



Thyrobulletin

Autumn 2014

Graves' Disease

- What is *Graves' Disease*?
- What causes it?
- When will I get better?
- Symptoms
- Diagnosis
- Treatment
- Two *Graves' Disease* stories



Also in this issue:

- Thyroid and nutrition
- Let's light a tree for the holidays
- Thyroid autoimmunity

SAVE THE DATE:

June 13, 2015 – Kingston ON
Thyroid Foundation of Canada
Annual Meeting

*Celebrating 35 years of helping thyroid patients
across Canada and around the world*





FOUNDER / FONDATRICE

**Diana Meltzer Abramsky, C.M., B.A.
1915 - 2000**



**We are the Voice and Face
of Thyroid Health in Canada**

**Thyroid Foundation of Canada
La Fondation canadienne de la Thyroïde**

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2014-2015**

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Important Notice: The information contained within is for general information only and consequently cannot be considered as medical advice to any person. For individual treatment or diagnosis consult your health care professional.

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**Thyroid Foundation of Canada
P.O. Box 298, Bath ON, KoH 1Go**

www.thyroid.ca • 1.800.267.8800



A message from the president



Un message de votre présidente

□ **D**ear Readers and TFC members:

The Thyroid Foundation of Canada was founded by Diana Meltzer Abramsky who suffered from Graves' Disease and wanted to make sure that other thyroid patients were able to access information and get the medical care they needed.

Almost thirty-five years later, the Thyroid Foundation continues to provide information to patients and, in this issue, we share the stories of two patients who have had their own challenges with Graves' Disease. We are also pleased to include an article by Dr. Jack Wall, Diana's physician, who was instrumental in helping her find solutions to her medical condition, and who encouraged her to establish the Foundation.

The Foundation is addressing the need to evolve with advances in technology – both for effective medical care and for communication. Our website www.thyroid.ca attracts a lot of activity: almost 100,000 visitors last month. Patients and their families turn to the website 24 hours a day to find good information and answers to their questions. Pharmacists can also now access an accredited training course on the website.

Now is the time to renew your membership for 2015 (a membership form can be found on page 12) and while you are thinking of us, please remember to support the Foundation by making a generous donation! Just like Diana, think of the many other patients who still need to access information and good medical care. □

Donna Miniely

□ **C**hers lecteurs et membres de la FCT:

La Fondation canadienne de la Thyroïde a été fondée par Diana Meltzer Abramsky qui souffrait de la maladie de Graves et voulait s'assurer que d'autres patients de la thyroïde ont été en mesure d'accéder aux informations et obtenir les soins médicaux dont ils avaient besoin.

Près de trente-cinq ans plus tard, la Fondation de la thyroïde continue de fournir des informations aux patients et, dans ce numéro, nous partageons les histoires de deux patients qui ont eu leurs propres problèmes avec la maladie de Graves. Nous sommes également heureux d'inclure un article du Dr Jack Wall, le médecin de Diana, qui a joué un rôle important pour qu'elle trouve des solutions à son état de santé, et l'a encouragée à créer la Fondation.

La Fondation tient à souligner le besoin d'évoluer selon les progrès de la technologie - à la fois pour les soins de santé efficaces et de communication. Notre site web www.thyroid.ca attire une grande activité : près de 100,000 visites le mois dernier ont été reçues. Les patients et leurs familles se tournent vers notre site web 24 heures par jour pour trouver de l'information pertinente et des réponses à leurs questions. Les pharmaciens peuvent désormais accéder à un cours de formation agréé sur le site.

Il est maintenant temps de renouveler votre adhésion pour 2015 (un formulaire d'adhésion se trouve à la page 12) et alors que vous pensez de nous, s'il vous plaît n'oubliez pas de soutenir la Fondation en faisant un don généreux! Tout comme Diana, pensez aux nombreux autres patients qui ont encore besoin d'accéder aux informations et de bons soins médicaux! □

Donna Miniely



AGM 2014

ANNUAL GENERAL MEETING TORONTO - MAY 31, 2014

□ This AGM, held on May 31, 2014 in Toronto celebrated 34 years of providing much needed information to thyroid patients all across Canada, North America and around the world. Members were pleased to learn of the various activities that are growing within the organization.

The Kitchener-Waterloo chapter reported on their continued success with Education and Awareness meetings. The attendance at those meetings is tremendous with most seats filled and on occasion having to limit how many can be accommodated. There are always great speakers on various aspects dealing with Thyroid Disease.

The London *Friends of TFC* had another most successful Fashion Show fundraiser in 2013. The success of this event is a result of Judy Duncan's dedication and interest in the Thyroid Foundation of Canada, and in research and information on thyroid disease and patients for many years. TFC is very thankful to Judy and her friends for their contributions. So if you're ever in London in the Spring, check to see if this event is happening – you'll be ever so pleased you did. It's well organized, elegant and very entertaining.

Other activities that have taken place across Canada were Information Sessions, displays, participation in Wellness Fairs, etc. Already plans are underway for various sessions in Quebec, Manitoba, Alberta, Ontario and more.

One highlight of the AGM was a talk given by Dr. George Awad on Thyroid Disease and Mental Health. He spoke of the importance of knowing that a malfunction of the thyroid gland can often result in various degrees of mental illness. We were very fortunate to hear what he had to say.



2014-2015 Board of Directors

From left: Frances Salvaggio , Donna Miniely (President), Ashok Bhasen (Past President), Mabel Miller, Melanie Solmon, Josie Frano, Gabriela Albarracin, Angela O'Connell, Dan Fey (Vice President)
Absent: Rinda Hartner (Treasurer)

We welcome some newcomers to the National Board, Josie Frano (Toronto), Dan Fey (Ottawa) and Angela O'Connell (Halifax). We are enthused by their interest and the expertise they bring to TFC. No matter how many are involved with the work of TFC, we are always glad to see more join us. If you feel you have something to offer, consider getting in touch with us.

As Ashok Bhasen steps down as President, Donna Miniely takes his place as TFC's new President. With a long family connection to the TFC (her mother, Marjorie Miniely, founded the London Chapter), she is well equipped and we look forward to the new Board's plans for the coming year. □

Our Aims

Awareness - To awaken public interest in, and awareness of, thyroid disease.

Support - To lend moral support to thyroid patients and their families.

Research - To assist in fund raising for thyroid disease research.



Nos objectifs

Sensibilisation - Accroître l'intérêt du public envers les maladies thyroïdiennes et le sensibiliser davantage à ce problème.

