Fall, 2006

Canadian HEMOCHROMATOSIS SOCIETY

Iron Filings

Member in the Spotlight

Calgary HH Advocate Takes Her News to the Games

Anne Stang, tireless HH educator, is our featured Member in the Spotlight for the Fall 2006 Iron Filings edition. Thanks to Anne for spreading the HH word not only amongst her own family – initiating multiple new diagnoses – but also in her hometown of Calgary and within her German Russian ethnic population.

At the Bunnock Tournament in Macklin, Saskatchewan. Anne Stang (l) hands a CHS brochure to her cousin, Kay Zerr.

"Hemo what?" I suspect I am not the only person who has had that reaction on being told about hemochromatosis. Fortunately for me, I was diagnosed from a routine blood test by my GP in February 2005. Hearing that my serum ferritin level was 458 ng/ml and that I had two copies of the C282Y mutation in the HFE gene didn't mean much back then. My first stop on the way home that day was to get the Iron Disorders Institute's book on HH to educate myself.

Since my diagnosis came as a result of a routine test, I was only aware of symptoms in retrospect. I was stiff at times, and tired, but I attributed that to my age (now 66). I also had mood swings, but they were not that frequent and I don't think they were noticeable by others. I had ten phlebotomies (I call them my "leech" sessions) in twelve weeks in the spring of 2005 to bring my ferritin level back to normal. I have had three phlebotomies since, as part of maintenance, and have regular blood tests to follow my ferritin levels. Typical blood donations through CBS are unfortunately not an option for me because I had rheumatic fever at age ten, and have travelled to malaria-exposed countries. I have also modified my diet at home. I choose not to eat red meat and I got rid of iron-rich products like Bran Flakes and Cheerios. It remains to be seen

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INSIDE

The Newsletter of the Canadian Hemochromatosis Society

HEMOCHROMATOSIS

Very common • Virtually unknown • Potentially fatal • Easily treatable

What is it?
The excess storage of iron in the body.

What is the cause?
Primarily hereditary.

Most common symptoms
Chronic fatigue, joint pain, irregular heart beat, mood swings, confusion, bronzing of the skin, loss of libido and abdominal pain.

Most common complications
Liver and heart disease, diabetes, arthritis and hormonal irregularities.

Tests required for diagnosis
Serum ferritin, transferrin saturation percentage and genetic testing.

Treatment
Phlebotomy treatments (bloodletting) which are ongoing for life.

Reference reading
The Bronze Killer; Ironic Health; The Iron Elephant; Iron Disorders Institute Guide to Hemochromatosis.

From the President

We 'Googled' to the Top

Our web master recently told us that a Google search of the word "hemochromatosis" now has our website at the top of the list, ahead of the National Institute of Health in the U.S. and several other great sites.

The Mayo Clinic site, well down the page on this same Google search, lists hemochromatosis as a liver disorder. Way back in our history – when the Society was in danger of being absorbed by the Liver Foundation of Canada after we approached them about possible cooperation between our organizations – we were concerned that focusing just on the liver would be too narrow a focus, especially when the clinical manifestations of hemochromatosis are so much broader in scope.

I believed then and now that our concerns were justified, but ironically, current research on iron metabolism is focusing on the liver as the centre of the cause for hereditary hemochromatosis. The protein which produces the hormone hepcidin, has been identified as perhaps *the* central player in the regulation of iron absorption, and it is produced in the liver. In recognition of this, we have included two articles in this edition of the newsletter that feature information about the liver, tests done on the liver and what they mean, and more specifically, information about hepcidin. We count ourselves very fortunate to have access locally to medical experts in this field – Dr. Sigfried Erb and Dr. Sam Krikler – who have once again contributed their time and expertise in illuminating these topics. Our editor, Julie MacFarlane is responsible for the question and answer format that helps ensure that the kind of questions our readers ask are covered by the doctors in their handling of the topic.

Our "Member in the Spotlight" features yet another one-woman dynamo, from Calgary this time. I am constantly amazed at not only the energy but the incredible variety of interesting backgrounds of our members that we hear about when we do these features.

Elizabeth Minish, President,
Canadian Hemochromatosis Society

Anne Stang introduces us to a unique ethnic group *and* a game I had never heard about with her story of turning a family reunion into an incredible hemochromatosis awareness opportunity.

