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AMERICAN THYROID ASSOCIATION

Optimal Thyroid Health for All

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Mooijjaart SP et al 2019 Association between levothyroxine treatment and thyroid-related symptoms among adults aged 80 years and older with subclinical hypothyroidism. *JAMA*. Epub Oct 30:1–11. PMID: 31664429.

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Iodine deficiency is a world-wide health problem affecting about 30% of the population. An unintentional consequence of iodine supplementation in iodine deficient populations has been a report of an increase of both hyperthyroidism and hypothyroidism. This study examined the rate of hyperthyroidism and hypothyroidism in a group living in an area in the north of Denmark a few years after the mandatory supplementation with iodine in the salt took effect.

Petersen M et al. 2019 Changes in subtypes of overt thyrotoxicosis and hypothyroidism following iodine fortification. *Clin Endocrinol (Oxf)*. Epub 2019 Aug 9. PMID: 31400012.

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There are two surgery choices for people diagnosed with low risk papillary thyroid cancer: 1) hemithyroidectomy to remove just that part of the thyroid that contains the thyroid cancer or 2) total thyroidectomy. The purpose of the study reviewed here was to compare quality of life for people who underwent a hemithyroidectomy for low risk papillary thyroid cancer to quality of life for people who underwent total thyroidectomy.

Bongers PJ et al. 2020 Differences in long-term quality of life between hemithyroidectomy and total thyroidectomy in patients treated for low-risk differentiated thyroid carcinoma. *Surgery* 167:94–101

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Derakhshan An et al 2019. Association of urinary bisphenols and triclosan with thyroid function during early pregnancy. *Environ Int* 133(Pt A):105123. PMID: 31521814.

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Calcaterra V et al 2019 Anti-gastric parietal cell antibodies for autoimmune gastritis screening in juvenile autoimmune thyroid disease. *J Endocrinol Invest*. Epub 2019 Jul 1. PMID: 31264142.

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Clinical Thyroidology for the Public

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EDITOR'S COMMENTS

As spring nears, welcome to another issue of *Clinical Thyroidology for the Public*. In this journal, we will bring to you the most up-to-date, cutting edge thyroid research. We also provide even faster updates of late-breaking thyroid news through [Twitter](#) at [@thyroidfriends](#) and on [Facebook](#). Our goal is to provide patients with the tools to be the most informed thyroid patient in the waiting room. Also check out our friends in the [Alliance for Thyroid Patient Education](#). The [Alliance](#) member groups consist of: the *American Thyroid Association*, *Bite Me Cancer*, *the Graves' Disease and Thyroid Foundation*, *the Light of Life Foundation*, *MCT8 – AHDS Foundation*, *ThyCa: Thyroid Cancer Survivors' Association*, *Thyroid Cancer Canada*, *Thyroid Cancer Alliance* and *Thyroid Federation International*.

We invite all of you to join our [Friends of the ATA](#) community. It is for you that the American Thyroid Association (ATA) is dedicated to carrying out our mission of providing reliable thyroid information and resources, clinical practice guidelines for thyroid detection and treatments, resources for connecting you with other patients affected by thyroid conditions, and cutting edge thyroid research as we search for better diagnoses and treatment outcomes for thyroid disease and thyroid cancer. We thank all of the *Friends of the ATA* who support our mission and work throughout the year to support us. We invite you to help keep the ATA mission strong by choosing to make a donation that suits you — it takes just one moment to give online at: www.thyroid.org/donate and all donations are put to good work. The ATA is a 501(c)3 nonprofit organization and your gift is tax deductible.

March is [Medullary Thyroid Cancer Awareness Month](#).

In this issue, the studies ask the following questions:

- Does treatment of hyperthyroidism cause obesity?
- Is treatment of subclinical hypothyroidism indicate in the elderly?
- Does iodine supplementation in populations that are iodine deficient cause an increase in thyrotoxicosis?
- Does the effect of surgery for low risk papillary thyroid cancer affect quality of life?
- Does BPA exposure affect thyroid hormone levels in pregnant women?
- Are children with Hashimoto's thyroiditis or Graves' disease at risk for developing autoimmune gastritis?

We welcome your feedback and suggestions. Let us know what you want to see in this publication. I hope you find these summaries interesting and informative.

— Alan P. Farwell, MD, FACE



HYPERTHYROIDISM

Treatment for hyperthyroidism increases the risk of obesity in some patients

BACKGROUND

Hyperthyroidism occurs when there is too much thyroid hormone in the blood. The most common cause of hyperthyroidism in the United States is Graves' disease. A common symptom of hyperthyroidism is weight loss without trying. Sometimes this can even happen despite increased appetite. Treatment of hyperthyroidism commonly results in weight gain, but the extent of weight gain is not well known. Patients may regain the weight they had lost or may overshoot and become obese. The aim of this study was to find out whether treatment of hyperthyroidism is associated with increased risk of developing obesity. Authors designed the study to assess the amount of weight gain and they also examined the effect of treatment method on weight gain risk.

THE FULL ARTICLE TITLE

Torlinska B et al 2019 Patients treated for hyperthyroidism are at increased risk of becoming obese: findings from a large prospective secondary care cohort. *Thyroid* 29:1380–1389. PMID: 31375059.

