# Iron Filings

**The Newsletter of the Canadian Hemochromatosis Society** **Spring, 2005**

## Awareness Week Project Does the Job


The Toronto support group set up an info booth during Awareness Week 2004 at the Don Mills Centre. More than 250 people stopped by and collected literature. Before the both was completely set up, a young woman read one of the plaques on the counter (listing symptoms) and said "Tha's me! I've got all but two of those." Adele Patterson and her daughter spoke with her and by the time she left, she was "shaking with hope."

On the last evening, a woman stopped to look over the info. Her husband had been told he had hemochromatosis. When asked what treatment he was getting, she said "Nothing!"

The group felt it was a worthwhile exercise and will do it again this year. Kay's son David had his company print the banner and plaques, designed by Steve Traino, a member of the group. Radio Shack at the mall provided equipment for the video.

Thanks to all the members who sat at the booth, and special thanks to Linda Finlay and husband Scott who converted the CHS public service announcement into a useable format for radio and TV stations.

*Photo l-r: Kay Easun, Morley Patterson, Kimberly Morrison, Adele Patterson, Jean Saleranti.*

## Inside



## Hemochromatosis

heem-ah-chrom-ah-TOE-sis

**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 Editor*

# New Research Raises New Questions

Researchers identified the first gene for hemochromatosis in 1996. Patients and practitioners alike hoped the discovery would lead to earlier diagnosis of HHC through family screening and even broad population screening. Much has happened in genetic research since then, but instead of the picture getting clearer, it is getting more blurry by the year.

Several different mutations of the HFE gene have now been identified, as have four additional types of hemochromatosis caused by defects on different genes. Each type seems to affect a slightly different aspect of the iron absorption mechanism, and perhaps because of this, the irregularities in iron profile tests vary.

Until recently, HHC has been considered a recessive genetic disorder: you need two copies of the defective gene, one from each parent to develop hemochromatosis. New research indicates that Type 4 hemochromatosis is dominant, so you only need one copy of the gene to be at risk for loading iron.

All this is further complicated by the fact that not everyone who has two defective copies of the gene for hemochromatosis actually goes on to manifest symptoms, and that some people who only have one do develop the disorder. Clearly, further research is required.

We try to shed some light on the genetics of hemochromatosis in this issue of the newsletter. Early diagnosis has always been a primary goal of the CHS, and we attempt to sort out how this new information may help or hinder that cause.

This issue of the newsletter also features information about and ideas for our annual Awareness Week at the end of May. Of course our campaign to educate people about hemochromatosis is ongoing, but the last week of May gives us the opportunity to focus our attention and efforts for maximum impact. We also give notice of our other rite of spring, the AGM, which is undergoing a bit of a format change this year.

In our on-going attempt to let our members and donors know how vital they are to the important work of the society, we have some information from our Treasurer about where our revenues come from and how we spend the money we receive.

Thank you for your interest and support.

*Elizabeth Minish, President, CHS*

## Volunteer Contacts

We need contacts in outlying areas. We have no contacts in PE, NT, or NU, so please call us if you can help.

### MEETINGS

**Ottawa-Gatineau Support Group**

April 13, May 11, June 8, 2005, Riverside Hospital Boardroom, 1967 Riverside Dr., Ottawa. Parking is $4.50. Call Marjorie Lounder at 613-739-9277 (jlounder@magma.ca) or Elaine Robinson at 613-521-5897.

**Toronto Support Group**

April 28: A geneticist will speak to the group. Meetings are held at Kay Easun's home in downtown Toronto. Call 416-598-5248 for more information.

**Richmond Support Group**

The Richmond Support Group requires a new leader. Please contact our offices.

*Newsletter produced by Chris Petty*

## Iron Filings in French, Too

*Iron Filings* is available in French translation for our members who prefer their serious reading in the language of Voltaire.

Our translator, Frédéric Achi, originally from France, is a 26 year-old with two passions in life: languages and travel. He has travelled all over the world, even spending a year in Japan. He arrived in Canada in 2003, "to discover a country I always wanted to visit since I was a child." He feels so good here, he says, that he just might stay.

About *Iron Filings*, he says, "I always wanted to volunteer for a good cause. That's why I am very happy to help the CHS spread information about the disorder."

Thanks, Frédéric. For more information on how you can get a copy in French, call or email our office.