Speaking of awareness activities, if you read the spring newsletter you would be aware of a success story from the Maritimes in delivering timely information from a variety of specialists in the hemochromatosis field to a large number of people through the 3rd annual information session held in Halifax. We have been so impressed with this model of cooperation and efficiency that we have decided to attempt to recreate the event in British Columbia next spring in conjunction with Awareness Month. We hope that, eventually, this may spread to all the major centres across Canada. In the meantime, we are looking for volunteers to help coordinate this event, including a project leader.

The fall newsletter is also the edition during the year where we take time to say thank you to our donors. We are always amazed at your generosity and count ourselves very fortunate indeed to have such a dedicated group who support our vital work year after

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Iron Filings

Editor
Julie MacFarlane

Canadian Hemochromatosis Society
#272 - 7000 Minoru Boulevard
Richmond, BC Canada V6Y 3Z5
604-279-7135
fax 604-279-7138
email office@toomuchiron.ca
Toll Free 1-877-BAD-IRON

www.toomuchiron.ca

Member of the International Association of Hemochromatosis Societies
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The Disorder

Hemochromatosis is the most common genetic disorder afflicting Canadians. It is a potentially fatal condition caused by iron overload in organs, joints and tissues. The complications caused by the disorder are preventable.

Our Purpose

The society is dedicated to preventing the unnecessary suffering and death caused by hemochromatosis by promoting awareness and early diagnosis while supporting those affected by the disorder.

Up Close and Personal with Your Liver

Two specialists explain the impact of hemochromatosis on the liver

This issue of **Iron Filings** *focuses on the liver – the central organ involved in hemochromatosis – and the recently identified protein, hepcidin, which is produced in the liver and now found to play a key role in the pathogenesis of the disease. We have asked two physicians: Dr. Siegfried Erb, Hepatic (liver) specialist and Dr. Sam Krikler, Hematopathology (blood) specialist, to help us understand how the liver is affected by the iron accumulation central to hemochromatosis, and how hepcidin functions in the body as part of the signaling process for iron.*

Dr. Siegfried Erb is a liver specialist with the Division of Gastroenterology, Department of Medicine, VGH and the BC Transplant Society

What blood tests are done by the specialist and what do they tell about my liver or my hemochromatosis?

The blood tests as a group tell us what's wrong with the liver, how well the liver is working, and any complications. The blood tests can be divided into four types: the first are liver injury tests, also known as liver enzymes; the second are true liver function tests; the third are disease-specific markers used to determine the cause of liver disease; and the fourth are miscellaneous tests.

Which blood tests would my doctor do first?

Initially, he would do an ALT, iron studies, and viral markers to see if you've been exposed to, or are protected against, hepatitis A, B or C. If not, you would need vaccination.

If my ALT is abnormal, does that mean I have cirrhosis?

ALT is an enzyme predominantly found in liver cells. Abnormally elevated ALT levels tell you that you have ongoing damage to your liver. It tells you nothing about cirrhosis. It often returns to normal after you've been de-ironed. The ALT has nothing to do directly with hemochromatosis.

The reason we measure ALT is because it's found in the liver cell about 1,000 times the concentration found in the blood. If a few liver cells are being killed off, it leaks into the blood. So ALT is a good indicator for acute liver cell damage, and acts as a good predictor before other symptoms occur such as jaundice (yellowing of the skin).

What are the true liver function tests and do they tell you about cirrhosis?

The true liver function tests include: the direct bilirubin (a breakdown product of heme and excreted from the liver), albumin (protein made exclusively in the liver) and INR (International Normalized Ratio, related to coagulation proteins produced in the liver). The tests are not very sensitive but they are specific. If they are abnormal, this indicates that there has been a significant injury to the liver. Fortunately, in the vast majority of hemochromatosis patients, these tests are normal.

You mentioned disease-specific markers as the third type of blood test. What tests should someone have to diagnose hemochromatosis?

The screening test for hemochromatosis should be a total iron binding capacity (TIBC) which is calculated into a transferrin saturation percentile (TS%). If the TS% is more than 60 per cent on one occasion, or more than 45 per cent on two occasions, then you should have (HFE) genetic testing done to confirm hemochromatosis (unless your ethnicity is non-Caucasian, such as Asian). These tests are good at determining whether you have primary (genetic) hemochromatosis, or a secondary form of hemochromatosis perhaps related to alcohol or hepatitis C.

