be abnormally elevated in various conditions such as Gaucher’s disease, congenital cataracts, rheumatoid arthritis, malignancies, hepatitis and liver injury or alcohol abuse, transferrin saturation is the best first measure to focus on when looking for hereditary hemochromatosis.

Individuals with transferrin saturation  $> 40\%$  and serum ferritin  $> 150\text{ng/mL}$  are typically suffering from iron overload/iron storage in the body due to some reason, even if not

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<sup>2</sup> Baer DM, Simons JL, Staples RL, Rumore GJ, Morton CJ. Hemochromatosis screening in asymptomatic ambulatory men 30 years of age and older. *Am J Med.* 1995;98:464-8.

<sup>3</sup> Phatak PD, Sham RL, Raubertas RF, Dunnigan K, O’Leary MT, Braggins C, et al. Prevalence of hereditary hemochromatosis in 16031 primary care patients. *Ann Intern Med.* 1998;129:954-61.

<sup>4</sup> Niederau C, Niederau CM, Lange S, Littauer A, Abdel-Jalil N, Maurer M, et al. Screening for hemochromatosis and iron deficiency in employees and primary care patients in Western Germany. *Ann Intern Med.* 1998;128:337-45.

<sup>5</sup> Feldman: Sleisenger & Fordtran's Gastrointestinal and Liver Disease, 6th ed., 1998 W. B. Saunders Company.

hemochromatosis, and doctors will normally treat them with phlebotomies regardless of whether or not DNA test results confirm a hemochromatosis diagnosis.

Why?

Because if someone is proven to have excess iron in their system, then they are in danger of organ damage and premature death if left untreated. Therefore, they should undergo phlebotomies in order to reduce the iron levels in their body, and we'll discuss this and other options for reducing the body's iron stores in the next chapter.

Now what are the other iron measures that suggest hemochromatosis?

As seen in the chart below, the serum ferritin level will also usually be elevated in patients with hemochromatosis.

<b>SERUM</b>	<b>NORMAL</b>	<b>HEMOCHROMATOSIS</b>
<b>Transferrin Saturation (%)</b>	20-50%	55-100%
<b>Ferritin (ng/mL)</b>		
* Males	20-200	300-3000
* Females	15-150	250-3000
<b>Iron</b>		
* mug/dL	60-180	180-300
* mumol/L	11-32	32-54

## **Genetic Tests**

If these blood tests are abnormally high, then the next step is to perform a genetic test for the mutations in the HFE gene.<sup>6</sup> Typically, anywhere from 85-98% of patients with clinical iron overload will show the presence of the hemochromatosis mutations.

What is this exact mutation?

Hereditary (or genetic) hemochromatosis is mainly associated with a defect in a gene called *HFE*, whose purpose is to help regulate the amount of iron we absorb from the foods we eat. There are two known important mutations in *HFE*, named Cys282Y and His63D, and DNA tests to confirm hemochromatosis look for these specific mutations.

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<sup>6</sup> This gene is the result of a single base change in which tyrosine is substituted for cysteine at position 282 of the HFE protein (Cys282Y).

The DNA gene testing that can confirm hemochromatosis must test for both HFE mutations (Cys282Y & His63D, also known as C282Y and H63D) and is called **HLA-H**, or more commonly the HFE or Hfe test. Remember that if you want these tests, you have to ask for these tests by name – the **Cys282Y and His63D mutation tests**.

Not all labs test for both hemochromatosis mutations so that should be the main question when considering lab tests. These genetic tests are commercially available and cost about \$200.

Cys282Y is the most important defect to look for. When the Cys282Y abnormality is inherited from both parents, dietary iron is usually over-absorbed and hemochromatosis can result. His63D usually does cause a little increase in iron absorption, but a person with His63D from one parent and Cys282Y from the other will rarely develop hemochromatosis.

You must also understand that HLA-typing (the HFE test) is not the same as genetic DNA testing, so be sure not to get confused about some older forms of testing that many labs still offer. Rather, to confirm hemochromatosis, be sure to ask for DNA testing by the specific name of the mutations: **cys282** (pronounced "siss two eighty two) and **his63** (pronounced "hiss sixty-three) and mention you're looking to confirm hemochromatosis, to make sure you are getting the correct test.

If the test results come back positive, then it's also important to test any children for the gene mutations so that you can assess their risks for hemochromatosis before any damage occurs. Early tests can help determine whether there's anyone else in the family who might be at risk for storing excessive iron in the future.

The solution?

Act now!

## **Liver Biopsy**

A patient's physician may also want to test liver enzymes (usually they'll be elevated) and investigate the family history in order to confirm a diagnosis of the genetic disorder.

A liver biopsy can also show exactly how much excessive iron the liver is storing, and the extent of damage to the liver in advanced cases of hemochromatosis. Among other things, the liver biopsy is normally used to diagnose primary liver cancer, and liver cancer is a possible outcome of advanced hemochromatosis.

Depending on whether there is evidence of liver damage, your doctor may suggest a liver biopsy should be done to assess the damage to your liver. In a liver biopsy, a tiny piece of liver tissue is removed and examined under a microscope to reveal how much iron has accumulated in the liver and determine whether the liver is damaged, and to what degree.

Liver biopsies are invasive procedures, and you can tell your doctor that a biopsy might not be necessary if you cite certain research.

You can point your doctor to a 1998 study in *Gastroenterology*<sup>7</sup> that examined 197 French hemochromatosis patients who had the Cys282Y homozygous gene. The purpose of the study was to determine if there were any noninvasive predictors of severe liver fibrosis, which is a complication usually involving cirrhosis.

The study found that simple biochemical and clinical variables -- **serum aspartate aminotransferase, serum ferritin, and hepatomegaly** --were just as predictive as invasive liver biopsies except for the diagnosis of severe fibrosis.

What were the results of this study, and others?

It was derived in a French population and validated in a Canadian population. Only 1 of 105 patients (0.9%) with a ferritin level of 1000 µg/L or less had cirrhosis. In combination with a normal aspartate aminotransferase (AST) level and no hepatomegaly, 0 of 94 patients had cirrhosis. Findings in the validation population were similar. The more recent follow-up report<sup>8</sup> found that ferritin levels greater than 1000 µg/L, platelet counts less than  $200 \times 10^9$  cells/L, and elevated AST levels led to a correct diagnosis of cirrhosis in 77% of the Canadian participants and in 90% of the French participants who were tested. Morrison and colleagues<sup>9</sup> found that patients with ferritin levels less than 1000 µg/L were unlikely to have cirrhosis on liver biopsy (1 of 93 patients). These 3 studies strongly suggest that patients who are at high risk for hereditary hemochromatosis (homozygous C282Y mutation) with serum ferritin levels of 1000 µg/L or less are unlikely to have cirrhosis.<sup>10</sup>

Let's summarize this in English.