# Iron Filings

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## The Disorder

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

## Our Purpose

The society is dedicated to the dissemination of information about the disorder, and its early diagnosis and treatment.

# Mutated Genes, Your Parents, and Too Much Iron

## A Hemochromatosis Primer

Hereditary Hemochromatosis (HHC) is an inherited genetic disorder that causes the body to absorb too much iron from a normal diet. Iron is absorbed in body tissues (i.e., liver, heart, pancreas) and cannot be excreted. Over time, this iron overloading can damage vital organs, causing such diseases as cirrhosis, heart failure, diabetes, arthritis, thyroid disease and many others.

The gene associated with most cases of HHC is named HFE and was discovered in 1996. Normally, this gene creates a protein that manages the transfer of iron between the blood and bone marrow, organs and other cells. The protein creates mechanisms that block iron absorption when adequate supplies are present, and facilitates absorption when more is needed to produce blood or support tissue health. When changes or mutations occur within this gene, it produces a protein that is ineffective in blocking iron absorption, so iron builds up in the body.

The two most common mutations in the majority of patients with HHC have been named (with typical scientific obscurity for the layperson) C282Y and H63D, with C282Y causing greater problems than the other form. Originally, researchers thought just these two mutations of the HFE gene were to blame, but recent research has turned up additional mutations that might contribute to activating HHC, while even newer research suggests that mutations in genes other than HFE cause other types of HHC (see chart page 5). Studies are underway to determine how significantly these new mutations contribute to the severity of problems of iron metabolism.

**Genes and Their Impact**

Genes produce protein. Each gene or gene pair produces a particular protein designed for a specific job, from building body structures to managing minute processes. Researchers have been able to identify the genes that make up the human genome, and know what most of them do. How they work, what triggers them and why they fail is still a mystery. It's like giving a copy of the Oxford Dictionary to space aliens: they might possess the language, but they have no idea how it works.

**Homozygotes**

Every person has two copies of each gene, one from each parent. In a recessive genetic disorder such as Type 1 hemochromatosis, a person must receive two copies of the mutated gene to develop the disease. Such a person is said to be *homozygous* for the condition e.g., C282Y/C282Y, or, H63D/H63D.

**Heterozygotes**

A person who inherits one faulty copy and one correct copy of the gene for hemochromatosis (e.g., C282Y/normal or H63D/normal) is called a *heterozygote* carrier of the disease. Researchers believe that heterozygotes with one copy of the C282Y gene may have mildly increased iron levels but are at extremely low risk of developing HHC. And if they had one copy of the lesser mutation (e.g., H63D), their chances are even lower because the remaining normal copy of the HFE gene is sufficient in most carriers to prevent the development of hemochromatosis.

But even if a carrier doesn't develop the condition, they still may pass the mutation on to his or her children. If two carriers have children together, each of their children has a 25% chance of inheriting two mutated genes and a 50% chance of inheriting one.

**Compound Heterozygotes**

A person who inherits a combination of two different faulty copies of the gene for hemochromatosis (C282Y/H63D) is called a *compound heterozygote*. Researchers estimate that fewer than 2% of these individuals will develop symptoms of hemochromatosis.

**Triggering Factors**

The Centre for Genetics Education Fact Sheet states that "while genetic carriers of HHC would usually show no signs … they still may develop it if they have diabetes, have a high alcohol intake or have some other triggering factors."

Other triggering factors outlined in ***"Exposing the Hidden Dangers of Iron"*** by Dr. E. Weinberg of the Iron Disorders Institute include a recently discovered hepcidin protein, diet, a non-identified mutation of HFE that

### Glossary

**Autosomal** Humans have 26 pairs of chromosomes; 23 have nothing to do with sexual characteristics and are called 'autosomal.'

**C282Y** Refers to the most common mutation of the HFE gene. The C282Y mutation results in a change in the amino acid in the protein produced, and affects how the protein 'folds,' thereby limiting its ability to perform its task.

**Carrier** A person who carries a gene that causes a disorder, but does not show symptoms.

**Compound Heterozygotes** Individuals who inherited two different mutated versions of a gene, one on each of their chromosomes.