Why don't non-Caucasians need to be tested?

Ninety-two per cent of hemochromatosis patients have the type 1 form, caused by mutations in the HFE gene. This is typically seen in Caucasians, especially Northern Europeans. HFE-hemochromatosis is very rare in Asians. But there are other forms of hemochromatosis. The type 2 form of hemochromatosis (called juvenile hemochromatosis) occurs earlier in life in all races, and the type 3 form of the disease can often occur in Asians. *(See the Society's webpage for more information on the different types of hemochromatosis. Ed.)* If the genetic markers for the HFE type 1 form are negative, and no other cause can be found for the high iron levels detected in the blood, then a liver biopsy is indicated.

I get the impression that most patients do well after their hemochromatosis diagnosis, and that significant liver complications are now less of a problem than they used to be.

That's correct. This is due to a better awareness of hemochromatosis both in the medical profession and with the public.

How does hepcidin function in the iron metabolism pathway?
Hepcidin acts together with several iron-regulating proteins . . . but the precise mechanism by which these proteins talk to each other is not yet known. What is now clear is that hepcidin is the key signalling protein for the other proteins in the iron metabolism pathway.

Is a liver biopsy indicated for type 1 patients like me?
Some experts say yes. The risk of developing liver cancer is much higher in patients with cirrhosis, and since the true liver function tests are not very sensitive, I can see the argument for having a biopsy. On the other hand, scar tissue on the liver is progressive and not an all or none phenomenon. Additionally, liver biopsy is not the best way to look for scar tissue. Also, liver cancer has rarely been reported in hemochromatosis patients without cirrhosis. As such, all my hemochromatosis patients are enrolled in a screening program to watch for cirrhosis first.

What do you do to screen for cirrhosis and liver cancer?
I do an AFP (alpha-fetoprotein), a blood test, and an ultrasound or CT scan. I do it every six to eight months if I think they have cirrhosis and yearly if they don't.

What if my tests are normal? Once I'm deironed do I need follow-up tests?
Yes, you're a patient for life. You need annual tests which would include a CBC (complete blood count), liver enzymes, iron studies, an AFP and often an ultrasound.

Can my GP order these tests or do I need to see a specialist?
It will depend on your GP and your specialist. Some GPs won't be comfortable with ordering and interpreting these tests, and so therefore, you should continue to have follow-up tests with your specialist.

Is there a correlation between liver tests and genetic markers?
There are many factors that determine iron overload apart from genetics, such as age and gender. We don't know why some individuals develop a more severe form of hemochromatosis involving the liver while others have a milder form. There may be environmental co-factors such as alcohol. Having said that, compound heterozygotes (HFE C282Y/H63D) generally do not develop as much iron overload as affected homozygotes (HFE C282Y/C282Y). Also, type 2 juvenile hemochromatosis patients develop iron overload at a much earlier age, and are more likely to have significant liver and heart problems.

I get the impression that most patients do well after their hemochromatosis diagnosis, and that significant liver complications are now less of a problem than they used to be.
That's correct. This is due to a better awareness of hemochromatosis both in the medical profession and with the public. This has lead to better medical guidelines for earlier diagnosis, aggressive treatment, and ongoing follow-up for patients with hemochromatosis.

Dr. Sam Krikler is Director, Hematopathology Division, Department of Laboratory Medicine, Surrey Memorial Hospital, British Columbia.

What is hepcidin?
Hepcidin is a small protein (a hormone) responsible for the regulation of iron recycling and iron balance in the body. Hepcidin regulates iron absorption from the intestine and the release of iron from macrophages (macrophages are storage cells involved in the immune system for bug-fighting).

Why is hepcidin making such big news in hemochromatosis research?
Hepcidin may be the long sought-after iron stores regulator. Hepcidin may be the key protein that senses iron levels in the body and signals for other processes to take place, either the body needing more iron, or the body needing less iron, depending on the situation.

Where does hepcidin function in the body?
Hepcidin is produced by the liver in response to inflammatory stimuli and iron. It then circulates in the blood in a manner similar to other hormones.

