

Thyroid and antithyroid agents

Two pharmacologically opposite toolkits sharing one page - levothyroxine (and briefly liothyronine) replace a hormone the gland can no longer make enough of, while the thionamides methimazole and propylthiouracil block a gland that is making too much, and the two toolkits are monitored by the same feedback loop but never mixed up with each other.

Online guide: <https://www.medguideglobal.com/pharmacology/endocrine-and-metabolic/thyroid-and-antithyroid-agents>

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Quick revision

One Liner: Levothyroxine replaces thyroid hormone and is titrated against TSH over weeks; methimazole and propylthiouracil block new hormone synthesis in an overactive gland and share one safety concern - agranulocytosis - that both labels ask patients to watch for by symptom, not by routine blood count alone.

1. Levothyroxine (T4) is the standard replacement for hypothyroidism of any cause and has a narrow therapeutic index, so its own label specifically flags brand and formulation consistency as clinically meaningful. [1]
2. Several common substances reduce levothyroxine absorption enough to matter: calcium and iron supplements, proton pump inhibitors, sucralfate and antacids, plus soybean flour, cottonseed meal, walnuts and high-fibre meals - the label directs dosing at least 4 hours apart from calcium or iron and on an empty stomach roughly 30-60 minutes before breakfast. [1]
3. Dose changes are followed by a TSH recheck no sooner than 6 to 8 weeks later in adults, because levothyroxine's own half-life of roughly 6-7 days in euthyroid patients means the new steady state takes weeks to show up. [1]
4. Liothyronine is synthetic T3 - faster-acting and shorter-lived than T4 - used far less often than levothyroxine and mentioned here mainly to contrast with it, not as a routine first-line replacement. [4]
5. Methimazole and propylthiouracil (the thionamides) both inhibit thyroid peroxidase, the enzyme that iodinates and couples tyrosine residues to build new T4 and T3 - they block new hormone synthesis, they do not destroy hormone already stored in the gland. [4]
6. Agranulocytosis is the safety concern both thionamide labels lead with: patients are told to report fever or sore throat immediately rather than to rely on a scheduled blood count, because onset can be abrupt. [2][3]
7. Propylthiouracil carries a boxed warning for severe, sometimes fatal liver injury that methimazole's label does not carry in the same boxed form, which is the main reason methimazole is generally preferred once a thionamide is indicated. [3]
8. The one place propylthiouracil is specifically favoured over methimazole is the first trimester of pregnancy, because methimazole carries its own rare but distinct teratogenic risk in that window; guidance generally supports switching back to methimazole for the second and third trimesters. [2][3]
9. Radioactive iodine ablation is a non-drug alternative that gradually destroys thyroid tissue and almost always ends in lifelong levothyroxine replacement; it is mentioned here for context only, not as a dosing or treatment recommendation. [6]

Overview

1. This class page covers two pharmacologically opposite toolkits under one taxonomy entry because they are managed by the same feedback axis and are frequently taught, and confused, together. On one side is thyroid hormone replacement - levothyroxine as the default, with liothyronine as a faster, shorter-acting synthetic T3 used far less often. On the other side are the thionamides, methimazole and propylthiouracil, which slow down a gland that is producing too much hormone. Both toolkits are ultimately titrated against thyroid-stimulating hormone (TSH) and, where relevant, free T4, but the direction of titration and the drugs used are opposite, which is exactly the distinction

exam questions like to probe. [4]

2. Levothyroxine's defining pharmacological trait is not its mechanism, which is simply supplying the hormone the gland is not making enough of, but its narrow therapeutic index combined with a slew of ordinary substances that blunt its oral absorption. Calcium and iron supplements, proton pump inhibitors, sucralfate, antacids, soy-based foods, high-fibre meals and even the timing of breakfast can all measurably lower how much of an oral dose actually reaches the circulation, which is why the label is specific about empty-stomach dosing and 4-hour separation from calcium or iron. Because bioequivalence between formulations is not automatically guaranteed at this narrow a margin, staying on one specific brand or generic product, rather than switching casually between them, is treated as a genuine clinical safeguard rather than a convenience preference. [1]

3. Liothyronine supplies T3 directly rather than relying on the body's own peripheral deiodination of T4, so it acts faster and clears faster, and its narrower dosing window makes it a less forgiving agent for routine long-term replacement than levothyroxine. It appears on this page mainly as the contrast case that clarifies why levothyroxine, not liothyronine, is the default: predictable, once-daily, long-half-life dosing is what makes an oral hormone replacement practical for lifelong use. [4]

4. The thionamides work by a shared mechanism - acting as preferential substrates for thyroid peroxidase and thereby blocking the iodination and coupling steps that build new thyroid hormone - but they diverge sharply on safety.