**DNA** Short for deoxyribonucleic acid, DNA is the molecule that carries genetic information.

**Ferritin** A protein that stores iron. High ferritin levels in the blood can be one indication of hemochromatosis.

**Gene** A portion of DNA that contains instructions for making a protein.

**H63D** A mutation of the HFE gene that can cause hereditary hemochromatosis.

**HFE** The gene on chromosome 6 that encodes the HFE protein. People with Type 1 hereditary hemochromatosis have abnormal HFE proteins due to mutations in the HFE gene.

**HHC** Abbreviation for hereditary hemochromatosis.

**Heterozygotes** Individuals who inherited one normal and one mutated version of the same gene, one on each of their chromosomes.

**Homozygotes** Individuals with two identical mutations of a gene, one on each of their chromosomes.

**Phlebotomy** Blood-letting, the common treatment for hemochromatosis.

**Transferrin** A protein that circulates in the bloodstream and binds to iron thus "ferrying" iron to other parts of the body. Transferrin is normally 30% bound to iron.

## Types of Hereditary Hemochromatosis

| | Type 1 | Type 2A | Type 2B | Type 3 | Type 4 |
|---|---|---|---|---|---|
| **Name** | Classic HHC | Juvenile Hereditary Hemochromatosis | | T*f*R2-related HHC | Ferroportin-related iron overload |
| **Gene** | HFE | HJV | HAMP | T*f*R2 | SLC40A1 |
| **Protein Produced** | HFE | Hemojuvelin | Hepcidin | Transferrin receptor 2 | Ferroportin (iron regulatory protein) |
| **Inheritance** | autosomal recessive | autosomal recessive | autosomal recessive | autosomal recessive | autosomal dominant |
| **Function** | interruption of transferrin-bound iron, possible modulation of hepcidin expression | unknown; possible hepcidin modulation | regulation of iron release in intestinal and blood cells | possible interference of iron uptake by liver cells | possible interference of iron export from intestinal, liver and placental cells |
| **Organ damage** | variable | high | high | variable | low |
| **Decade of onset of symptomatic organ disease** | 4th or 5th | 2nd or 3rd | 2nd or 3rd | 4th or 5th | 4th or 5th |

Adapted from New England Journal of Medicine, 350;23, June 3, 3004:
Classifications as defined by OMIM (Online Mendelaian Inheritance in Man)

There may be other as yet unknown functions related to iron overload. The listed functions do not, at least at this time, always account for the known pathophysiological features associated with gene mutation.

investigators have named R6S, mutations in the protein transferrin (which transports iron in the blood) or to transferrin receptors, or to any other of the iron-absorption/excretion mechanisms in the body.

**Testing**

Testing is important because symptoms are nonspecific and can resemble other medical problems. Iron tests are not ordered routinely by physicians. Patients who are heterozygote or compound heterozygote, should have their blood tested for iron levels every 2-3 years. If there is a significant rise in ferritin levels or a rise in transferrin saturation levels, then testing may be required more frequently.

With undiscovered genes and other unknown contributing factors involved, it is essential that each person take charge of his or her health and stay on top of medical issues.

**Preventive Care**

Genetic illnesses are not, as yet, preventable, but making a few lifestyle changes can make a difference:

- don't take iron supplements, or vitamins with iron in them
- avoid taking vitamin C (especially with food), which enhances iron absorption
- don't eat raw shellfish
- reduce alcohol intake, as metabolizing alcohol enhances iron absorption and can stress an already compromised liver.
- reduce the consumption of red meat.
- stop smoking. Tobacco has high levels of iron.
- avoid or reduce enriched and fortified foods (i.e., breakfast cereals, flour, pasta)
- don't use iron cookware as it will add iron to food

**Conclusion**

Although statistics for risk to hemochromatosis carriers is rated low, evidence is mounting that iron loading may occur in heterozygotes and compound heterozygotes. New research is inconclusive about the statistical likelihood that any category of carrier is more or less susceptible to developing the disorder, as more mutations have been observed on HFE and other genes responsible for some aspect of iron metabolism.

Research continues to advance our knowledge of genetically-linked disorders at an amazing rate. Nonetheless, the more we know, the less sure we are. It's important that all carriers be suspicious of their own iron levels, and insist on monitoring them closely.