Soutien - Offrir un soutien moral aux personnes atteintes d'une maladie thyroïdienne et à leur famille.

Recherche - Contribuer à recueillir des fonds pour la recherche sur les maladies thyroïdiennes.



Awards



**Former President
Mabel Miller
Appreciation for service**



**Past President
Ashok Bhaseen
For inspirational guidance and leadership**



**Judy Duncan
Volunteer of the Year**



**Barry Hayter, General Manager,
Abbott Laboratories
2013-2014
Thyroid Patient Initiative
Recognition Award**

The AGM is the time we show our gratitude to the volunteers who make TFC's goals possible. Awards are given to those people who have extended their efforts beyond expectations. This year's recipients were Mabel Miller who has served on and off the National Board for many years, Ashok Bhaseen

who has taken on the role of President twice, and Judy Duncan whose work is featured on p.7. This year a Corporate award was presented to Abbott Canada for its outstanding contribution toward thyroid health for patients. We are very grateful to all our volunteers for their caring support.

Public forum

Following the AGM, a Patient Forum was held featuring Dr. George Awad, Professor Emeritus of Psychiatry from the University of Toronto.

Dr. Awad spoke about "Thyroid Function in Health and Psychiatric Disorders", covering the spectrum of thyroid disorders from hypothyroidism to thyroid cancer. He described the many body functions that thyroid hormone impacts and factors that can lead to abnormal thyroid states. He then focused on hyperthyroidism, hypothyroidism and subclinical hypothyroidism.

After this overview of the general considerations, the attendees were invited to submit questions to Dr. Awad in writing. He graciously took the time to read and respond to each question. As a result of this personal touch, the attendees were quite satisfied with the outcome of the Forum. The attendees received a handout of Dr. Awad's slide presentation, which has now been posted on the TFC website: www.thyroid.ca

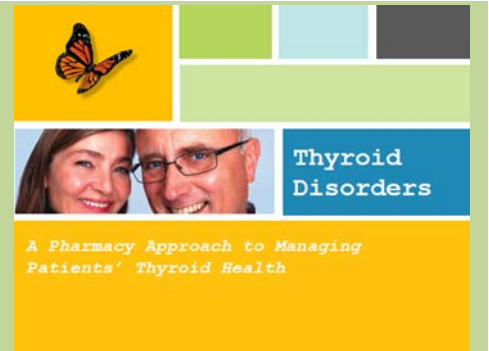


Dr. George Awad and Board Member Frances Salvaggio

The psychiatric disorders that accompany thyroid conditions can be quite challenging for patients and their families. The Thyroid Foundation of Canada is very grateful to Dr. Awad for the insights he provided at the Patient Forum on May 31, 2014. □



Thyroid disorders – A pharmacy approach to managing patients’ thyroid health



The Thyroid Foundation of Canada’s mandate is to provide thyroid education and awareness, not only to thyroid patients, but also to the professionals who deliver our health care. We are pleased to announce a new opportunity to help us in this endeavour. TFC has partnered with Abbott Laboratories in two exciting projects.

The first project, **Thyroid disorders – A Pharmacy Approach to Managing Patients’ Thyroid Health**, developed an online accredited training course for pharmacists to more effectively manage the health of their patients with thyroid disease. The objectives are to help pharmacists to:

- ✚ Recognize the different types of thyroid disease and their typical symptoms
- ✚ Know how hypothyroidism is diagnosed and treated
- ✚ Understand how pregnancy affects management of hypothyroidism

The course is accessed through the TFC website and is followed by a *Thyroid Disorders Learning Assessment Quiz*. On successful completion, the pharmacist will receive a **Statement of Completion** certificate.

When all the pharmacists at a pharmacy complete the training, the pharmacy will receive a certificate of recognition from TFC.

This training for pharmacists has been accredited by the Canadian Council on Continuing Education for Pharmacists (CCCEP). The views presented in the training may not necessarily represent the views of the Thyroid Foundation of Canada.

Our second project, **Pharmacy Thyroid Health Days**, will take place in various pharmacies across Canada and are open to the public. A registered nurse will be on hand and educational material on display.

Find out more about the various forms of thyroid disease, pick up some helpful materials, fill out a questionnaire to pinpoint your symptoms. There will be an opportunity to consult the nurse, so bring your questions!

The first event took place in Nepean, Ontario on November 15th. Other dates and locations are listed on TFC’s website www.thyroid.ca, and will be updated regularly so be sure to check back often to see if one is coming to your area.



Pharma Choice Gabriel Drugs in Nepean hosted the first of Thyroid Health Days
LT to RT: Ricki Samuels (patient referred to nurse by pharmacist), Carolyn Goodfellow (TFC Ottawa Chapter), Tina Perkins (nurse)

We hope to see you at one of our **Pharmacy Thyroid Health Days**.

The two projects are made possible through an educational grant from Abbott Laboratories.



Fundraising with fun and flair

□ Judy Duncan has been organizing the very successful “*A Touch of Spring*” Fashion Show and fundraiser event for the Thyroid Foundation of Canada in London Ontario since 2000.

Judy’s connection with thyroid disease began 19 years ago when her husband, Bruce, was diagnosed with thyroid cancer. Bruce’s father had had the same diagnosis and other family members have also had some form of thyroid disease.

Luckily Bruce recovered and when Judy retired from her job as AVP, Human Resources at TD Canada Trust, she decided to attend a Public Education Meeting of the Thyroid Foundation of Canada in London to learn more about thyroid disease. She met Barb Cobbe, the London Chapter President, who was planning to hold the very first Thyroid Foundation fashion show to raise funds for thyroid education and research. Judy offered to help and since then has helped organize 14 Fashion Shows for TFC.

The first show was a dessert and coffee affair and raised about \$500. It was such a success that for the second year they moved the venue to the Hellenic Centre to accommodate more people, and the dessert and coffee became a full-course dinner. The 2014 show was a sellout and raised \$13,500, the highest amount ever.

Local businesses donate the models’ clothes, the prizes or their services. All the models are volunteers, and most have a connection to thyroid disease. They also contribute by selling tables.

Everyone has a great time participating in the event and although many hours are spent organizing everything, Judy feels very proud of their achievements. Even her two daughters, Kayla and Krista, participate and both have acted as treasurer, great experience for young people. Judy says what makes it so successful is that everyone helps. “Having the thyroid connection and the passion to make a difference is also helpful.” At this year’s AGM, Judy was awarded “Volunteer of the Year” for the second time.