It appears to interact with receptors on the surface of intestinal cells and macrophages. In this way, it regulates iron metabolism.

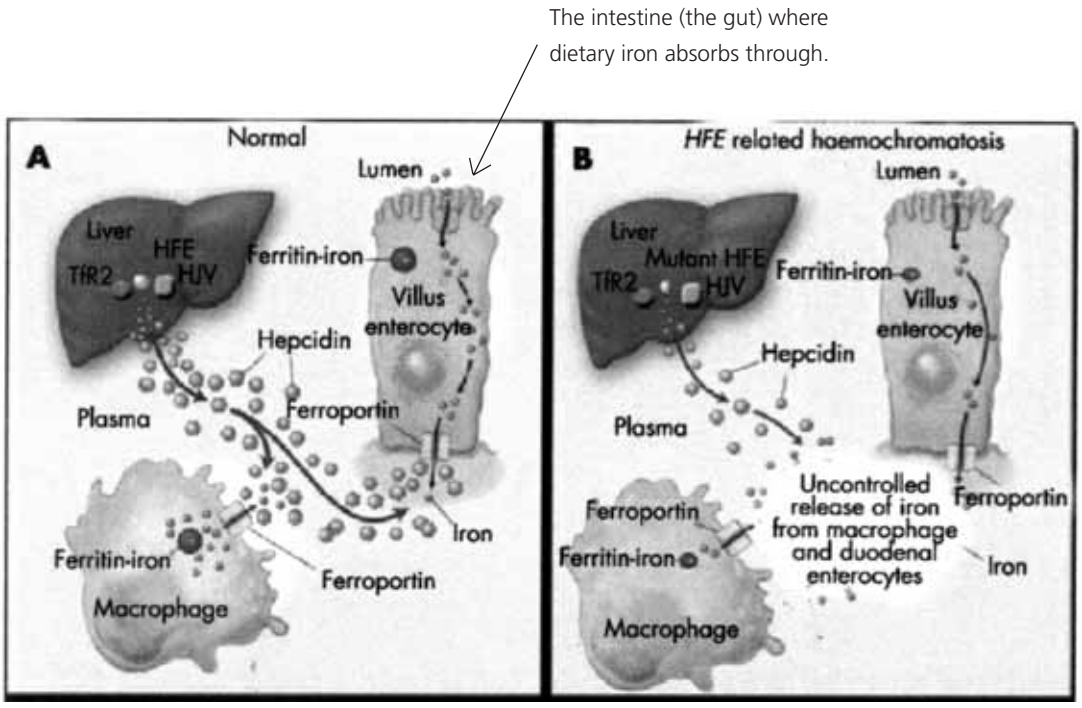
How does hepcidin function in the iron metabolism pathway?
Hepcidin acts together with several iron-regulating proteins, including ferroportin, transferrin receptor 2, hemojuvelin and HFE, but the precise mechanism by which these proteins talk to each other is not yet known.

What is now clear is that hepcidin is the key signalling protein for the other proteins in the iron metabolism pathway. When all the iron-regulating proteins function correctly and they receive the correct signal from hepcidin, the amount of iron transferred into the blood will be appropriate to body needs, and excessive iron deposition in tissues will be avoided. Too much or too little hepcidin, however, results in problems cascading down this iron pathway: hepcidin deficiency results in iron overload, whereas hepcidin excess results in anemia.

One possible scenario of how hepcidin and the other iron regulating proteins interact is shown in the diagram on page five *(adapted from A. Pietrangelo. "Molecular Insights into the Pathogenesis of Hereditary Hemochromatosis. Gut 2006; 55:564-568).*

So is it the hepcidin gene or the hepcidin protein that is abnormal in adult-onset HH?

The common form of hemochromatosis is *not* due to an abnormality in the hepcidin gene. HH is caused, in the majority of cases, by mutations in the *HFE* gene. Presumably, *HFE* plays a role in regulating hepcidin production in the liver because in patients with hemochromatosis, levels of hepcidin protein measured in the blood are found to be inappropriately low. Low levels of hepcidin in the blood lead to excessive iron absorption from the gut and release of iron from safe storage sites in macrophages.