*Thanks to Anna Kyle for researching this article, and for providing references. Accuracy of the information contained herein is the responsibility of the editor.*

References

**Exposing the Hidden Dangers of Iron**, pp 36, 37, 48, Iron Disorders Institute
In addition to the known mutations of HFE, investigators have discovered several other gene abnormalities that modify iron homeostasis.

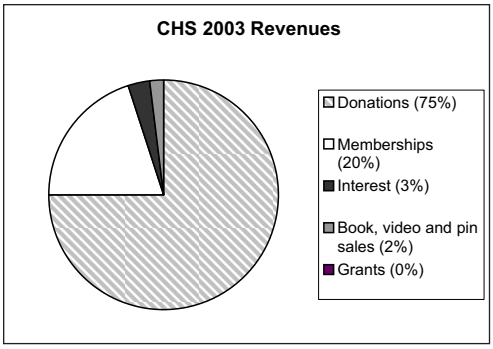
**CDC Genomics and Disease Prevention**, Oxford University Press (2004), Gene-gene and Gene-environment Interactions: Findings suggest that non-genetic influences, additional HFE mutations, or variation of additional genes affecting iron metabolism may also modulate iron overload.

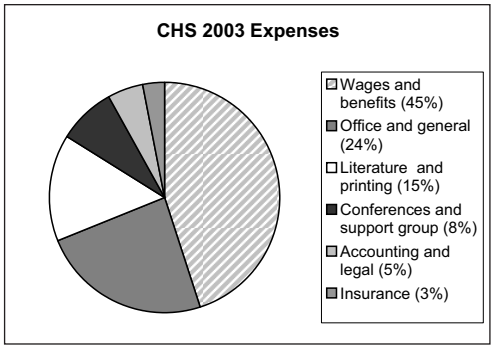
**Cancer Epidemiol Biomarkers**
Prev. 2004 Feb; 13(2):205-12
Altered iron metabolism in C282Y carriers may promote the development of breast cancer and/or more aggressive forms of the disease.

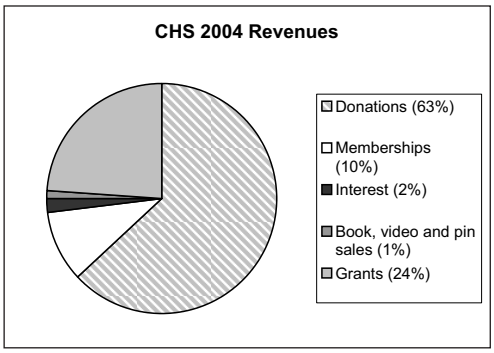
**Nutrition Review** Vol. 61, #2, Feb. 2003
Health Implications of Iron Overload: The Role of Diet and Genotype
Heterozygotes may have poorer control of iron absorption when presented with large amounts of highly bioavailable iron.

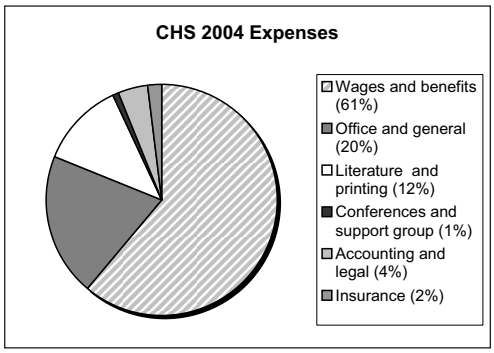
**Journal of the National Cancer Institute** Vol. 95, #2, 154-159, January 15, 2003
Association Between HFE Mutation Carrier Status and the Risk of Colon Cancer
HFE gene mutations are associated with an increased risk of colon cancer. Cancer risk is greatest in mutation carriers who are older or consume high quantities of iron.

**Genetic Health**, November 15, 2000
Inheriting Just One Copy of a Mutated HFE Gene Can Be a Problem. People who have inherited just one HFE gene with the C282Y mutation may be at risk for cardiovascular disease due to small increase in iron storage.




# Numbers Tell the Story

The graphs above indicate sources of revenue and expenses for the last two years of operations of the Canadian Hemochromatosis Society. In both years, donations made up by far the largest portion of our revenues, while wages and benefits of our staff were our major expenses.

In 2004, the society hired a part time, professional executive director in the person of Agnes Papke to streamline office procedures and help develop our fund raising potential. This hiring accounts for the increase in wages and benefits.

