



GENESIS
FUNCTIONAL WELLNESS

A GENESIS FUNCTIONAL WELLNESS PATIENT GUIDE

Hashimoto's & Hypothyroidism

How thyroid autoimmunity connects to your energy, gut, hormones, fertility, and metabolism — often before standard testing reveals the full picture.

Genesis Functional Wellness

Elverson, Pennsylvania & telehealth across PA
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EDUCATIONAL GUIDE
NOT MEDICAL ADVICE

The Complete Hashimoto's & Hypothyroidism Guide

How autoimmunity can affect your thyroid, gut, hormones, fertility, blood sugar, and metabolism — often before standard testing reveals the full picture.

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Could this be more than a thyroid problem?

You are tired, but you cannot explain why. Your weight is changing though your habits have not. Your hair is thinning, your skin is dry, you feel cold when everyone else is comfortable. You struggle with constipation, brain fog, anxiety, low mood, irregular cycles, infertility, high cholesterol, blood-sugar problems, or symptoms that worsen around major hormonal transitions.

Perhaps you were told your thyroid tests were normal. Perhaps you were diagnosed with hypothyroidism, prescribed medication, and expected to feel better — but you still do not feel like yourself. Perhaps you have Hashimoto's antibodies

but were told nothing needs to be done until your thyroid “fails.” Or perhaps you have pursued separate treatments for your digestion, cycles, fertility, blood sugar, mood, and metabolism without anyone asking whether these problems could be connected.

The thyroid does not work in isolation. Its hormones influence energy, metabolism, digestion, temperature, cholesterol, brain function, menstrual cycles, ovulation, pregnancy, and how the body handles glucose. When thyroid function declines, the effects may be felt throughout the body. Hashimoto’s adds another layer: it is not simply a low-thyroid condition, but an autoimmune disease in which the immune system has lost tolerance to the thyroid — a process that may begin long before the gland can no longer keep up.

WHAT THIS GUIDE WILL HELP YOU UNDERSTAND

- Hashimoto’s vs. hypothyroidism
- Why oxidative stress matters
- Why TSH alone may not reveal the picture
- The thyroid–gut–SIBO connection
- Ovaries, fertility & pregnancy
- Why medication is often not optimized
- How autoimmune thyroid disease develops
- What TPO & thyroglobulin antibodies mean
- How ultrasound adds information
- Thyroid, PCOS & blood sugar
- What research says about gluten & dairy
- What a comprehensive evaluation includes

Inside you’ll also find a **quick self-assessment** to help you spot patterns worth discussing, plus a checklist of questions to ask your clinician.

The goal is not to blame every symptom on the thyroid. It is to make sure the thyroid has been thoroughly evaluated before its effects are overlooked.

01 Hashimoto's and hypothyroidism are not the same

The terms are often used interchangeably, but they describe two different problems. **Hashimoto's thyroiditis** is an autoimmune disease: the immune system begins treating thyroid proteins as targets and creates an inflammatory response within the gland. The antibodies most associated with it are thyroid peroxidase (TPO) and thyroglobulin (Tg) antibodies, and the broader process also involves T cells, B cells, inflammatory messengers, and immune-cell infiltration.

Hypothyroidism means the body isn't getting enough thyroid hormone to meet its needs. Hashimoto's is the most common cause in iodine-sufficient areas, but not the only one — surgery, radiation, certain medications, congenital conditions, iodine deficiency or excess, pituitary/hypothalamic disorders, and other thyroiditis can all cause it. A person can have Hashimoto's antibodies while hormone production remains adequate, or hypothyroidism without Hashimoto's.

Hashimoto's is the autoimmune process. Hypothyroidism is one possible result of that process.