A rule to predict the presence of liver cirrhosis (damage sometimes caused by hemochromatosis) has already been developed and validated, and can help your doctor avoid the need for a biopsy. From this research, the following is the profile of an individual *unlikely* to have cirrhosis:

- Serum ferritin level < 1000 µg/L without hepatomegaly and
- Normal AST level

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<sup>7</sup> Guyader D, Jacquelinet C, Moirand R, Turlin B, Mendler MH, Chaperon J, et al. Noninvasive prediction of fibrosis in C282Y homozygous hemochromatosis. *Gastroenterology*. 1998;115:929-36.

<sup>8</sup> Beaton M, Guyader D, Deugnier Y, Moirand R, Chakrabarti S, Adams P. Noninvasive prediction of cirrhosis in C282Y-linked hemochromatosis. *Hepatology*. 2002;36:673-8.

<sup>9</sup> Morrison ED, Brandhagen DJ, Phatak PD, Barton JC, Krawitt EL, El-Serag HB, et al. Serum ferritin level predicts advanced hepatic fibrosis among U.S. patients with phenotypic hemochromatosis. *Ann Intern Med*. 2003;138:627-33. [PMID: 12693884].

<sup>10</sup> <http://www.annals.org/cgi/content/full/143/7/522?ck=nck>

Individuals with a high probability of liver cirrhosis will typically show:

- Serum ferritin levels > 1000 µg/L
- Platelet counts < 200 x 10<sup>9</sup> cells/L, and
- Elevated AST levels

The earlier your diagnosis of hemochromatosis, the less your chances of ever needing a liver biopsy, which just serves to point out the importance of early screening for HH. If you must have a liver biopsy performed, ask your doctor to use ultrasound or CT to help guide the biopsy.

Patients at high risk for liver cancer because of hemochromatosis should be screened periodically with various blood tests<sup>11</sup> for the rest of their lives.

The big moral of the story is that hemochromatosis is not a diagnosis arrived at by one test. First there are blood tests, and then genetic tests looking for the confirmation of gene mutations.

If one is identified early enough, there are various treatments for hemochromatosis that do involve mild discomfort and inconvenience, but an individual can live a long and normal life with these treatments.

So let's turn to the conventional treatments, and then the alternative treatments that can help with this condition.

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<sup>11</sup> Alpha fetoprotein and PIVKA-II

## How Do Doctors Normally Treat Hemochromatosis?

Because hemochromatosis can affect a number of organs in the body, treatment is usually handled by any number of professionals including a hepatologist (liver disorder specialist), gastroenterologist (specialist in digestive disorders), and hematologist (specialist in blood disorders).

Several other specialists may also help in treating the condition including cardiologist (because of the heart problems), endocrinologist, or rheumatologist (because of joint problems). Internists and family practitioners can also treat the disease.

What is the main treatment for hemochromatosis?

Hemochromatosis is conventionally treated through **phlebotomy treatments**, also known as blood letting or donating blood.

In other words, the normal treatment for hemochromatosis is that you donate blood on a frequent, regular basis just like you would do for a blood bank. It's simple, safe and inexpensive. Yes, it's a bit inconvenient and is mildly uncomfortable, but that's a very small price to pay for successful treatment.

Here's how it works. Usually an individual with hemochromatosis "gives" blood (sometimes weekly) until measures of iron stores are reduced to a safe level, and then maintenance donations are given on a less frequent schedule *throughout life*.

If anemia is also present, however, drugs in the form of iron chelators may also be prescribed by doctors.

### Phlebotomies

The number of phlebotomies necessary to "de-iron" the body varies, depending on the severity of the disease discovered at diagnosis. The target of the phlebotomies is to reduce the blood ferritin levels to the very low end of normal and then keep them there.

As stated, phlebotomies are usually needed periodically throughout life in addition to the ones necessary to bring iron down to a safe level within the body.<sup>12</sup>

When someone is identified as having iron overload hemochromatosis, usually one to two pints of blood (which contains iron locked away in the hemoglobin of red blood

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<sup>12</sup> Borch-Iohnsen, Primary hemochromatosis and dietary iron(in Norwegian), Tidsskr Nor Laegeforen (Norway)Oct 10 1997, 117(24)p3506-7.

cells) are initially removed on a weekly basis until the iron stores drop to a normal level. Naturally the phlebotomy treatments (frequency and amount of blood removed) are individualized to each patient and take into account the age, sex, size, weight, and stage of hemochromatosis.

At this rate of phlebotomy therapy, and taking into account the amount of iron absorbed from the diet, patients undergoing two 500-milliliter phlebotomies per week will usually lose about 50 mg of iron per day, or about 18 grams per year. Usually a person loses about 25- 50 micrograms of ferritin per liter of blood serum. On this particular topic, University of Utah researchers wrote:

Massive iron stores of 20 to 30 grams can be normalized in 12 to 18 months of twice-weekly phlebotomy. Because the time-dosage toxic threshold of iron that results in irreversible organ damage is known, iron stores should be completed completely and quickly. Phlebotomy performed at a rate of less than 500 milliliters every month may be counterproductive, as the rate of iron absorption from the diet may exceed the rate of iron depletion.<sup>13</sup>

The entire sequence of blood lettings for hemochromatosis patients may take anywhere from several months to several years to remove much of the excess iron, but if a person is treated early they can look forward to a completely normal life. You only need to look at two studies to get a feel for this:

- In 1976, one study of brain-syndrome patients with hemochromatosis found that those whose iron stores were depleted through phlebotomy or chelation lived an average of 63 months after diagnosis, while untreated patients survived an average of only 18 months.<sup>14</sup>
- In 1985, another study of 163 hemochromatosis patients found that those without cirrhosis or diabetes at the time of diagnosis and treatment lived a normal life expectancy. Of the hemochromatosis patients who underwent iron therapy, 92% were alive after 5 years, 76% after 10 years, 59% after 15 years and 49% after 20 years.<sup>15</sup>

**Remember**, after the iron levels are reduced to normal, the situation isn't over. A hemochromatosis patient still needs to undertake maintenance phlebotomy treatments four to six times each year to prevent a re-accumulation of iron.