Picture A: Normally, hepcidin protein produced in the liver is released into the bloodstream and interacts with the other iron-regulating proteins to signal for a normal balance of iron absorption through the gut and normal iron release from macrophages.

Picture B: In hemochromatosis caused by mutations in HFE, hepcidin levels are lower and this signals for excess absorption of iron through the gut, and uncontrolled release of iron from the macrophages.

So in adult-onset hemochromatosis, gene mutations in *HFE* result in an abnormal *HFE* protein. This abnormal *HFE* protein (along with the other iron-regulating proteins) communicates with hepcidin, sending the message to hepcidin that the body mistakenly needs more iron. So hepcidin then in turn interacts with the gut, and the resulting low amount of hepcidin signals for more iron absorption. This signaling feedback loop eventually results in the iron overload of hemochromatosis.

So the pathway is intact, but it is receiving the wrong message and not being regulated properly.

If hepcidin is now considered the key regulator of iron metabolism, is hepcidin going to affect the current or future management of hemochromatosis? Can knowing about hepcidin help in the diagnosis of HH, or for predicting the amount of iron-loading a patient may have?

At this time, measuring the amount of hepcidin in a patient's blood does not appear to be necessary or useful for the diagnosis of the common type of hemochromatosis. Currently, serum ferritin and transferrin saturation are still the best screening tools for helping to diagnose the common type of hemochromatosis (*HFE* – HH). But measuring hepcidin may be helpful in sorting out puzzling cases of iron overload that are not attributed to mutations in the HFE gene. Accurate hepcidin measurement both in the blood and in the urine are still being fine-tuned in the research setting.

With respect to predicting the rate or extent of iron-loading in hemochromatosis and helping with ongoing management of the disease, simpler monitoring tests such as serum ferritin, transferrin saturation, and hemoglobin are still more useful and more specific at this time. Hepcidin is still at the research level, so it remains to be seen if and how it can be best incorporated into clinical practice both for iron overload and anemia.

Ed note: the Canadian Hemochromatosis Society will keep you posted in future newsletters and on our website about how hepcidin research and its effect on hemochromatosis are evolving.

Message from the President

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year. This spring we received our largest single donation to date from the estate of a friend of the late Eugene Boyko. I am certain Eugene would be delighted to know that his legacy lives on in this way. This gift also serves to remind us of a trend in charitable giving that focuses on estate planning. To that end, we have reprinted some information on this subject from an earlier newsletter in the hope of providing yet another tool to our members who are interested in maximizing the effectiveness of their charitable giving. "Planned Giving Benefits the Giver and the Receiver" appears on page seven.

I hope you enjoy your read, and as always, when you're finished with your copy, hand it to a friend, or if you are receiving this electronically, forward it to a friend.

Elizabeth Minish, President

Anne Stang: Member in the Spotlight

continued from page 1

what the long-term effects of my phlebotomies and diet changes will be. The doctor does not think I have sustained any tissue damage. I still get stiff of course, but my energy level has been much higher for almost a year and the mood swings are gone. Some visible weight loss was a welcome side-effect! Overall though, the HH diagnosis has not changed my life much except to make me even busier. I am a retired teacher librarian and I occupy my time with reading, gardening, sewing, cooking (even with tofu), cycling, cross-country skiing, travelling, going to operas and other concerts, singing, and volunteering.

I have a large family with eight living siblings, and my doctor suggested to me that I inform them about hemochromatosis. They have all had their ferritin checked now, and several have had the genetic test. One older sister also has HH and two sisters are confirmed carriers. I suspect another sister and an older brother are also carriers, and the other three are unaffected. My sister with HH has had monthly phlebotomies, and one of her daughters has also had several as she inherited the H63D gene variation from her father. My sister, who is a carrier, has had four phlebotomies and is awaiting another appointment with a hematologist to see if she needs more. The suspected carriers in the family are either on the wait-list for genetic counselling or are contemplating the referral.

Because my parents were both from very large families, and two of mom's sisters married two of dad's brothers, I felt that it was extremely important to reach further than my sibling branch of the family tree, and inform as many relatives as possible. I talked a lot at family gatherings and sent some letters and emails. The first news of an affected relative came from the daughter of a "double" cousin. She was diagnosed at 22. Since then, I have found out that her mother (my cousin's wife) and brother also have HH. So the hemochromatosis family history is growing. We all now use the CHS website and have become society members.

