“Reflect on your present blessings, of which every man has many; not on your past misfortunes, of which all men have some.”

Charles Dickens, author

**IRON AND DIABETES: AN OVERVIEW**
By Cheryl Mellan
Marietta, OH

Hereditary hemochromatosis is recognized as a very common inherited disorder. It is defined as a disorder of varying penetrance in which patients can display a wide range of clinical signs and symptoms caused by mutations of genes that control iron metabolism. Inappropriately increased intestinal iron absorption leads to progressive body iron accumulation and the generation of oxidative stress in tissues. This results in significant cellular damage, induction of inflammation, and fibrosis. Liver cirrhosis, hepatocellular carcinoma, diabetes mellitus, and cardiac insufficiency are diagnosed in the advanced stages of the disorder. Early diagnosis and treatment protecting against disease progression and multi-organ insufficiency is possible.

Primary iron overload (inherited) and secondary iron overload (acquired) have similar consequences, which make it essential to determine the cause of iron overload for a complete diagnosis, and determination of an appropriate treatment plan which includes initial therapy as well as preventative monitoring. Iron overload is confirmed with a combination of clinical and laboratory findings often prompted by abnormal routine blood work and the patient’s complaints of symptoms.

Although symptoms of excess iron are rarely specific, chronic fatigue is among the first complaints of patients with iron overload whether the cause is primary or secondary. Besides chronic fatigue, individuals with excess iron due to hereditary hemochromatosis are often seen with non-inflammatory joint pain and hypogonadism early on, with liver disease, diabetes and cardiac problems later. Conversely, a thalassemia major patient with iron overload due to hemolysis and transfusion dependence might initially display heart trouble and hypogonadism, with liver disease and diabetes thereafter.

Diabetes mellitus represents a group of disorders that have one common feature: abnormally high levels of glucose (sugar) in the blood. Normally, blood sugar levels are kept within a narrow range by several hormonal and neuronal mechanisms, especially by the hormone insulin, which is produced by the beta cells of the pancreas. Beta cells are found in specialized clumps of cells in the pancreas called islet cells. When defects in insulin production, insulin action, or both are present, high blood sugars can result.

Iron can cause damage to tissues of vital organs by changing metabolism, awareness and facilitation

I met Claudette Goulbourne, (See page 2.) a remarkable lady who has beaten the odds, at a health fair sponsored by the James R. Clark Memorial Sickle Cell Foundation in Columbia, SC. Claudette was volunteering her services that day. I later had the opportunity to chat with her during lunch, and hear her triumphs and challenges as a person with sickle cell disease, and also her thoughts on the Exjade™ clinical trial that she is currently participating in. Meeting Claudette was definitely a pleasure as well as motivation to continue the work needed to educate the minority community on iron related diseases. Sheila Dogan Director, Minority Health Issues
Iron Disorders Institute

**IDI’s mission is to reduce pain, suffering and unnecessary death by disorders of iron through education, awareness and facilitating research**

**THE GENETIC COUNSELING CORNER**
Lisa Kessler

**IRON PATIENTS – THEIR OWN STORIES – SCA**
Claudette Goulbourne

**STAFF & VOLUNTEER PROFILES**
Cheryl Mellan

**IRON PATIENTS – THEIR OWN STORIES**

5 LINKS TO DIABETES

**IDI’s way of keeping in touch with you!**

**What tests can be used to determine pre-diabetes?**
See page 4.
Benefits to having HFE mutations

As many of you know, being a carrier of an HFE gene mutation is quite common. 1 in 10 people in the US carry a gene change related to hemochromatosis. The DNA Direct site contains more statistics about carrier frequency and lots of information about hemochromatosis.

http://genesanddrugs.dnadirect.com/patients/tests/hemochromatosis/should.jsp

If they are so common, then how did these gene mutations develop?

The story is similar to another well known genetic condition. You may already be aware of a connection between sickle cell anemia and an unrelated benefit. Folks who are carriers for this genetic condition have protection against malaria.

So where did the HFE mutations come from? Several researchers, including Dr. Eugene Weinberg, support the following theory:

During the past 15-20 centuries, there were countless outbreaks of plague, tuberculosis and typhoid fever. These medical conditions thrive on iron-loaded macrophages (a type of white blood cell that takes in foreign material). Interestingly, individuals with hemochromatosis have low iron in their macrophages which made them less susceptible to plague, TB and typhoid fever, allowing them to survive these specific illnesses.

According to Sharon Moalem, a neurogeneticist and evolutionary biologist “many genetic diseases may have been selected for as evolutionary compromises that have helped human survival” (NewScientist, 2007)

Learning how these gene mutations survived is fascinating, but how about for the present and for looking forward, what other benefits are there?

There are both biological and psychosocial benefits. One recent study found that patients who have hepatitis C virus infection and have HFE mutations have a significantly better response to treatment compared to those with hepatitis C without HFE mutations.

Some patients have told me that there are other benefits to having genetic conditions such as stronger family bonds and a deeper appreciation for everything in life. Other patients have said that they developed lasting relationships with friends with the same condition who they otherwise would not have met.

Have you ever thought of any positives that have come out of having hemochromatosis? This is a particular area of interest for me, and I would love to hear about them. As always, I am also available to help with your genetics questions, so please feel free to contact me at:

lkessler@dnadirect.com
Or
1-877-321-0077

Lisa Kessler is a certified genetic counselor with DNA Direct; http://www.dnadirect.com

IRON PATIENTS – THEIR OWN STORIES

“Education is the most powerful weapon which you can use to change the world.”

CLAUDETTE GOULBOURNE
SICKLE CELL ANEMIA AND EXJADE™

I am presently participating in a four year oral chelation study because of very high iron overload resulting from blood transfusions for sickle cell anemia. Monthly transfusions were the solution to relieve years of experiencing acute chest syndrome, which later developed into pulmonary hypertension. It was imperative that I do something to relieve my system of this excess iron before the iron damaged any of my organs.

My physician suggested using a Desferal® infusion for removing the excess iron. My thinking at that time was that Desferal® was invasive and too time consuming. My physician continued to stress the danger that existed because of high iron level. I was later informed about a study by Novartis for the oral chelation ICL670, which seemed much more reasonable to work with because it is taken orally rather than intravenously.

I realize that every study has its pros and cons, which must always be considered. Some of my concerns were changes in vision, hearing, and possible kidney and liver damage. I have noticed that over the years I have been in the study there have been changes in my vision and hearing.