SUMMARY OF THE STUDY

Patient information was obtained from Thyroid Clinic Database at the University Hospitals Birmingham NHS Foundation Trust. Patients were treated for newly diagnosed hyperthyroidism using either anti-thyroid medications, radioactive iodine therapy (RAI) or a combination of the two methods between 2000 and 2014. None of the patients had thyroid surgery. Patients that were included in the study had at least 6 months of follow up after treatment, had a minimum of four recorded weight measurements and they had to have successful treatment of hyperthyroidism. Successful treatment was either having normal thyroid hormone levels or requiring thyroid hormone replacement. Comparison group was from the Health Survey for England participants. There were 1373 patients, 573 were treated with anti-thyroid medications and 800 were treated with RAI or

a combination. The comparison group without thyroid disease had 10,984 participants.

Before treatment patients with hyperthyroidism weighed less than the comparison group. After treatment men were 1.7 times and women were 1.3 times more likely to develop obesity. Weight gain occurred mostly in the first 6 months of treatment but continued until 24 months. A total of 65% of patients gained 5% of their weight and 38% gained 10% or more. Men gained on average 17.6 lbs and women gained about 12.1 lbs. Average weight gain was about 11.8 lbs for patients who were treated with medication, 12.3 lbs for those who had RAI treatment without developing hypothyroidism, and 15.6 lbs for those who had RAI treatment and developed hypothyroidism. Other risk factors for more weight gain were Graves' disease as the cause of the hyperthyroidism, an elevated TSH after treatment, or the requirement of thyroid hormone replacement. The amount of weight gain was associated with the severity of hyperthyroidism at time of diagnosis and patients who had reported weight loss prior to treatment had more weight gain.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Treatment of hyperthyroidism with RAI or anti-thyroid medications is associated with an increased risk of gaining weight and even developing obesity. This risk is slightly higher with RAI therapy compared to medications. Patients who had surgery were not included in the study so we do not have a comparison. The risk of weight gain can be very scary, however, hyperthyroidism needs to be treated otherwise it can become very severe and even lead to deadly complications. The most important step to control weight gain from any cause is to increase the awareness of providers and the patients. Strategies to prevent weight gain should be a part of the initial treatment plan for hyperthyroidism.

— Ebru Sulanc, MD, FACE



HYPERTHYROIDISM, continued

ATA THYROID BROCHURE LINKS

Hyperthyroidism (Overactive): <https://www.thyroid.org/hyperthyroidism/>

Graves' Disease: <https://www.thyroid.org/graves-disease/>

Thyroid and Weight: <https://www.thyroid.org/thyroid-and-weight/>

ABBREVIATIONS & DEFINITIONS

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

Thyroid hormone therapy: patients with hypothyroidism are most often treated with Levothyroxine in order to return their thyroid hormone levels to normal. Replacement therapy means the goal is a TSH in the normal range and is

the usual therapy. Suppressive therapy means that the goal is a TSH below the normal range and is used in thyroid cancer patients to prevent growth of any remaining cancer cells.

TSH: thyroid stimulating hormone – produced by the pituitary gland that regulates thyroid function; also the best screening test to determine if the thyroid is functioning normally.

Radioactive iodine (RAI): this plays a valuable role in diagnosing and treating thyroid problems since it is taken up only by the thyroid gland. I-131 is the destructive form used to destroy thyroid tissue in the treatment of thyroid cancer and with an overactive thyroid. I-123 is the non-destructive form that does not damage the thyroid and is used in scans to take pictures of the thyroid (Thyroid Scan) or to take pictures of the whole body to look for thyroid cancer (Whole Body Scan).



HYPOTHYROIDISM

Levothyroxine treatment of subclinical hypothyroidism does not improve symptoms in patients 80 years of age or older

BACKGROUND

Subclinical hypothyroidism is a mild form of hypothyroidism in which the level of Thyroid Stimulating Hormone (TSH) is increased, but the thyroid hormone level is within the normal range. This is compared to overt hypothyroidism where the TSH is increased and the thyroid hormone levels are low. Subclinical hypothyroidism much more common than overt hypothyroidism in adults, and its rate increases with age. Some studies suggest that subclinical hypothyroidism may affect up to 25% of individuals over the age of 75. Treatment of subclinical hypothyroidism is controversial as beneficial effects are hard to show in many studies.

Studies have shown that many elderly patients with subclinical hypothyroidism complain of fatigue, poor memory, dry skin, depressed mood and other symptoms that are non-specific. Further, these symptoms may also be seen commonly in the elderly population who do not have a thyroid problem. Several studies had been conducted to evaluate if prescribing thyroid hormone in adults with subclinical hypothyroidism may improve these symptoms. The current study was designed to examine the effect of treating subclinical hypothyroidism in patients who are 80 or older.

THE FULL ARTICLE TITLE

Mooijjaart SP et al 2019 Association between levothyroxine treatment and thyroid-related symptoms among adults aged 80 years and older with subclinical hypothyroidism. JAMA. Epub Oct 30:1–11. PMID: 31664429.

SUMMARY OF THE STUDY

The authors analyzed the data from two previous randomized clinical trials done in Europe, but focused their analysis specifically on individuals who were 80 or

older. They all had abnormal TSH levels, ranging from 4.6 to 19.9 mIU/L in two or more blood tests which were 3 months apart. People who were taking thyroid hormone, lithium and amiodarone or had recent thyroid surgery, radioactive iodine therapy as well as individuals with terminal illness and severe heart disease were excluded. Patients were divided into two groups; one received thyroid hormone therapy and the other placebo. The dose of thyroid hormone was increased gradually to achieve a normal TSH result.