02 What the thyroid does

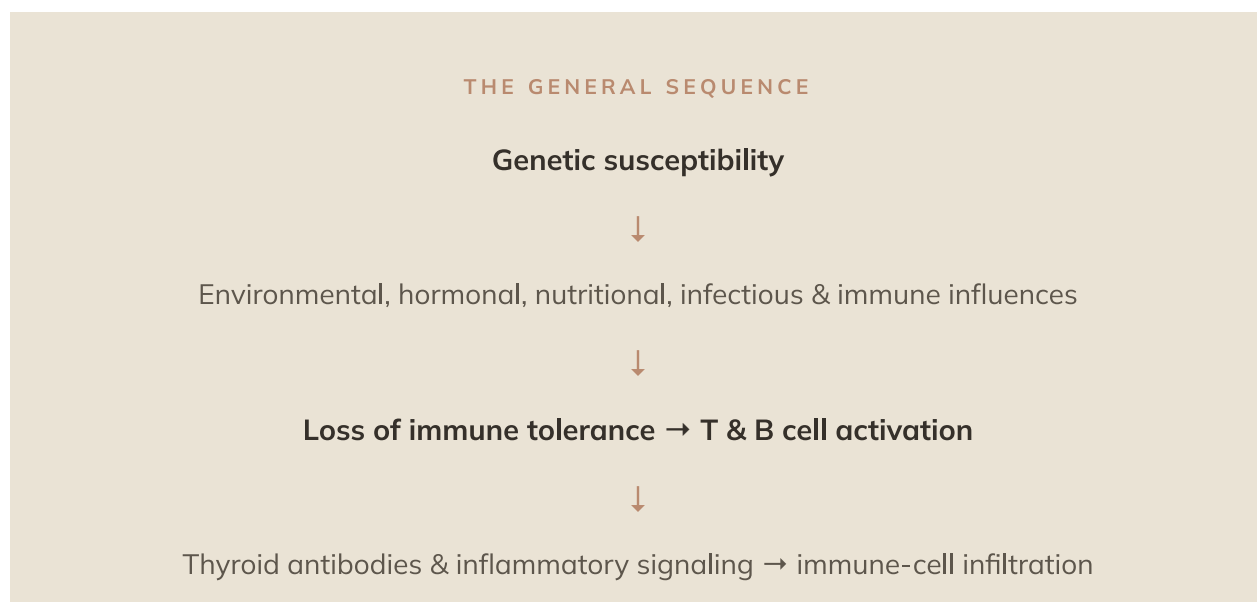
The thyroid is a small, butterfly-shaped gland at the front of the neck. It makes mostly thyroxine (T4) plus a smaller amount of triiodothyronine (T3); T4 acts largely as a circulating precursor converted into the more active T3 within tissues.

Thyroid hormones influence nearly every system: resting energy expenditure, body temperature, heart rate, cholesterol, glucose use, digestion and intestinal movement, brain function and mood, skin and hair, muscle and bone, menstrual regularity, ovulation, fertility, and pregnancy.

When thyroid hormone availability drops, processes slow — contributing to fatigue, cold intolerance, constipation, dry skin, hair loss, puffiness, weight gain, high cholesterol, muscle discomfort, cognitive changes, depression, and heavy or irregular cycles. None of these are exclusive to hypothyroidism, so they should be read alongside history, lab trends, medication, antibodies, and other findings.

03 How an autoimmune condition develops

The immune system is built to recognize threats while leaving the body's own tissue unharmed — the ability to tell “self” from “non-self,” called immune tolerance. In Hashimoto's, that tolerance breaks down. A person may inherit susceptibility; hormonal changes, immune stressors, excess iodine, infections, nutrition, gut factors, medications, and environmental exposures may help determine whether and when it becomes clinically expressed. No single trigger explains every case.



↓
Repeated thyroid-cell stress & injury → gradual loss of reserve

↓
Subclinical, then overt hypothyroidism

T cells play a major role in thyroid-cell injury; B cells become antibody-producing cells; inflammatory messengers recruit more immune cells. Over time, thyroid follicles may be damaged, replaced by inflammatory tissue, and eventually become fibrotic and atrophic.

Are the antibodies themselves destroying the thyroid? TPO and thyroglobulin antibodies are important markers, but they are not the entire disease. TPO antibodies may participate in injury, but destruction is also driven by T-cell activity, inflammatory signaling, infiltration, and programmed cell death. An antibody number should not be read in isolation as a precise measure of how much damage is occurring.

04 Oxidative stress and the thyroid

Making thyroid hormone requires iodine and controlled oxidative reactions, including hydrogen peroxide. Normally, antioxidant systems — glutathione and glutathione peroxidase, selenium-dependent enzymes, catalase, superoxide dismutase, and thioredoxin pathways — protect the gland. Oxidative stress occurs when reactive oxygen species exceed the body's ability to neutralize them or repair the damage. Because hormone production is inherently oxidative, the thyroid may be especially vulnerable when antioxidant protection is inadequate or inflammation is high. Research links oxidative stress, immune dysfunction, Hashimoto's, and PCOS — though it is one contributor, not the sole cause of either.

This can become a feedback loop: normal oxidative activity → overwhelmed defenses → stressed thyroid proteins and cells → altered proteins become more visible to the immune system → more inflammation → more reactive oxygen species → further injury. Contributors to oxidative burden include poor blood-sugar control, insulin resistance, smoking, chronic inflammation, inadequate sleep, nutrient deficiencies, low antioxidant intake, excess iodine, certain infections, environmental exposures, mitochondrial dysfunction, and ongoing gut inflammation. Supporting antioxidant capacity may help, but it does not replace thyroid hormone when the gland can't keep up, nor guarantee antibodies will normalize.