Late last year Agnes organized an onsight analysis for the board to discuss the future mission of the CHS, and how we might go about raising more money to bring that mission to life.

At present the society is largely dependant on member donations for all its funding requirements. We appreciate members' support very much and would not be able to carry out the society's work without it. Your support is vital to our effort.

With new research about genetically-related illnesses coming out daily, our awareness campaign is more challenging now than ever before.

The board and staff will continue to investigate every avenue for funding so we can get the word out, convince more physicians to test for hemochromatosis, and be a support for victims of this disorder.

We invite all members to attend our Annual General Meeting on Tuesday, April 5 at 5:00 pm at the Richmond Caring Place.

### Donate Your HBC Reward Points

Zellers, The Bay, and Home Outfitters now issue HBC Rewards points. Help us by donating your points to the Society. Use our card #593 471 099 under the name CHS. Be sure to tell the rewards centre that you want to keep your own card active when donating points, or they will cancel it.

### Good Donations

You can donate online through our website. Visit www.canadahelps.org. Search "hemo," then click "Donate now." This is a secure site. You can use your credit card with confidence.

### Matching Gifts

Does your employer have a matching gift program? If so, please indicate the company name on your donation. If you aren't certain, just send us your employer's name and we can follow up. Many firms will match some portion of their employee's charitable donations.

### When sending money . . .

. . . such as a cheque or Visa number, be sure to let us know what it is for. Money will be automatically entered as a donation unless you specifically tell us it is for a membership or in memory of a loved one.

## *From the Fundraising Chair*

Like all of you, I was shocked and saddened by the devastation caused by the tsunami in Asia. However, I take some consolation in our response to this terrible tragedy.

As a Canadian, I am proud of the way we have rallied to provide moral and financial support to those affected by this international crisis.

This spirit of giving comes as no surprise to me. As the Canadian Hemochromatosis Society's Fundraising Chair, I am privileged to see first-hand just how generous people can be.

Everyday, CHS supporters make a lasting impact through their volunteerism and financial support. Thank you for your kindness to the newly diagnosed families who rely on CHS education programs such as the new website under development.

Thanks to those of you who made our seasonal campaign so successful once again.

Awareness Week (May 25- 31) is coming up fast. What a great time to become a member of the CHS, or perhaps throw a party to raise awareness of hemochromatosis or place brochures and posters in your community.

I salute everyone who makes a difference, both at home and abroad. I urge you all to continue your good work.

- Margaret Campbell

### Enjoy your newsletter!

When you have finished with your newsletter, please pass it on. Our newsletter is also available online on our website. If you would rather read it electronically, or if you don't want future newsletters, let us know and we'll take you off the list.

### Speak Up!

When leaving a message on our toll-free line, 1-877-BAD-IRON, leave your full name and address (spell them out) and your 10-digit number.

# Awareness Week 2005

The week of May 25 - 31 is Hemochromatosis Awareness week in Canada. Please help us get the word out.

There are many things you can do. If you have a Canadian Blood Services unit in your community, call all your friends and do a mini blood drive.

Hand out HHC information at the blood drive, and drop off brochures at doctors' offices, drug stores, pharmacies, and other medical offices. Contact your local paper and let them know about Awareness Week and about hemochromatosis. Chances are they've never heard of it!

Call your local school or college and arrange to give a talk or to pass out brochures to students in medical programs. Give a talk at your local Rotary or Lion's club, or at the Legion.

Or, like the Toronto group, set up an information booth at a local mall or supermarket. You will be surprised how much interest it will generate.

Call our offices for more ideas and for the resources you need.

*Correction*

## Paralytic Shellfish Poisoning Not the Culprit

In "The Great Sushi Debate," (Fall, 2004 *Iron Filings)* we published some erroneous information about how raw fish and shell fish such as is found in sushi (and sashimi) can pose a risk to hemochromatosis patients. We incorrectly attributed the risk to the bacteria that causes paralytic shellfish poisoning (PSP).

While PSP can kill you as easily as it can non-HHC sufferers, that isn't the bacteria that HHC patients need to especially wary of. There are two, *vibrio vullificus* and *salmonella enteriditis*, both of which occur in raw shellfish or seafood, particularly oysters. These bacteria like an iron rich environment, like the blood of hemochromatosis patients. Individuals with compromised livers who get infected have a 50% mortality rate.