A total of 2,989 individuals were screened for inclusion in this study. A total of 1883 individuals (63%) had an abnormal TSH result initially that returned to normal in future tests. A total of 251 patients with the average age of 85 years were included in the study. Almost half (47%) were women. After careful analysis, the rate of tiredness, other symptoms and heart related events were not different between the two groups.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study shows that the vast majority of individuals over the age of 80 with an initial increased TSH with normal thyroid hormone levels will have a normal TSH on follow up testing. In those patients with persistent subclinical hypothyroidism, treatment with thyroid hormone did not improve fatigue or other non-specific symptoms. This confirms prior studies that many symptoms like tiredness, problems with memory and changes in mood are common in older adults, but are not specific to thyroid disease. Thus, this study shows there is no clear benefit of treatment of elderly patients with subclinical hypothyroidism with thyroid hormone.

— Shirin Haddady, MD

ATA THYROID BROCHURE LINKS

Hypothyroidism (Underactive): <https://www.thyroid.org/hypothyroidism/>

Older Patients and Thyroid Disease: <https://www.thyroid.org/thyroid-disease-older-patient/>



HYPOTHYROIDISM, continued

ABBREVIATIONS & DEFINITIONS

Subclinical Hypothyroidism: a mild form of hypothyroidism where the only abnormal hormone level is an increased TSH. There is controversy as to whether this should be treated or not.

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

TSH: thyroid stimulating hormone – produced by the pituitary gland that regulates thyroid function; also the best screening test to determine if the thyroid is functioning normally.

Thyroid hormone therapy: patients with hypothyroidism are most often treated with Levothyroxine in order to return their thyroid hormone levels to normal. Replacement therapy means the goal is a TSH in the normal range and is the usual therapy. Suppressive therapy means that the goal is a TSH below the normal range and is used in thyroid cancer patients to prevent growth of any remaining cancer cells.



AMERICAN THYROID ASSOCIATION
Optimal Thyroid Health for All

MARCH Medullary Thyroid Cancer Awareness Month



IODINE AND THE THYROID

Iodine supplementation lowers the long-term risk of thyrotoxicosis without increasing hypothyroidism

BACKGROUND

Low iodine in the diet (iodine deficiency) is a world-wide health problem affecting about 30% of the population around the world. Iodine deficiency can cause goiters (thyroid enlargements) and hypothyroidism which can lead to neurological problems in children. Iodine supplementation in areas of the world where dietary iodine is low has been very effective in eliminating this problem. However, an unintentional consequence of iodine supplementation in populations that were initially iodine deficient has been a report of an increase of both hyperthyroidism (overactive thyroid) and hypothyroidism (underactive thyroid).

This study was done in Denmark, a country with mild to moderate iodine deficiency that underwent a mandatory iodine fortification program in 2001 (iodine was added to all salt used for commercial bread making and to the salt used for household consumption). This study looked at the thyroid levels in a group of about 300,000 people living in an area in the north of Denmark during 2014-2016, a few years after the mandatory supplementation with iodine in the salt took effect. They identified all cases of hyperthyroidism and hypothyroidism. They also compared the results with those found in an identical survey done during 1997-2000.

THE FULL ARTICLE TITLE

Petersen M et al. 2019 Changes in subtypes of overt thyrotoxicosis and hypothyroidism following iodine fortification. Clin Endocrinol (Oxf). Epub 2019 Aug 9. PMID: 31400012.

SUMMARY OF THE STUDY

The authors identified all cases of hyperthyroidism and hypothyroidism diagnosed during 2014-2016 in a group of about 300,000 people living in an area in the north of Denmark. The authors reviewed the medical records of the patients with abnormal thyroid levels to look for the specific causes of hyperthyroidism and hypothyroidism. They compared these results with those from an identical study done during 1997-2000, just before the mandatory iodine supplementation took effect.

The study found that, during 2014-2016, the rate of hyperthyroidism in this population was about 50 in 100,000 individuals. When they compared these results with the prior survey (before iodine supplementation), they found that the rate of hyperthyroidism was 50% lower. The decrease of hyperthyroidism was mostly due to a decrease in the rate of toxic nodular goiters. The chances of hyperthyroidism decreased in all age groups, but it was most significant in middle age and older individuals. The rates of underactive thyroid disease were not affected.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The results of this study confirm that programs that mandate iodine supplementation in the form of iodized salt are safe and effective. Further, over the long term, iodine supplementation may also lower the chances of hyperthyroidism, presumably by lowering the stimulus to thyroid growth and nodule formation. Finally, the addition of iodine did not increase the chances of hypothyroidism.

— Susana Ebner MD

ATA THYROID BROCHURE LINKS

Iodine Deficiency: <https://www.thyroid.org/iodine-deficiency/>

Goiter: <https://www.thyroid.org/goiter/>

Hypothyroidism (Underactive): <https://www.thyroid.org/hypothyroidism/>

Hyperthyroidism (Overactive): <https://www.thyroid.org/hyperthyroidism/>



IODINE AND THE THYROID, continued

ABBREVIATIONS & DEFINITIONS

Goiter: a thyroid gland that is enlarged for any reason is called a goiter. A goiter can be seen when the thyroid is overactive, underactive or functioning normally. If there are nodules in the goiter it is called a nodular goiter; if there is more than one nodule it is called a multinodular goiter.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

Iodine: an element found naturally in various foods that is important for making thyroid hormones and for normal thyroid function. Common foods high in iodine include iodized salt, dairy products, seafood and some breads.

Toxic nodular goiter: characterized by one or more nodules or lumps in the thyroid that may gradually grow and increase their activity so that the total output of thyroid hormone in the blood is greater than normal.