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<sup>13</sup> Edwards, C.Q.; Griffen, L.M.; Kushner, J.P. "Disorders of excess iron." *Hospital Practice* 26(3):30-26, April 1991.

<sup>14</sup> Bomford, A and Williams R. "Long term results of venesection therapy in idiopathic hemochromatosis." *Queensland J Med.* 45:611, 1976.

<sup>15</sup> Niederau C, et al. "Survival and causes of death in cirrhotic and non-cirrhotic patients with primary hemochromatosis." *New England J. Med.* 313: 1256, 1985.

This lifetime maintenance is recommended because otherwise, any individual with hereditary hemochromatosis who neglects the blood letting may become iron overloaded again in a few years once again.

Now if the hemochromatosis condition has progressed to a stage where it has caused liver cirrhosis, the situation is more serious than just an accumulation of excess of iron in the system. The statistics show that liver cancer – because of the iron promoting free radical activity - can occur in up to 30% of these patients, which is why those for risk of liver cancer should be periodically tested. Those who have suffered liver damage are also advised to see a good naturopath and nutritionist who can help build up the liver to normal.

Hemochromatosis that damages the pancreas can also result in diabetes mellitus while damage to other organs may cause heart problems, chronic fatigue, joint pain, loss of body hair, loss of libido and infertility or impotence. As previously stated, in some advanced cases of hemochromatosis, the patient may develop an enlarged liver, liver cirrhosis or spleen enlargement.

In addition to the phlebotomies, the conventional treatments for hemochromatosis also include chelation therapy and dietary restrictions for patients. To avoid liver damage, a hemochromatosis sufferer will also usually be told to avoid alcohol.

Additionally, to avoid excessive iron uptake, when doctors are on the ball they will advise sufferers to avoid taking vitamin C with meals. Vitamin C increases your uptake of dietary iron but many internists and family practitioners don't know this to warn patients.

They do know to warn patients to avoid eating iron heavy foods such as breakfast cereals fortified with 100% of the RDA of iron, eating raw shellfish (such as clams, oysters and shrimp), and cooking with cast-iron cookware.

We'll cover all these rules in a subsequent chapter of this book. We'll also cover the fact that a hemochromatosis patient might also choose to drink tea with meals in order to help block the uptake of iron from the foods they eat, and thereby delay the frequency of phlebotomies.

None of the natural treatments to help with hemochromatosis “cure” the condition or get rid of the need for phlebotomies. When effective, they only help delay the need for phlebotomies but they are still necessary.

Although the standard medical treatment cannot cure the conditions associated with established hemochromatosis, it will help most of them. The main exception seems to be arthritis, which does not improve even after the excess iron is removed from the body.

Again, once normal iron levels are re-established for a hemochromatosis sufferer, they can be maintained by periodic blood removal (2 to 6 times a year depending on the individual). The treatment of phlebotomies, or blood donation, will prevent damage that would have been caused by excess iron accumulation in the body, and once again you must remember that this treatment is ongoing for life.

## **Chelation Therapy**

Chelation therapy is also sometimes used in cases of hemochromatosis, though phlebotomies are the recommended first line procedure for removing excess iron from the body.

Let's discuss how chelation works...

In chelation therapy, you're hooked up with an IV solution containing a special chelating chemical agent that, together with other nutrients, circulates throughout all the blood vessels in your body. A chemical chelating agent in the solution grasps certain minerals – such as iron, lead, mercury, cadmium, manganese -- and then escorts most of these toxic metals out of your body through your kidneys as urine.

In chelation therapy, your body gets rid of all sorts of accumulated metallic pollutants (not just iron), radiation particles, and assorted foreign elements that tend to cause cellular breakdown. It's often referred to as a rejuvenating therapy in the natural health field because of all the benefits won through the removal of heavy metals from the body.

If chelation therapy is a second line way to attack the condition, the logical question to ask is, "When is chelation therapy preferred over phlebotomy?"

The answer is: when a hemochromatosis patient suffers from angina or bone marrow suppression. These individuals usually do better receiving intravenous infusions of EDTA or **deferoxamine (Desferal)**, both of which are iron chelators. In cases of iron detoxification or transfusion dependent anemias, most doctors will use Desferal (deferoxamine) for iron chelation.

Remember, even if you'd like an "alternative therapy," doctors usually don't administer chelation for primary hemochromatosis because phlebotomy is the current standard procedure. Yes, iron stores are indeed depleted through chelation therapy, but at a much slower rate than through phlebotomy. The phlebotomies are recommended.

Today there are a variety of oral chelating agents available on the market, and even rectal suppositories of products like Detoxamin ([www.detoxamin.com](http://www.detoxamin.com)) that are used for chelation under a doctor's supervision.

You might want to ask your doctor to check into these various products and their capabilities if you want to try chelation therapy without the trouble of an intravenous drip. This is not a decision for you to make alone, as you cannot depend on alternative therapies for this condition, however much you may like them.

The big thing is to start removing the excess iron from the body, once diagnosed with hemochromatosis, and to prevent a further uptake of excess iron that will contribute to the condition. This means that it's best to eliminate high iron content foods from the diet (Not eliminating iron entirely) and to possibly consume various supplements or food that will bind dietary iron so that it's not available for uptake.

This is where the field of alternative medicine excels with its recommendations, so now on to the dietary recommendations for hemochromatosis patients.

## **The Hemochromatosis Diet**

The main dietary rule to help manage hemochromatosis is simple – decrease your consumption of foods that are rich in iron, but don't avoid iron foods entirely or you're likely to become sick, weak or ill.

Phlebotomy (bloodletting) is the treatment of choice for hemochromatosis and it does remove excess iron from the body. However, when you are undergoing frequent phlebotomies, you should eat a well balanced diet to keep up your strength, or it may be hard to continue your treatment.

That's why you don't eliminate iron completely from the diet. If you do, you'll quickly become weak and your diet will be rather unbearable to maintain.

Hence, you can go “cold turkey” and try to eliminate the foods from your diet that contain iron, but even altering your diet in this way does not cure hemochromatosis or prevent hemochromatosis. You should not do this. Yes, cut down on the frequency of high iron foods, but do not eliminate them entirely if you want to maintain your strength and ability to recover from any of the damage already done by the condition.