Methimazole is the generally preferred agent because it is dosed once daily, reaches control faster, and does not carry the boxed hepatotoxicity warning that propylthiouracil's label does. Propylthiouracil is reserved for a small number of specific situations: the first trimester of pregnancy, thyroid storm (where its additional inhibition of peripheral T4-to-T3 conversion is clinically useful), and patients who cannot tolerate methimazole and are not candidates for surgery or radioactive iodine. Radioactive iodine ablation itself is mentioned here only as the non-drug alternative that exists alongside these two drug options, not as a treatment recommendation with a dose or protocol. [3][4][6]

Classification and drug examples

Intro: The class splits cleanly along direction of effect: hormone replacement raises circulating thyroid hormone toward normal, while thionamides lower an overactive gland's own output. A third, non-pharmacological option - radioactive iodine ablation - sits alongside the thionamides as an alternative route to the same goal and is included here only for orientation.

1. Name: Thyroid hormone replacement (T4, and T3)

1. Note: Oral synthetic hormone that restores what an underactive or absent thyroid is not producing; titrated against TSH rather than symptoms alone.

1. Name: Levothyroxine

1. Synthroid

2. T4

3. L-thyroxine

1. Route: Oral (also IV for select acute settings)

1. Note: The default replacement agent; narrow therapeutic index and multiple absorption interactions make dosing consistency and timing clinically important. [1]

2. Name: Liothyronine

1. T3

2. synthetic triiodothyronine

2. Route: Oral

2. Note: Faster onset and shorter half-life than levothyroxine; used selectively rather than as routine first-line replacement, and covered here mainly by contrast. [4] [1]

2. Name: Thionamides (antithyroid drugs)

2. Note: Block new thyroid hormone synthesis at the thyroid peroxidase step; both members share an agranulocytosis warning but differ sharply on hepatotoxicity risk and pregnancy trimester preference.

1. Name: Methimazole

1. Tapazole

2. thiamazole

1. Route: Oral

1. Note: Generally the preferred thionamide outside the first trimester of pregnancy; once-daily dosing and no boxed hepatotoxicity warning. [2]

2. Name: Propylthiouracil

PTU

2. Route: Oral

2. Note: Reserved for the first trimester of pregnancy, thyroid storm, and methimazole intolerance where surgery or radioactive iodine are not options; carries a boxed warning for severe liver injury. [3] [4]

3. Name: Non-drug alternative (context only)

3. Note: Not a medicine, and no dose or protocol is given here - included only so the drug options above are understood against the full set of choices a clinician weighs.

Name: Radioactive iodine ablation (I-131)

1. radioiodine therapy

2. RAI

Route: Oral capsule or liquid

Note: Gradually destroys thyroid tissue; most patients become hypothyroid afterward and need lifelong levothyroxine, which is why this class page and the ablation pathway ultimately converge on the same replacement drug. [6] [6]

Mechanism of action

Summary: The two toolkits act at opposite ends of the same hormone axis. Levothyroxine and liothyronine simply supply the hormone (T4 or T3) that an underactive gland is not making enough of, restoring the negative-feedback signal to the pituitary. Methimazole and propylthiouracil instead enter an overactive gland and act as preferential substrates for thyroid peroxidase (TPO), the enzyme that iodinates tyrosine residues on thyroglobulin and couples them into new T4 and T3 - blocking that step reduces new hormone synthesis without touching hormone already stored in colloid, which is why thionamide onset is not immediate.

Target: Replacement: the thyroid hormone receptor pathway generally (via exogenous T4/T3). Thionamides: thyroid peroxidase (TPO) inside thyroid follicular cells.