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THYROID CANCER

The Effect of Surgery Extent for Low-Risk Thyroid Cancer on Quality of Life

BACKGROUND

Papillary thyroid cancer is the most common type of thyroid cancer and, although this cancer generally has a very good survival rate/prognosis, thyroid surgery is still needed in most cases. Most papillary thyroid cancers are 'low risk', meaning that they are small (< 4 cm in diameter), are completely contained within the thyroid and do not spread into neighboring tissues or to distant body sites. There are two surgery choices for people diagnosed with low risk papillary thyroid cancer: 1) removal of just that part of the thyroid that contains the thyroid cancer (this is called a hemithyroidectomy or partial thyroidectomy) or 2) removal of the whole thyroid gland (this is called a total thyroidectomy).

There are two advantages of hemithyroidectomy. First, the part of the thyroid that is left in place (is not surgically removed) will continue to make thyroid hormone, which is the normal job of the thyroid and is needed for controlling how the body uses energy (metabolism). For this reason, many people who undergo hemithyroidectomy do not need to take a thyroid hormone replacement pill. On the other hand, people who have had all of their thyroid tissue removed (total thyroidectomy) must take this pill every day following surgery. Second, hemithyroidectomy is a smaller surgery than is total thyroidectomy and, for this reason, the risk of complications from surgery are also smaller. Recent studies also suggest that, because hemithyroidectomy is a smaller surgery than total thyroidectomy and with a lower risk of complications, overall quality of life after surgery may be better for people who undergo a hemithyroidectomy.

Hemithyroidectomy does have disadvantages, however. First, the thyroid tissue that is not surgically removed generally must be monitored over time for formation of new thyroid cancer and, if this does happen, another surgery might be needed to remove this cancer. Second, it can be more difficult for doctors to tell if a thyroid cancer recurs (comes back) if the whole thyroid gland is not removed.

The purpose of the study reviewed here was to compare quality of life for people who underwent a hemithyroidectomy for low risk papillary thyroid cancer to quality of life for people who underwent total thyroidectomy.

FULL ARTICLE TITLE

Bongers PJ et al. 2020 Differences in long-term quality of life between hemithyroidectomy and total thyroidectomy in patients treated for low-risk differentiated thyroid carcinoma. *Surgery* 167:94–101

SUMMARY OF THE STUDY

The authors identified 529 patients who underwent either hemithyroidectomy or total thyroidectomy at their hospital between 2005 and 2016. A survey designed to gather information about quality of life was then mailed to each patient and, of the 529 patients in the group, 270 (51%) filled out and returned the survey. A total of 59 of these patients had undergone hemithyroidectomy and the remainder (211 patients) had undergone total thyroidectomy.

When the surveys were analyzed, the study team ultimately found no difference in quality of life between people who had undergone hemithyroidectomy compared to those having had a total thyroidectomy. They did, however, find that people who had undergone hemithyroidectomy worried more about their cancer coming back (recurring) than did people who underwent total thyroidectomy. There was no actual difference in the rate of cancer coming back between the two groups during the study time frame, however.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The study suggests that the overall quality of life after surgery for people who underwent a hemithyroidectomy for low risk papillary thyroid cancer, compared to those who underwent a total thyroidectomy, was the same. This is an important finding because less surgery (hemithyroidectomy) is becoming much more common. However,



THYROID CANCER, continued

patients who underwent hemithyroidectomy did worry more that their cancer could come back compared to people for whom the whole thyroid was removed. This is important and should be included in the discussion of the

risks and benefits of both more and less surgery in patients diagnosed with low risk papillary thyroid cancer to help each patient choose their best surgery option.

— Jason D. Prescott, MD PhD

ATA THYROID BROCHURE LINKS

Thyroid Cancer (Papillary and Follicular): <https://www.thyroid.org/thyroid-cancer/>

Thyroid Surgery: <https://www.thyroid.org/thyroid-surgery/>

ABBREVIATIONS & DEFINITIONS

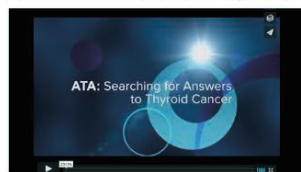
Papillary thyroid cancer: the most common type of thyroid cancer. There are 4 variants of papillary thyroid cancer: classic, follicular, tall-cell and noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP).

Total thyroidectomy: surgery to remove the entire thyroid gland.

Hemithyroidectomy: surgery that removes only part of the thyroid gland (usually one lobe with or without the isthmus).

Cancer recurrence: this occurs when the cancer comes back after an initial treatment that was successful in destroying all detectable cancer at some point.

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Anaplastic Thyroid Cancer – Support Research for Treatments
April 17, 2016



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THYROID AND PREGNANCY

Early pregnancy exposure to Bisphenol A may affect thyroid hormone levels

BACKGROUND

In recent years, there has been increased awareness of the effect of chemicals in our environment on body processes. Those that interfere with the endocrine glands and hormone levels are called endocrine disruptors. Two such endocrine disruptors are bisphenols and triclosan. Bisphenols are a group of chemicals used to make commonly used plastics such as those for food and water containers, receipts, CDs, DVDs, toys, and plastic bags. Triclosan is an antibacterial chemical frequently used to make hand sanitizers or other personal care products. They are commonly detected in environments, and consequently, in humans.

Animal studies have shown that bisphenols and triclosan may affect thyroid function. Bisphenol A (BPA) may affect uptake of iodine into thyroid gland, which is required to make thyroid hormone. It may also affect deiodinase enzymes that convert T_4 to T_3 . However, these actions of BPA have not been shown in humans. BPA use has been since restricted in many countries, and now substituted with other bisphenols such as bisphenol S (BPS) and bisphenol F (BPF). There are not many studies on the effects of BPS, BPF, or triclosan on human thyroid function, especially in pregnancy.

