NMDC221 Session 26: Endocrine System Disease Part I
Recommended reading

Topic Summary

Endocrine System Disease: Part I

- Principles and considerations in nutritional medicine management of the endocrine system - Thyroid
- Review anatomy & physiology of the endocrine system

Nutritional management of specific endocrine conditions with consideration of drug-nutrient interactions

- Hypothyroidism
- Hyperthyroidism
Endocrine System

- Hypothalamus
- Pituitary gland
- Pineal gland
- Thyroid gland
- Trachea
- Thymus
- Heart
- Stomach
- Kidney
- Liver
- Adrenal glands
- Pancreas
- Small intestine
- Uterus
- Ovary
- Testes
- Scrotum
- Skin
- Lung

PARATHYROID GLANDS (behind thyroid glands)
Principles of feedback control in the endocrine system

Different mechanisms of cell signaling

- Intracrine
- Autocrine
- Paracrine
- Endocrine
- Neuroendocrine

Source: https://www.ncbi.nlm.nih.gov/books/NBK20/
Chemical structure of hormones and their synthesis

Hormonal Activation & Protein Synthesis

1. Lipid-soluble hormone diffuses into cell
2. Activated receptor–hormone complex alters gene expression
3. Newly formed mRNA directs synthesis of specific proteins on ribosomes
4. New proteins alter cell's activity

(Tortora & Grabowski, 2003)
THYROID GLAND
Autoimmunity and molecular mimicry

Molecular Mimicry

Antibodies bind to the specific protein sequences of antigens. While gluten, casein, and your own tissues may all be different, they share some of the same protein sequences. A cross reaction occurs when your immune system cannot distinguish between these molecules.
Mimicry

- Each time your body is exposed to a possible trigger, your immune system memorizes its structure, specifically its protein sequence, so that it can develop the perfect defense to that pathogen and recognize it in the future.

- However, our immune system’s recognition system isn’t perfect; as long as a molecule’s structure and protein sequences are similar enough, the immune system can be fooled into attacking look-a-like molecules that are actually your body’s tissue, causing autoimmune disease. Unfortunately for the thyroid, it has two common risks - gluten and casein made worse by leaky gut.
Thyroid Gland

(Tortora & Grabowski, 2003)
Thyroid factor in control of:

- Basal metabolic rate (including respiratory rate, heart rate and body temperature).
- Up-regulates β receptor sensitivity to catecholamines (adrenalin & noradrenalin)
- Stimulate protein synthesis, triglyceride breakdown and glucose metabolism for cellular metabolism
- Enhance cholesterol excretion in bile
- Growth & development

(Tortora & Grabowski, 2003)
## Thyroid Hormone Levels

<table>
<thead>
<tr>
<th>Condition</th>
<th>Concentrations of binding protein</th>
<th>Total Plasma T4, T3, RT3</th>
<th>Free Plasma T4, T3, RT3</th>
<th>Plasma TSH</th>
<th>Clinical State</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperthyroidism</td>
<td>Normal</td>
<td>High</td>
<td>High</td>
<td>Low</td>
<td>Hyper-thyroid</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>Normal</td>
<td>Low</td>
<td>Low</td>
<td>High</td>
<td>Hypo-thyroid</td>
</tr>
<tr>
<td>Estrogens, methadone, heroin, major tranquillisers, clofibrate</td>
<td>High</td>
<td>High</td>
<td>Normal</td>
<td>Normal</td>
<td>Euthyroid</td>
</tr>
<tr>
<td>Glucocorticoids, androgens, danazol, l-asparaginase</td>
<td>Low</td>
<td>Low</td>
<td>Normal</td>
<td>Normal</td>
<td>Euthyroid</td>
</tr>
</tbody>
</table>