05 TPO and thyroglobulin antibodies

Thyroid peroxidase (TPO) is an enzyme used in hormone production; TPO antibodies are found in most — but not all — people with Hashimoto's.

Thyroglobulin is a protein that serves as the framework for hormone production; thyroglobulin antibodies are less consistently present but can identify autoimmune thyroid disease when TPO is negative.

Positive antibodies may indicate the immune system has recognized thyroid proteins, that autoimmune thyroid disease is present or developing, that there is greater risk of future dysfunction, and that monitoring should consider more than the current TSH. A person can have positive antibodies and normal hormone levels — sometimes called euthyroid autoimmune thyroiditis.

WHAT THE ANTIBODY NUMBER DOES *NOT* TELL YOU

Exactly how much tissue is damaged · how quickly it will progress · whether symptoms are entirely thyroid-related · whether a specific food is causing the antibodies · whether medication is required · whether you will definitely become hypothyroid. Levels fluctuate, and symptom severity doesn't always track the number.

o6 How Hashimoto's may progress

Hashimoto's doesn't follow one identical timeline, but a common progression runs: genetic susceptibility → immune dysregulation / loss of tolerance → antibodies and/or tissue changes → structural changes on ultrasound → symptoms or a shift from your previous baseline → subclinical hypothyroidism → overt hypothyroidism. The order can vary — antibodies may appear before ultrasound changes, ultrasound changes can occur in antibody-negative disease, and symptoms may precede overtly abnormal levels.

It can progress slowly over years, and some people stay euthyroid a long time. Others decline more noticeably after pregnancy, illness, or major hormonal change. As **thyroid reserve** falls, the gland has less ability to adapt during pregnancy, illness, major stress, postpartum changes, perimenopause, significant weight change, or shifts in iodine exposure — eventually producing subclinical and then overt hypothyroidism.

o7 Thyroid ultrasound: what it can and cannot tell us

An ultrasound evaluates the gland's *structure*; it does not measure how much hormone the gland produces. Findings that may be associated with autoimmune thyroiditis include reduced echogenicity (a darker gland), heterogeneous texture,

enlargement, altered blood flow, pseudonodular changes, true nodules, and later atrophy. Ultrasound can sometimes reveal thyroiditis patterns while hormone levels are still in range, and can help when antibodies are negative but suspicion is high.

Ultrasound can be a valuable early-detection tool. By showing what's actually happening structurally in the gland, it helps build a fuller picture — and it can help confirm trends seen on lab work. In some cases it reveals structural changes consistent with thyroiditis before overt hypothyroidism is obvious on standard testing, which is why it can add meaningful information alongside antibodies and hormone levels.

A clinician may consider ultrasound with thyroid enlargement, an irregular or firm gland, a palpable lump, neck pressure, trouble swallowing, persistent hoarseness, positive antibodies, a suggestive family history, abnormal thyroid tests, or suspected nodules. In our practice, a thyroid ultrasound is a routine part of a thorough evaluation, because it adds structural information that lab values alone cannot show.

o8 Why TSH alone may not reveal the whole picture

TSH (thyroid-stimulating hormone) is made by the pituitary, not the thyroid. The pituitary monitors circulating thyroid hormone and adjusts TSH to tell the thyroid how hard to work — when the thyroid makes less, TSH often rises. It's a valuable, sensitive marker of that feedback system, but it does not directly measure thyroid autoimmunity, antibodies, structure, nodules, medication absorption, every tissue's hormone exposure, or the cause of symptoms.

TSH may remain normal during early thyroid autoimmunity, and it cannot identify antibodies or structural changes. Its interpretation becomes more informative combined with history, trends, free T4, and appropriate additional testing.

A fuller evaluation may include TSH, free T4, free T3, TPO and thyroglobulin antibodies, a thyroid ultrasound, a review of past trends, medication timing and formulation, and pregnancy- or age-specific interpretation. Reverse T3 is sometimes ordered in complex cases but isn't a routine first-line test and shouldn't be used alone to diagnose a thyroid problem or set treatment.

09 “Normal” results vs. the individual picture

Reference intervals are designed to include most results in a selected population, and they're necessary for identifying disease and treating safely. But many factors shift thyroid ranges — age, sex, pregnancy, time of day, iodine intake, assay method, medications, illness, antibody status, and the reference population itself — and there is real variation between individuals. One person's usual set point may occupy a narrow slice of the population range.

“Within range” does not automatically mean “unchanged,” “ideal for this individual,” or “unrelated to symptoms” — but symptoms shouldn't be used to force results into an arbitrary narrow range either.

There is no single validated “optimal range” that fits every adult regardless of age, pregnancy, medication, and context. Good care weighs the lab result, history, medication, trends, symptoms, reproductive stage, and other possible explanations together.