Normal thyroid hormone levels in pregnancy is critical for baby's development, so this study was done to evaluate potential association of BPA, BPS, BPF, and triclosan levels in pregnant women and their thyroid hormone levels.

THE FULL ARTICLE TITLE

Derakhshan An et al 2019. Association of urinary bisphenols and triclosan with thyroid function during early pregnancy. *Environ Int* 133(Pt A):105123. PMID: 31521814.

SUMMARY OF THE STUDY

A total of 1996 pregnant women in Sweden were included in the study between September 2007 and March 2010. None had twin pregnancy or thyroid disease, or took

thyroid medications. Blood and urine samples were collected at their first prenatal visit, which was on average at 10 weeks of pregnancy. Several different measures of thyroid function (thyroid stimulating hormone (TSH), thyroxine (T_4), and triiodothyronine (T_3)), and thyroid antibody levels were measured from blood sample. The majority of thyroid hormone made by thyroid gland is in the form of T_4 , which is changed to a more active form, T_3 in the body. BPA, BPS, BPF, and triclosan levels were measured from urine samples.

BPA, BPS, BPF and triclosan were detected in most of pregnant women in the study (99% for BPA, 80% for BPS, 88% for BPF, and 93% for triclosan). However, the levels were overall low. Higher BPA levels were associated with lower T_4 levels, but not with changes in TSH. In very early pregnancy (at average of 7 weeks of pregnancy), higher BPA levels were also associated with higher T_3 levels. This was not true a little later in pregnancy (at average of 12 weeks of pregnancy). However, higher BPA levels were also associated with lower $T_4:T_3$ ratios, suggesting that it may be associated with higher conversion of T_4 to T_3 . There were no associations between BPS, BPF, or triclosan levels and thyroid hormone levels. Thyroid antibody levels did not have any effect on the findings.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study shows that exposure to BPA may affect thyroid hormone levels in very early pregnancy. BPA was associated with lower T_4 levels, but also lower $T_4:T_3$ ratios, suggesting higher conversion from T_4 to T_3 . However, exposure to BPF, BPS, or triclosan did not have significant effect on thyroid hormone levels in this study.

Overall, the amount of bisphenols and triclosan measured in urine samples was low, about 1,000 times lower than the safe levels of exposure recommended by the U.S. Environmental Protection Agency or the European Food



THYROID AND PREGNANCY, continued

Safety Authority. At this lower level of exposure, there may not be significant effect on thyroid hormone levels in pregnancy in humans. However, further studies are

needed to confirm these findings and find out how these chemicals affect thyroid hormone levels in humans.

— Sun Lee, MD

ATA THYROID BROCHURE LINKS

Thyroid Disease in Pregnancy: <https://www.thyroid.org/thyroid-disease-pregnancy/>

Thyroid Function Tests: <https://www.thyroid.org/thyroid-function-tests/>

ABBREVIATIONS & DEFINITIONI

Bisphenols: a group of chemicals used to make commonly used plastics such as those for food and water containers, receipts, CDs, DVDs, toys, and plastic bags. Common bisphenols are bisphenol A, S and F. BPA use has been since restricted in many countries.

Triclosan: an antibacterial chemical frequently used to make hand sanitizers or other personal care products.

Endocrine disruptors: chemical pollutants in the environment that can affect the action of endocrine glands. Examples include bisphenol A (BPA), polychlorinated biphenols (PCBs), perfluoroalkyl substances (PFAs) and organochlorines (OCs).

TSH: thyroid stimulating hormone – produced by the pituitary gland that regulates thyroid function; also the best screening test to determine if the thyroid is functioning normally.

Thyroxine (T₄): the major hormone produced by the thyroid gland. T₄ gets converted to the active hormone T₃ in various tissues in the body.

Triiodothyronine (T₃): the active thyroid hormone, usually produced from thyroxine.

Deiodinase enzymes: these enzymes convert T₄ to T₃ on the cellular level by removing an iodine molecule from T₄.



AUTOIMMUNE THYROID DISEASE

Children and adolescents with Hashimoto's thyroiditis or Graves' disease are at increased risk for autoimmune gastritis

BACKGROUND

Autoimmune thyroid disease occurs when proteins produced by the body's immune system (antibodies) get confused and attack the thyroid. If the antibodies block or destroy the thyroid, hypothyroidism developed, called Hashimoto's thyroiditis. If the antibodies turn on the thyroid, hyperthyroidism results, called Graves' disease. Patients with autoimmune thyroid disease are at risk of having another autoimmune disorder, for example type 1 diabetes, rheumatoid arthritis or celiac disease, although most patients only have the thyroid involved. One such autoimmune disorder that occasionally occurs in patients with autoimmune thyroid disease is autoimmune gastritis, a chronic inflammatory condition in which parietal cells of the stomach are destroyed by antibodies. Patients with autoimmune gastritis have problems with iron and vitamin B12 absorption. A decrease in vitamin B12 uptake can lead to pernicious anemia (low red blood cell) and problems with their nerves. The presence of parietal-cell antibodies can be used to screen for autoimmune gastritis. The current study examined the presence of parietal-cell antibodies in children with autoimmune thyroid disease and the development of autoimmune gastritis.

THE FULL ARTICLE TITLE

Calcaterra V et al 2019 Anti-gastric parietal cell antibodies for autoimmune gastritis screening in juvenile autoimmune thyroid disease. J Endocrinol Invest. Epub 2019 Jul 1. PMID: 31264142.