Spring 2015 will be the 15th Annual “*A Touch of Spring*” Fashion Show. Judy is ready to hand over the reins to someone else. If you would like to help with this year’s show, learn the ropes and take on future fashion shows, or if you’d like to hold one in your area, please let us know by telephone: 1-800-267-8822 or by email: info@thyroid.ca □

Thank you Judy for your dedication, hard work - and for making a difference!



Krista, Judy, Bruce and Kayla



My Graves' story

Linda Vorano



□ **I** am a Medical Research Administrative Assistant and I was diagnosed with Graves' Disease in 2007. At the time of diagnosis I was experiencing stress in the workplace.

I normally had routine thyroid tests for other medical reasons. In January 2007, my thyroid tests were normal. In April, I began experiencing symptoms of illness and in June 2007, my thyroid tests, T3 and T4 were only mildly elevated, i.e. T3 9.0, T4 29, but my TSH was less than 0.01. My symptoms were rapid heart rate and dry, irritated eyes. My diagnosis was confirmed with antibody blood tests, thyroid ultrasound and radioactive iodine (RAI) uptake test. I was started on 10 mg of Methimazole (brand name Tapazole). Though this addressed the symptoms of rapid heart rate and normalized my T3 and T4 fairly quickly, my TSH did not budge from less than 0.01 for two years and my eyes remained irritated. I still had antibodies and did not feel like myself, though I know I felt better than some Graves' patients who had had treatments of either surgery or RAI and replaced with synthetic T4.

Then one day, I came across a medical article by Dr. Salvatore Benvenga, an endocrinologist in Italy, who in 2001 did a research study with hyperthyroid patients using L-carnitine, an amino acid that is normally found in the body. The results were astounding! I showed the article to my boss (a Genetic Metabolic specialist who routinely used L-Carnitine for some of the diseases he specializes in) and he said, "Well, it won't hurt you". I

then went to my local health food store and bought some 500 mg L-carnitine tablets and started on 1 or 2 tablets a day. At my next visit, my TSH had risen for the first time to 0.05. I felt more normal. I was able to lower the Methimazole to 7.5 mg. I then tried adding more L-carnitine and combining different types of carnitines (Regular L-carnitine and Acetyl-L-Carnitine). My TSH rose to 0.78 and I attributed that to the addition of Acetyl-L-Carnitine. Then the Canadian government banned the sale of Acetyl-L-carnitine in Health Food stores and my TSH rapidly dropped to 0.12. I continued on the regular L-Carnitine (purchased through a doctor's prescription) along with continuing the Methimazole. I was able to drop my Methimazole down to 5.0 mg daily. Then L-carnitine was allowed to be sold again in Canada in Health Food Stores and I stopped the Regular L-Carnitine and started on Acetyl-L-Carnitine, 1000 mg a day with 5 mg of Methimazole and my next TSH was 1.4!!!. My T3 and T4 remained in the normal range throughout all of this. My dry and irritated eyes were treated with collagen plugs inserted in 5 minutes in my ophthalmologist's office which blocked the tear ducts. I have shared my experience with other Graves' patients on other Patient Boards. Some of them had far more impressive results than I did. They have tried Acetyl-L-Carnitine and/or regular L-carnitine and report feeling normal for the first time in a long time and are seeing a rapid improvement in their blood results and symptoms of brain fog and muscle weakness. The only "problem" I have with the



Acetyl-L-carnitine is that it is too good! I have decreased my dose down to 250 mg a day along with 5 mg of Methimazole and my TSH was 2.4. So my endocrinologist has now decreased my Methimazole down to 2.5 mg. My eye symptoms improved dramatically at this level.

It was important for me during all of this to continue to be monitored by my physician and of course to have frequent blood test monitoring. Before starting the L-carnitine I had my free carnitine level checked and it showed I was carnitine deficient. I was also Vitamin D deficient. I believe that most Graves' patients become carnitine deficient because of their disease and are Vitamin D deficient; replacing these two elements greatly aids recovery and healing from this disease. I no longer have antibodies. I believe L-Carnitine was so beneficial to myself and other Graves' patients because hyperthyroid patients rapidly lose L-Carnitine from their muscles due to their disease and need to replace it. I have posted my results on some Patient Boards, specifically (www.patient.co.uk) and heard back from other patients who have tried the L-Carnitine and Acetyl-L-Carnitine.

One woman reported about her 18-year-old daughter who had Graves' for 5 years and still had negative symptoms such as weight gain on the antithyroid drugs, sleeplessness, poor energy, etc. She reported after 2 weeks of starting on Acetyl-L-carnitine and vitamin D: *"Her last lab test result was TSH <0.01, T4F 24.4, T3F 10.0...her lab test result from today is TSH 0.01, T4F 23.6, T3F 8.3. (her TSH has not budged in over a year until now...even when she took a high dosage of Tapazole). We knew her test was going to change for the better because of her behavior...more energy, more talkative, no more sleep problems, no more moodiness, more*

laughing and kidding around with us...it's been amazing! We hope to continue to experiment with the dosage".

And after a further two weeks (1 month in all) she reported, *"You're not going to believe this....my daughter's FT4 and FT3 are both within the range. This marks the first time she has been in range without having to increase her Tapazole dosage. In fact, in March, we decreased Tapazole by 2.5 mg and increased the Acetyl L-carnitine and her results have dramatically changed...not just a little dramatically. She went from FT4 24.3 to 15.6, FT3 8.2 to 5.81...her TSH remains at .01, but I was told that that doesn't matter as much. I can't tell you how excited I am. This will mean I can lower the Tapazole by another 2.5.mg until she is stable. Thanks for spreading the L-carnitine news and people out there who are struggling...please give this a try!"*

The safe dose is up to 3 grams a day or 3,000 mg, of the regular L-Carnitine, however one of the side effects reported at higher doses is stomach hyperacidity. For the Acetyl-L-Carnitine, I have never had to take over 1,000 mg.