IO Genetics and family patterns

Hashimoto's has a strong genetic component. Twin and family studies suggest inherited factors may explain roughly 65–75% of susceptibility in some populations. Heritability doesn't mean 65–75% of a given person's disease was "caused by genes" — it describes how much variation in susceptibility within a studied population relates to genetic differences. Susceptibility doesn't guarantee disease; environment, hormones, nutrition, and immune factors also shape expression.

Parents, children, and siblings of people with Hashimoto's have increased risk of thyroid antibodies or disease, and large studies show substantial familial clustering. It would be too strong to say most children of an affected parent will develop it, but a strong family history should lower the threshold for evaluating fatigue, constipation, cold intolerance, dry skin, hair loss, growth or school-performance changes, mood changes, menstrual irregularity, delayed puberty, unexplained cholesterol elevation, thyroid enlargement, or another autoimmune disease. Screening decisions should be made with a pediatrician or provider.

II The thyroid–ovary connection

Thyroid hormones interact with reproduction at several levels: brain–pituitary–ovary communication, follicular development, ovulation, sex hormone-binding globulin, prolactin, estrogen metabolism, progesterone after ovulation, cycle regularity, fertility, and pregnancy maintenance. With hypothyroidism, cycles may become heavy, irregular, infrequent, or absent, and ovulation less reliable; some develop elevated prolactin or altered ovarian signaling.

Do thyroid antibodies affect the ovaries directly? This is an active research question. Some studies associate TPO or thyroglobulin positivity with lower AMH, lower antral follicle count, diminished ovarian reserve, infertility, miscarriage, or differences in assisted-reproduction outcomes; others find no meaningful association in euthyroid women. The research isn't settled.

Thyroid autoimmunity may be associated with ovarian and reproductive changes in some women, even when TSH is in range — but it has not been proven that TPO antibodies directly damage every antibody-positive woman's ovaries.

I2 Fertility, pregnancy, and miscarriage

Adequate thyroid hormone matters before conception and throughout pregnancy, which increases hormone requirements. Someone with limited reserve may keep up before pregnancy but struggle once pregnant. Thyroid autoimmunity has been associated with higher risks of developing hypothyroidism in pregnancy, miscarriage, preterm birth, and certain fertility difficulties — sometimes even in euthyroid women with positive antibodies, though the mechanism and the benefit of treating every such woman remain debated.

Anyone pregnant, planning pregnancy, in fertility treatment, or experiencing recurrent loss should get individualized medical thyroid evaluation — pregnancy uses different treatment thresholds and monitoring than the non-pregnant state.

I3 The Hashimoto's–PCOS connection

PCOS is a complex endocrine and metabolic condition involving some combination of irregular or absent ovulation, elevated androgens, polycystic ovarian morphology, insulin resistance, metabolic dysfunction, inflammation, and genetic susceptibility. Hashimoto's and autoimmune thyroid disease occur more often among women with PCOS than in controls across many studies; a systematic review and meta-analysis found a higher prevalence of Hashimoto's among women with PCOS, though reported prevalence varied widely by population.

Both conditions can contribute to irregular cycles, infertility, hair loss, fatigue, weight changes, mood changes, elevated cholesterol, insulin resistance, difficulty losing weight, inflammation, and sleep problems — so hypothyroidism can be missed in someone assumed to have “only PCOS,” and vice versa. **Does Hashimoto's cause PCOS?** Not in every case — PCOS is multifactorial. But hypothyroidism may intensify a PCOS pattern (reduced energy expenditure, weight gain, lower insulin sensitivity, cholesterol changes, altered SHBG, higher prolactin in some, irregular ovulation), and the two may share inflammatory, immune, genetic, and oxidative-stress pathways.

I4 Metabolism and blood sugar

Thyroid hormones influence how the body produces, uses, and stores energy: resting metabolic rate, glucose production and uptake, insulin sensitivity, lipid metabolism, mitochondrial energy, exercise tolerance, intestinal motility, and body composition. When thyroid function declines, the body may burn energy more slowly and become less metabolically flexible, which can make existing insulin

resistance more noticeable and worsen PCOS features. But not every blood-sugar problem is caused by Hashimoto's — genetics, sleep, muscle mass, diet, medications, stress hormones, menopause, liver function, and activity also matter.

Lean patients can still have metabolic dysfunction. You don't have to be overweight to have insulin resistance, reactive blood-sugar symptoms, PCOS, elevated androgens, or abnormal glucose tolerance. Body weight alone shouldn't decide whether metabolic evaluation is appropriate.

15 The thyroid–SIBO–PCOS network

These aren't a formal triad that occurs in every patient — they're a network that can overlap and reinforce one another. **SIBO** (small intestinal bacterial overgrowth) occurs when excessive or inappropriate bacteria populate the small intestine. Hypothyroidism can slow gut motility, and when movement is reduced, bacteria may be cleared less effectively, favoring overgrowth.

