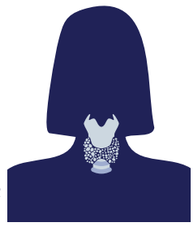
Hashimoto's thyroiditis (HT) and the gut-immune interface

fx Medicine® BioCeuticals

HT is the most common cause of **hypothyroidism**[2]



10-20% of AUSTRALIANS have elevated **thyroid antibodies**[1]

HT is 10x more common in women than in men[2]

Up to **1/3 of patients receive INADEQUATE hypothyroid treatment**[4-6]

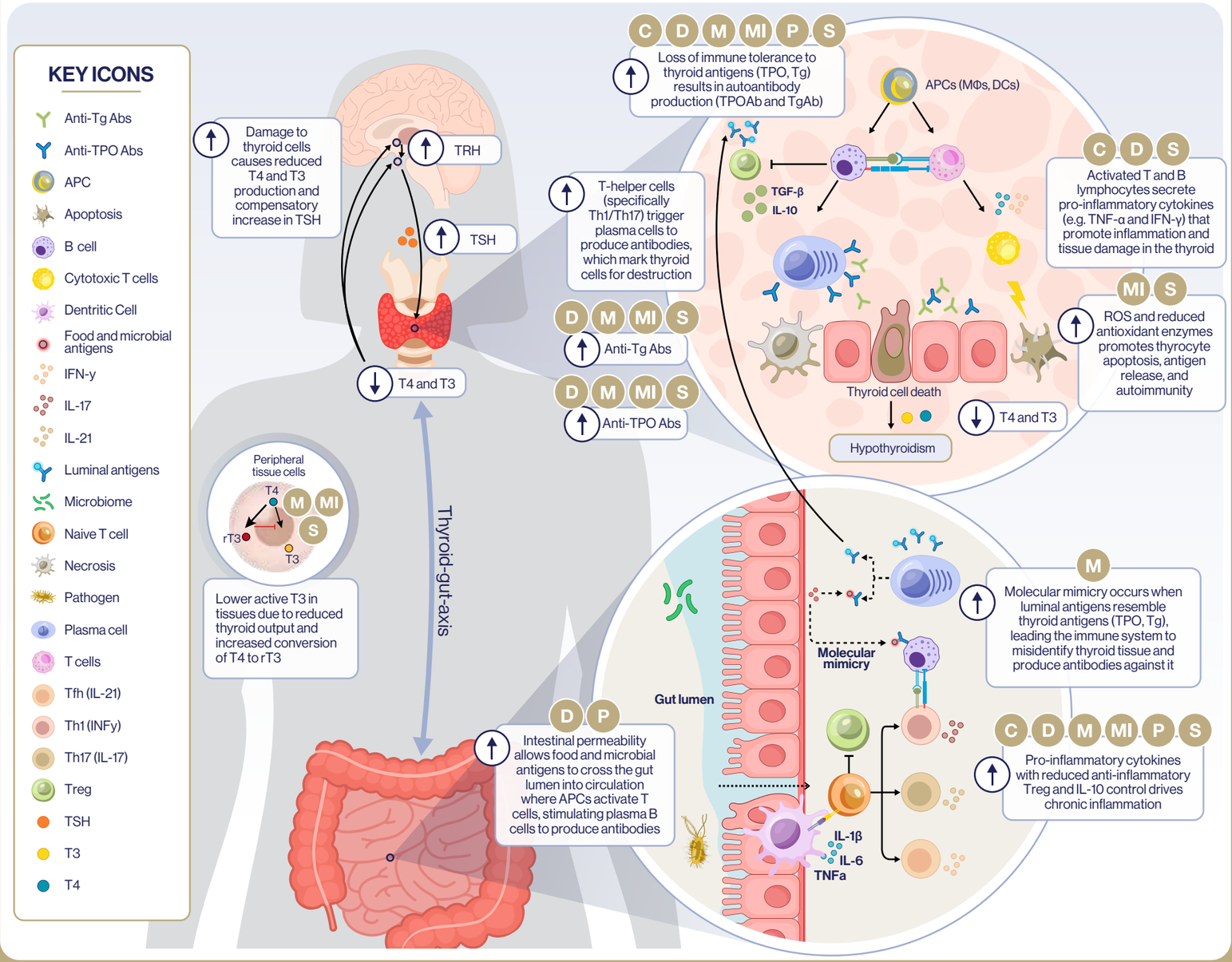
RISK AND CONTRIBUTING FACTORS

- Female sex
- Age - most common in 30-50 yrs
- Genetic predisposition
- Familial autoimmunity
- Polyautoimmunity
- Overweight/obesity
- Environmental factors, including pollution
- Endocrine-disrupting chemicals
- Smoking/vaping
- Alcohol
- Bacterial and viral infections
- Intestinal dysbiosis
- Increased intestinal permeability (IP)
- Chronic stress
- Postpartum period
- Diet e.g. ultra-processed food and excessive animal/saturated fat intake
- Nitrates and nitrites
- Malnutrition
- Iodine deficiency or excess
- Other micronutrient deficiencies e.g. selenium, iron, copper, magnesium, vitamin B12, vitamin D, zinc
- Certain medications e.g. lithium, interferon-alpha, tyrosine kinase inhibitor[2,7-32]

KEY

APC: antigen-presenting cells; **B cell:** B lymphocyte; **CD:** cluster of differentiation; **DC:** dendritic cell; **HT:** Hashimoto's thyroiditis; **IFNγ:** interferon-gamma; **IL:** interleukin; **IP-10:** interferon gamma-induced protein 10; **MΦ:** macrophages; **ROS:** reactive oxygen species; **rT3:** reverse T3; **T cell:** T lymphocyte; **Tfh:** T follicular helper; **Tg:** thyroglobulin; **TgAb:** thyroglobulin antibodies; **TGF-β:** transforming growth factor-beta; **Th:** T-helper; **TNF-α:** tumour necrosis factor-alpha; **TPO:** thyroid peroxidase; **Treg:** regulatory T cells; **TRH:** thyrotropin-releasing hormone; **TSH:** thyroid-stimulating hormone; **T3:** triiodothyronine; **T4:** tetraiodothyronine

HT pathophysiology and therapeutic interventions[1-71]