I would recommend that patients tell their doctors that they have heard or read that Graves' patients are carnitine-deficient and that they would like to be tested for this. The test is for Free carnitine and Total carnitine but is not covered by Ontario Health Insurance Plan. The cost is approximately \$40.00 (it still needs to be ordered by their doctors). If they are found to be deficient (as I was and as most Graves' patients will be) then they can justify to their doctors adding the L-Carnitine or Acetyl-L-carnitine to their medications. This makes more sense to their doctors because they will have proof it is needed. □

Related medical articles, p. 10



Related medical articles:

Usefulness of L-Carnitine, A Naturally Occurring Peripheral Antagonist of Thyroid Hormone Action, in Iatrogenic Hyperthyroidism: A Randomized, Double-Blind, Placebo-Controlled Clinical Trial; Salvatore Benvenega, Rosaria M. Ruggeri, Antonia Russo, Daniela Lapa, Alfredo Campenni, and Francesco Trimarchi; The Journal of Clinical Endocrinology & Metabolism 86(8):3579–3594; http://press.endocrine.org/doi/pdf/10.1210/jcem.86.8.7747

Effects of L-carnitine on Thyroid Hormone Metabolism and on Physical Exercise Tolerance; S. Benvenega, Horm Metab Res 2005;37:1-6

Carnitine and Thyroid Disease; Why Hyperthyroid Patients Need Carnitine; Julium G. Goepp, MD; Life Extension Magazine, Dec. 2007 http://www.lef.org/magazine/2007/12/report_thyroid/page-01

A word on the use of L-carnitine

Prakash Chandra, MD MSc DABIM

Hyperthyroidism is one of the common endocrinological conditions where there is an excess release of thyroid hormones triiodothyronine (T3) and thyroxine (T4) from the thyroid gland into the blood. These excess hormones excite the receptors inside body cells which usually results in several concerning symptoms including weight loss, tremors, anxiety, sweating and muscle weakness.

Hyperthyroidism is thought to deplete the body stores of L-carnitine and result in muscular weakness and other symptoms. The entry of inhibitors, both triiodothyronine (T3) and thyroxine (T4), into the cell prevents the hormones from activating its receptors in the cell¹. There have been some studies published which have supported this useful effect of L-carnitine in certain hyperthyroid conditions.

A widely quoted research study² was published about a decade ago in the prestigious *Journal of Clinical Endocrinology & Metabolism*. Researchers compared the effect of L-carnitine use on patients who are having symptoms of hyperthyroidism due to treatment from thyroid hormone. It seems patients who received this supplement at a dose of 2 grams or 4 grams of L-carnitine daily protected participants' bones and reduced symptoms of hyperthyroidism. L-carnitine was also found to be

helpful in the treatment of patients with thyroid storm (a severe hyperthyroid state usually requiring hospitalization) together with an anti thyroid drug³.

Current professional guidelines like the American Association of Clinical Endocrinologists medical guidelines for the clinical use of dietary supplements and nutraceuticals⁴ does acknowledge the role of L-carnitine in the treatment of hyperthyroidism but it's unclear how widely it is being used in clinical practice to treat hyperthyroidism

-
- 1 *Ann NY Acad Sci.* 2004 Nov;1033:158-67
 - 2 *J Clin Endocrinol Metab.* 2001 Aug;86(8):3579-94
 - 3 *Am J Med.* 2003 Oct 1;115(5):417-8
 - 4 *Endocr Pract.* 2003 Sep-Oct;9(5):417-70

Note: This information is not medical advice and is intended for general information purposes only and should not be considered or treated as medical advice. Patients should consult their physicians if they have any specific questions and before making any decision about using this information for medical treatment of their medical condition, or before discontinuing any medical treatment.



It's our 4th Annual

Let's Light-a-Tree for the Holidays!



Add a star to the tree and your name to our list of **TFC Stars** or dedicate your star to someone special. Add as many as you like - or just make a donation.

Information to appear on website

(Leave blank if you prefer to make an anonymous donation)

Name/initials:

City/Province:

Name/initials:

City/Province:

Name/initials:

City/Province:

Name/initials:

City/Province:

(include a separate list if you have more stars to add!)

Your stars will be added to our Holiday Tree at www.thyroid.ca



Be a **TFC Star** by completing and mailing this form with your payment of **\$20 per star** – or visit our website and click on the star to visit our Holiday Tree to make your contribution.

Each \$20 adds one star for the TFC Tree

Number of Stars:

Total \$:

Please add your total donation to the form on page 12



Proceeds go directly to the work of the Thyroid Foundation of Canada

Official tax receipts are issued for all donations and memberships



TFC Membership terms run from January 1st to December 31st.



If your membership expires in December 2014, please complete the membership form below and send with payment by December 31, 2014 - or visit our website and pay online: www.thyroid.ca



Thyroid Foundation of Canada Membership and Donation Form / La Fondation canadienne de la Thyroïde Formulaire d'adhésion et dons

Name/Nom: [] New /Nouvelle [] Renew /Renouvellement
Address/Adresse: Postal Code postal:
Tel.: Email/Courriel :

Membership options table with columns for ONE YEAR / UN AN and TWO YEAR / DEUX ANS, including Regular, Senior, Student, and Family rates.

Payment section including Membership/Adhésion, General Donation/Don général, Let's Light-a-Tree Donation/Don, and Total payment/Total paiement. Includes payment method options like Cheque, Visa, and MasterCard.

Receipt/Reçu and Thyrobuletin options: [] by regular mail/par courrier, [] to email above/par courriel ci-dessus, [] no Thyrobuletin /pas de Thyrobuletin

Please send completed form to/ S'il vous plaît envoyer le formulaire dûment rempli à: Thyroid Foundation of Canada / La Fondation canadienne de la Thyroïde, PO Box 298, Bath, ON K0H 1G0

Thank you for your support! / Nous vous remercions de votre soutien! (Accompanied by a butterfly graphic)

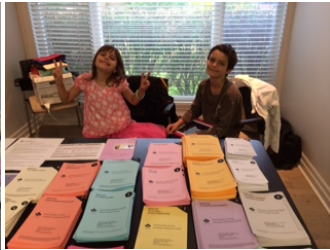


News and events

MONTREAL



Rinda's helpers at the Health & Wellness Fair



Rinda being interviewed by Elizabeth Stafiej, host of CJAD Radio show *Living Better*

TFC board member Rinda Hartner, participated in the **Health & Wellness Fair** held in Pointe Claire QC (near Montreal) on October 18, 2014. Many interested visitors stopped by the thyroid booth and display of thyroid educational materials.

KITCHENER-WATERLOO



Ron Yochim, Pharmacist

Pharmacist Ron Yochim of Reipert Pharmacy, PharmaSave, in Kitchener, spoke on April 15, 2014 on the topic of *What Affects your Medication*.

medications close to this. He discussed foods and the affects on the thyroid and medications.

He advised everyone on the correct time and method to take thyroid medication. He also explained the effects of taking other

The audience had the opportunity to ask questions. Everyone was very impressed and appreciative of all his helpful information.



Thinking of selling or trading in that old clunker?

Why not **DONATE** it instead and support TFC's programs?