Research repeatedly links hypothyroidism and SIBO. A 2025 study found SIBO in about 33% of participants with hypothyroidism versus 15% of controls, and in a large records analysis, hypothyroidism and autoimmune thyroiditis were each associated with more than twice the subsequent risk of SIBO. Levothyroxine treatment was associated with reduced SIBO risk, though observational findings don't prove medication alone prevents it. SIBO symptoms can include bloating, distention, gas, constipation, diarrhea, discomfort, food intolerance, nutrient deficiencies, and fatigue, and it may affect nutrient and medication absorption.

Gut-microbiome changes have also been associated with insulin resistance, inflammation, and hyperandrogenism in PCOS, though dysbiosis hasn't been proven to be the sole cause. At the center of the cycle sit shared amplifiers: oxidative stress, inflammation, insulin resistance, immune dysregulation, nutrient

insufficiency, poor sleep, and hormonal disruption. The presence of one condition doesn't prove the others — testing and treatment should follow the person's actual symptoms and findings.

I6 Gluten, celiac disease, and Hashimoto's

The clearest established gluten connection is the overlap between autoimmune thyroid disease and celiac disease — an autoimmune condition where gluten causes immune-mediated intestinal injury. People with autoimmune thyroid disease have a higher prevalence of celiac disease than the general population, and for someone with celiac, strict lifelong gluten avoidance is medically necessary.

Hashimoto's without celiac? Some people report improved digestive or systemic symptoms after removing gluten, and small studies report changes in antibodies or other markers — but results are inconsistent, samples are small, and undiagnosed celiac or other diet changes may influence findings. Current research does not establish that every person with Hashimoto's must permanently avoid gluten. A trial may be more relevant with diagnosed celiac, positive celiac serology, a strong family history, unexplained iron deficiency, chronic GI symptoms, unexplained nutrient deficiencies, dermatitis herpetiformis, or a reproducible response to gluten. When possible, complete celiac testing *before* removing gluten, since eliminating it first makes testing less reliable.

I7 Molecular mimicry and food cross-reactivity

Molecular mimicry occurs when a foreign protein resembles part of a human protein closely enough that an immune response may react with both. It's a recognized potential mechanism in autoimmunity — but showing antibody binding

in a lab does not prove that eating a food causes autoimmune disease in a living person. Cross-reactivity can exist without being clinically important.

A 2017 laboratory study by Kharrazian, Herbert, and Vojdani tested antibodies against thyroid-axis targets against 204 purified food proteins. It found **no** food reactions with antibodies against TPO, the TSH receptor, or thyroid-binding globulin; some reactions with antibodies against T3 or T4; one involving thyroglobulin; and one involving type II 5-deiodinase. Importantly, it did **not** show that a long list of foods cross-reacts with the TPO antibodies most commonly measured in Hashimoto's — nor that eating those foods raised antibodies, caused more destruction or symptoms, or improved on removal. It was an in-vitro experiment meant as an early step.

A reasonable approach: evaluate celiac when appropriate; identify clear digestive and immune symptoms; use a structured, time-limited elimination when justified; reintroduce foods systematically; and avoid removing nutritious foods indefinitely without evidence they're problematic *for you*. This does not justify placing every person with Hashimoto's on a permanent, highly restrictive diet.

I8 What about dairy?

The evidence that dairy proteins routinely mimic thyroid tissue is weaker than commonly claimed. Dairy may still be a problem for some because of lactose intolerance, milk-protein allergy, individual sensitivity, GI inflammation, acne or congestion, or medication timing. Lactose intolerance appears relatively common in some Hashimoto's populations and may make levothyroxine harder to stabilize; lactose restriction has reduced TSH in some lactose-intolerant patients on levothyroxine — likely by improving absorption rather than stopping autoimmunity. Calcium-containing dairy shouldn't be taken at the same time as levothyroxine,

since calcium reduces absorption. Dairy removal should be individualized: a temporary elimination followed by reintroduction gives more useful information than assuming dairy is harmful for everyone.

19 Nutrients involved in thyroid function

Several nutrients contribute to hormone production, conversion, immune regulation, and antioxidant protection: iodine, selenium, iron, zinc, vitamin D, vitamin B12, folate, magnesium, protein, and tyrosine. Adequacy matters, but supplementation should be based on diet, labs, medications, pregnancy status, and history.