SUMMARY OF THE STUDY

Parietal-cell antibodies were measured in children at an Italian medical center at time of diagnosis of their

thyroid disorder and then followed yearly. Patients testing positive for parietal-cell antibodies underwent further testing to confirm the diagnosis of autoimmune gastritis. The study included 220 children with autoimmune thyroid disease and a majority of this patients had Hashimoto's thyroiditis while the rest of the patients had Graves' disease. The average age at diagnosis was ~11 years. Participants were followed for an average of ~7 years. At diagnosis, 1 patient had parietal-cell antibodies. During an average monitoring period of 2.7 years, parietal-cell antibodies were detected in 10 patients. The likelihood of detecting parietal-cell antibodies was not different based on the thyroid diagnosis (Hashimoto's thyroiditis compared to Graves' disease). Overall, 5 of 10 patients positive for parietal-cell antibodies had abnormal lab findings such as low iron levels, low vitamin B12 levels, and/or anemia (low red blood cells). The remaining 5 patients with positive parietal-cell antibodies and normal lab findings and no stomach symptoms underwent endoscopy (evaluation of the digestive tract) and were diagnosed with autoimmune gastritis.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study suggests that children with Hashimoto's thyroiditis or Graves' disease are at increased risk for the development of parietal-cell antibodies, B12 deficiency and autoimmune gastritis. The authors suggest screening and testing for parietal-cell antibodies in children and adolescents with Hashimoto's thyroiditis or Graves' disease.

— Priya Mahajan, MD

ATA THYROID BROCHURE LINKS

Thyroiditis: <https://www.thyroid.org/thyroiditis/>

Graves' Disease: <https://www.thyroid.org/graves-disease/>



AUTOIMMUNE THYROID DISEASE, continued

ABBREVIATIONS & DEFINITIONS

Antibodies: proteins that are produced by the body's immune cells that attack and destroy bacteria and viruses that cause infections. Occasionally the antibodies get confused and attack the body's own tissues, causing autoimmune disease.

Autoimmune thyroid disease: a group of disorders that are caused by antibodies that get confused and attack the thyroid. These antibodies can either turn on the thyroid (Graves' disease, hyperthyroidism) or turn it off (Hashimoto's thyroiditis, hypothyroidism).

Autoimmune disorders: A diverse group of disorders that are caused by antibodies that get confused and attack the body's own tissues. The disorder depends on what tissue the antibodies attack. Graves' disease and Hashimoto's thyroiditis are examples of autoimmune thyroid disease. Other Autoimmune disorders include: type 1 diabetes mellitus, Addison's disease (adrenal insufficiency), vitiligo (loss of pigment of some areas of the skin), systemic lupus erythematosus, pernicious anemia (B12 deficiency), celiac disease, inflammatory bowel disease, myasthenia gravis, multiple sclerosis, and rheumatoid arthritis.

Pernicious anemia/B12 deficiency: caused by antibodies that destroy the cells in the stomach that produce a protein that is needed for the body to absorb vitamin B12, causing a severe anemia (low blood count).

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

Hashimoto's thyroiditis: the most common cause of hypothyroidism in the United States. It is caused by antibodies that attack the thyroid and destroy the gland.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.



Clinical Thyroidology® for the Public

ATA Alliance for Thyroid Patient Education

GOAL The goal of our organizations is to provide accurate and reliable information for patients about the diagnosis, evaluation and treatment of thyroid diseases. We look forward to future collaborations and continuing to work together toward the improvement of thyroid education and resources for patients.



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www.thyroid.org

ATA Patient Resources:

www.thyroid.org/thyroid-information/

Find a Thyroid Specialist: www.thyroid.org

(Toll-free): 1-800-THYROID

thyroid@thyroid.org

Bite Me Cancer

www.bitemecancer.org

info@bitemecancer.org

Graves' Disease and Thyroid Foundation

www.gdatf.org

(Toll-free): 877-643-3123

info@ngdf.org

Light of Life Foundation

www.checkyourneck.com

info@checkyourneck.com

MCT8 – AHDS Foundation

mct8.info

Contact@mct8.info

Thyca: Thyroid Cancer Survivors' Association, Inc.

www.thyca.org

(Toll-free): 877-588-7904

thyca@thyca.org

Thyroid Cancer Alliance

www.thyroidcanceralliance.org

www.thyroidcancerpatientinfo.org

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JOIN US

PLEASE JOIN OUR JOURNEY TO ADVANCED DISCOVERIES AND TREATMENT FOR THYROID DISEASE AND THYROID CANCER

As patients with thyroid disease navigate the challenges to their quality of life and researchers and physicians look for more effective directions, we at the ATA have our own destination—**funding for critical thyroid research, prevention, and treatment.** For 94 years, the ATA has led the way in thyroidology. It's a daily obstacle course to find new drugs, better treatments, advanced surgical methods, and more rapid diagnoses for the 20 million Americans who have some form of thyroid disease.

“The ATA was a valuable resource for our family when my dad was diagnosed with Anaplastic Thyroid Cancer. When you're faced with a detrimental diagnosis where even a few days can make the difference in life or death, understanding your options quickly is critical. The ATA website offers a one-stop shop for patients and caregivers to find specialists, current clinical trials, general thyroid cancer information, and links to other patient support groups and information.”