Phlebotomies will help you lower your iron load gradually, and as long as you don't fight this tendency to reduce your iron load by always eating high iron foods, you'll gradually reduce your iron stores. The important thing is not to emphasize foods in the diet that contain concentrated amounts of highly absorbable iron.

Now ... can the diet really help?

Sure!

It's a well proven fact that various dietary rules can help individuals manage their iron levels. For instance, dietary changes in a well individual can produce anemia in a very short period of time – in as little as 120 days. They can also produce a state of iron overload, with symptoms appearing in as little as 60 days. Lowering cholesterol in the diet can produce dramatic changes as well.

So what's normal for dietary iron intake?

The RDA (recommended daily allowance) for iron is 10 mg/day for adult men and for post-menopausal women. For pre-menopausal women, the RDA is 15 mg/day.

Because the intestines of a hemochromatosis patient absorb more iron than usual and because they already have high iron stores when they have the condition, they should not exacerbate this problem by constantly favoring high iron foods. The iron absorption rates

from food vary widely - from 1% to nearly 100% -- so the key is not to make a highly absorbable, high iron food a frequent, standard staple of the diet.

What are these iron rich foods?

## **Cutting Down on Iron Rich Foods**

The iron rich foods are just what you'd expect – **red meats** (steak, beef liver, etc.), **poultry, fish** and **seafood** (including clams, oysters, shrimp, etc.).

These are the richest sources of dietary iron. The iron within meats is also problematical because it comes in a form (heme iron) that is more readily absorbed by the body than the iron found in plants.

Now, whether or not it's related to iron consumption, restricting the consumption of red meat has been shown in various studies to reduce the risk of contracting colon cancer.<sup>16</sup> So that's one reason you can tell yourself when you are fighting to cut down on meat consumption, if you choose to do so.

As to seafood, the big rule for hemochromatosis patients is never to eat raw seafood (cooked seafood is okay) if you are suffering from liver damage because it's possible to cause fatal liver infections. Therefore, no raw oysters or clams – especially since they are extremely heavy with iron content.

One should also not drink alcohol if there has been any sort of liver damage as well. Alcohol consumption easily damages one's liver and if the liver is already ill, you don't want to further damage it. Moderate alcohol consumption usually doesn't pose a problem with iron absorption, but excessive alcohol consumption is associated with iron overload.

There are plant foods that are high in iron as well that you should make note of. These include **enriched and whole grain cereals and breads** (such as iron fortified breakfast cereals), **tofu, dry beans** and **peas** (black beans, kidney beans, lima beans, pinto beans, lentils, chickpeas, baked beans, etc.), and **dark green leafy vegetables** (spinach, collard greens, swiss chard etc.).

The iron in these foods is in a form that is not as well absorbed by the body as the iron in meat, poultry, and fish (non-heme iron). Typically, only about 5% of the iron found in plant foods is available to the body versus 30-50% of iron available from red meats.

**Blackstrap molasses** is another food also rich in iron.

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<sup>16</sup> Kampman E, et al. Meat consumption, genetic susceptibility, and colon cancer risk. *Cancer Epid Biomarker Prev* 1999;8:15-24.

Interestingly enough, there are also certain spices that promote iron absorption from the diet. These include: **Olive oil** and spices such as **anise, caraway, cumin, licorice** and **mint**. If you cook with these spices, you are encouraging iron absorption, so you might wish to look at any tendencies in your food recipes to see whether you are contributing to the condition in this way.

Once again, strictly following these various rules does not prevent hemochromatosis. They only help you to manage the condition.

Are there any other related rules related to cooking and the diet that are relevant?

Yes -- **Avoid cast iron or stainless steel cooking pots**, because they will increase the amount of iron you absorb. If you cook food in iron pots, naturally you'll actually increase your consumption of free iron.

And remember -- no fortified processed foods, especially iron fortified breakfast cereals. Check your cereal boxes and grain-food boxes for their iron content. Fortified Raisin Bran cereal is usually a typical high iron cereal, but you have to check the label as it varies by manufacturer.

Remember, strictly following these dietary rules does not prevent the disease. It only helps to manage the condition of hemochromatosis, and that's all.

### **Iron Content of Selected Vegan Foods**

<b>FOOD</b>	<b>AMOUNT</b>	<b>IRON (mg)</b>
Soybeans, cooked	1 cup	8.8
Blackstrap molasses	2 Tbsp	7.0
Lentils, cooked	1 cup	6.6
Tofu	4 oz	0.7-6.6
Quinoa, cooked	1 cup	6.3
Kidney beans, cooked	1 cup	5.2
Chickpeas, cooked	1 cup	4.7
Lima beans, cooked	1 cup	4.5
Pinto beans, cooked	1 cup	4.5
Veggie burger, commercial	1 patty	1.1-4.5
Black-eyed peas, cooked	1 cup	4.3
Swiss chard, cooked	1 cup	4.0
Tempeh	1 cup	3.8
Black beans, cooked	1 cup	3.6
Bagel, enriched	3 oz	3.2
Turnip greens, cooked	1 cup	3.2
Prune juice	8 oz	3.0
Spinach, cooked	1 cup	2.9
Beet greens, cooked	1 cup	2.7
Tahini	2 Tbsp	2.6

Raisins	1/2 cup	2.2
Cashews	1/4 cup	2.0
Figs, dried	5 medium	2.0
Seitan	4 oz	2.0
Bok choy, cooked	1 cup	1.8
Bulgur, cooked	1 cup	1.7
Apricots, dried	10 halves	1.6
Potato	1 large	1.4
Soy yogurt	6 oz	1.4
Tomato juice	8 oz	1.4
Veggie hot dog	1 hot dog	1.4
Almonds	1/4 cup	1.3
Peas, cooked	1 cup	1.3
Green beans, cooked	1 cup	1.2
Kale, cooked	1 cup	1.2
Sesame seeds	2 Tbsp	1.2
Sunflower seeds	1/4 cup	1.2
Broccoli, cooked	1 cup	1.1
Brussels sprouts, cooked	1 cup	1.1
Millet, cooked	1 cup	1.0
Prunes	5 medium	1.0
Watermelon	1/8 medium	1.0

Sources: USDA Nutrient Data Base for Standard Reference, Release 12, 1998.  
Manufacturer's information.