(Pizzorno & Murray, 2006)
Hyperthyroidism
# Hyperthyroidism

Hyperthyroidism is the overproduction of thyroid hormones

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Serum Investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accelerated metabolism, hyperactivity</td>
<td>↓ TSH (0.4 – 5.0 mIU/L)</td>
</tr>
<tr>
<td>Nervousness, irritability, weakness</td>
<td>↑ T3 (triiodothyronine)</td>
</tr>
<tr>
<td>Appetite with weight loss, thirst</td>
<td>↑ T4 or normal levels (thyroxine)</td>
</tr>
<tr>
<td>Increased body temperature, vasodilation, sweating, heat intolerance</td>
<td>(Kumar &amp; Clark, 2009)</td>
</tr>
<tr>
<td>Tachycardia, full pulse</td>
<td>Serum tests require TSH, T3 and T4 levels to be tested, not just TSH alone.</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td></td>
</tr>
<tr>
<td>Frequent bowel motions</td>
<td></td>
</tr>
<tr>
<td>Reproductive hormone disturbances</td>
<td></td>
</tr>
<tr>
<td>Muscle weakness, tremors, stiffness</td>
<td></td>
</tr>
<tr>
<td>Exophthalmos &amp; stare (Grave’s disease)</td>
<td></td>
</tr>
</tbody>
</table>

(Kumar & Clark, 2009)
Hyperthyroidism

Graves’ Disease

- Auto-immune (most common hyperthyroid condition) where antibodies mimic TSH activity. As this is not regulated by normal negative feedback mechanisms, the thyroid gland is constantly stimulated to grow & produce hormones. Uncontrolled production of TSH leads to the characteristic presentation of goitre.

- Exophthalmos (protruding eyes – oedema behind eyes) is the characteristic of Grave’s disease.

- Other common symptoms of hyperthyroidism present

- Pathology presents with TSH receptor autoantibodies (Habra & Sarlis, 2005; Kumar & Clark, 2009)
Hyberthyroidism

Sub Acute Thyroiditis

- Symptoms usually follow URTI of viral origin
- Symptoms include asthenia, malaise, pain over thyroid or referred to lower jaw, ear, or occiput.
- Less common, acute onset with severe pain over thyroid, fever, sometime thyrotoxicosis.
- Symptoms may continue (smoulder) for weeks/months & eventually subside with return to normal thyroid function.

(Kumar & Clark, 2009)
Hyperthyroidism

Female Reproductive Hormones

- Sudden changes to female reproductive hormones (menarche, pregnancy, oral contraceptive use) can cause a transient rise in thyroxine-binding globulin (TBG) and TSH.
- This in turn increases total T4 and T3 levels.
- This can result in thyroid enlargement and hyperthyroid symptoms.

(Dal Maso et al. 2009)
Hyperthyroidism

Nutritional Considerations

Iodine
- Kelp and iodine containing supplements and drugs (iodinated contrast agents, amiodarone) are substrates for thyroxine synthesis. This is usually evident in individuals that present with sub-acute hyperthyroidism or an underlying goitre
  (Habra & Sarlis, 2005; Kumar & Clark, 2009)

Tyrosine
- Avoid Tyrosine in excessive concentration – a substrate for Thyroxine (T4) production
  (Pizzorno & Murray, 2006)
Hyperthyroidism

Treatment Aims

- Reduce high cortisol;
  - exogenous – food intolerances, stimulants
  - endogenous – immune dysregulation, imbalances in neurotransmitters, inflammatory markers)

- Support of overactive metabolic pathways, organs and systems through nutrient-dense foods, antioxidants, energy-production cofactors and other specific nutrients as indicated e.g. calcium

- Support co-morbid organ dysfunction that may present

- Minimize hyper-stimulation to thyroid

(Sarris & Wardle, 2010)
Hyperthyroidism

Treatment Aims

- Support the breakdown of T4 via phase II detoxification (glucuronidation)
- Assess protein status \[IW \times 0.9 \times EF \ (1.1-1.5)\]
- Assess fat status:
  - High consumption of (W3) EFA rich food, moderate MUFA and PUFA and decreased SFA’s.
- Assess carbohydrate status
- Assess required kilojoule intake given BMR and REE
  \(\text{(Pizzorno & Murray, 2006; Gropper et al. 2009)}\)
# Hyperthyroidism