Visit: charitycar.ca

or go to: thyroid.ca and follow the links

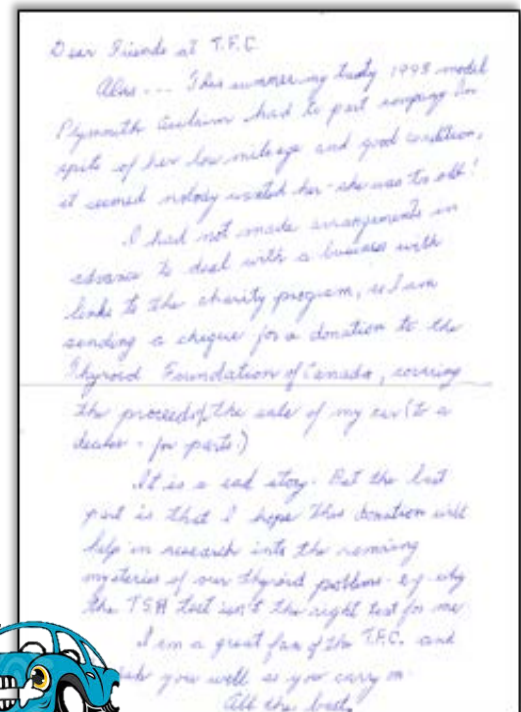
Dear friends at TFC:

Alas... This summer my trusty 1993 model Plymouth Acclaim and I had to part company. In spite of her low mileage and good condition it seemed nobody wanted her – she was too old! I had not made arrangements in advance to deal with a business with links to the charity program, so I am sending a cheque for a donation to the Thyroid Foundation of Canada, covering the proceeds of the sale of my car (to a dealer – for parts)

It is a sad story. But the best part is that I hope this donation will help in research into the remaining mysteries of our thyroid problems, eg. why the TSH test isn't the right test for me.

I am a great fan of the TFC and I wish you well as you carry on.

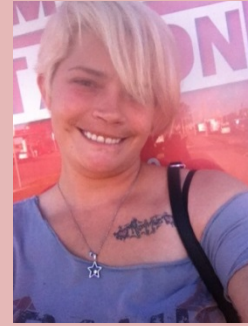
All the best, MM





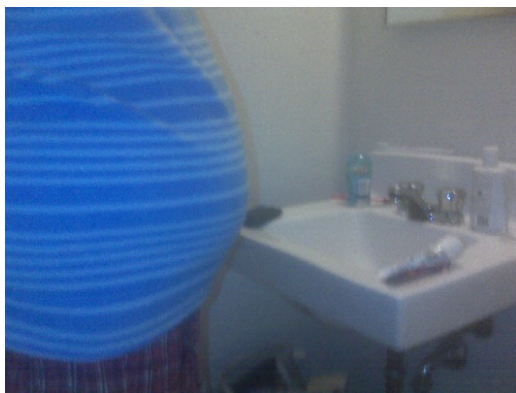
My story living with Graves' Disease

Traci Slauenwhite



I was diagnosed about 9 years ago with Graves' disease. I was sick for a very long time. Doctors over the years had diagnosed and treated me for depression and extreme anxiety. The treatments didn't work however.

I was also diagnosed with acalculous cholecystitis. The problem with that is normally people who have been in ICU for extended periods get it, not normally healthy people. My gallbladder was removed and the doctor told me it might not help. At my check-up he told me my red blood cells were enlarged. I told my family doctor. He said it was nothing to worry about and did no tests to find out why my red blood cells were enlarged. I believe that was 2009. They were enlarged because my thyroid was low. I have test levels that came back profoundly low in 2011. That doctor said it was ok just to take my pills. I did everything I could do.



My stomach after being diagnosed with acalculous cholecystitis

One day I had a very sore throat. I went to emergency and Dr. Toft looked at my neck. He said he wanted to do a blood test, which confirmed the goitre I had and didn't realize it; it was indeed from Graves' disease.

That man changed my life forever. He gave me hope where I had absolutely none. I owe him my life.

My T4 count was the second highest my specialist had ever seen. He wasn't new either. I was in my mid 20's and I was pulled out of work, taken off salt and processed food. I was at a very real risk of developing congestive heart failure.

It was a very scary time and yet I felt reborn. After my radiation treatments I had no racing heart, no sweats. I felt better for a while. I went back to school, got my Ontario Secondary School Diploma and became a hair stylist.

For the past few years I've been having increasing body pain, muscle cramps, and extreme PMS. I was diagnosed as being bipolar as well.

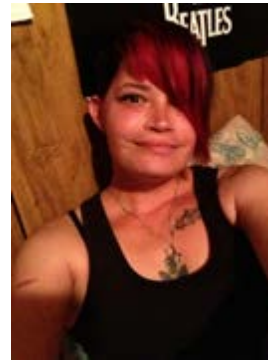
Today I learned I am extremely hypo. So hypo my red blood cells are apparently huge! I've had my thyroid levels tested and I was told they were ok. Dr. Gilbert told me it would have taken years to become like this. This is Canada - how does all this happen to one person here? I'm not sure how to feel. Most of my adult life has been hampered by this. I've lost jobs, relationships and almost my life more than once.

cont...



Please stop this from happening to anyone else.

In closing I'd just like to add that I'm really looking forward to getting to know who I really am. I'm just sorry I had to wait till almost 40 to do it. I could write a book with everything I've been through. I'm just one person but I want to save people from this. They deserve it. ☐



Traci

Upcoming events

Winnipeg

Tuesday December 2, 2014 6:30-8:30 pm

Gut Instinct: The critical connection between a healthy gut and a healthy brain

Tara Maltman-Just, B.Sc.(Pharm), RPh
ABAAHP, FAARFM
Caboto Centre, 1055 Wilkes Ave., Winnipeg

Free registration for **TFC members**
who register by Dec. 1st

Register at:

www.vitalityintegrativemedicine.com/RSVP.html
and mention that you are a TFC member

Kitchener-Waterloo

Monday, April 20, 2015

Thyroid Nodules

Dr. Deric Morrison, MD FRCPC
Division of Endocrinology, Department of Medicine,
University of Western Ontario

Kitchener Public Library, 85 Queen Street North
Education Table from 6:00 pm
Business meeting 6:30 pm
Speaker 7:00 pm - 8:45 pm

Please call to register - Phone (519) 743-0271

New this year – Thyroid health days!