Cidal Static: Not applicable in the cidal/static sense - replacement restores a physiological hormone level, and thionamides suppress an enzymatic synthesis step rather than killing a cell population.

Kill Kinetics: Replacement: steady state and clinical effect build over the drug's own half-life, roughly 6-7 days for levothyroxine, so dose changes are not re-assessed for 6-8 weeks. Thionamides: onset is delayed because pre-formed hormone stored in the gland must first be depleted before new-synthesis blockade lowers circulating levels.

1. Step: 1

1. Title: Hormone replacement: exogenous T4 (or T3) enters circulation

1. Detail: Levothyroxine is absorbed in the small intestine and behaves pharmacologically like endogenous T4; roughly 80% of a daily T4 dose is deiodinated peripherally to active T3 or inactive reverse T3. [1]

2. Step: 2

2. Title: Circulating hormone suppresses TSH via negative feedback

2. Detail: As T4/T3 levels rise toward normal, pituitary TSH secretion falls proportionately, which is the basis for using TSH as the monitoring target rather than T4 level alone in primary hypothyroidism. [1]

3. Step: 3

3. Title: Thionamides are taken up by thyroid follicular cells

3. Detail: Methimazole and propylthiouracil concentrate inside the thyroid gland rather than acting systemically on stored hormone. [5]

4. Step: 4

4. Title: Thionamides act as preferential TPO substrates, blocking iodination and coupling

4. Detail: By competing for the peroxidase enzyme, they prevent iodide from being incorporated into tyrosine residues and prevent those residues from coupling into T4 and T3, halting new hormone production. [4][5]

5. Step: 5

5. Title: Propylthiouracil adds a second, peripheral action at higher doses

5. Detail: Beyond its thyroidal TPO effect, propylthiouracil also inhibits the peripheral enzyme that converts T4 to the more potent T3, an extra action methimazole lacks and the reason PTU is favoured in thyroid storm specifically. [4]

Major clinical uses

1. Drug: Levothyroxine

1. Organism: Not applicable

1. Indication: Hypothyroidism of any cause (primary, secondary, tertiary, congenital or acquired) as replacement therapy; adjunct to surgery and radioiodine in thyrotropin-dependent well-differentiated thyroid cancer

1. Line: first-line replacement

1. Note: The default agent for essentially all chronic thyroid hormone replacement. [1]

2. Drug: Liothyronine

2. Organism: Not applicable

2. Indication: Hypothyroidism, used selectively rather than as routine first-line replacement

2. Line: selective / adjunctive

2. Note: Faster onset and offset than levothyroxine; not the default for long-term maintenance because of its narrower dosing window. [4]

3. Drug: Methimazole

3. Organism: Not applicable

3. Indication: Graves' disease and toxic multinodular goiter, including preparation for thyroidectomy

3. Line: generally preferred first-line thionamide

3. Note: Preferred over propylthiouracil outside the first trimester of pregnancy because of its once-daily dosing and lack of a boxed hepatotoxicity warning. [2]

4. Drug: Propylthiouracil

4. Organism: Not applicable

4. Indication: Hyperthyroidism specifically in the first trimester of pregnancy, in thyroid storm, and in patients intolerant of methimazole for whom surgery or radioactive iodine are not appropriate

4. Line: reserved / second-line

4. Note: Its label states it is not recommended for pediatric patients except in rare cases where alternative therapies are unsuitable. [3]

Pharmacokinetics

1. Drug: Levothyroxine

1. Route: Oral (IV for select acute uses)

1. Absorption: Absorbed in the small intestine; reduced by calcium, iron, PPIs, sucralfate, antacids, soy and high-fibre meals

1. Csf Penetration: Not a relevant parameter for this drug class

1. Metabolism: Peripheral deiodination of roughly 80% of the daily dose to T3 or reverse T3

1. Elimination: Hepatic and renal; half-life approximately 6-7 days in euthyroid patients, longer in hypothyroidism

1. Dose Adjust: Elderly patients and those with cardiovascular disease start below full replacement dose because of atrial fibrillation and other cardiac risk; separate dosing by at least 4 hours from calcium or iron [1]