Mary Catherine Petermann

- Father who was diagnosed with Anaplastic Thyroid Cancer in 2006
- He was treated at Mayo Clinic
- He has clean scans as of October 2016

The ATA has paved the way with management guidelines for clinicians who diagnose and treat thyroid disease. For physicians treating pregnant women diagnosed with thyroid disease, our recent publication presents 97 evidence-based recommendations making sure that best practices are implemented with the latest, most effective treatment.



Through your generous support and donations, research takes the lead and hope is on the horizon. **Will you join us** in our campaign to raise **\$1.5 million** for thyroid research, prevention, and treatment? Your compassionate, tax-deductible gift will provide funds for:

- Research grants that pave the way for 1,700 ATA physicians and scientists who have devoted their careers to understanding the biology of and caring for patients affected by thyroid disease.
- Patient education for individuals and families looking for life-changing clinical trials, the best thyroid specialists, and cutting edge treatment and drugs.
- Professional education that offers a wealth of knowledge and leading-edge research for trainees and practitioners.
- A website that is the go-to resource for thyroid information for patients and practitioners alike. In 2016 alone, there were more than 3,700,000 website views of ATA's library of online thyroid information patient brochures.

Donations **of all sizes** will change the future for thyroid patients. You will make a direct impact on patients like Mary Catherine's father as he deals with Anaplastic Thyroid Cancer. You will help scientists like ATA Associate Member Julia Rodiger, Ph.D., a scientist at the National Institutes of Health, as she analyzes thyroid hormones for intestinal stem cell development.

Medullary Thyroid Cancer

WHAT IS THE THYROID GLAND?

The thyroid gland is a butterfly-shaped endocrine gland that is normally located in the lower front of the neck. The thyroid's job is to make thyroid hormones, which are secreted into the blood and then carried to every tissue in the body. Thyroid hormone helps the body use energy, stay warm and keep the brain, heart, muscles, and other organs working as they should.

CANCER OF THE THYROID

Thyroid cancer is relatively uncommon compared to other cancers. In the United States it is estimated that in 2016 approximately 64,000 new patients will be diagnosed with thyroid cancer, compared to over 240,000 patients with breast cancer and 135,000 patients with colon cancer. However, fewer than 2000 patients die of thyroid cancer each year. In 2013, the last year for which statistics are available, over 630,000 patients were living with thyroid cancer in the United States. Thyroid cancer is usually very treatable and is often cured with surgery (see [Thyroid Surgery brochure](#)) and, if indicated, radioactive iodine (see [Radioactive Iodine brochure](#)). Even when thyroid cancer is more advanced, effective treatment is available for the most common forms of thyroid cancer. Even though the diagnosis of cancer is terrifying, the prognosis for most patients with [papillary and follicular thyroid cancer](#) is usually excellent.

MEDULLARY THYROID CANCER

Medullary Thyroid Cancer (MTC) accounts for 1%–2% of thyroid cancers in the United States. MTC is different from other types of thyroid cancers (which are derived from thyroid follicular cells – the cells that make thyroid hormone), because it originates from the parafollicular C cells (also called “C cells”) of the thyroid gland. These cells do not make thyroid hormone and instead make a different hormone called calcitonin.

MTC can, and frequently does, spread to lymph nodes and can also spread to other organs. MTC is likely to run in families (inherited forms) in up to 25% of diagnoses, and inherited forms can be associated with other endocrine tumors, in syndromes called Multiple Endocrine Neoplasia (MEN) 2A and MEN 2B. In addition to MTC, patients with MEN2A may have tumors of the adrenal glands called pheochromocytomas or in the parathyroid glands (parathyroid adenomas). Patients with MEN2B, have MTC, pheochromocytomas and neuromas (typically a benign growth or tumor of nerve tissue) in the lining of the mouth and/ or gastrointestinal track.

Patients with an inherited form of MTC usually have a mutation in a gene called the RET proto-oncogene. This mutation is present in all of the cells in their body (a germline mutation) and these mutations cause the development of MTC. This is important because in family members of a person with an inherited form of MTC, a blood test for a mutation in the RET proto-oncogene can lead to an early diagnosis of MTC and, to curative surgery to remove it. However, in the majority of patients (~ 75%) a germline mutation is not found - indicating that MTC is not an inherited or inheritable condition. In these cases, MTC is called sporadic.

Whether MTC is sporadic or familial can be determined by a blood test for the RET proto-oncogene. Anyone diagnosed with MTC should have this test run to determine whether the MTC is familial (meaning other family members may also have MTC that has not yet been diagnosed) or sporadic.



Medullary Thyroid Cancer

WHAT ARE THE SYMPTOMS OF MEDULLARY THYROID CANCER?

Medullary thyroid cancer usually presents as a lump or nodule in the thyroid. It may be noted by the patient or discovered during routine neck examination by the doctor. Sometimes, the nodule is discovered incidentally by imaging studies done for other unrelated reasons (CT of the neck, PET scan, or carotid ultrasound). The nodule may cause no symptoms, but in some cases the tumor may have spread to lymph nodes in the neck, which may be enlarged on physical examination.

Patients with advanced MTC may complain of pain in the neck, jaw, or ear. If a nodule is large enough to compress the windpipe or the esophagus, it may cause difficulty with breathing or swallowing. Hoarseness can be present if the cancer invades the nerve that controls the vocal cords.

MTC is usually more aggressive than the other more common types of thyroid cancer (See *Thyroid Cancer-papillary and follicular- brochure*), and it is usually easier to treat and control if it is found before it spreads to lymph nodes in the neck or other parts of the body.

Thyroid function tests such as TSH are usually normal, even when MTC is present.

If you have a family history of MTC and have tested positive for the RET mutation, then you should see an endocrinologist to help determine how best to follow you or treat you.