- **Iodine — more is not always better.** It's essential, but both deficiency and excess disrupt thyroid function. In susceptible people, excess iodine may increase oxidative stress, alter thyroglobulin, and contribute to autoimmune activity. High-dose iodine shouldn't be used casually in Hashimoto's without clinical reasoning and monitoring.
- **Selenium** supports glutathione peroxidase, antioxidant protection, deiodinase enzymes, and hormone metabolism. It has reduced antibody levels in some trials, though evidence it consistently changes progression, medication needs, symptoms, or quality of life is less certain — and excess can be harmful.
- **Iron** is needed for TPO activity, and deficiency can independently cause fatigue, hair loss, poor exercise tolerance, restless legs, cognitive symptoms, and pregnancy complications — which may be mistaken for persistent hypothyroidism.

20 Potential triggers and contributors

Hashimoto's has no single universal root cause. Potential contributors include genetic susceptibility, pregnancy and postpartum immune changes, perimenopause and other hormonal transitions, celiac disease, other autoimmune conditions, excess iodine, nutrient deficiencies, smoking, infections, chronic inflammatory conditions, gut dysfunction, certain medications, radiation exposure, environmental exposures, poor sleep, and prolonged physiologic stress.

Identifying a contributor doesn't necessarily mean removing it will reverse established thyroid damage. The goal is to identify modifiable factors *while also* replacing thyroid hormone when production is inadequate.

21 Before chasing every symptom: has the thyroid actually been optimized?

Many people with hypothyroidism are prescribed medication and told the thyroid is "handled." When fatigue, constipation, weight changes, brain fog, hair loss, low mood, irregular cycles, high cholesterol, infertility, or cold intolerance continue, they start searching for entirely different explanations. Sometimes another condition is contributing — but taking thyroid medication is not automatically the same as having adequately managed hypothyroidism.

Treatment may still need attention when the dose is too low or too high; follow-up relied on incomplete information; testing wasn't repeated after a dose change; medication is taken inconsistently; the pharmacy changed manufacturers; the formulation isn't absorbed reliably; medication is taken too close to food, coffee,

calcium, iron, fiber, or antacids; a GI condition affects absorption; or requirements changed with pregnancy, postpartum, perimenopause, menopause, weight change, or illness.

Under-replacement

Fatigue, constipation, cold intolerance, dry skin, hair loss, weight gain, puffiness, high cholesterol, heavy or irregular cycles, low mood, slower thinking, reduced exercise tolerance.

Over-replacement

Palpitations, fast or irregular heartbeat, anxiety, tremor, sweating, heat intolerance, insomnia, diarrhea, muscle loss, bone loss, higher risk of atrial fibrillation.

Absorption matters. Levothyroxine has a narrow therapeutic window and is sensitive to how it's taken — coffee, food, calcium, iron, soy, fiber, antacids, and some medications can reduce or destabilize it, and GI conditions can raise requirements. A reasonable prescription can act like an inadequate dose when it isn't consistently absorbed.

Levothyroxine (T4) is the standard first-line treatment and works well for most people. Still, some remain symptomatic after TSH normalizes — a real-world study found about **1 in 4** levothyroxine-treated patients reported persistent or new symptoms despite a normal TSH. That doesn't prove all remaining symptoms were from inadequate treatment, but it confirms they shouldn't simply be dismissed. T4/T3 combination or desiccated thyroid isn't automatically better — fourteen clinical trials haven't shown consistent population-wide superiority — though expert consensus supports further research into carefully selected, supervised trials for some persistently symptomatic patients after other causes are considered.

The point isn't that everyone should change medication. It's that the medication, dose, formulation, administration, absorption, lab response, symptoms, and safety should all be reviewed before concluding the thyroid has been fully optimized. Never start, stop, or change thyroid medication on your own — work with your prescribing clinician.

22 Don't skip the thyroid to chase downstream symptoms

When replacement remains inadequate, its effects show up everywhere — constipation, high cholesterol, insulin resistance, weight gain, menstrual irregularity, anovulation, fertility problems, depression, brain fog, hair loss, muscle pain, fatigue, poor exercise tolerance, cold intolerance. Those deserve evaluation, but increasingly complicated protocols can get built around downstream symptoms while a major hormone deficiency stays incompletely managed.

A MORE LOGICAL SEQUENCE

1. Confirm whether hypothyroidism is present. •
2. Identify the likely cause. •
3. Review whether medication is taken and absorbed correctly. •
4. Determine whether the dose is safe and adequate. •
5. Reassess whether the formulation gives an acceptable response. •
6. Evaluate autoimmunity and structure. •
7. Investigate nutritional, digestive, metabolic, reproductive, and sleep contributors. •
8. Reassess symptoms after thyroid problems are corrected.

The thyroid may not explain everything — but it shouldn't be declared “fine” simply because a prescription was written or one test read in range.