Thanks to Bill F., one of our constant Discussion Groups participants, for setting us straight.

## Normal Iron Absorption and Storage

**Iron facts**

- All body cells need iron. It is crucial for oxygen transport, energy production, and cellular growth and proliferation.
- The human body contains an average of 3.5 g of iron (males 4 g, females 3 g).
- The typical daily diet contains 10–20 mg.
- Only about 10% of dietary iron is absorbed (1–2 mg/day).

**Iron absorption**

- Mainly absorbed in the upper intestine.
- A protein, divalent metal transporter 1 (DMT1), facilitates iron transfer across intestinal cells.
- Normally, individuals absorb less than 10% of dietary iron, or 1–2 mg/day
- Most absorbed iron is used in bone marrow to form red blood cells.
- Iron homeostasis is closely regulated via intestinal absorption.
- Once iron is absorbed, there is no physiologic mechanism for excretion of excess iron other than blood loss (i.e., pregnancy, menstruation), and the normal daily loss through skin sloughing, sweating, etc.

**Iron transport**

- Most absorbed iron is transported in the bloodstream bound to the glycoprotein transferrin.
- Transferrin is a carrier protein that plays a role in regulating the transport of iron from the site of absorption to virtually all tissues.
- Normally, 20–45% of transferrin binding sites are filled (measured as percent transferrin saturation [TS]).

**Iron use in the body**

- 75% of absorbed iron is bound to proteins such as hemoglobin that are involved in oxygen transport.
- About 10% to 20% of absorbed iron goes into a storage pool that is also recycled in the creation of red blood cells, so storage and use are balanced.

**Iron storage**

- Iron is initially stored in ferritin molecules.
- A single ferritin molecule can store up to 4,000 iron atoms.
- When excess dietary iron is absorbed, the body responds by producing more ferritin to facilitate iron storage.

## Hemochromatosis on the Internet

If you find yourself with time on your hands and speedy internet line, visit these sites and learn new things about HHC.

**www.has-ironoverload.co.uk/main.html**
Trust the Brits to have us on. Good humour and good info.

**www.ygyh.org/hc/whatisit.htm**
Genetic info and great graphics from the Dolan DNA Learning Centre.

**http://cmmyakman.tripod.com/HFE/hemochromatosis.htm**
Everything you ever wanted to know about HHC, with diagrams.

**www.phd.msu.edu/DNA/HH_family5.html**
From the MSU DNA Diagnostic Program, with info on genetic illness.

**http://home.vicnet.net.au/~johnlee/Hemo/**
An Australian site with great information about iron absorption and its function in the body, with lots of HHC info.

**www.csu.edu.au/learning/ncgr/gpi/odyssey/hemo/**
Another great Australian site, with humour and info.

**www.cdc.gov/hemochromatosis/training/index.htm**
American Centre for Disease Control, hemochromatosis site for health care providers.

Canadian Hemochromatosis Society

# Annual General Meeting

Tuesday, April 5, 2005, 5:00 pm
Richmond Caring Place
7000 Minoru Boulevard
Richmond, BC

Presentation of our annual report, election of board members, recognition of retiring board members and plans for the future.

Refreshments will be served.

RSVP to 604-279-7135 or office@cdnhemochromatosis.ca
Toll Free 1-877-BAD-IRON

Can't make the meeting?
Be part of the proceedings on our conference call telephone!
Call toll free 1-866-888-6959.
then enter password 7135 followed by the # sign.
Call at 5:00 pm
and enjoy the AGM in the comfort of your home!

# Blood Donor Clinic

May 28, 2005

Canadian Blood Services is holding a special clinic in support of Awareness Week, May 25 – 31

4750 Oak Street, Vancouver
Saturday, May 28, 2005
11:00 am – 1:00 pm

Call 1.800.TO DONATE to make an appointment. The clinic is open to everyone, so bring family and friends. Refreshments will be served.

## Contact us!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| Fax | 604-279-7138 |
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❑ **I have HHC** ❑ **A blood relative has/had HHC**

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❑ As a member/donor, I grant permission to publish my name in the CHS newsletter.

❑ Do not publish my name in any CHS media.

Send me ___ brochures and __ information packages.

❑ Payment enclosed ❑ Please charge my VISA

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Please return to:
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THANK YOU!

March, 2005