2. Drug: Methimazole

2. Route: Oral

2. Absorption: Rapidly absorbed; peak plasma concentration within 1-2 hours; bioavailability approximately 80-95%

2. Csf Penetration: Not a relevant parameter for this drug class

2. Metabolism: Hepatic, involving CYP1A2 and CYP2C9

2. Elimination: Predominantly renal; short elimination half-life of roughly 4-6 hours despite once-daily dosing being adequate clinically

2. Dose Adjust: Discontinue if transaminases rise beyond three times the upper limit of normal; avoid where possible in the first trimester of pregnancy [5]

3. Drug: Propylthiouracil

3. Route: Oral

3. Absorption: Readily absorbed from the gastrointestinal tract

3. Csf Penetration: Not a relevant parameter for this drug class

3. Metabolism: Extensive hepatic metabolism

3. Elimination: Renal; roughly 35% of a dose appears in urine within 24 hours

3. Dose Adjust: Not recommended in pediatric patients except when alternatives are unsuitable; hepatic function is monitored closely given the boxed warning [3]

1. This page gives no dose regimens by design. Selecting a starting dose, titrating it, or switching between levothyroxine formulations is a decision for the treating clinician working from a current prescribing reference and the individual patient's labs.

2. Levothyroxine's long half-life is what makes a single daily tablet practical for lifelong replacement, while both thionamides act mainly by depleting a pre-existing hormone store rather than by their own short plasma half-lives - which is why clinical effect for either drug family takes weeks to fully show, not hours. [1][5]

3. Methimazole's renal elimination and PTU's extensive hepatic metabolism mean the two thionamides are cleared quite differently despite sharing the same target enzyme, which is one more reason they are not simply interchangeable milligram-for-milligram. [5][3]

Adverse effects

1. Effect: Overdose-pattern symptoms with levothyroxine

1. Detail: Excess replacement produces effects that mirror hyperthyroidism itself - arrhythmia risk, tremor, nervousness, insomnia, heat intolerance and muscle weakness - because the drug is simply supplying more of the same hormone the body would otherwise overproduce. [1][7]

2. Effect: Mild rash, arthralgia or gastrointestinal upset with thionamides

2. Detail: Minor hypersensitivity-type reactions are reported with both methimazole and propylthiouracil and do not necessarily require stopping the drug, unlike the serious reactions below. [2]

1. Effect: Agranulocytosis

1. Detail: The safety concern both thionamide labels centre on; propylthiouracil's label quotes an incidence around 0.2% to 0.5%, typically arising within the first three months of treatment, and methimazole's label describes the same pattern of sudden-onset leukopenia.

1. Action: Patients are told to report fever or sore throat to their clinician immediately rather than waiting for a scheduled complete blood count, since onset can be abrupt. [2][3]

2. Effect: Severe liver injury (propylthiouracil)

2. Detail: Propylthiouracil carries a boxed warning for severe liver injury and acute liver failure, in some cases fatal or requiring transplantation; the label notes that routine liver function monitoring is not expected to reliably predict this because onset can be rapid and unpredictable.

2. Action: Patients starting propylthiouracil are counselled on symptoms of liver injury and the drug is stopped if these appear, rather than relying solely on scheduled labs to catch it early. [3]

3. Effect: Hepatotoxicity (methimazole)

3. Detail: Methimazole is also associated with liver injury, but of a cholestatic pattern rather than the acute hepatocellular failure described for propylthiouracil, which is one of the reasons methimazole does not carry the same boxed warning.

3. Action: Liver enzymes are checked if symptoms of hepatic dysfunction appear during methimazole therapy. [2]

4. Effect: Teratogenic effects specific to each thionamide

4. Detail: Methimazole exposure in the first trimester has been linked to rare congenital defects including aplasia cutis and craniofacial, gastrointestinal and umbilical anomalies; propylthiouracil's own teratogenic profile is considered less severe in that window, which is the basis for the trimester-specific drug choice described above.

4. Action: Pregnancy status and trimester are established before starting or continuing either thionamide. [2]

5. Effect: Vasculitis (propylthiouracil)

5. Detail: Rare ANCA-associated vasculitis has been reported with propylthiouracil, occasionally severe enough to cause glomerulonephritis or pulmonary haemorrhage.