23 Why symptoms may continue after treatment is optimized

Even after careful thyroid management, some people remain symptomatic. Possible non-thyroid or overlapping causes include iron deficiency, anemia, vitamin B12 or D deficiency, sleep apnea, insomnia, blood-sugar instability, PCOS, perimenopause or menopause, low reproductive hormones, depression or anxiety, chronic inflammation, celiac disease, SIBO, autoimmune gastritis, medication side effects, chronic infection, another autoimmune condition, inadequate calorie or protein intake, overtraining, a sedentary lifestyle, or chronic pain. Persistent symptoms call for a broader evaluation — one that *complements* appropriate thyroid management rather than replacing it.

24 Hashimoto's across a woman's lifespan

- **Adolescence** — fatigue, constipation, growth changes, puffiness, dry skin, hair loss, heavy/irregular or missed periods, mood changes, declining concentration, elevated cholesterol. Because these are easily attributed to adolescence, family history is especially important.
- **Fertility years** — may affect ovulation, cycle regularity, prolactin, ovarian signaling, implantation, and pregnancy.
- **Pregnancy** — requirements increase; medication often needs prompt adjustment and closer monitoring.
- **Postpartum** — a major immune shift; postpartum thyroiditis can cause a hyperthyroid phase, a hypothyroid phase, or both. Some recover; others develop persistent hypothyroidism.

- **Perimenopause & menopause** — symptoms overlap heavily (fatigue, brain fog, weight changes, sleep disruption, anxiety, depression, hair thinning, palpitations, temperature dysregulation, menstrual changes). Both thyroid and reproductive hormones may need evaluation, especially since thyroid disease becomes more common with age.
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25 Conditions that may coexist

People with one autoimmune disease are more likely to develop another. Conditions associated with Hashimoto's include celiac disease, type 1 diabetes, autoimmune gastritis, pernicious anemia, vitiligo, alopecia areata, rheumatoid arthritis, Sjögren syndrome, lupus, Addison disease, and other autoimmune endocrine disorders. It may also coexist with non-autoimmune conditions such as PCOS, insulin resistance, dyslipidemia, SIBO, sleep apnea, iron deficiency, endometriosis, and perimenopausal hormone changes. Coexistence doesn't prove one condition caused the other.

26 A comprehensive thyroid evaluation

Not everyone needs every test — testing should follow symptoms, history, medication, pregnancy status, exam, and prior results. A thorough workup considers the full history (family and personal autoimmune history, childhood symptoms, pregnancy and postpartum, cycles, fertility, PCOS or endometriosis, digestion, blood sugar, weight, sleep, stress, medications, supplements, iodine exposure, prior imaging, past lab trends, and response to previous medication).

THE THYROID PANEL WE RECOMMEND

The complete 9 thyroid tests — TSH, Total T4, Free T4, Total T3, Free T3, Reverse T3, T3 uptake (or TBG), TPO antibodies, and thyroglobulin antibodies — **plus a thyroid ultrasound**. In our practice, Free T3 and ultrasound are always included, not optional add-ons.

RELATED TESTING THAT MAY FIT

CBC · metabolic panel · ferritin & iron · B12 & folate · vitamin D · lipids · fasting glucose & A1c · fasting insulin · celiac screen (before gluten removal) · reproductive hormones & prolactin · SIBO testing · sleep-apnea evaluation

A comprehensive evaluation doesn't mean ordering every possible test — it means selecting the tests most likely to answer your actual clinical questions.

27 Could this be more than a thyroid problem? A self-assessment

This does not diagnose anything — it can help you spot patterns worth discussing with a clinician. Check any that apply.

ENERGY & METABOLISM

- ☐ Persistent fatigue
- ☐ Never fully rested
- ☐ Unexplained weight gain
- ☐ Difficulty losing weight
- ☐ Feeling unusually cold
- ☐ Reduced exercise tolerance
- ☐ Muscle weakness or aching
- ☐ Elevated cholesterol

BRAIN & MOOD

- ☐ Brain fog
- ☐ Poor concentration
- ☐ Forgetfulness
- ☐ Depression
- ☐ Anxiety
- ☐ Slower thinking
- ☐ Low motivation

SKIN, HAIR & APPEARANCE

- ☐ Dry skin
- ☐ Hair loss
- ☐ Thinning outer eyebrows
- ☐ Brittle nails
- ☐ Puffy face or eyelids
- ☐ Fluid retention

DIGESTION

- ☐ Constipation
- ☐ Bloating or distention
- ☐ Diarrhea
- ☐ Food intolerance
- ☐ Reflux
- ☐ History of SIBO
- ☐ Iron or B12 deficiency

MENSTRUAL & REPRODUCTIVE

- ☐ Heavy periods
- ☐ Irregular or missed periods
- ☐ Painful cycles
- ☐ Inconsistent ovulation
- ☐ Infertility
- ☐ Recurrent miscarriage
- ☐ PCOS or endometriosis
- ☐ Worsened postpartum
- ☐ Marked perimenopausal change

THYROID-SPECIFIC HISTORY

- Known positive TPO or Tg antibodies
- Thyroid enlargement or nodules
- Abnormal thyroid ultrasound
- Family history of Hashimoto's /
hypothyroidism
- Another autoimmune condition
- Symptoms despite medication
- Only TSH routinely checked
- Medication taken with coffee, food, iron, or
calcium

The more areas involved, the more important it becomes to evaluate the thyroid as part of an interconnected system rather than one isolated number.