THERAPEUTICS

C — CoQ10/Ubiquinol
Antioxidant, anti-inflammatory, immunomodulator
- ATP producer (required for immune regulation)
- Neutralises ROS and NF-κB-driven inflammation
- Regenerates other antioxidants (Vit C and E)
- Regulates immune function e.g. T and B cells[36-43]

D — Vitamin D
Immunomodulator, tolerogenic, anti-inflammatory
- Restores Treg/Th17 and Th1/Th2 balance
- Reduces pro-inflammatory cytokines (e.g. IL-6)
- Induces expression of antimicrobial peptides
- Reduces B cell activation and autoantibody production[2,14,15,23,44-49]

M — Magnesium
Anti-inflammatory, antioxidant, immunomodulator
- Catalyses conversion of T4 to T3
- Regulates target tissue thyroid hormone receptor (THR) sensitivity
- Modulates Th cell activity and immune homeostasis
- Inhibits pro-inflammatory cytokines (e.g. TNF-α)
- Essential for vitamin D synthesis and distribution[17,23,32,50-54]

MI — Myo-inositol
Cytoprotective, immunomodulator, anti-inflammatory
- Required for T4 to T3 synthesis
- Regulates TSH and TSH receptor signalling
- Inositol lipids regulate T-, B-, and Treg-cell behaviour
- Reduces IP-10 and pro-inflammatory cytokines[7,55-61]

P — Probiotics
Antimicrobial, nutritive, immunomodulator
- Improves nutrient absorption and bioavailability
- Increases SCFAs and Tregs
- Improves Treg/Th17 balance
- Strengthens tight junctions and reduces IP, antigen translocation, and excessive inflammasome activation
- Lower *Bifidobacteria, Lactobacilli* and *Prevotella* and higher *Bacteroides* and *Enterococcus* are commonly observed with HT[62-71]

S — Selenium
Immunomodulator, antioxidant, anti-inflammatory
- Reduces pro-inflammatory Th1/Th17 cytokines
- Reduces ROS, lipid peroxidation and oxidative damage
- Crucial for T3 synthesis (T4 to T3 conversion)
- Stabilises T3 levels and rT3 production[10,15,29,31-35,62]

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