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>5000-10000 iu</td>
<td>Deficiency alters the pituitary-thyroid axis and effects peripheral thyroid hormone metabolism.</td>
</tr>
<tr>
<td>Vitamin B6</td>
<td>5-50mg</td>
<td>Hormone synthesis. Reduces androgenic &amp; estrogenic transcription response</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>2-4gm</td>
<td>Linked to increased lipid peroxidation in skeletal &amp; cardiac muscle</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>100-1,000 iu</td>
<td>Deficiency presents in hyperthyroidism. Found to attenuate alterations to heart rate reduce free radical overexpression in the liver</td>
</tr>
<tr>
<td>Coenzyme Q10</td>
<td>100-300mg</td>
<td>Deficiency presents in hyperthyroidism</td>
</tr>
</tbody>
</table>

(Coulston, 2001; Zimmerman et al. 2004; Osiecki, 2006; Venditti & Di Meo, 2006; Subudhi & Chainy, 2010)
# Hyperthyroidism

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<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
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<tbody>
<tr>
<td>Manganese</td>
<td>2-50mg</td>
<td>Aids in the production of manganese superoxide dismutase.</td>
</tr>
<tr>
<td>Copper</td>
<td>2-5mg</td>
<td></td>
</tr>
<tr>
<td>Magnesium</td>
<td>300-1000mg</td>
<td>Homeostasis of calcium, regulation of body temperature</td>
</tr>
</tbody>
</table>

(Coulston, 2001; Habra & Sarlis, 2005; Thiel & Fowkes, 2005; Pizzorno & Murray, 2006; Venditti & Di Meo, 2006)
# Hyperthyroidism

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<th>Therapeutic Actions</th>
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<tr>
<td>Glycine</td>
<td>4-30g</td>
<td>Supports phase II glucuronidation pathway for T4 metabolism</td>
</tr>
<tr>
<td>Taurine</td>
<td>250-2000mg</td>
<td></td>
</tr>
<tr>
<td>Cysteine</td>
<td>200-500mg</td>
<td></td>
</tr>
<tr>
<td>Glutathione</td>
<td>100-500mg</td>
<td>Deficiency in liver and muscle were present in hyperthyroidism</td>
</tr>
<tr>
<td>Turmeric</td>
<td>Inhibits LT, TH &amp; PG production. Found to attenuate overexpression of free radical in the liver (mitochondria) due to the overstimulation by T4</td>
<td></td>
</tr>
<tr>
<td>Indoles</td>
<td>Induce protective enzymes that deactivate estrogen</td>
<td></td>
</tr>
</tbody>
</table>

(Coulston, 2001; Pizzorno & Murray, 2006; Venditti & Di Meo, 2006; Subudhi & Chainy, 2010)
## Hyperthyroidism

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Side Effects</th>
<th>Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anti-thyroid Drugs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbimazole</td>
<td>Blocks the organic binding of iodine through inhibition of iodination of tyrosine. Has some affect on peroxidase - required catalyst in thyroxine synthesis.</td>
<td>Nausea, headache, bone marrow depression, hematological disturbances, myopathy, arthralgia, pruritus, urticaria, alopecia, hepatic effects.</td>
<td>None listed</td>
</tr>
<tr>
<td><strong>Anti-thyroid Drugs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propylthiouracil</td>
<td>Blocks peripheral conversion of thyroxine ((T_4)) to triiodothyronine ((T_3)) by inhibiting incorporation of iodide into tyrosine.</td>
<td>Haematopoietic effects, hypothyroidism, thyrotoxicosis, itching.</td>
<td>None listed</td>
</tr>
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(Bryant & Knights, 2011)
# Hyperthyroidism

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<tr>
<td>Radioactive Iodine</td>
<td>Oral administration of radioactive iodine that is taken up by the thyroid that irreversibly damages thyroid nodule function.</td>
<td>Transient damage to mouth, throat &amp; GIT, Nausea, vomiting, Irritability, fatigue, Symptoms pass within 2-4 weeks</td>
<td>None listed</td>
</tr>
</tbody>
</table>