My advocacy about HH has included talks to my ethnic group and to a senior's group, as well as several short articles in my ethnic and community newsletters. My ethnic group is called the Germans from Russia (GRs). They are the Germans who migrated to Russia between the 1760s and 1840s, and then to the Americas from about 1870 to 1930. They lived in their own communities in Russia, and often did that as well for the first years in Canada, the United States and South America. Consequently, there were not a lot of marriages outside of the community for as much as two hundred years. The GR population eventually numbered well over 1 million, and given the history, I wonder if the incidence of HH is higher among the GRs than in the general Caucasian population.

It was this speculation that prompted me to visit Macklin, Saskatchewan, where many of my relatives live, for the annual Bunnock Tournament. Bunnock is a game that was brought to Canada by the GR ancestors. I knew the tournament brought many visitors to the area, so I arranged to have a table at their fair to explain HH and to distribute brochures. Because I am related to many families in the area, I received more than a polite reception and many promised to talk to their doctors. I hope the doctors in the area will be inundated with some of the 400 brochures I handed out!

I have met very few people in Calgary with HH, but I am hearing more informed responses when I tell people about my condition. Just this week, my massage therapist told me that one of his clients complained about her diabetes and other symptoms. He remembered our conversations and gave her one of the brochures I had left there. A week later, she called the therapist to say she had been diagnosed with HH. A little effort from all of us to spread the word can make a difference for so many others.

Anne Stang

What's New in HH Literature?

Risk of iron overload in carriers of genetic mutations associated with hereditary haemochromatosis: UK Food Standards Agency workshop. Mamta Singh et al., British Journal of Nutrition (2006), 96, 770–773

The UK Food Standards Agency convened a group of expert scientists to review current research investigating diet and carriers of genetic mutations associated with HH. The workshop concluded that C282Y heterozygotes (HFE carriers) have a slightly higher transferrin saturation level compared with healthy individuals with no mutations in the HFE gene, but they do not have a higher serum ferritin concentration. Absorption of dietary iron, and iron stores, do not appear to be significantly higher in C282Y heterozygotes (carriers) than in wild types. HFE carriers appear to respond normally to dietary iron and do not need to alter their diet to avoid increased levels of stored iron.

Editor note: this UK recommendation is for HFE carriers only. The literature suggests that HH patients (those with 2 mutations in the HFE gene) should reduce intake of foods that are iron-rich. The effect of heme iron intake (from meat, fish, and poultry) is two times greater on C282Y homozygotes than other groups, so these types of foods should be minimized. (Non-heme or plant-based iron such as in beans and grains has little effect so they are less of a problem.)

Coming Up in Iron Filings, Spring 2007

Part of our role at the CHS is to raise hemochromatosis awareness in the medical community.

In our Spring, 2007 newsletter we will outline the newly developed Alberta medical protocol for screening for iron overload and hemochromatosis. We will also review other Canadian and US screening procedures and discuss who should be tested and when.

Enjoy your newsletter!

Please pass it on. Our newsletter is also available online on our website. If you would rather read it electronically let us know and we'll take you off the list.

Anna Kyle Recognized

Congratulations to CHS new Board Member Anna Kyle for being honoured with a civic award in Ottawa due to all her volunteer efforts for health charities, including the Canadian Hemochromatosis Society.

Xenon Research Funding Approved

Congratulations to Xenon Pharmaceuticals Inc. in Burnaby, BC for the approval of the 2nd half of their 3-year co-funded federal government research grant from Genome BC / Genome Canada. Xenon's iron metabolism program involves the development of better screening tests and therapeutic options for hemochromatosis.

Speed up the diagnosis: Educate your GP!

Experience has taught us that the first hurdle we encounter in getting a diagnosis for hereditary hemochromatosis is our family doctor. You can help to change this fact. The next time you visit your family doctor, ask if he/she will display our brochure in their office. Just let us know the number of brochures that you need. Alternatively, we can mail them directly to the doctor's office if you prefer – all we need is a name and address! Become part of the network of HH educators!

On the look-out for HH friendly Doctors…

If your family doctor or specialist is accepting new patients and you have found them to be supportive in helping you or a family cope with HH, please let us know. We would like to build a Canadian list of HH friendly Doctors to be able to pass on their names to those seeking medical attention.