5. Action: New joint pain, blood in urine or breathlessness during propylthiouracil treatment warrants prompt clinical evaluation. [3]

6. Effect: Cardiac effects of over-replacement (levothyroxine)

6. Detail: Excess levothyroxine dosing increases risk of atrial fibrillation and other cardiac adverse reactions, particularly in elderly patients or those with existing cardiovascular disease.

6. Action: This is why the label directs starting below full replacement dose in these groups and rechecking TSH at defined intervals rather than adjusting dose more frequently. [1]

1. Drug: Levothyroxine

1. Effect: Contraindicated in uncorrected adrenal insufficiency, because raising metabolic rate before glucocorticoid replacement is addressed can precipitate an adrenal crisis. [1]

2. Drug: Methimazole

2. Effect: The label states it is not recommended in the first trimester of pregnancy unless the benefit is judged to outweigh the risk, given its distinct teratogenicity profile. [2]

3. Drug: Propylthiouracil

3. Effect: Its own label restricts pediatric use to rare cases where methimazole is not tolerated and surgery or radioactive iodine are not appropriate, reflecting the liver-injury risk seen even in children. [3]

Contraindications, precautions and interactions

1. Levothyroxine: uncorrected adrenal insufficiency, and known hypersensitivity to the product. [1]

2. Methimazole and propylthiouracil: known hypersensitivity to the respective drug or its excipients. [2][3]

1. Levothyroxine is not used for weight loss or obesity treatment in euthyroid patients; doses above genuine hormonal need can cause serious, even life-threatening toxicity. [1]

2. Elderly patients and those with cardiovascular disease are started on lower levothyroxine doses because of an increased risk of cardiac adverse reactions during titration. [1]

3. Pregnancy trimester is specifically weighed when choosing between the two thionamides, given their differing teratogenicity and hepatotoxicity profiles. [2][3]

4. Any fever or sore throat during thionamide therapy is treated as a possible agranulocytosis warning sign until a blood count says otherwise, not dismissed as a routine infection. [2][3]

5. Baseline and symptom-triggered liver function assessment is part of routine care with either thionamide, though the labels are explicit that this cannot reliably prevent propylthiouracil's rapid-onset liver injury. [3]

1. With: Calcium and iron supplements

1. Effect: Reduce levothyroxine absorption; the label directs separating dosing by at least 4 hours. [1]

2. With: Proton pump inhibitors, sucralfate and antacids

2. Effect: Can lower levothyroxine absorption enough to affect TSH control, supporting consistent dosing timing relative to these agents. [1]

- 3. With: Soy-based foods, high-fibre meals, walnuts and cottonseed meal
- 3. Effect: May bind levothyroxine in the gut and decrease its absorption, which is part of why the label recommends taking it on an empty stomach. [1]
- 4. With: Other hepatotoxic drugs
- 4. Effect: Concomitant use alongside propylthiouracil adds to hepatic risk and is weighed by the treating clinician against the reason PTU was chosen in the first place. [3]

Comparison tables

Title: Methimazole vs propylthiouracil: same target, different safety trade-off

Caption: Both thionamides inhibit thyroid peroxidase, but the boxed hepatotoxicity warning, dosing frequency and pregnancy-trimester preference separate them sharply. Agent choice belongs to the treating clinician.

- 1. Key: drug
- 1. Label: Drug
- 1. Is Row Header: true
- 2. Key: dosing
- 2. Label: Dosing frequency
- 3. Key: hepatic_risk
- 3. Label: Hepatic risk framing
- 4. Key: pregnancy
- 4. Label: Pregnancy positioning
- 1. Drug: Methimazole
- 1. Dosing: Once daily once controlled, despite a short 4-6 hour plasma half-life
- 1. Hepatic Risk: Cholestatic-pattern injury reported; no boxed warning
- 1. Pregnancy: Avoided where possible in the first trimester because of its own teratogenicity profile; preferred for the second and third trimesters [5][2]
- 2. Drug: Propylthiouracil
- 2. Dosing: Typically multiple daily doses
- 2. Hepatic Risk: Boxed warning for rapid-onset, sometimes fatal acute liver failure
- 2. Pregnancy: Preferred specifically in the first trimester, when its hepatotoxicity is judged the lesser risk relative to methimazole's teratogenic profile [3]