I am tested every six months to determine if organ damage has occurred from the Exjade™. My weight is measured so that the appropriate dose of Exjade™ can be administered. Additionally, I have monthly blood work to measure decreases in iron.

I know I have benefited from this study because my iron overload [ferritin] has decreased more than two thousand points. However, because I must continue with chronic blood transfusion, my concern is whether my ferritin level will be normalized.

The "side effects" are what I like least about the study. However, sometimes it seems like a double edged sword – one must take this to treat one problem but what can develop from taking it? Four or five other problems! Maybe!

The FDA has finally approved Exjade™ for treating sickle cell patients who are suffering from iron overload. Although Exjade™ has been approved, the study is ongoing. All the patients in the study are given the option to continue, and I have chosen to remain on the study. This is the only way I can determined what the long term effects will be when this has been accomplished, so that many more patients will benefit.

VISIT THESE WEB SITES TO LEARN MORE ABOUT SICKLE CELL ANEMIA

The James R. Clark Memorial Sickle Cell Foundation
http://www.midnet.sc.edu/jrcsc/

The American Sickle Cell Association
http://www.ascaa.org

How long does a person with sickle cell disease live?

See answer on page 14.
oxygen into a form known as a “free radical”, which leads to increased oxidative stress. Free radical activity can wreak havoc on cells throughout the body. It may be possible that this is how iron destroys the beta cells, causing diabetes. Beta cells or islet cells have very low levels of the enzymes that break down free radicals. Hence any agent that increases free radical production – including iron – could result in destruction of pancreatic cells.

The liver plays a unique role in controlling carbohydrate metabolism by maintaining glucose concentrations in a normal range. This is achieved by a tightly regulated system of enzymes and kinases regulating either glucose breakdown or synthesis in hepatocytes. This process is under the control of glucoregulatory mediators among which insulin plays a key role. In type 2 diabetes, as well as in liver disease, alterations in hepatic glucose metabolism like an increased post-absorptive glucose production together with diminished glucose uptake following carbohydrate ingestion occur, implying insulin resistance as a central pathological principle. Among its functions, the liver produces bile, which emulsifies fats; metabolizes carbohydrates, and filters harmful substances such as ammonia, alcohol, drugs, and toxic chemicals. The liver stores cholesterol, vitamins and clotting factors. Everything we eat or drink, including many medications, is processed by the liver. Our livers can become diseased in several ways. Viral hepatitis, alcoholism, drugs, exposure to toxic chemicals, excessive consumption of fats, nonalcoholic steatohepatitis (NASH), diabetes, defects in metabolism, and excessively high iron levels as are sometimes seen in hemochromatosis can damage this organ. Although steatohepatitis can be induced by an excessive intake of alcohol, it can also arise through various other causes, in which case it is known as non-alcoholic fatty liver disease (NAFLD). NAFLD is classified into two categories: simple fatty liver with a favorable clinical outcome, and non-alcoholic steatohepatitis (NASH), which is intractable and progressive. The liver is the major site for storage of excess iron in the body. Any process or organ dependent upon liver function is then compromised.

Metabolic syndrome is a cluster of conditions that occur together, increasing your risk for heart disease, stroke and diabetes. Having just one of these conditions — increased blood pressure, elevated insulin levels, excess body fat around the waist or abnormal cholesterol levels — contributes to your risk of serious disease. In combination, your risk is even greater. Because insulin resistance tends to run in families, we know that genes are partly responsible. Excess weight also contributes to insulin resistance because too much fat interferes with muscles’ ability to use insulin. Lack of exercise further reduces muscles’ ability to use insulin. Many people with insulin resistance and high blood glucose have excess weight around the waist, high LDL (bad) blood cholesterol levels, low HDL (good) cholesterol levels, high levels of triglycerides (another fat in the blood), and high blood pressure, all conditions that also put the heart at risk. This combination of problems is referred to as the metabolic syndrome, or the insulin resistance syndrome (formerly called Syndrome X). Doctors have talked about this constellation of risk factors for years and have called it many names. Whatever it’s called, it’s becoming very prevalent.

According to the Mayo Clinic, tobacco and alcohol use, often issues of interest to hemochromatosis patients are also factors that can be associated with diabetes. The pancreas releases the hormone insulin, which helps regulate blood sugar (glucose). Heavy alcohol use can cause chronic inflammation of the pancreas (pancreatitis). This can lead to permanent damage to the pancreas and impair its ability to secrete insulin, which can result in diabetes. Smoking not only increases the risk of pancreatic cancer, but can also increase blood sugar levels and reduce your body’s ability to use insulin. In addition, the chemicals in tobacco can damage blood vessels, muscles and organs. This may also increase your risk of diabetes. Pregnant women who smoke have an increased risk of diabetes during pregnancy (gestational diabetes). Both alcohol and tobacco enhance iron absorption and contribute to iron in excess.

You have a higher risk for diabetes if you have any of the following:
- Family history of diabetes
- Hemochromatosis
- Low activity level
- Poor diet
- Excess body weight (especially around the waist)
- Age greater than 45 years
- High blood pressure
- High blood levels of triglycerides (a type of fat molecule)
- HDL cholesterol of less than 35
- Impaired glucose tolerance (identified by your doctor)
- Diabetes during a previous pregnancy, or a baby weighing more than 9 pounds
- Certain ethnicities -- African-Americans, Hispanic-Americans, and Native Americans all have high rates of diabetes

Everyone over 45 should have their blood glucose checked at least every 3 years. Regular testing of random blood glucose should begin at a younger age, and be performed more often, if you are at higher risk for diabetes.

Source: Medline Plus:

Apples or Pears?

Q: I know that obesity is a risk factor for diabetes. But I’ve been told that body shape also plays a role. Is this true?

A: Yes, it’s true. People who carry most of their excess weight around their waist (often called “apples”) are at greater risk of diabetes than those who carry most of their excess weight below their waist (often called “pears”).

The more fatty tissue you have, the more resistant your body’s cells become to the effects of your own insulin. But this appears especially true if your weight is concentrated around your abdomen.

To determine whether you’re carrying too much weight around your abdomen, measure the circumference of your waist at its smallest point, usually at the level of your navel. Using a flexible, cloth-like tape measure is best. A measurement of more than 40 inches in men and more than 35 inches in women indicates increased health risks.

The good news is that you can lower your risk of diabetes by achieving and maintaining a healthy weight.