## **Nutritional Supplements and Vitamin C**

Perhaps the biggest mistake people with hemochromatosis make is taking multi-vitamin/multi-mineral supplements that contain iron. That's just adding fuel to the fire, so-to-speak. But before we get to the figures, let's depart from our strict discussion of hemochromatosis a bit, and just talk about regular iron overload and see what the topic suggests to us about meal servings.

In a recent study of elderly Americans,<sup>17</sup> 13% of the participants had high serum ferritin iron stores, which were defined as serum ferritin levels over 300 µg/L in men and over 200 µg/L in women. We're not talking about hemochromatosis - just high iron stores.

Naturally, red meat consumption and the consumption of fruit or fruit juice (because of the vitamin C content, which we'll discuss below) were identified as powerful risk factors for accumulating high iron stores.

Here's where the numbers come in handy for us: people who consumed three or more servings of fruit/fruit juice a day had a much higher risk of high iron stores than those who consumed two servings a day. This gives us our first indication of how much fruit/fruit juice is TOO MUCH for a hemochromatosis patient. If these amounts are too

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<sup>17</sup> American Journal Clinical Nutrition December 2002 76:1375-1384.

much for ordinary individuals because they lead to higher iron stores, they are certainly good guidelines for hemochromatosis patients.

In this study, it was also found that those who consumed more than four servings of red meat a week had three times the risk of high iron stores than participants who ate four servings a week. However, in this study, eating light meats such as poultry and seafood, did not affect the risk which suggests that poultry, even with its high heme-iron content, is a preferable meat than beef in terms of iron uptake.

Once again, it's a matter of frequency and quantity rather than eliminating meats altogether. You don't have to cut out red meat consumption entirely when you have hemochromatosis; it's just that "less is better than more, and some is better than none."

In a Norwegian study of hemochromatosis patients who wished to reduce the frequency of phlebotomies by regulating their diet, the study arrived at the conclusion that the following foods should be avoided:

- Ascorbic acid-rich fruit juice (particularly when taken with meals)
- Ascorbic acid-rich fruit (particularly when taken with meals)
- Alcohol
- Meat (in limited quantities)

This Norwegian dietary study also suggested that hemochromatosis patients follow a diet rich in the following:

- Non-iron fortified Bread and cereals
- Fruits (non-ascorbic acid varieties)
- Fresh vegetables

The consumption of whole grains was also found to decrease the risk of accumulating high iron stores in the body. Those who consumed more than seven servings of whole grains per week had a 77% lower risk of high iron stores than those who did not eat whole grains. Researchers speculated that this association was due to the inhibitory effect of fiber on the absorption of non-heme iron, which we'll also discuss in time, so keep that fact in the back of your mind ...

Now here's the major point I wanted to get to: one of the most significant risk factors for developing high ferritin levels was consuming iron-containing supplements, which brings us back to hemochromatosis again. Those individuals who consumed between 12 and 30 mg of iron a day, which is an amount commonly found in multivitamins, had the strongest risks of high iron stores.

The moral of this all?

Don't take a multivitamin, multi-mineral supplement that contains iron if you suffer from hemochromatosis!

If you are taking a multivitamin, go right now and check if the label contains iron. If so, it would be prudent to replace your supplement with an iron free brand. The least thing you want to be doing right now is supplementing with the mineral whose accumulation may kill you!

Another thing to do is cut down on your total vitamin C consumption that you get through supplements because vitamin C increases iron absorption. If you wish to continue taking vitamin C supplements, take it between meals on an empty stomach. In that way, it will not increase dietary iron absorption.

Remember, you cannot and should not try to eliminate vitamin C entirely from your diet altogether. What a recipe for catastrophe. There are lots of reasons for this:

- Vitamin C is needed for many body process – a deficiency will cause scurvy.
- In particular, published research shows that vitamin C acts as an antioxidant against lipid peroxidation (fats spoiling) when the blood is iron rich.
- While vitamin C does improve the absorption of dietary iron, it's also required to move iron out of ferritin tissue stores, so it's needed to help remove iron out of your tissues naturally. In other words, if you don't have enough antioxidants such as vitamin C and E, the iron in ferritin tissues cannot be transferred onto transferrin plasmin.

So you do not eliminate your intake of vitamin C, you just learn to be cognizant of it and not overdo vitamin C consumption. That's where people make the error.

Many people do not realize that with all the supplements they are taking (usually people take them with meals), they may be ingesting several thousand milligrams of vitamin C that increases iron absorption!

For one client I had, we added up the total amount of vitamin C they were taking on a daily basis through of all the supplements they were consuming, and found several thousand milligrams per day! Simply by cutting down on these supplements, we were able to cut down on his iron absorption and return things to normal in a short period of time.

Once again, don't take this rule to an extreme and avoid vitamin C altogether. You can and should eat vitamin C-containing fruits and drink fruit juices, but do this away from meals that contain a high concentration of iron. Also, don't overdo your consumption of these foods or supplements.

Vitamin C should not be avoided altogether because of its many benefits, including the fact that studies show vitamin C consumption is associated with a decreased risk for heart disease, cancer, cataracts and other disorders. You can check into the Linus Pauling Foundation for more information on the many benefits of vitamin C.

In short, the two big supplement rules are:

1. Reduce your intake of vitamin C – supplements and fruit/fruit juices – with meals. Rather, take any fruits or juices containing vitamin C apart from meals.
2. Reduce your intake of iron supplements

If you follow these rules, you're doing a lot to limit your iron absorption and helping to reduce the frequency of phlebotomies, which most hemochromatosis patients complain about.

Are there other dietary rules to follow that can help?

That's what we're covering next...

## **Using Calcium and Other Foods to Block Iron Absorption**

One of the other ways of lowering the amount of iron you absorb from food is to interfere with its absorption by eating other substances at the same time. You can do this by ingesting fiber and calcium with your meals.

Let's discuss calcium's role in this first.

Calcium interferes with iron absorption. It's a plain and simple fact, and therefore a strategy you can use to help with hemochromatosis.

The American Journal of Clinical Nutrition stated that if you eat 300 mg of calcium with a meal, you would reduce the amount of iron you normally absorb by 40%. Therefore, this is a simple way to help reduce the iron in your blood,<sup>18</sup> though once again it does not eliminate the need for phlebotomies.

How do you obtain 300 mg of calcium with a meal?

Simple – just take a calcium supplement that contains 300 mg of elemental calcium, which you can easily determine by reading supplement labels. According to published scientific studies that took accurate measurements, the maximum amount of calcium that will inhibit iron absorption is around 300 mg with each meal.