Thank you to Donors

The CHS sends out a heart-felt thank you to all those who have already generously donated to the society this year. A very special appreciation goes out to the estate of Mrs. Jeanne Mary Fairn of Langley, BC from whom the CHS recently received $53,900.

We have attached a list of this year's donors, and to you we extend our heartfelt thanks. Because of your generosity, we are able to provide the best service we can in supporting those affected by hemochromatosis.

Planned Giving Benefits the Giver and the Receiver

Many individuals consider planned giving as a way to be remembered, as a way of giving to their favourite charity and as a way of lessening taxes and probate fees. Financial planning can address the ongoing needs of a family, and provide a lasting contribution to the Canadian Hemochromatosis Society.

Many financial instruments exist that allow donors to take advantage of this giving method.

- Bequests in a will
- A gift of property
- Life insurance
- Gifts of securities and stocks
- Endowment funds
- Charitable remainder trusts
- Charitable gift annuities
- Gifts of residual interest

Bequest In A Will

A lump sum or a percentage of an estate can be left to CHS. A receipt will be issued to offset estate taxes and taxes arising from capital gains.

Gift of Property

Any property can be donated through a will.

Life Insurance

An individual can purchase life insurance with CHS as beneficiary, with tax receipts issued for premiums paid. Current policies with cash value can also be donated and a tax receipt given for current value. Life insurance is not taxable when paid out as a death benefit and may be used to offset taxes owing.

Endowment Funds

Charities establish endowments to provide a source of future income. Donors may stipulate that a gift is be used for the charities endowment fund.

Charitable Remainder Trusts

Donors can transfer cash, securities, property, bonds etc to a trust. Income generated by the trust is then paid to the charity. When the trust ends or the donor dies, the remainder is distributed to the charity. The donor receives a receipt when the trust is established, based on the market value at the end of the trust. These assets do not form part of the donor's estate, and will reduce probate fees.

Charitable Gift Annuities

Annuities can generate a regular income for a donor. The charity is given a gift of capital, and buys a lifetime annuity for the donor. The income from this annuity is payable to the donor and is tax free.

Gifts of Residual Interest

An individual can deed a property to charity, and retain the use of the property for life, or other predetermined time, and receive a charitable receipt for the present market value of the residual interest. The advantage to the donor is reduced taxes and probate fees.

You can help the Canadian Hemochromatosis Society by making a planned gift to the Society. We recommend that you consult with your financial advisor, lawyer and life insurance agent. Call our office for more information.

Husky-Mohawk Loyalty Card

Thanks to all of you who are now using the Husky-Mohawk Loyalty Card. For every purchase from a Husky-Mohawk gas station and convenience store, CHS receives a percentage of your purchase. This is a very convenient way to support the Society. So far, $128.20 has been donated back to the CHS. So keep filling those gas tanks!
Please call or email our office to receive your free loyalty card. Please note that Husky-Mohawk retail outlets are located only in the Yukon, Western Canada & Ontario. To find a location list, go to www.huskyenergy.ca.

Did you know?

- You can donate securities to CHS without incurring a capital gains tax on profits? Ask your financial advisor.
- All of your membership fee is tax deductable?

How Can You Help the CHS?

There are many ways in which you can help CHS inform and build awareness about the most common yet not well known genetic disorder.

- Take brochures with you to drop off at your library, community centre, and health care office (doctor, dentist, testing lab, local hospital, walk-in clinics, Canadian Blood Services Clinics).
- Refer family and friends to our website www.toomuchiron.ca
- Pass on your newsletter when you have finished with it.
- Volunteer to assist during Awareness Month at information tables in malls, hospital lobbies, and schools.
- Are you a member of a service organization? We can send you a power point presentation for a presentation to fellow members.
- Do you live in the Greater Vancouver area? We can always use volunteer help with data entry, and clerical tasks in our office. Call us at 604-279 – 7135, or email office@toomuchiron.ca
- Donate to the CHS.

Contact us!

Canada Post	#272 - 7000 Minoru Boulevard Richmond, BC Canada V6Y 3Z5
Telephone	604-279-7135
Fax	604-279-7138
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THANK YOU!

November, 2006