Source: Maria Collazo-Clavell, M.D.; www.mayoclinic.com
Like many diseases, type 2 diabetes can result from a varied combination of factors, both environmental and genetic. While environmental factors might include such things as physical inactivity, poor dietary choices, tobacco use, alcohol abuse and obesity, those who turn to the Iron Disorders Institute for guidance on issues related to iron out of balance are often more attune than the average health information consumer to the importance of genetics to our health. We already know that diabetes is thought to be a familial disease. According to “Genetics Home Reference”, a service of the US National Library of Medicine”, researchers are learning that nearly all conditions and diseases have a genetic component. Some disorders, such as sickle cell anemia and cystic fibrosis, are caused by mutations in a single gene. The causes of many other disorders, however, are much more complex. Common medical problems such as heart disease, diabetes, and obesity do not have a single genetic cause—they are likely associated with the effects of multiple genes in combination with lifestyle and environmental factors. Conditions caused by many contributing factors are called complex or multifactorial disorders.

Although complex disorders often cluster in families, they do not have a clear-cut pattern of inheritance. This makes it difficult to determine a person’s risk of inheriting or passing on these disorders. Complex disorders are also difficult to study and treat because the specific factors that cause most of these disorders have not yet been identified. By 2010, however, researchers predict they will have found the major contributing genes for many common complex disorders.”

If reading this article makes you feel as if you’re suddenly being sucked into the vortex that will transport you to the Twilight Zone you’re in good company. The crosstalk between diabetes and iron is significant. It becomes very clear it’s time patients with disorders of iron metabolism take note – and action! Do you know what your numbers are – just how are you “at risk”?

“There is a fifth dimension, beyond that which is known to man. It is a dimension as vast as space and as timeless as infinity. It is the middle ground between light and shadow, between science and superstition. And, it lies between the pit of man’s fears and the summit of his knowledge. This is the dimension of imagination. It is an area which we call... the Twilight Zone.”

Journey on with us... read the experiences of some members of the Excess Iron Discussion Group who have graciously offered to share their story. (See pages 6 - 8.) Each one is unique, with iron being a common thread.

Visit These Web Sites to Learn More About Diabetes

American Diabetes Foundation
http://www.diabetes.org

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)
http://www2.niddk.nih.gov/
Iron Disorders Institute National Headquarters Staff and Volunteers

IRON PROFILES

"What makes a person extraordinary? It's what they do, not how they're born"  
Jody Williams, 1997 Nobel Peace Prize recipient

Cheryl Mellan  
Volunteer  
Excess Iron List Moderator

“There is no such thing as a ‘dumb’ question; everyone is a beginner at some point.”

Along with being a wife, mother, grandmother and a well-known figure in her Southern Ohio community, Cheryl is also a well-known name in cyberspace, where for seven years she has devoted countless hours to helping people with iron disorders. As a volunteer Ambassador for Iron Disorders Institute, Cheryl moderates the online Excess Iron Discussion List. People from all walks of life have benefited from her helpful answers to their questions about hemochromatosis, iron deficiency anemia, anemia of chronic disease and other disorders of iron. Says Cheryl, “There is no such thing as a “dumb” question; everyone is a beginner at some point. It is easy to understand how they feel, because I remember what it was like when my husband Dale was diagnosed with hemochromatosis. I was frantic for information; all I could find was that his condition was fatal. That was just not good enough! Not only did I feel compelled to learn as much as I could about this condition, I knew that I needed to share what I learned with others.”

The following comments from current Excess Iron members demonstrate the power of the list to help people:

“I am so thankful for the information. I am overwhelmed almost to tears by the support. I know that sounds a little extreme but I feel so relieved to know that I am not in this alone. I want to understand hemochromatosis. I want to reach a healthy iron balance. I want to help my children avoid the health problems I have endured.”

“I have learned more in just one month than with any study group I’ve ever been in. You are amazing! “…I love you folks, though I’ve never met you, and I am just really grateful that my husband found this list and signed on!”

Cheryl has also actively participated and contributed immensely towards the success of IDI’s Patient Conferences. She was awarded IDI’s “stars, in our eyes” award in 2003 for her positive and proactive influence for her untiring contributions.

Take time to meet Cheryl for yourself. Sign on and start asking questions; you WILL get helpful answers and make friends!

Joining “Excess Iron” is easy and anyone with an interest in iron out of balance is welcome – you can do so directly from the IDI website, or by sending a blank e-mail to ExcessIron-on@mail-list.com

Jim Hines  
Volunteer  
Id-in Touch Editor

“Diagnosis, treatment and diet are nothing more than ‘common sense’ protocols for managing iron overload.”

Jim is dedicated towards sharing his experiences and the experiences of other patients with hemochromatosis so that patients and their families can benefit from gained knowledge. “Active involvement through participation and education are fundamental towards ensuring future generations do not endure the unnecessary suffering and humiliation that HHC patients have in the past,” explains Jim as to why he devotes his time to Iron Disorders Institute.

His record indicates he is no stranger to volunteering his time and skills to benefit others. Jim has been volunteer with IDI since 2001 participating in patient conferences at NIH, assuming technical responsibilities for publishing IDI’s newsletter in January, 2004, increasing newsletter publication from 4 times per year to 6, and has been instrumental in several web site modifications.

Currently, he volunteers his time instructing senior citizens how to use computers in Chesapeake, VA. He has also served as Treasurer, Board of Directors for a community service organization in Clinton, MA, and as a reading tutor in Nashua, NH. Additionally, Jim has edited and published several newsletters for military organizations.

“I willingly share my time because I have had the good fortune to encounter open-minded doctors knowledgeable in treating hemochromatosis with the wisdom to allow me to actively participate in my own iron management. I wish the same for future patients.”

“I realize that volunteerism is all about altruism, but when I give my time to a worthwhile health organization whose primary goal is education, I know that I will personally be reaping many intangible rewards. When an HHC patient realizes that hemochromatosis is not as daunting a disorder as initially appeared, when an HHC patient successfully convinces other family members to seek medical advice and possible diagnosis, or when an HHC patient or family member becomes sufficiently confident in their knowledge that they too can begin to educate others by volunteering their skill and time and sharing their iron experiences with others, I am rewarded beyond measure.”

“Patient experiences and ideas serve to better educate not only others in the ‘Iron Community’, but the medical community as well.”