*Coming to communities across
Canada*



Speak to a Health Professional

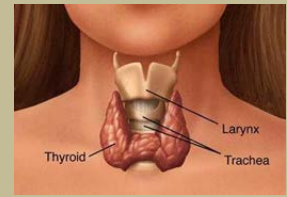
Check back for dates and locations at:

www.thyroid.ca



Thyroid and nutrition

Dr. Phoebe Bishara

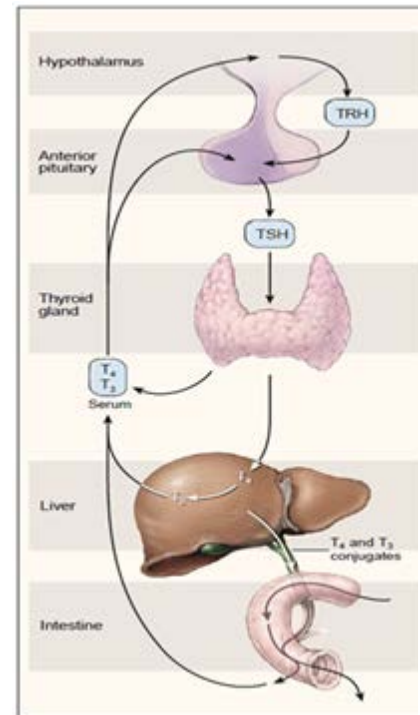


The Thyroid gland is a butterfly-shaped very vascular organ at the front of the lower part of your neck. The thyroid gland secretes mainly levothyroxine (T4) and a small amount of liothyronine (T3). Thyroid hormone secretion is under the control of a pituitary hormone which is called thyroid stimulating hormone (TSH) and hypothalamic hormones called thyroid releasing hormone (TRH). Thyroid hormones are involved in every cell function in every organ of our body. The thyroid gland is a very dynamic and reactive organ. It reacts to any physical or psychological stress, any physical illness, and any exogenous supplements.

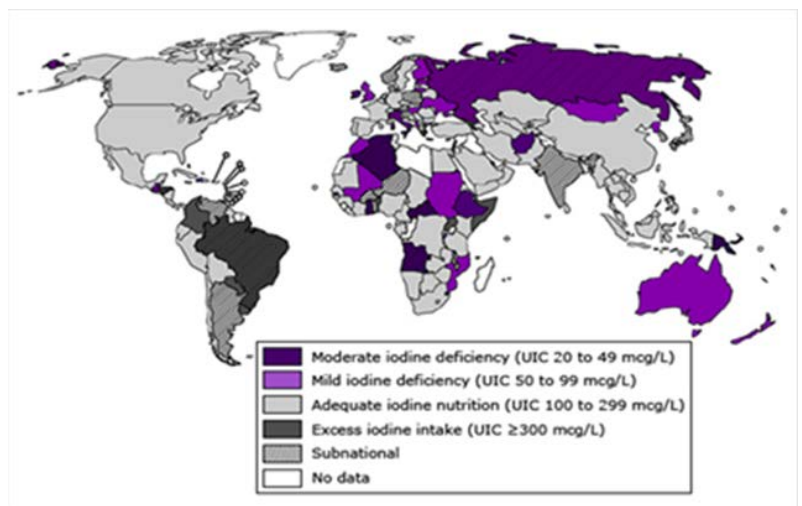
Thyroid hormones are activated mainly in the liver, gastro-intestinal tract and peripheral tissue cells. Several supplements can affect thyroid hormone secretion but the two most studied nutritional supplements are iodine and selenium.

Iodine:

The total daily dose (TDD) recommended for adults is 150 mcg daily for non-pregnant adults, 220 for pregnant women and 290 for lactating women. The thyroid gland requires 52 mcg of iodine daily in order to function normally. In the US, iodized salt contains 76 mcg of iodine/g. If you are healthy and eat everything in moderation, there is no need for additional iodine supplement.



- Iodine deficiency remains the world's most prevalent cause of thyroid disease but not in North America. Normal subjects can adapt to iodine intake.
- The major question in our mind is, if the thyroid needs iodine to form thyroid hormones, does it help if we take more iodine supplements? If you live in an iodine sufficient area, it does not help and it may even affect your thyroid function in a negative way because a normal thyroid gland has the ability to regulate itself. Sudden exposure to excess serum iodide inhibits organification of iodide by inhibiting its trapping in thyroid cells, thereby diminishing hormone biosynthesis; this phenomenon is called the Wolff-Chaikoff effect. In contrast, if your thyroid has a tendency to become overactive, a small amount of iodine intake (even 300-500 mcg) can cause excessive thyroid hormone secretion. This effect was first described by Basedow and it is called Jod-Basedow effect.
- There are several foods considered very rich sources of iodine and everyone living in North America, especially if thyroid disease runs in the family





should avoid eating an excessive amount of them e.g. Seaweed, and oral kelp (wrack and bladderwrack). It is estimated that 600 mg of kelp contains up to 6 mg of iodine, i.e. forty times the amount recommended as a total daily need. Many foods are considered rich in iodine and they are not good if eaten excessively. These include almonds, cauliflower (any vegetable that falls into the broccoli family is a goitrogen and shouldn't be eaten more than twice a week if you have hypothyroidism), millet, pears, turnips, brussels sprouts, corn, mustard, pine nuts, cabbage, kale, peaches, soy (Isoflavones block iodine), canola oil, peanuts, spinach. As iodine deficiency is not a common cause of thyroid disease, then iodine supplements provide no benefit and may cause abnormalities in thyroid function.

Selenium:

The recommended daily allowance (RDA) of selenium in adults is 55 mcg. Pregnant and nursing women need slightly higher doses. Selenium is involved in restoration of antioxidants and may be involved in thyroid hormone activation. Selenium deficiency is not common as soil is considered sufficient. In some countries, selenium is fortified in fertilizers. Selenium deficiency can be induced

by working around chemicals, eating foods with too many chemicals on them, or by breathing air with too much pollution in it. If you eat healthy and have no pre-existing health condition; you may not need any supplements of selenium. Selenium is one of the common trace elements in all multivitamins. The maximum recommended dose of supplements is 100 mcg daily, but the usual dose in multivitamins is 33 mcg. Any dose higher than 100 mcg is not well studied.

If you have hypothyroidism is there anything you should watch in your diet?