High-yield exam pearls

- 1. Pearl: Thyroid hormone replacement and thionamides act in opposite directions on the same axis, but both are ultimately re-checked against TSH.
- 1. Why: It is the organising idea for the whole class and the reason both toolkits are taught together despite having opposite pharmacological goals. [4]
- 2. Pearl: "PTU in the first trimester, methimazole otherwise" is the classic trimester rule for thionamide choice in pregnancy.
- 2. Why: It resolves a genuine trade-off - methimazole's teratogenicity risk versus PTU's hepatotoxicity risk - rather than being an arbitrary preference. [2][3]
- 3. Pearl: Agranulocytosis is taught as a symptom-driven warning (fever, sore throat), not a scheduled-labs-only safeguard.
- 3. Why: Both thionamide labels tell patients to report symptoms immediately because onset can be abrupt enough that routine monitoring intervals could miss it. [2][3]
- 4. Pearl: PTU's extra trick is blocking peripheral T4-to-T3 conversion, which methimazole does not do.
- 4. Why: It is the pharmacological reason PTU, despite its worse hepatotoxicity profile overall, is specifically favoured in thyroid storm. [4]

5. Pearl: Levothyroxine's absorption interaction list (calcium, iron, PPIs, soy, fibre) is long enough that timing, not just dose, is a monitoring variable.
5. Why: An unexplained TSH change is often an absorption or adherence question before it is a true dose question. [1]
6. Pearl: Radioactive iodine ablation usually converts hyperthyroidism into permanent hypothyroidism.
6. Why: It is the conceptual bridge between the two halves of this class page: the alternative to antithyroid drugs typically ends in needing thyroid hormone replacement anyway. [6]

Common exam traps

1. Trap: Assuming methimazole is simply "the safer thionamide" in every situation, including early pregnancy.
1. Correction: Methimazole is generally preferred overall, but propylthiouracil is specifically favoured in the first trimester because methimazole's own teratogenic risk profile is judged worse in that particular window. [2][3]
2. Trap: Treating agranulocytosis monitoring as equivalent to a routine periodic blood count schedule.
2. Correction: Both labels emphasise immediate symptom reporting (fever, sore throat) precisely because onset can be too sudden for a fixed monitoring interval to reliably catch it first. [2][3]
3. Trap: Expecting a levothyroxine dose change to show up on the next TSH drawn a week or two later.
3. Correction: Because of the drug's roughly 6-7 day half-life, the label specifies waiting 6-8 weeks after a dose change before rechecking TSH in adults. [1]
4. Trap: Believing PTU's boxed liver warning means methimazole carries no hepatic risk at all.
4. Correction: Methimazole is also associated with liver injury, of a cholestatic pattern, but that risk has not been judged to warrant the same boxed warning as PTU's acute hepatocellular failure risk. [2]

Self-test questions

1. Question: Why does levothyroxine's label direct separating its dose from calcium or iron supplements by at least 4 hours?
 1. Calcium and iron increase levothyroxine toxicity
 2. Calcium and iron reduce levothyroxine's oral absorption
 3. Levothyroxine reduces calcium and iron absorption instead
 4. There is no pharmacological interaction, only a taste conflict
1. Explanation: The label lists calcium and iron supplements among substances that reduce levothyroxine absorption, so dosing is separated by at least 4 hours to protect efficacy. [1]
2. Question: Which thionamide is specifically preferred in the first trimester of pregnancy, and why?
 1. Methimazole, because it has no teratogenic risk at all
 2. Propylthiouracil, because its own teratogenic profile is judged less severe than methimazole's in that window
 3. Methimazole, because it crosses the placenta less readily
 4. Neither is used in pregnancy under any circumstances
2. Explanation: Methimazole carries a distinct first-trimester teratogenicity risk (including aplasia cutis and craniofacial anomalies), so propylthiouracil is specifically favoured in that trimester despite its own hepatotoxicity risk. [2][3]
3. Question: What safety concern do methimazole and propylthiouracil share, and how are patients told to respond to it?
 1. Hearing loss; patients are told to get annual audiometry
 2. Agranulocytosis; patients are told to report fever or sore throat immediately
 3. Weight gain; patients are told to reduce caloric intake
 4. Photosensitivity; patients are told to avoid direct sunlight
3. Explanation: Both thionamide labels warn of agranulocytosis and instruct patients to report fever or sore throat right away rather than waiting for a scheduled blood test, because onset can be abrupt. [2][3]
4. Question: Why does propylthiouracil have a specific role in thyroid storm that methimazole does not share?
 1. It has a longer half-life than methimazole