You can email your suggestions or contributions to jedwhines@irondisorders.org.
I was diagnosed with hereditary hemochromatosis in 1999 at the age of 48; serum ferritin (SF) 639 ng/mL, transferrin-iron saturation percentage (TS%) 94.5%. Genetic testing revealed I am a C282Y homozygote. I did choose to have a liver biopsy; it revealed extensive iron deposition, inflammation and mild portal fibrosis. It confirmed also there was no fat present.

I was de-ironed with 34 weekly phlebotomies, and have continued to be faithful to monitoring iron levels in maintenance and having therapeutic phlebotomies as needed in a timely manner which, as it turns out appears to be only approximately every four months. Not too bad! I have also overcome my thinking to the idea of a complete annual physical being a non-necessity. Active preventative maintenance has become a primary focus in life – not an obsession, but rather more an “insurance”. Having baseline test results allows me to monitor for trends or concerns so that they can be addressed if and when they arise early on in the process. My wife’s willingness to advocate on my behalf has been a tremendous plus; she is always reading something and has been great about staying on top of new information as it becomes available, changes in protocols and diet management. She has more than once been the first to recognize a potential problem in the making.

My youngest sibling, a brother 10 years younger, was diagnosed with HHC as a result of my diagnosis. His serum ferritin level was almost twice my own, and he remains under treatment. Neither of my sisters exhibit iron overload, but monitor iron status annually.

The overwhelming fatigue I had prior to diagnosis is completely resolved, as has the “fuzzy brain” and difficulties in short term memory recall, moodiness and depression. I am left with arthritic concerns, and am a candidate for bilateral knee replacements. At 54, I feel well and am once again active and content.

We’ve been monitoring my serum glucose levels with greater attention recently. My family history includes a paternal uncle with insulin dependant diabetes and an older sister who is now on oral diabetic medication. In reviewing my records we can see a continual pattern of elevating serum glucose levels which have gone from mid-range ten years ago to now consistently hovering just inside or just above the upper limit of the normal range. Knowing that family history AND iron put me at increased risk for diabetes we’re not taking a potential red flag for granted.

We’re taking advantage of our knowledge by intensifying our monitoring, making use of more definitive testing than just serum glucose and learning all we can about insulin resistance, pre-diabetes and diabetes. By making small changes now – food choices, increasing exercise – along with that 15-20 pounds I’m told I will be losing, we hope to minimize potential problems with diabetes, if not completely avoid it.

I knew something was very, very wrong in December, 2005. A routine physical indicated only a decreased testosterone level and my serum glucose was slightly elevated. I was 44 with a testosterone level of 22 ng/dl (this number would hopefully be in the 400 range) and overwhelmed with fatigue. A medication was given, along with the reassurance that additional testing would be done if this didn’t resolve my concerns. I remember an endoscopy being mentioned. I became more ill.

In February, 2006 I was diagnosed as pre-diabetes, to be controlled hopefully with diet and exercise. I became more ill. By May, 2006 I was put on oral diabetes medication. An MRI revealed a mass on my pituitary. I became more ill. By August, 2006 I was an insulin dependant diabetic.

I called the doctor’s office and asked to go in for that tube down my throat, but was instructed instead to go directly to ER, that visit culminating in a 10 day hospital admission. My heart was working at 21% efficiency. I had an enlarged liver. The close association between heart disease and kidney function was now in play. The kidney is the key organ in our body for excretion of waste products. In about three months I’d “gained 30 pounds” from fluid retention (45 pounds being lost following about six months of treatment.) My serum ferritin (SF) was 5,740 ng/mL and my transferrin-iron saturation percentage (TS%) was 99%. A liver biopsy was performed and it did reveal cirrhosis of the liver. I am a C282Y homozygote.

Because of my weakened condition I was unable to do phlebotomies and was begun on chelation therapy. Chelation therapy is much less effective than blood loss for depleting iron stores, but gradually I did begin to feel better and regain some strength; the chelation was discontinued and aggressive therapeutic phlebotomies was begun. I’m making good progress with 44 phlebotomies behind me, doing them twice weekly. My current ferritin is 2,049 ng/mL. I’ve been “doing the math” and I’m hoping that with my next round of monitoring testing I’ll be in the 1,500 range, maybe even “done” by late summer. My pituitary is completely non-functioning. I am hoping too, that de-ironing might bring some positive function back.
I am one of seven children; four of the seven of us have hemochromatosis, my siblings being diagnosed as a result of my diagnosis. Two others are presently in treatment. Like most people I'm sure, I have regrets. Had I been tested six months earlier and HHC discovered I might not have spent 10 days in the hospital. We can only speculate about how different my life might have been if iron level testing was “routine” and my HHC was diagnosed six years ago.

“I’ m ready to get off the roller coaster and get on with life.”
Clyde Salsgiver, Wheeling, IL

I was diagnosed with hereditary hemochromatosis in May, 2003 at the age of 46. I had been participating in a special work project at that time that included eight weeks of educational classes in addition to my regular schedule. It seemed especially rough keeping up the pace. Not only was I suffering from what I assumed was “just fatigue”, I was experiencing a relentless pain in my right side for which no cause could be found after repeated testing. I was diagnosed with elevated cholesterol and blood pressure and began taking both a statin and blood pressure lowering drug. Neither improved either the fatigue or abdominal pain. At the suggestion of my sister who is a nurse living in another state I requested iron levels be checked. My ferritin was in the 800 range.

My HHC diagnosis was confirmed with a liver biopsy that revealed only slight amounts of fat deposit. Genetic testing was done; it confirmed that I am a C282Y homozygote. I was de-ironed over a period of one year with bi-weekly phlebotomies. Unfortunately, we overshot our mark and I sustained a prolonged iron deficiency anemia at the end of my de-ironing lasting just over one year.

With the iron deficiency anemia, it was a disappointment when the newfound energy I’d been enjoying during de-ironing disappeared. I was aware of the symptoms of anemia, but the shortness of breath I was having even with things like going down stairs was almost debilitating. One night after coming home from work I sent my doctor an e-mail asking if this “were normal” or if he thought I needed to be seen. That e-mail was quickly responded to with a phone call directing me to report to ER immediately. On December 1, 2004, three stents were inserted during an angiogram to clear the right coronary arteries due to blockage.

I am one of six siblings – the only male in the crowd, coming fifth in line. Although my sisters do monitor their iron status, none to date have experienced iron overload. Three of my sisters have diabetes; one on insulin, two on oral agents. My dad died at the age of 50 with a heart attack complicated by high blood pressure and diabetes. My mom is still living and has been an insulin dependant diabetic for more than twenty years.