(Bryant & Knights, 2011)
## Hyperthyroidism

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<tr>
<th>Drug</th>
<th>General Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Contraceptive Pill</td>
<td>May cause a transient rise in thyroxine-binding globulin (TBG) and TSH linked to thyroid enlargement and hyperthyroidism.</td>
</tr>
<tr>
<td>Tyrosine</td>
<td>Tyrosine in excessive concentration – a substrate for Thyroxine (T4) production</td>
</tr>
<tr>
<td>Iodine</td>
<td>Kelp, iodine containing supplements and drugs (iodinated contrast agents, amiodarone) can increase T4 synthesis. Responses are greater in those individuals that present with sub-acute hyperthyroidism or an underlying goitre.</td>
</tr>
</tbody>
</table>

(Habra & Sarlis, 2005; Kumar & Clark, 2009; Pizzorno & Murray, 2006; Dal Maso et al. 2009)
Hypothyroidism
Hypothyroidism

Under Active Thyroid Function

Precursors to hypothyroidism:

- Surgical intervention of the thyroid gland (hyperthyroid)
- Radioactive iodine or radiation (hyperthyroid)
- Iodine deficiency
- Environmental toxicity
- Auto-immune disorders

(Habra & Sarlis, 2005; Pizzorno & Murray, 2006)
Hypothyroidism

Primary causes;
- Iodine deficiency
- Autoimmune disease: Hashimoto’s thyroiditis
- Iatrogenic (radiation, surgery and drugs)

Main secondary cause;
- Hypopituitarism

(Hywood, 2004)
## Hypothyroidism

### Symptoms

- Reduced metabolism – fatigue, cold intolerance, weight gain/ inability to lose weight
- Depression, psychosis/ dementia, poor memory,
- Constipation
- Reproductive hormone dysfunction, poor libido
- Goitre (Hashimoto’s thyroiditis, Iodine deficiency)
- Hypotension, bradycardia, anaemia
- Dry brittle hair, dry, coarse skin, loss of eyebrows
- Myalgia, muscle weakness, arthralgia

### Serum Pathology

<table>
<thead>
<tr>
<th></th>
<th>Serum Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑</td>
<td>TSH (0.4 – 5.0 mIU/L)</td>
</tr>
<tr>
<td>↓</td>
<td>T3 (triiodothyronine)</td>
</tr>
<tr>
<td>↓</td>
<td>T4 (thyroxine)</td>
</tr>
</tbody>
</table>

(Kumar & Clark, 2009)

TSH, T3 and T4 should be measured, not just TSH or TSH and T4 as T3 can be elevated with normal T4.
Hypothyroidism

Hashimoto’s Disease

- An autoimmune antibody presentation to thyroid peroxidase enzyme (responsible for liberating iodine for combination with tyrosine).
- Characteristic goitre presentation is due low levels of T4 & T3 not negatively feeding back to the pituitary to attenuate TSH production. This high level of TSH causes swelling of the thyroid tissue.
- Usual characteristics of hypothyroidism present.

(Kumar & Clark, 2009)
Hypothyroidism

Iodine Bioavailability Factors

Goitrogens

- Block the binding of iodine to thyroglobulin. Food sources include raw brassica vegetables, tobacco, millet, soy and catechins from tea.

  (Gruner T, in: Sarris and Wardle 2015)
Hypothyroidism

Iodine Bioavailability Factors

Lithium

- “Lithium therapy can cause hypothyroidism in 5–15% of patients and goitre in as many as 37%.”

Iodine-containing drugs - iodinated contrast agents, amiodarone, and iodine-containing supplements

- High levels of iodine containing drugs can cause transient suppression of thyroid hormones.