In other words, if you take more than 300 mg of calcium with your meal, it won't cause any additional interference with iron absorption. Therefore, only 300 mg is necessary; taking more is of no benefit in terms of blocking or interfering with iron absorption.

**M**ost calcium supplements tell you how much elemental calcium they provide on the label, so all you have to do is look at the label to see what it says.

If you don't even want to look at labels, the easiest way to provide about 300 mg of elemental calcium is to take one or two 1000-mg capsules of calcium citrate with every meal that contains iron. So if you plan on eating red meats with lots of iron, you would supplement your meal with a calcium tablet.

The reason I recommend 1000-mg calcium citrate capsules is because each one provides about 220 mg of elemental calcium. Other types of calcium and other calcium

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<sup>18</sup> Hallberg L, Does calcium interfere with iron absorption? Am J Clin Nutr 1998 Jul;68(1):3-4.

supplements will provide a different amount of calcium per capsule dosage, which you can usually read off the label.

**CAUTION:** Calcium consumption is not a strategy that you can count on using forever because some people become tolerant to calcium-induced, iron-absorption blockage after several months. In other words, it may stop working over time as your body gets used to it, which means you have to test to see if it's working by having regular blood tests that monitor your iron levels.

Drinking milk is also known to inhibit the absorption of iron. Part of the reason is because milk contains calcium, but another reason is because it contains lactoferrin, which tends to bind iron as we'll find in another chapter.

Are there any other substances that can inhibit the absorption of iron like calcium does?

Sure. There are a whole list of foods that help limit iron absorption because they bind to it or interfere with its uptake:

- Soluble fibers such as psyllium seed husks (Metamucil), guar gum, and the pectins also help to block iron (and other mineral) absorption.
- Antacids, eggs and soy are known to reduce the availability of dietary iron.
- Milk and dairy products (Cheese and yoghurt) because they contain lactoferrin.
- Carbonates, oxalates, and phosphates are also iron blockers and the foods that contain them include, cranberries, rhubarb, spinach, and soda.
- Phytic acid (a component of whole grains and seeds such as sesame) binds to iron and other minerals in the gastric tract and help to limit iron availability; eating whole grain breads and cereals therefore helps bind dietary iron and reduce its absorption from foods.
- Bioflavonoids (found in berries, coffee, green tea, pine bark, quercetin and the rind of citrus fruits, particularly blueberry, cranberry, elderberry and grape seed) also tend to bind to iron and prevent its uptake into the body.

The benefit of eating foods rich with bioflavonoids and phytic acid is that if these substances don't bind to minerals in the digestive tract, they will get absorbed into the bloodstream. Once in the bloodstream, they will tend to bind to any free iron they find and act as iron chelators for the blood.

Therefore, just as we want to consume any vitamin C and fruits apart from meals, it's good to know that the maximum iron chelation abilities of these other foods for the blood is also achieved when these iron binders are consumed apart from meals.

So remember this little fact and use it to your benefit.

## **Avoiding Extra Manganese**

While we're discussing the mineral calcium, a word about **manganese** and iron ...

The absorption of iron in the body is actually dependent on the mineral manganese (Mn). Many minor iron-deficiency situations can actually be reversed by manganese supplements without any need for iron supplementation! Therefore, the presence of manganese in your nutritional supplements when you have hemochromatosis is not necessarily a good thing because it might help to increase your iron absorption.

Now some research studies say that manganese supplementation can protect against the free radical damage from excess iron<sup>19</sup> and some sources claim that manganese lowers iron levels, but this is mostly a theoretical consideration that would only happen under unusual circumstances.

In actual clinical settings, you'll rarely if ever see a patient's iron (ferritin) levels decline as a result of taking a manganese supplement, even when very high doses of manganese are taken on an ongoing basis. This is still a matter under study by researchers, however, nevertheless I thought it pertinent to bring to your attention.

The point is that if you wish to take a multivitamin supplement, take a good look at the label for its iron, calcium, vitamin C and manganese contents.

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<sup>19</sup> *Free Radical Biology and Medicine*, 1992; 13: pp.115-20.

## Tea Drinking

Another option for reducing your iron absorption is drinking tea with each meal.

That's right – drinking tea!

A study in the British medical journal *Gut* revealed that drinking **tannin-rich black teas** with meals can reduce your iron absorption, and thus tea drinking is of benefit to those with hemochromatosis.

In the study, a control group of individuals drank water with their meals. The other group drank tea with their meals. Researchers then measured the intestinal iron absorption for each group by studying the test subjects' blood figures for serum iron binding capacity and serum ferritin.

The results?

There was a significant reduction in the iron absorption for the tea drinking group as compared to the water drinkers.

Also, in 1982 another study<sup>20</sup> was performed designed to determine the effect of various drinks on iron absorption. Test subjects were fed a standard meal consisting of a hamburger, string beans, mashed potatoes and water.

When **green tea** was substituted for the water, measurements showed that iron absorption was reduced by 62%. Milk and beer seemed to have no effect on iron absorption in this study (a different finding than in other studies, as the milk would normally limit iron consumption), but **coffee** reduced iron absorption by 35%.

As one would expect, orange juice – because it contains vitamin C – had the opposite effect and actually increased iron absorption by 85%. We already know that vitamin C usually increases iron absorption, which is why hemochromatosis patients should limit their intake of vitamin C in nutritional supplements.

In another British study, the inhibition of iron uptake by black tea was found to be 79-94%, peppermint tea 84%, pennyroyal 73%, cocoa 71%, vervain 59%, lime flower 52% and chamomile 47%.<sup>21</sup>

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<sup>20</sup> Hallberg L, Rossander L. Effect of different drinks on the absorption of non-heme iron from composite meals. *Hum Nutr Appl Nutr* 1982;36:116-23.

<sup>21</sup> Hurrell RF, Reddy M, Cook JD. Inhibition of non-haem iron absorption in man by polyphenolic-containing beverages. *Br J Nutr*. 1999;81(4):289-295.

The logical strategy that falls out of these studies is to use the drinking of coffee or tea with meals to slow your iron uptake and thereby reduce the frequency of phlebotomies necessary to maintain your health. In delaying the absorption of iron, this makes it easier for hemochromatosis patients to manage their rate of iron absorption from foods.

Nevertheless, the frequency for phlebotomies is something that your doctor should determine even if you choose this routine, and should not be something you choose yourself.