It was a disappointment, but not too much of a shock when my own diabetes was diagnosed within just months of my HHC diagnosis. I started on one oral drug to control my fasting glucose level, and after about a year another oral drug was added due to rising fasting glucose levels. In November 2005 I progressed to daily injections for long term glucose control, and we are presently looking at increasing that dosage for better control.

With diabetes, heart disease, hereditary hemochromatosis and one look at my family history, it’s not too hard to understand the sudden significant interest in “genetics”; it’s also been interesting to learn about how one condition affects the other. It’s been a challenge putting all the pieces of the puzzle together, but I’m feeling better already and looking forward to still further improvement in my health. I’m ready to get off the roller coaster and get on with life.

“I reflect often on my good fortune.”
Jan Townsend, Diamond Bar, CA

Ironically, my hereditary hemochromatosis and type 2 diabetes diagnoses hit almost simultaneously in 2003 at the age of 53. Having not been feeling well for some time and suspecting I might have the symptoms for diabetes I scheduled an office visit. Diabetes is present in both my maternal and paternal family histories, I was aware of the importance of early diagnosis, treatment and control. Shortly after that visit I received a call from my physician. While testing did confirm type 2 diabetes, she also wanted additional tests run. She said “I think you may have something else also.” Those tests revealed a serum ferritin (SF) of 1,167 ng/mL and transferrin-iron saturation percentage (TS%) of 55%.

This is the first time I had heard the word “hemochromatosis” spoken. My only comment was a wonder as to why I cannot ever have something I know how to spell! I thought my fibromyalgia was just interesting enough. I had no idea the journey my life path was about to take me on.

My life was suddenly consumed with medically related referrals, testing, and education.

I began a five week diabetes education program offered by Kaiser Permanente at the recommendation of my endocrinologist and began oral diabetes medication. I was scheduled by my gastroenterologist for a liver biopsy, the results of which indicated fatty liver, but thankfully no further damage. I was genetically tested by my hematologist, the results of which indicate that I am compound heterozygous with the C282Y/H63D mutations. I began therapeutic phlebotomies on a schedule of four weeks on, one week off with monthly progress monitoring testing and was de-ironed with 12 phlebotomies over a 15 or 16 week period. Unfortunately, I experienced over-bleeding ending with hemoglobin of 7 g/dL, not a state I would recommend.

Despite the challenges I have needed to meet, I feel so very fortunate. I remember a time when I sincerely felt I was dying a little, every single day. Today, I am more than twenty...
pounds lighter than I was in 2003. I am de-ironed and have good control of my iron levels. I have the drive and ability to continue learning about iron out of balance, and thankfully many resources from which to learn. My oral diabetes medication was halved once during the course of my de-ironing, and discontinued a year later in light of my continual positive test results. I remain aware of the importance of my iron and glucose maintenance levels, my weight, the need for exercise and my diet.

I reflect often on my family history. The uncle who died in his 40's when I was a youngster, amid whispers of cirrhosis and "what he must have drank." I think of my dad, who with his round belly and tanned face was so often at the doctor's office for his belly pain, his heart palpitations and his migraines. I can see his hand, contracted into a fist and how he rubbed those first two knuckles so often saying "Oh, my hand is just killing me today." My mom passed away in 2005 after an extended battle with emphysema. It was not until immediately prior to her death that a mass on her liver was identified.

I asked my doctor once to tell me how she ever came to diagnose the hemochromatosis. She had one other patient with both diabetes and HH and learning what she had, she has made it her policy to routinely test liver enzymes in her diabetic patients. Those results alerted her to the need to look further. I reflect often on my good fortune.

You are in good company!

Almost everyone knows someone who has diabetes. An estimated 18.2 million people -- 6.3 percent of the population -- have diabetes, a serious lifelong condition. About 5.2 million people have not yet been diagnosed. Each year, about 1.3 million people age 20 or older are diagnosed with diabetes.

Source: National Diabetes Information Clearinghouse and the Centers for Disease Control and Prevention

A Few Famous People with Diabetes

Ernest Hemingway - 20th Century Novelist
Jackie Robinson - Baseball
Minnie Pearl - Comedienne
Thomas Edison - Inventor
Dizzy Gillespi - Jazz Pioneer
Ella Fitzgerald - Premier Jazz Vocalist
Jackie Gleason, Actor/Comedian
Halle Berry, Actress
Menachem Begin, Israeli Leader
Jerry Mathers, Actor "Beaver"
Anwar Sadat, Egyptian leader
Andrew Lloyd Webber, Broadway composer

Dan Wilson, Clarksville, TN

"I am feeling well relieved to finally 'know' what it is I have to deal with, and the peace of mind I now have has made all the trouble and pain worthwhile."

I was diagnosed with diabetes in August 2001 and I am presently taking Metformin, an oral diabetes medicine that helps control blood sugar levels. Both of my parents have type 2 diabetes; my mom being insulin dependant. Shortly after my diabetes diagnosis, I was also officially diagnosed with high blood pressure.

I was diagnosed with hemochromatosis/iron overload in June 2004 at the age of 44; serum ferritin (SF) 719ng/ml, transferrin-iron saturation percentage (TS%) 97%. Genetic testing revealed that I am a H63D heterozygote. A trial of therapeutic phlebotomies was begun and my excess iron was depleted in 13 weekly phlebotomies.

To the best of my recollection March 1998 was the first time I’d been noted to have elevated liver enzymes. I was tested at that time for possible causes, including viral hepatitis and cancer. Thankfully these tests were negative, but the liver enzyme problem did not resolve. In October 2000 I became ill with a cough and upper right quadrant pain. Radiological tests were inconclusive, liver enzymes remained elevated. In May 2004 a new physician investigating my now chronic cough thankfully looked further. An ultrasound revealed an enlarged liver, and the elevated iron levels were discovered. I was advised against a liver biopsy and initially accepted that advice. However, after consideration and a little education I did request the biopsy. I was a young man with a wife and two children, I had learned the hard way not to take health for granted – and I wanted a REASON why I had elevated liver enzymes for more than eight years without resolution.

My liver biopsy revealed only trace iron (by this time I had already completed by phlebotomies to normalize iron levels), and no fibrosis or cirrhosis. However, the diagnosis of nonalcoholic steatohepatitis (NASH) was confirmed. It resembles alcoholic liver disease, but occurs in people who drink little or no alcohol. The major feature in NASH is fat in the liver, along with inflammation and damage. Many patients with NASH have elevated blood lipids, such as cholesterol and triglycerides, and many have diabetes or pre-diabetes, but not every obese person or every patient with diabetes has NASH.