(Habra & Sarlis, 2005)
Hypothyroidism

Iodine Bioavailability Factors

Tricyclic Antidepressants

Can alter:

- iodine uptake by thyroid cells
- form non-bioavailable complexes with inorganic iodine making it unavailable for TH synthesis
- inhibit the activity of thyroid peroxidase and enhance the conversion of T4 to T3—or to rT3—by stimulation of deiodinases

(Habra & Sarlis, 2005)
Hypothyroidism

Other Factors

Increased Oxidative Stress
- Poor liver and peripheral metabolism of ethanol can result in enhanced free radical generation.
- This has been found to cause “…HPT axis dysfunction and peripheral thyroid hormone reduction.”
  (Valeix et al. 2008)

Saturated Fats
- Saturated fats decrease T3 levels and promote body fat accumulation.
  (Pizzorno & Murray 2006)
Hypothyroidism

Other Factors
Elevated IL-6
- Elevated serum IL-6 concentrations were also correlated with...alterations in TH levels..." (Habra & Sarlis, 2005).
- IL-6 has been found to “...inhibit 5’-deiodinase.” (Sarris & Wardle, 2010, p. 329)

Smoking
- Smoking can decrease thyroid hormone secretion and its peripheral circulation.  
  (Pizzorno & Murray, 2006)
Hypothyroidism

Other Factors

Hypercortisolism

Excess cortisol has been found to partially suppress TSH secretion.

Elevated cortisol leads to:

- Reduction in active T3 and increases in reverse T3
- Reduced peripheral hormone metabolism by inhibition of the enzyme 5’-deiodinase
- Potential to present with autoimmune disease, such as Hashimoto’s disease

(Hywood, 2004; Habra & Sarlis, 2005)
Hypothyroidism

Other Factors
Manganese

- TSH binding to thyroid plasma membranes is strongly inhibited by manganese.

- Through negative feedback, the increased circulation of TSH would inhibit further TSH from the pituitary resulting in a subsequent reduction in T4 production.

- Manganese has been linked to changes in regulating the deiodinase enzymes through unknown mechanism

(Soldin & Aschner, 2007)
Hypothyroidism

Alcohol

- Valeix et al. (2008) found that moderate alcohol consumption levels of 3 standard drinks for males and 1.5 standard drinks for females statistically increased the risk for thyroid volume enlargement.
- This was present in individuals that were not iodine deficient.

Mercury

- Environmental mercury vapour exposure is linked to reduced T3 and increased reverse T3. This is exacerbated with iodine deficiency.

(Sarris & Wardle, 2010)
Hypothyroidism

Treatment Aims

- Ensure precursors (tyrosine, iodine) and cofactors (selenium, zinc) for thyroid hormone production are replete.
- Review of food intolerances/gut dysfunction – gluten, candida
- Treat autoimmune components
- Support optimal energy production
- Support co-morbid organ dysfunction that may present
- Assess protein status \[\text{IW} \times 0.9 \times \text{EF (1.1-1.5)}\]
- Assess fat status:
  - High consumption of (W3) EFA rich food, moderate MUFA and PUFA and decreased SFA’s.

(Groper et al. 2009; Sarris & Wardle, 2010)
Hypothyroidism

Treatment Aims

- Reduce high cortisol;
  - exogenous – food intolerances, stimulants
  - endogenous – immune dysregulation, imbalances in neurotransmitters, inflammatory markers
- Increase thyroid hormone precursors and cofactors in production and conversion
- Assess and support dysfunction caused by drug usage
- Support of antioxidant defense system

(Sarris & Wardle, 2010)
# Hypothyroidism

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tyrosine</td>
<td>39mg/kg</td>
<td>Substrate for T4</td>
</tr>
<tr>
<td>Garlic, Ginger</td>
<td></td>
<td>Inhibits prostaglandin, thromboxane and leukotriene synthesis.</td>
</tr>
<tr>
<td>Turmeric</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quercetin, Bromelain</td>
<td></td>
<td>COX – 2 Inhibitor (Bradykinin inhibitor) Proteolytic enzyme</td>
</tr>
<tr>
<td>Omega 3</td>
<td>2-6gm</td>
<td>Reduces inflammation and stimulate the immune system, cell membrane fluidity and integrity</td>
</tr>
<tr>
<td>Iodine</td>
<td>100-1000mcg</td>
<td>Substrate for T4. Synthesis of thyroid hormones</td>
</tr>
</tbody>
</table>