2. It additionally inhibits peripheral conversion of T4 to the more potent T3
3. It is the only thionamide that can be given intravenously
4. It works faster because it does not need to inhibit thyroid peroxidase
4. Explanation: Beyond its thyroidal TPO-inhibiting action shared with methimazole, propylthiouracil also blocks peripheral T4-to-T3 conversion, a second mechanism useful in the acute setting of thyroid storm. [4]
5. Question: How soon after a levothyroxine dose change should TSH typically be rechecked in an adult?
 1. The next day
 2. After 1 week
 3. After 6 to 8 weeks
 4. TSH is never rechecked once a stable dose is found
5. Explanation: Levothyroxine's label specifies rechecking TSH 6 to 8 weeks after a dosage change in adults, reflecting the drug's roughly week-long half-life and the time needed to reach a new steady state. [1]
6. Question: What typically happens to thyroid function after radioactive iodine ablation for hyperthyroidism?
 1. Thyroid function usually normalises permanently without further treatment
 2. Most patients become hypothyroid and need lifelong levothyroxine replacement
 3. It has no lasting effect on thyroid hormone levels
 4. It converts hyperthyroidism into thyroid cancer in most patients
6. Explanation: Radioactive iodine gradually destroys thyroid tissue, and most patients subsequently become hypothyroid and require lifelong levothyroxine replacement - the same drug this page opens with. [6]

Frequently asked questions

1. Question: Why can't I just switch between generic levothyroxine brands freely?
 1. Answer: Levothyroxine has a narrow therapeutic index, and the label notes that bioavailability can differ slightly between formulations. For most patients this margin matters enough that staying on one specific product, and rechecking TSH if a switch does happen, is the safer approach rather than treating all levothyroxine tablets as interchangeable. [1]
 2. Question: If I take calcium or iron supplements, does that mean I can't take levothyroxine?
 2. Answer: No - it means timing matters. The label directs taking levothyroxine on an empty stomach and separating it from calcium or iron supplements by at least 4 hours, because both can meaningfully reduce how much levothyroxine is actually absorbed. [1]
 3. Question: Is methimazole always safer than propylthiouracil?
 3. Answer: Generally, yes - methimazole is once daily and does not carry propylthiouracil's boxed warning for severe liver injury. The one specific exception is the first trimester of pregnancy, where methimazole's own distinct teratogenic risk makes propylthiouracil the preferred choice for that window only. [2][3]
 4. Question: Why do both antithyroid drugs warn about sore throat and fever specifically?
 4. Answer: Those are the early symptoms of agranulocytosis, a rare but serious drop in infection-fighting white blood cells that both methimazole and propylthiouracil can cause, sometimes abruptly. Reporting these symptoms immediately, rather than waiting for a scheduled blood test, is how this risk is actually caught in practice. [2][3]
 5. Question: Does radioactive iodine treatment replace the need for thyroid medication?
 5. Answer: Not usually. Radioactive iodine is a non-drug alternative to thionamides for hyperthyroidism, but it works by gradually destroying thyroid tissue, and most patients end up hypothyroid afterward and need lifelong levothyroxine - so it tends to lead back to the replacement side of this page rather than away from medication altogether. [6]

References

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[6] Radioactive Iodine (I-131) Therapy for Hyperthyroidism and Thyroid Cancer. StatPearls Publishing / NCBI Bookshelf 2025. <https://www.ncbi.nlm.nih.gov/books/NBK557741/>

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