For the first time since my problems with elevated liver enzymes began, these levels are now normal. That chronic cough is gone. Although there is no specific “treatment” for NASH, I have made very good use of my education about current recommendations with regard to weight control, diet, and lifestyle choices. I am feeling well relieved to finally "know" what it is I have to deal with, and the peace of mind I now have has made the trouble and pain all worthwhile.

To date, I have not reloaded iron and have required no maintenance phlebotomies, but I assure you that learning what I have about iron balance throughout this journey, it is my intention to monitor these numbers, taking action accordingly for the rest of my life.

"I am feeling well relieved to finally ‘know’ what it is I have to deal with, and the peace of mind I now have has made all the trouble and pain worthwhile."

Dan Wilson, Clarksville, TN

I was diagnosed with hemochromatosis/iron overload in June 2004 at the age of 44; serum ferritin (SF) 719ng/ml, transferrin-iron saturation percentage (TS%) 97%. Genetic testing revealed that I am a H63D heterozygote. A trial of therapeutic phlebotomies was begun and my excess iron was depleted in 13 weekly phlebotomies.
When Sierra’s 7th grade science class started their genetics unit, Sierra was the first to raise her hand and share her story of having hemochromatosis. Each student in the class was assigned a genetic disease to research and prepare a project but Sierra was asked if she would like to do her report on HHC. She was thrilled. Sierra has done reports on HHC for classes in elementary school, Girl Scouts, and the science fair. However, this report proved to be different as her maturity level opened up new questions and curiosities for research and discussion. For example, the “I can’t have HHC because I’m anemic” statement was one Sierra and her Mom spent a great deal of time on.

Sierra chose a PowerPoint demonstration for her report format and she spoke as she presented the material. She didn’t even use note cards. Her classmates seemed interested in her report and amazed at the statistics.

Sierra’s mom, Linda informs id-in Touch, “We highly recommend HHC projects to anyone out there. For children with HHC, it can make a disorder much less frightening. It will also empower the kids to feel like they might make a difference in someone’s life even save one someday. Sierra will continue to share her story every chance she gets and hopefully somewhere down the line when one of her fellow classmates get diagnosed, has a family member or friend with HHC, or even becomes a doctor, they will remember her story.”

Sierra Randall’s Genetics Project PowerPoint Presentation

Note: Sierra’s project is best viewed by increasing your Adobe Reader’s magnification. 150% or greater is highly recommended.
Reminder: Sierra’s project is best viewed by increasing your Adobe Reader’s magnification. 150% or greater is highly recommended.

IDI will publish a student’s school project in *id-in Touch* that raises awareness of Iron-Out-of-Balance™. The subject may be on an iron-loading disease or disorder, anemia, genetics, or any sub-topic relating to iron. Send a recommendation to Newsletter Editor, c/o of IDI, or send email to jedwhines@irondisorders.org.
Reminder: Sierra’s project is best viewed by increasing your Adobe Reader’s magnification. 150% or greater is highly recommended.

Slide Photograph Legend

Slide 15: December 1999. Dr. Elliott Vichinsky, Hematologist, Dr. Paul Harmatz, Gastroenterologist, Sierra Randall, & Linda Randall, (left to center) along with Laurice Levine Thalassemia Outreach Coordinator (far right) proudly accept a $195,000 check from 3 representatives of a corporate donor for Children’s Hospital Oakland’s ferritometer.

Slide 16: October, 2000. Ferritometer at Children’s Hospital, Oakland, CA. Learn more about the ferritometer in the March/April, 2006 issue of IDI’s newsletter, id-in Touch, and technical discussions at Tristan Technologies, Inc. (See links below.)

Slide 20: February, 2000. Dr. Roland Fischer from Hamburg, Germany and Sierra Randall, age 5, examine the ferritometer being prepared for shipment to Torino, Italy during Sierra’s visit to Tristan Technologies in San Diego, CA.

Slide 21: February 2000. Linda and Sierra Randall meet with Tristan Technologies’ scientists. Front row, l. to r: Sierra, Laurice Levine, Dr. Roland Fischer, Douglas Paulson, Ph.D., President. Back row, l. to r: Linda, Dr. Paul Harmatz, Huuythong Nguyen and Robert Fagaly, Ph.D., Vice-president, (far r.)

Slide 22: October, 2000. Sierra Randall stands in front of the ferritometer at CHORI.


Ferritometer links:

“The Randall Family’s Journey with Hemochromatosis” page 3.
“Liver And Tissue Iron Measurement Methodologies” page 8.
http://www.irondisorders.org/Newsletter/ma06.pdf
Tristan Technologies, Inc.
http://www.tristantech.com/prod_large_array.html
http://www.chori.org/Current_News/Archives/01_feb_ferritometer.html

Rose Penrod and Sierra Randall share an HHC moment in front of CHORI’s ferritometer.
Almost 16 million Americans have diabetes mellitus—that’s approximately one in every 15 people in the USA. Among these 16 million, about one-third are not aware of their condition. Advanced diabetes can lead to blindness, and gangrene of the toes and fingers requiring amputation. Diabetes can also lead to heart disease, kidney failure, and premature death—all of which might be prevented.

Diabetes mellitus represents a group of disorders that have one common feature: abnormally high levels of glucose (sugar) in the blood. Normally, blood sugar levels are kept within a narrow range (70-130 mg/dL) by several hormonal and neuronal mechanisms, especially by the hormone insulin, which is produced by the beta-cells of the pancreas. Beta cells are found in specialized clumps of cells in the pancreas called islet cells. When defects in insulin production, insulin action, or both are present, high blood sugars can result.

Diabetes is usually divided into two broad categories: type I diabetes and type II diabetes. Type I is caused by a deficiency of insulin production by the beta-cells in the pancreatic islets possibly due to viral infections or autoimmune insulinitis (inflammation of the beta-cells). Type I is most common in children and young adults and is often called early-onset or juvenile diabetes. Type II diabetes is caused by a combination of reduced insulin effectiveness (insulin resistance) and insulin production. Individuals with type II initially have too much insulin in the bloodstream or hyperinsulinemia. Eventually, however, this type diabetes can result in pancreatic exhaustion and develop into insulin-dependent diabetes. In adults the vast majority of diabetes (about 90%) is type II diabetes.