If you have hypothyroidism and you are on thyroid hormone replacement, make sure you take your medication regularly. Try to keep your stomach empty for one hour before and after and avoid taking your thyroid hormone at the same time as walnuts, soybean flour, cottonseed meal, iron supplements or multivitamins containing iron, calcium supplement, antacids that contain aluminum or magnesium, some ulcer medications, such as sucralfate (Carafate), some cholesterol-lowering drugs, such as cholestyramine (Questran) and colestipol (Colestid). To avoid potential interactions, eat these foods or use these products several hours before or after you take your thyroid medication. □

Hyperthyroidism, Thyrotoxicosis & Thyroid Storm

□ **H**yperthyroidism and thyrotoxicosis produce similar symptoms but they have different causes. Hyperthyroidism is a result of producing too much thyroid hormone and thyrotoxicosis is a result of receiving too much.

Hyperthyroidism is one cause of thyrotoxicosis but thyrotoxicosis can also occur without hyperthyroidism.

Some people develop thyrotoxicosis due to inflammation of the thyroid gland, which can lead to excessive release of thyroid hormone already stored in the gland (without the accelerated hormone production that characterizes hyperthyroidism).

Thyrotoxicosis can also occur after ingestion of excessive amounts of thyroid hormone in the form of thyroid

hormone supplements. This sometimes happens when TSH levels change and the patient needs a lesser dosage.

Thyroid storm is a rare, but life-threatening complication of Graves' disease. It is also known as accelerated hyperthyroidism or thyrotoxic crisis. It is more likely to occur when severe hyperthyroidism is untreated or is treated inadequately.

According to the Mayo Clinic website, "The sudden and drastic increase in thyroid hormones can produce a number of effects, including fever, profuse sweating, vomiting, diarrhea, delirium, severe weakness, seizures, markedly irregular heartbeat, yellow skin and eyes (jaundice), severe low blood pressure, and coma. Thyroid storm requires immediate emergency care." □

<http://www.mayoclinic.org/diseases-conditions/graves-disease/basics/complications/con-20025811>



Thyroid autoimmunity

Jack R. Wall

Understanding our Immune System

The immune system

□ **T**he immune system, which has evolved to protect us from foreign proteins such as bacteria and viruses, is complex, comprising antibodies in the blood which are produced by one type of white blood cells called B lymphocytes. These cells mature into plasma cells and various other white blood cells including T lymphocytes, killer cells, suppressor cells and regulating cells, which reside in the lymph nodes and bone marrow. Although serum antibodies are good markers of the autoimmune process - since they can be easily measured in the blood in a variety of antibody tests such as ELISA, it is usually the white blood cells, in particular the T lymphocytes which cause cell and tissue damage. T lymphocytes, including the cytotoxic T lymphocytes, produce a variety of proteins and other soluble peptides which act in a complex manner to lead to cell death.

Autoimmunity

Hashimoto's thyroiditis, which leads to hypothyroidism, and Graves' disease, which leads to hyperthyroidism and the associated eye changes, (referred to as ophthalmopathy), are "organ-specific autoimmune disorders" of the thyroid gland. With these disorders, the immune system reacts against the body's own antigens. In the case of Hashimoto's thyroiditis, this leads to a focused destructive reaction within the thyroid gland. In the case of Graves' disease, it leads to stimulation of the thyroid cells due to the production of a unique antibody called TSH-receptor antibody.

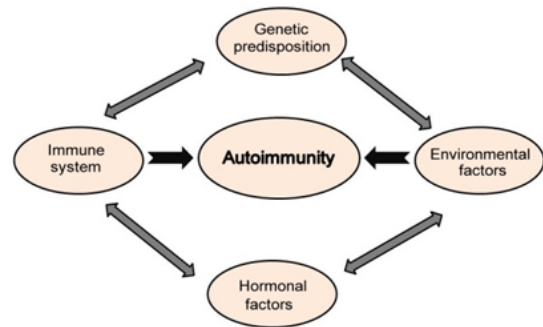
Other organ specific autoimmune disorders include:

- Addison's disease, in which destruction of the adrenal gland leads to deficiency of cortisone
- type 1 diabetes where antibodies and lymphocytes target those pancreatic islet cells which produce insulin, leading to insulin deficiency, increased blood sugar and the characteristic featurer of diabetes of youth
- psoriasis
- vitiligo and
- celiac disease

When the immune reactions affect more than one tissue or cell type this is termed "multi-system autoimmune disease" and these include systemic lupus erythematosus (SLE),

rheumatoid arthritis, scleroderma and Sjogren's disease.

The autoimmune reaction, of which thyroid autoimmunity is a good model, was originally considered to be impossible but in reality, this process is very common. For example, Hashimoto's thyroiditis affects about 8% of all adult women and 1% of men and Graves' disease affects about 1% of adults. There are combinations of these organ-specific autoimmune disorders; for example 25% of patients with type 1 diabetes also have autoimmune thyroid disease and there are well known associations between Addison's disease, hypothyroidism, pernicious anaemia and type 1 diabetes. However, for most patients with autoimmune thyroid disease, their lifetime chance of developing one or other of these other disorders is very small.



Thyroid autoimmunity is not genetically determined, but the tendency to develop Graves' disease and Hashimoto's thyroiditis (and other autoimmune conditions) runs in families. The onset of autoimmune disease involves a second factor, such as stress, infection and other environmental factors that trigger the autoimmune reactions in the thyroid gland in predisposed subjects. Despite the central role of genetic predisposition in this process, interplay between genetic and non-genetic factors seems essential for the development of the full-scale autoimmune reaction. The presence of one factor without the other may not be sufficient for the development of full-scale human autoimmunity.

The role of stress

As demonstrated above, the development of autoimmunity in humans is the endpoint of a complex process involving the



immune system, hormonal factors and environmental factors in a genetically predisposed individual, hence the term 'mosaic of autoimmunity'. The evidence for stress contributing to the onset and course of autoimmune disease is circumstantial and the mechanisms by which stress affects autoimmune disease remain poorly understood. The best evidence for an effect of stress on the development of an autoimmune disorder is the well-known relationship between the clinical onset of Graves' hyperthyroidism, and its subsequent clinical course, and major stress in the preceding 2-3 months. When patients with Graves' disease are treated for a year with anti-thyroid drugs they become well and go into remission, and may remain well for many years, but sometimes the disease relapses, usually due to major stress. In the case of Hashimoto's thyroiditis, stress is probably important but less readily identified since the disease is slowly progressive and asymptomatic over many years before the development of hypothyroidism. Stress may be the major environmental factor in most patients who develop Graves' hyperthyroidism but there are other factors as well such as smoking, vitamin D deficiency and infections. Although future studies to tease out the role of all of these components will be difficult, they must be carried out and we need to be patient.