28 Questions to ask your clinician

1. Has the cause of my hypothyroidism been identified?
2. Have both TPO and thyroglobulin antibodies been checked?
3. How have my thyroid results changed over time?
4. Are my results appropriate for my age, pregnancy status, and situation?
5. Would a thyroid ultrasound add useful information?
6. Could my medication timing or supplements interfere with absorption?
7. Have I stayed on the same formulation or manufacturer?
8. When should labs be repeated after a dose change?
9. Could a GI condition be affecting absorption?
10. Are my symptoms consistent with under-replacement, over-replacement, or another condition?
11. Should iron, B12, vitamin D, blood sugar, cholesterol, or celiac be evaluated?
12. Could thyroid dysfunction be contributing to my menstrual or fertility problems?

13. Should PCOS, insulin resistance, or prolactin be evaluated?
 14. Could constipation, bloating, or SIBO be connected to reduced thyroid function?
 15. What would justify reconsidering my dose or formulation?
 16. What symptoms need more urgent evaluation?
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29 The thyroid–gut–ovary–blood-sugar connection

The thyroid

Metabolic rate, glucose use, cholesterol, intestinal movement, ovulation, cycles, energy production.

The gut

Nutrient & medication absorption, immune signaling, inflammatory burden, blood-sugar regulation, estrogen metabolism.

The ovaries

Ovulation, progesterone, estrogen, androgen production, cycles, fertility.

Blood sugar & insulin

Ovarian androgen production, inflammation, energy, appetite, weight regulation, PCOS expression.

SHARED AMPLIFIERS AT THE CENTER

Inflammation · Oxidative stress · Insulin resistance · Immune dysregulation · Nutrient insufficiency · Poor sleep

A problem in one area places greater demand on the others — which is why complex thyroid cases often require looking beyond one gland or one marker.

30 When thyroid symptoms need prompt care

Seek prompt medical care for: rapid thyroid enlargement or a new/growing neck mass; trouble breathing or swallowing; persistent unexplained hoarseness; chest pain; fainting; significant palpitations or a rapid/irregular heartbeat; severe weakness or confusion; symptoms of severe over-replacement; pregnancy with abnormal thyroid testing; significant unexplained weight loss; or a thyroid nodule with concerning features. **Do not stop or substantially change prescription thyroid medication without working with the prescribing clinician.**

THE GENESIS FUNCTIONAL WELLNESS APPROACH

Understanding what's actually driving your symptoms

We work with women experiencing complex, unresolved hormone and metabolic concerns — thyroid dysfunction, PCOS, fertility, endometriosis, perimenopause and menopause, gut health, and metabolic health. Many have been told their labs are normal while they continue to experience fatigue, weight changes, irregular cycles, infertility, mood changes, brain fog, digestive symptoms, or persistent inflammation.

Our approach builds a more complete picture by evaluating the connections among thyroid function and autoimmunity, medication management and response, reproductive hormones, blood sugar and insulin resistance, gut health and the microbiome, nutritional status, inflammation and immune activation, and environmental and lifestyle contributors. We don't assume every symptom comes

from the thyroid — and we don't assume the thyroid has been fully addressed simply because one test was normal.

A comprehensive evaluation can help determine whether your symptoms connect to Hashimoto's, inadequately managed hypothyroidism, PCOS, insulin resistance, SIBO or gut dysfunction, nutrient deficiencies, reproductive-hormone changes, or another overlapping condition.

[LEARN MORE OR REQUEST A CONSULTATION](#)

This guide is for general educational purposes only. It is not intended to diagnose, treat, cure, or prevent any disease, and it does not replace evaluation by a licensed medical professional. It should not be used to start, stop, or change thyroid medication, supplements, dietary restrictions, fertility treatment, or other medical care without appropriate professional guidance. Symptoms associated with thyroid disease can also occur with many other conditions. Testing, imaging, medication selection, and treatment should be individualized. Genesis Functional Wellness does not guarantee that addressing nutrition, gut health, lifestyle, or other contributors will normalize thyroid antibodies, regenerate damaged thyroid tissue, eliminate medication needs, or resolve every symptom.

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