Hypoglycemia—too little blood sugar—can occur in either type I or type II diabetes. Insulin-dependent diabetics can experience low blood sugars when too much insulin is administered or by failing to eat after insulin has been injected. Type II diabetics can experience low blood sugars during the "early" phase when insulin levels are abnormally high if other factors increase glucose utilization or decrease glucose production by the liver.

A combination of sugar and alcohol—such as in a gin & tonic—can precipitate hypoglycemia.

Although most mild or early diabetics have few or no symptoms whatsoever, symptoms of severe diabetes mellitus may include frequent and abundant urination, thirst, hunger, weight loss, and blurred vision. The cause of diabetes is not completely understood. Physical inactivity, obesity, and abdominal body fat distribution are all known risk factors for developing diabetes. Presence of diabetes in a family member also increases the risk of development of diabetes, which suggests that genetic factors play a role in causing the disease.

Hereditary hemochromatosis (HHC), a common genetic disorder of iron metabolism, has diabetes as one of its consequences. Patients with HHC absorb as much as four times more iron from their diets as do people with normal iron metabolism. Unneeded excess iron cannot be excreted and it eventually accumulates to toxic levels in vital organs. The impaired organs become unable to function properly. In most cases of HHC, this process is somewhat slow to develop—the damage shows up as a heart attack or liver failure or diabetes after about three to five decades of iron accumulation.

More than 90% of hemochromatosis patients with diabetes have Type II diabetes or are glucose intolerant and about 1/3 of these patients require insulin.

Iron can cause damage to tissues of vital organs by changing oxygen into a form known as a free radical-increased oxidative stress. Unopposed free radical activity can cause irreversible cell damage. It may be possible that this is how iron destroys the beta-cells causing diabetes. Beta cells or islet cells have very low levels of the enzymes that break down free radicals. Thus, agents that increase free radical production, such as iron, could result in destruction of pancreatic cells.

At this time, for those with iron loading disorders such as hemochromatosis, removal of excess iron from the body with therapeutic phlebotomy or periodic blood donation is the safest, cheapest and most effective way to lower excessive body iron stores.

If HHC is diagnosed before complications such as diabetes develop, maintaining a de-ironed status will significantly diminish the risk of iron-related diabetes and other disease. Research is underway to determine whether other, more expensive and complicated methods for removing iron will prove to be a benefit for those with established diabetes-related neuropathy. "Chelation therapy—inactivation and removal of metals pharmacologically by a special chemical that binds iron tightly—has been shown to slow or even reverse peripheral nerve damage in experimental animals with diabetes" according to Drs. Mingwei Qian and John Eaton of Baylor College of Medicine, Houston, Texas. "It appears that, in diabetes, there is an accumulation of metals such as iron and perhaps copper bound to blood vessel walls. These metal deposits prevent the normal relaxation of blood vessels which feed the nerves and this slowly starves—and ultimately kills—the nerves. This explains why administration of chelators may be able to preserve nerve function even in advanced diabetes, at least in experimental animals."

Chelation therapy for humans with diabetes is still experimental and calls for additional research such as the study currently in progress in the U.S.A., funded by the National Institutes of Health's Institute for Diabetes, Digestive and Kidney Disease (NIDDK). This study is intended to address important issues such as the following:

- Determining the relationships among body iron burden,
First Disease continued from previous page.

HFE gene mutations, especially Cys282Tyr and His63Asp, glucose tolerance, insulin sensitivity and diabetes

- Understanding the cellular and molecular mechanisms underlying the development of diabetes mellitus in (or associated with) iron overload disorders
- Determining the potential role of iron induced, free radical or lipid peroxidation mediated damage to the pancreatic beta-cell and to hepatocytes (liver cells)
- Examining the effects of increased body iron burden on insulin resistance and glucose transporter function in several insulin target cells: e.g., muscle, adipocytes (fat cells), hepatocytes (liver cells)
- Investigating how different genes behave (differential gene transcription/translation) in beta cells, hepatocytes and enterocytes in response to exposure to elevated iron
- Investigating the effectiveness of iron chelation therapy in preventing or reversing iron-overload mediated diabetes
- Investigating the role of proteins such as ceruloplasmin on the incidence of diabetes
- Studying the natural history of diabetes associated with hemochromatosis

In the announcement of its intent to fund such studies, scientists at the NIDDK commented as follows: “Since individuals at risk can be identified early, serial metabolic characterizations before and early in the disease to define the initial events in progression to diabetes are of interest. Insulin resistance is also under examination to learn if it is caused by hemochromatosis or if people with both conditions are more likely to progress to diabetes. ‘Studies are needed to determine’ the extent to which diabetes is reversible when iron overload is corrected. A number of reports suggest that increased iron leads to insulin resistance but the mechanisms responsible for this resistance are obscure.” For these reasons this important research is underway to better understand why so many individuals with hereditary hemochromatosis develop diabetes.

According to Dr. Frank Vinicor, Director of Diabetes Translation at the Centers for Disease Control and Prevention, “The exact mechanisms involved in diabetes with HHC are still unknown. Some data suggest that insulin-resistance and abnormal pancreatic beta-cell function appear well before insulin deficiency and are still reversible with iron depletion.”

As a preventive measure, if diabetes runs in your family, you might ask your physician to check your iron levels along with blood sugar levels. Other major symptoms that may suggest tissue iron levels are excessively high include abdominal pain, fatigue, tiredness, signs of liver damage, heart arrhythmias, impotence, joint pain, depression, and loss of menstrual periods.