HOW IS MEDULLARY THYROID CANCER DIAGNOSED?

A diagnosis of thyroid cancer is usually made by a *fine needle aspiration (FNA) biopsy* of a thyroid nodule, or after the nodule is surgically removed. Patients in whom the results of an FNA biopsy (or histopathology) are suggestive or indicative of MTC should be further evaluated with measurement of the proteins calcitonin and carcinoembryonic antigen (CEA) in the blood, which are typically elevated in patients with MTC. These tests are useful to confirm the diagnosis of MTC which can help ensure the surgeon plans the correct surgery, and also serve as tumor markers during long-term follow-up to detect any remaining disease or recurrence of the cancer.

WHAT IS THE RET MUTATION?

The RET proto-oncogene is located on chromosome 10. A genetic mutation in the RET oncogene is seen in all cells in the body in patients with the hereditary forms of MTC. Mutations in RET can also be seen only in the tumor cells in patients with sporadic MTC. Since the discovery of the RET oncogene, more than 100 different mutations have been identified in the gene in patients with MTC.

Genetic counseling and testing for RET gene mutations should be offered to patients diagnosed with MTC and first-degree relatives (parents, siblings and children of someone diagnosed with MTC) of all patients with proven germline mutations (hereditary MTC). If close relatives, especially children, are found to have the RET mutation on a blood test, the thyroid gland can be removed before MTC has a chance to develop or at least in its very early stages.

HOW IS MTC TREATED?

The primary treatment for MTC is surgery, and the currently accepted approach is to remove the entire thyroid gland (total thyroidectomy) (See *thyroid surgery brochure*). Often patients with MTC will have thyroid cancer present in the lymph nodes of the neck or upper chest. These lymph nodes are usually removed at the time of thyroid surgery or sometimes, at a later surgery if found subsequently. After surgery, patients need to take thyroid hormone replacement medication for life.

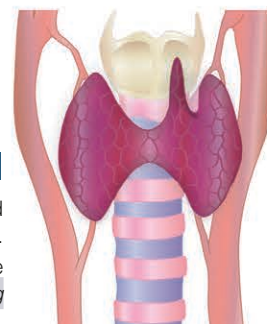
Unlike papillary and follicular thyroid cancer, medullary thyroid cancer does not take up iodine, and consequently radioactive iodine treatment is not a treatment option for patients with MTC.

Patients with MTC with very high levels of calcitonin should have imaging prior to surgery to determine whether the tumor has spread to sites outside the thyroid and/or outside the neck. If there is evidence of cancer outside the neck, surgery may be more palliative, aimed at reducing local complications caused by the tumor, rather than completely eliminating all tumor. Other treatment options (external beam radiation, or chemotherapy) may need to be used together with surgery after careful discussion with the patient.

FURTHER INFORMATION

Further details on this and other thyroid-related topics are available in the patient thyroid information section on the American Thyroid Association® website at www.thyroid.org.

For information on thyroid patient support organizations, please visit the *Patient Support Links* section on the ATA website at www.thyroid.org



Medullary Thyroid Cancer

New chemotherapeutic agents that have shown promise treating other advanced cancers are increasingly available for treatment of thyroid cancers. Two such agents, Vandetanib and Cabozantinib have been FDA approved for use by patients with MTC. These drugs do not cure advanced cancers that have spread widely throughout the body, but they can often slow down or partially reverse the growth of the cancer. These treatments are usually given by an oncologist (cancer specialist) and require care at specialized medical centers.

WHAT IS THE FOLLOW-UP FOR PATIENTS WITH MTC?

Periodic follow-up examinations are essential for all patients with MTC because the thyroid cancer can return, sometimes many years after successful initial treatment. These follow-up visits include a careful history and physical examination, with particular attention to the neck area. Neck ultrasound is also a very important tool to visualize the neck and look for nodules, lumps or enlarged lymph nodes that might indicate that the cancer has recurred.

Blood tests are also important in the follow-up of MTC patients. All patients who have had their thyroid glands removed require thyroid hormone replacement with levothyroxine. Thyroid stimulating hormone (TSH) should be checked periodically, and the dose of levothyroxine adjusted to keep TSH in the normal range. There is no need to keep TSH suppressed in patients with MTC.

Measurement of calcitonin and CEA are a necessary routine part of the follow-up of patients with MTC. Following thyroidectomy, it is hoped that calcitonin levels will be essentially undetectable for life. A detectable or rising calcitonin level should raise suspicion for possible cancer recurrence. Detectable calcitonin levels may require additional tests.

WHAT IS THE PROGNOSIS OF MEDULLARY THYROID CANCER?

The prognosis of MTC is usually not as favorable as differentiated thyroid cancers (*papillary and follicular cancer*). However, if discovered early, surgery can be curative. Even in cases where it is not caught early, MTC often progresses relatively slowly. Long-term survival depends on the stage of disease at the time of diagnosis. The blood levels of calcitonin or CEA over the first year after surgery can also be a predictor of a patient's survival.

ATA PARTNERING WITH MTC

The Medullary Thyroid Carcinoma (MTC) Registry Consortium* is partnering with the American Thyroid Association (ATA) to create a registry (list) of all new cases of MTC diagnosed in the United States over the next 10-15 years (the MTC Registry). The purpose of the MTC Registry is to help better understand what risk factors are associated with the development of MTC.

Click here for additional information:

<https://www.thyroid.org/professionals/partner-relations/medullary-thyroid-carcinoma-registry-consortium/>



FURTHER INFORMATION

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