These strategies only have the possibility of helping to prolong the gap of time between which one needs to give blood, but whether they actually work in this way has to be confirmed by blood tests.

Because green tea was found to be a potent iron-chelating agent, some nutritionists even recommend that you consume green tea extracts when you have hemochromatosis. This is based on a study where An extract of green tea taken by healthy women with a meal inhibited the absorption of non-heme iron (the form of iron found in plant foods, dairy products and iron supplements) by 26%.<sup>22</sup>

This is also a strategy you might consider, and based on studies, you'd want to be taking beverages or capsules that contain between 100-400 mg total polyphenols per serving in order to reduce iron absorption by 60-90%.

Actually, most people find that drinking tea seems to be a better and more enjoyable option than eating capsules, and it's the option encouraged when patients have hemochromatosis.

The time old thinking since Hippocrates is that it's usually better to manage health conditions through food rather than medicines or supplements, whenever possible, and because tea drinking is so easy and proven, versus taking capsules, it's I would recommend. However, the choices as always are up to you.

There are other supplements you can take to also help bind iron, and we're on to those next...

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<sup>22</sup> Samman S, Sandstrom B, Toft MB, et al. Green tea or rosemary extract added to foods reduces nonheme-iron absorption. *Am J Clin Nutr* 2001;73:607-12.

## Special Supplements

Another way to cut down on iron absorption is to consume various nutritional supplements that bind iron in your system.

Now you can do this by drinking teas or eating special foods, and this is the dietary approach to helping control your iron intake. That's the approach I personally favor.

Nevertheless, there are also special supplements you can take that have this desired property. Calcium, as you know, is one of them. IP-6 is another.

### IP-6 (Inositol Hexaphosphate)

The substance IP-6 (inositol hexaphosphate), also known as phytic acid, is made from rice bran. What is it? IP-6 is comprised of six phosphorus molecules and one molecule of inositol, which is a member of the B vitamin complex.

For years researchers ignored IP-6 because it impairs mineral absorption, but in our case that's exactly what we want. It's ability to bind to minerals makes it a very cheap and non-toxic form of iron chelation that can be done without a doctor's prescription, and it has some positive side benefits as a nutritional supplement as well, especially in the treatment of cancer.

Why?

Because it binds iron, and cancer cells need a lot of iron to replicate DNA when they divide in order to survive.

Now there's a little bit of confusion about IP-6, or phytic acid. When you supplement with phytic acid, which is derived from rice bran extract, it will bind to iron and other minerals in your digestive tract when you take it with food. When you take it apart from meals, however, it will enter your bloodstream and bind to any free iron and other minerals in the blood,<sup>23</sup> and then be eliminated through the kidneys as urine.<sup>24</sup>

Previously we mentioned chelation therapy as a way of binding iron and escorting it out of your system. Researchers speculate that phytic acid's ability to bind to iron will help it someday replace intravenous chelation therapy with EDTA or desferrioxamine (Desferal). That's how good it is at sequestering iron, which can come in handy during an infection when you want to deny iron's availability to bacteria, which need it to grow and thrive.

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<sup>23</sup> Phytic acid removes only excess or unbound minerals, not mineral ions already attached to proteins.

<sup>24</sup> [No authors listed] Phytic acid: new doors open for a chelator. *Lancet* 1987 Sept 19;2(8560):664-6.

You cannot just run out and start taking IP-6 carelessly. If you ever choose to design any nutritional supplement routine using the information provided such as this, always discuss things with your physician. They'll know how much you can take of any supplement, and will be able to monitor your condition to make sure you don't get into trouble.

The problem is that if IP-6 supplements are taken for more than several months then fatigue can set in as a result of iron deficiency!

As to other dangers, women should never take phytic acid supplements during pregnancy since it will deprive the developing fetus from the minerals necessary for healthy growth.

If someone with anemia takes phytic acid, they are also likely to feel weaker after the supplementation.

Some hemochromatosis patients take aspirin for their condition because aspirin produces a small loss of blood per day that consequently helps to control iron levels. However, you should never simultaneously use phytic acid with a daily aspirin regimen either.

Usually a three-month course of phytic acid will produce a quite noticeable amount of iron chelation, and for normal individuals prolonged daily supplementation may actually lead to iron-deficiency anemia. While anemic individuals who take phytic acid are likely to start feeling weak shortly after they begin consuming it, iron-overloaded individuals (such as hemochromatosis sufferers) are likely to feel increased energy.

A benefit of IP-6 is that it can help with cancer, which is one of the worries of hemochromatosis patients. IP-6 can more get into cancer cells to chelate iron than the pharmaceutical drugs designed to do so. So while I know of no studies done specifically on this, IP-6 supplementation might be one way for your doctor to help prevent liver cancer in advanced cases of hemochromatosis with liver damage. Discuss it with your doctor.

Regardless of its many benefits, remember that supplemental IP6 may slow down the amount of iron being absorbed from the digestive tract, but only specially formulated drugs or blood loss can remove iron from the body.

Tsuno Food & Rice Company of Wakayama Japan is the only manufacturer of IP6 in the world. Since any brand you purchased would ultimately come from this company, purchasing the least expensive brand at a store such as [www.iherb.com](http://www.iherb.com) is probably the way to go to get the best deal.

## **Lactoferrin**

Lactoferrin, which is a glycoprotein found in milk (and whey protein), has often been mentioned as a possible supplement to help bind iron and withhold it from bacteria during infections.

Lactoferrin belongs to the family of biological entities called cytokines, which coordinate the body's cellular immune defenses that protect us from most infections, tumors and cancers. Cytokines also boost the activity of T-cells and stimulate production of immunoglobulins within our bodies.

Since hemochromatosis sufferers are subject to higher incidents of infection and possible cancers, and since it binds iron, these various properties make lactoferrin of interest to us, including the fact that it is readily available as a nutritional supplement.

In particular, lactoferrin has the ability to bind 300 times more iron than serum transferrin. In studies, lactoferrin has also been found to remove free iron from synovial fluid aspirated from the joints of rheumatoid arthritic patients.<sup>25</sup>

Because of these properties, researchers have been working on producing recombinant human lactoferrin, which is indistinguishable from natural breast milk lactoferrin with respect to its iron binding properties, and it is now available.<sup>26</sup> Of course natural lactoferrin is available as well, but pharmaceutical firms cannot make money on natural supplements because they don't offer the possibility of patents. The whole idea behind producing a synthetic lactoferrin in the first place was to create a product that could become an addition to the de-ironing pharmaceutical products already available.