<table>
<thead>
<tr>
<th>DIABETES</th>
<th>TYPE I</th>
<th>TYPE II</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age at onset</strong></td>
<td>Early (before age 30)</td>
<td>Around age 40+</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td>Frequent &amp; abundant urination, thirst, weight loss, excessive hunger, ketoacidosis: abdominal pain, headache, rapid feeble pulse, decreased blood pressure, flushed, dry skin, irritability, nausea, vomiting, air hunger/shortness of breath, double or blurred vision</td>
<td>Frequent &amp; abundant urination, thirst, weight change, itching, peripheral neuropathy</td>
</tr>
<tr>
<td><strong>Therapy</strong></td>
<td>Insulin and diet</td>
<td>Diet, hypoglycemic drugs. Possibly insulin</td>
</tr>
<tr>
<td><strong>Islet Cell Antibodies</strong></td>
<td>Present at onset</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Insulin in Blood</strong></td>
<td>Little to none</td>
<td>Present</td>
</tr>
<tr>
<td><strong>Body Weight</strong></td>
<td>Normal/under</td>
<td>Obese (80%)</td>
</tr>
<tr>
<td><strong>Blood Glucose</strong></td>
<td>Elevated &gt; 200 mg/dl</td>
<td>Elevated &gt; 200 mg/dl</td>
</tr>
<tr>
<td><strong>Symptoms of HYPOGLYCEMIA</strong></td>
<td>Weakness, tremor, muscle twitching, nausea, vomiting, pallor (paleness), sweating, confusion, decreased blood pressure, decreased heart rate, palpitations, and air hunger (shortness of breath, sighing, hiccups).</td>
<td></td>
</tr>
</tbody>
</table>

**Consider donating to Iron Disorders Institute as a memoriam to your loved one**

You can obtain more information from our web site: [http://www.irondisorders.org/Donate/](http://www.irondisorders.org/Donate/)

Your contributions are fully tax-deductible to the amount allowed by law.

**SUGGESTED READING**

*Survival of the Sickest: A Medical Maverick Discovers Why We Need Disease*  
By Dr. Sharon Moalem with Jonathan Prince

Chapter 1 provides the reader with an insight into Aran Gordon’s struggle with hemochromatosis and eventually competing successfully in the *Marathon Des Sables* marathon.

Also included is background on Gene Weinberg’s discovery of how iron feeds bacteria and his quest in becoming one of the foremost experts on iron ingestion.
Discovery Health Channel – Mystery Diagnosis will air a 30 minute segment on Hereditary Hemochromatosis on:

APRIL 16, 2007 at 10:00 PM

IDI understands that Aran Gordon's story will be featured. Visit IDI's web site to learn more about Aran Gordon. http://www.irondisorders.org/Aran/

We need a volunteer to tape the segment. Contact IDI for details.

Phone: 864-292-1175
Email: Comments@irondisorders.org

The producer has also requested that viewers provide comments, thus several more volunteers. Send your email comments to: Comments@irondisorders.org.

NOTE: Please enter "Discovery Channel" in the subject line of your email. Thanks for your support!

RUSTY LOCATES MORE CLIVE CUSSLER AND HHC

After reading the six Clive Cussler novels I purchased from the library’s book sale, my wife purchased several more in paperback from our book store. Once again, Clive Cussler introduces hemochromatosis in Inca Gold while describing the current lifestyle of David Gaskill, a United States Customs Agent, who specializes in uncovering stolen art and antiquities.

“He led a lonely private life since his wife of twenty years died from a heart attack brought on by an iron overload disease known as hemochromatosis.”

Inca Gold: A Dirk Pitt Adventure by Clive Cussler; 1994

“Despite all the joint pain over the years I must laugh at myself.” – Patrick McKeever

TRIVIA ANSWERS

SCA: (From page 2.) The average life expectancy in America has improved. It is now in the mid 40 years of age range. Source: www.ascaa.org
Volunteer: President Richard Nixon, in 1974! Source: www.pointsoflight.org

WHO ARE VOLUNTEERS?

Volunteers are individuals who perform or offer to perform a service out of their own free will, often without payment.

To volunteer is to choose to act in recognition of a need, with an attitude of social responsibility without concern for monetary profit, going beyond what is necessary to one’s physical well-being.

IRON DISORDERS INSTITUTE’S 2007 AWARDS

Each year Iron Disorders Institute recognizes outstanding volunteer effort with two awards: “Making a Difference”, and “stars, in our eyes…”

Past year recipients are listed in the March/April, 2006 issue of id-in Touch on page 4.

http://www.irondisorders.org/Newsletter/ma06.pdf

The “Making a Difference” award is given to a medical doctor that has demonstrated outstanding efforts to educate the medical community about iron.

The “stars in our eyes…” award is given to a lay person who has made a unique contribution to the vision and mission of Iron Disorders Institute and especially to raise awareness about potentially deadly disorders of iron, such as hemochromatosis.

Send your recommendations to: comments@irondisorders.org
I would like to begin by giving a big thanks to Melodie Deselette, Executive Director of the James R. Clark Memorial Sickle Cell Foundation, for the invitation to their conference and her willingness to partner with IDI on this very crucial health issue in the minority population of South Carolina.

Sheila and Darlene handed out literature about iron overload and made many new contacts who expressed an interest in iron. Besides being very beneficial towards raising awareness about Iron-Out-of-Balance™ in the minority population, the conference was a day of sharing information, enjoying good food and lots of fun.

As I do my work and learn more about iron disorders in the minority population, the information continues to amaze me.

The past two months have been very busy. In February, Cheryl Garrison and I were on Capitol Hill visiting the offices of Senators Specter (R) and Casey (D) of Pennsylvania, Graham (R), DeMint (R) of South Carolina, and Isakson (R) and Chambliss (R) of Georgia. We provided information on the importance of iron disorders and how their help is needed to support iron related research. All the meetings went very well, but being stuck in Washington DC airport for two days due to the Saint Valentine ice storm wasn't much fun!

Cheryl Garrison, Executive Director, Vera Tanner, IDI Consultant and I facilitated a “Lunch & Learn” with Dr. Arlene Lester, DDS, MPH of the U.S. Department of Minority Health and Human Services in Atlanta. We shared information on the minority health issues with Dr. Lester and eight of her colleagues; all were excited about the information and the importance of the need for education and awareness about iron related health problems among minorities.

Greenville Hospital System and Morehouse School of Medicine have also demonstrated their concern for this under-recognized problem and have indicated their desire to partner with IDI to find ways to reach the minority communities.

On a fun note, Chef Manigault from Chef Manigault La Vieille Maison, Greenville SC has agreed to learn about iron and to help IDI demonstrate how to prepare iron-balanced meals for women who are iron deficient. Among minorities, iron deficiency anemia is up 5% rather than declining; the most at risk are African American and Hispanic women of childbearing age. The Center for Disease Control and Prevention (CDC) estimates that 20% of this population is iron deficient!

Our programs are developing around these issues but their success is dependent upon funding, as is always the challenge of any non-profit! If you have an interest in helping us get the word out to the minority population with either a donation, sponsorship or by volunteering time, contact me!

Phone: 864-292-1175
Fax: 864-292-1878
Email: sdogan@irondisorders.org

Iron Disorders Institute is a 501(c)3 voluntary health public interest organization with headquarters in Greenville, South Carolina.