Antibodies and Thyroid Disorders

Hashimoto's thyroiditis

In the case of Hashimoto's thyroiditis, antibodies react against two main proteins, thyroglobulin (TG), a large protein in the thyroid gland which stores thyroid hormones, and thyroid peroxidase (TPO), an enzyme located on the surface of the thyroid follicular cells. The antibodies are reliable clinical markers of the process but thyroid cell and tissue destruction in Hashimoto's thyroiditis is probably due to an effect of cytotoxic T cells, even though this has not been well studied nor convincingly demonstrated. The autoimmune process is progressive and extends over many years so that there may be long intervals between the detection of antibodies against TG and TPO and the development of hypothyroidism, which occurs when sufficient numbers of thyroid follicular cells have been destroyed by the inflammatory reaction leading to thyroid hormone deficiency. Real-time thyroid ultrasonography reveals the characteristic black spaces, scarring and so-called pseudo nodules in patients with end stage disease.

Because of the classical feedback relationship between pituitary thyroid stimulating hormone (TSH) and T4 and T3 produced by the thyroid gland, the pituitary recognises decreasing production of thyroid hormones and produces more TSH to "fire up" the failing thyroid gland. When patients

have reached a certain threshold, they develop symptoms of hypothyroidism such as tiredness, fatigue, tendency to gain weight / inability to lose weight and puffiness of the face. This process is progressive and the symptoms worsen if left untreated.

Graves' disease

Graves' disease is unique in that the disease is caused by an antibody which stimulates the TSH receptor, a protein on the surface of the thyroid follicular cell which binds TSH. TSH stimulates the thyroid cells to produce thyroid hormones while antibodies against the TSH receptor do the same thing, so they compete with TSH on the cell surface. They bind more intensely than TSH and eventually lead to overstimulation of the thyroid cells which become enlarged and divide and produce more and more thyroxine and T3, leading to the symptoms of hyperthyroidism which include increased heart rate, anxiety, heat intolerance, sweatiness, increase in appetite and weight loss.

Ophthalmopathy

Several associated abnormalities in Graves' disease as described by Robert Graves include "poppy eyes", or ophthalmopathy, a lumpy thickening of the skin called pretibial myxoedema and inflammation and swelling of the long bones in the hands and arms, and clubbing, called acropachy. These associations occur in patients with more severe disease, so that the great majority of patients with pretibial myxoedema and acropachy will have ophthalmopathy and severe hyperthyroidism. The immune reactions are different but somehow related to each other. The eye disorder is particularly important because some patients have distressing eye symptoms such as double vision, blurring of vision and cosmetic concerns. The appearance of "poppy eyes" (as seen above and on next page) are due to 1) upper eyelid retraction which results from the hyperthyroidism itself and 2) inflammation of the eye muscles and surrounding fatty tissues, or "true ophthalmopathy". Intense studies carried out over many years point to a reaction of antibodies against the TSH-receptor, which is also found in the orbital fatty tissue, although the evidence linking the development of the eye disorder with TSH-receptor antibodies is mainly circumstantial. The author has studied the role of antibodies against eye muscle proteins including calsequestrin and flavoprotein, which may be produced only after the eye muscle fibre has been damaged. Recent studies





suggest that TSH-receptor antibodies are not found in patients with “poppy eyes” who have normal thyroid function. This condition is referred to as ‘euthyroid Graves’ disease’.



Treatment

Studies carried out over decades addressing the different aspects of the autoimmune reaction are ongoing, but in the case of thyroid autoimmunity, few advances have been made because the treatment is straightforward and not directed towards the immune abnormalities themselves. Other organ-specific autoimmune disorders such as type 1 diabetes, myasthenia gravis and multiple sclerosis are much more problematic and research is necessary to lead to the discovery of specific therapies for these serious and often life-threatening diseases. Although research continues into aspects of the cause of the disease such as the effects of infection and stress, and the relationship between the brain and the immune reaction, treatment is limited to either replacing thyroxin, in hypothyroidism, or removing, blocking or destroying the thyroid gland, in Graves’ hyperthyroidism.

We do not treat with steroids or immunosuppressive agents because of side effects and, in the long term, it is unlikely that we would ever aim to treat the immune disorder itself unless very specific agents which block the thyroid autoimmune reaction are discovered. In ophthalmopathy, the autoimmune reaction is directed against the eye muscles and the surrounding connective and fatty tissue. This is a complex disorder, somehow linked to the thyroid reaction. In this serious disorder, we do sometimes treat with immunosuppressive drugs or steroids and even radiotherapy and surgery if there is loss of vision. Life-threatening diseases such as multiple sclerosis and type 1 diabetes require more intense and specific therapies with attempts to suppress the immune reaction which is causing tissue damage, whereas in Graves’ disease we use anti-thyroid drugs to block the overproduction of the thyroid

hormone and in Hashimoto’s thyroiditis we use thyroxin replacement to compensate for the decreased production of thyroid hormones.

Finally, one might ask – why does treatment of hyperthyroidism with Carbimazole or Propylthiouracil for one year or more lead to long-term remission of the Graves’ disease? Anti-thyroid drugs do have some immuno-suppressive role, but there is also a non-specific effect of the hyperthyroidism itself on the immune system which decreases as the hyperthyroidism is reduced with treatment and the patient may remain well for many years. On the other hand, radio-active iodine destroys, and thyroidectomy removes, the thyroid tissue including the proteins which are the target of the antibodies and cytotoxic white blood cells, so the autoimmune reactions cannot continue. Following these procedures, patients become hypothyroid and need to be given full replacement doses of thyroxin which are taken long term.

Sex hormones and autoimmunity

Thyroid autoimmunity, and indeed most other autoimmune disorders except type 1 diabetes, occurs more commonly in women than men. Adding to the complex mix of the factors which lead to the development of autoimmune disease are the androgens and estrogens (or sex hormones). Estrogens in females somehow favour the development of autoimmunity and androgens may protect, but this is much more complex and involves other factors as well, such as genes on the X chromosome. When thyroid autoimmunity occurs in males, the genetic factor tends to be more evident i.e. there is often a strong family history of thyroid autoimmunity, lupus, rheumatoid arthritis or other autoimmune disorders in their first degree relatives.

Conclusion

Thyroid autoimmunity is common but generally easily managed and we do not need to worry about the underlying cause or role of genetics. We should not have our children checked “for thyroid disease” unless they have symptoms and we must somehow teach them how to deal with major stresses. Good luck with that! ☐

With thanks to all members of our caring Thyroid community



and warm wishes for a healthy holiday season