The question this begs is whether hemochromatosis sufferers should supplement with lactoferrin for its various benefits. As of today, no studies have yet been done, so it's a matter for the researchers and doctors to ponder. You can ask your doctor about this possibility, and when studies are done it might soon be recommended as an addition to the standard medical protocols for hemochromatosis.

And if we want to look at other approaches to hemochromatosis that only doctors can recommend, time to turn to the next chapter ...

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<sup>25</sup> Guillen C, McInnes IB, Kruger H, Brock JH. Iron, lactoferrin-and iron regulatory protein activity in the synovium; relative importance of iron loading and the inflammatory response. *Ann Rheum Dis* 1998;57:309-14.

<sup>26</sup> Ward PP, Piddington CS, Cunningham GA, Zhou X, Wyatt RD, Conneely OM. A system for production of commercial quantities of human lactoferrin, a broad spectrum natural antibiotic. *Biotechnology* 1995;13:498-503.

## **Aspirin and Exercise**

The following strategy is not one that I recommend, but one which I feel you need to know about.

Most people know that some doctors will recommend taking an aspirin a day to prevent heart attacks and strokes. What happens though, is that taking an aspirin every day causes a small amount of blood loss via the digestive tract. It's a tiny amount on the order of about a tablespoon per day.

This blood loss results in iron loss, and by taking a baby aspirin per day, Raymond Hohl, M.D. (an assistant professor of internal medicine and pharmacology) at the University of Iowa says even chronic use of a baby aspirin may help to control iron. But remember, taking an aspirin a day also poses dangers, such as inducing iron-deficiency anemia.<sup>27</sup>

By exercising, a person also loses about 1 mg of iron through sweat.<sup>28</sup> Many men and women who engage in regular, intense exercise such as jogging, competitive swimming, and cycling end up having marginal or inadequate iron status, so exercise (if your doctor says so) may be one way to help control iron, too.

The possible explanations for this iron deficiency in athletes include increased gastrointestinal blood loss after running and a greater turnover of red blood cells. Also, red blood cells within the foot often rupture while running. Anyway, this is something that researchers must study before doctors will recommend exercise as a way to help control iron overload.

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<sup>27</sup> Bankhead C. In assessing anemia, doctors must decipher role of iron deficiency. *Med Tribune Clin Focus* 1997 Mar; 20:24.

<sup>28</sup> Vellar OD. Studies on sweat losses of nutrients. *Scand J Clin Lab Invest* 1968;21:157-67.

## Summary

All in all, you have to know that if hemochromatosis is discovered early before major organ damage has been done, with phlebotomies one can restore iron levels to normal and you are likely to live a very long life.

Nevertheless, blot letting (phlebotomy) does not cure hemochromatosis. You need to continue maintenance blood donations throughout life to prevent iron from rebuilding to dangerous levels.

A number of other practices can help reduce your iron intake, and thus reduce the frequency of phlebotomies once the basic condition is under control. These include:

- Dietary rules, such as reducing the consumption of red meats and seafood that contain highly absorbable heme iron, and reducing the intake of iron fortified foods such as breakfast cereals. However, one should not to try entirely restrict the consumption of iron from the diet.
- Reducing the consumption of vitamin C containing fruits and juices with meals, and eating them only between meals
- Reducing the intake of vitamin C supplements
- To stop cooking with iron cookware
- Eliminating iron and manganese supplements from the diet
- Eating foods that help to bind iron such as milk, dairy products, soluble fibers, antacids, eggs, soy, cranberries, rhubarb, spinach, whole grain breads and cereals, and blueberries
- Drinking green or black tea with meals to help bind iron
- Taking calcium supplements with meals to help bind iron (or green tea capsules)
- Changing cooking recipes that use spices such as anise, caraway, cumin, licorice and mint that increase iron uptake
- Checking with your doctor about the possibilities of using aspirin, IP-6, and substances like lactoferrin to bind iron

There are a variety of other rare sources or situations of iron consumption we haven't covered such as inhaling amosite, crocidolite, or tremolite asbestos, mining iron ore,

welding, grinding steel, or painting with iron oxide powder or working with iron oxides. Even cigarette smoking is a source of iron, because about 1-2  $\mu\text{g}$  iron are inhaled per cigarette pack.

All these things contribute to iron ingestion, and thus iron overload. The point is to decrease your exposure to iron in any form you can but remember not to totally eliminate iron from your diet. Let the phlebotomies do their work, and work with your doctor and nutritionist/naturopath to help regenerate any damage to organs already done because of iron overload.

I hope this has been a helpful primer on hemochromatosis and helping you navigate these waters. If you need more help, you can turn to the following organizations that can provide more information on this condition.

## **For More Information Contact These Organizations**

### **American Hemochromatosis Society Inc.**

4044 West Lake Mary Boulevard  
Unit #104, PMB 416  
Lake Mary, FL 32746-2012  
Phone: 1-888-655-IRON (4766) or 407-829-4488  
Fax: 407-333-1284  
Email: [mail@americanhs.org](mailto:mail@americanhs.org)  
Internet: [www.americanhs.org](http://www.americanhs.org)

### **American Liver Foundation (ALF)**

75 Maiden Lane  
Suite 603  
New York, NY 10038-4810  
Phone: 1-800-465-4837,  
1-888-443-7872,  
or 212-668-1000  
Fax: 212-483-8179  
Email: [info@liverfoundation.org](mailto:info@liverfoundation.org)  
Internet: [www.liverfoundation.org](http://www.liverfoundation.org)

### **Iron Disorders Institute Inc.**

P.O. Box 2031  
Greenville, SC 29602  
Phone: 1-888-565-IRON (4766) or 864-292-1175  
Fax: 864-292-1878  
Email: [publications@irondisorders.org](mailto:publications@irondisorders.org)  
Internet: [www.irondisorders.org](http://www.irondisorders.org)

### **National Organization for Rare Disorders Inc.**

55 Kenosia Avenue  
P.O. Box 1968  
Danbury, CT 06813-1968  
Phone: 1-800-999-6673 or 203-744-0100  
Fax: 203-798-2291  
Email: [orphan@rarediseases.org](mailto:orphan@rarediseases.org)  
Internet: [www.rarediseases.org](http://www.rarediseases.org)