(Thiel & Fowkes, 2005; Pizzorno & Murray, 2006; Braun & Cohen, 2010)
## Hypothyroidism

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<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>1000-1200mg</td>
<td>Calcitonin &amp; thyroid hormone release. Regulation of cell division and hormone secretion.</td>
</tr>
<tr>
<td>Magnesium</td>
<td>500-600mg</td>
<td>Homeostasis of calcium. Regulation of body temperature</td>
</tr>
<tr>
<td>Iron</td>
<td>15-50mg</td>
<td>Essential conversion co-factors in the phenylalanine to tyrosine for T4 &amp; T3 production.</td>
</tr>
<tr>
<td>Selenium</td>
<td>100-200mcg</td>
<td>Component of deiodinase enzyme for T4 to T3 and rT3 conversion. Hypothyroidism presents with increased free radicals and increased selenium requirements</td>
</tr>
<tr>
<td>Zinc</td>
<td>25-50mg</td>
<td>Activates deiodinase which catalyzes the conversion of T4 to T3 and rT3. Deficiency presents with reduced hypothalamic TRH.</td>
</tr>
</tbody>
</table>

(Coulston, 2001; Kohlmeier, 2003; Habra & Sarlis, 2005; Thiel & Fowkes, 2005; Osiecki, 2006; Pizzorno & Murray, 2006; Thiel & Fowkes, 2005; Sarris & Wardle, 2010)
Hypothyroidism

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<thead>
<tr>
<th>Nutrient</th>
<th>Dosage</th>
<th>Therapeutic Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>5000iu</td>
<td>Deficiency can exacerbate goitre presentation in iodine deficient hypothyroidism</td>
</tr>
<tr>
<td>Vitamin B6</td>
<td>5- 50mg</td>
<td>Hormone synthesis. Reduces androgenic &amp; estrogenic transcription response. Essential conversion co-factors in the phenylalanine to tyrosine for T4 &amp; T3 production.</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>1-2gm</td>
<td>Antioxidant. Essential conversion co-factors in the phenylalanine to tyrosine for T4 &amp; T3 production.</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>1000-10000iu</td>
<td>Regulation of PTH &amp; regulation of growth of parathyroid. Deficiency presents, exacerbated by thyroxine use</td>
</tr>
<tr>
<td>Folate</td>
<td>400-600mcg</td>
<td>Deficiency presents in hypothyroidism causing subsequent increases in homocysteine levels</td>
</tr>
</tbody>
</table>

(Barbé et al. 2001; Coulston, 2001; Kohlmeier, 2003; Zimmerman et al. 2004; Osiecki ,2006; Thiel & Fowkes, 2007)
Hypothyroidism

Therapeutic Actions

Iodine

- Iodine is required for T4 and T3 production. Seaweed sources are a good bioavailable source of iodine, and countries with traditionally low intakes of seaweed may present commonly with deficiencies.

  (Thiel & Fowkes, 2005)

Zinc

- “Circulating triiodothyronine and thyroxine are decreased in zinc deficiency, as is the hypothalamic thyroid-releasing hormone”

  (Thiel & Fowkes, 2005, p. 811)
Hypothyroidism

Therapeutic Actions

Selenium

- Selenium is an integral component of peripheral conversion enzymes for T4 to T3 or reverse (iodothyronine deiodinases). Deficiency of that mineral will exacerbate hypothyroidism.

- Hypothyroidism is linked to increased free radical presentation in the liver, depleting this mineral as it is funnelled into glutathione-peroxidase formation.

  (Thiel & Fowkes, 2005; Habra & Sarlis, 2005)
Hypothyroidism

Therapeutic Actions
Phenylalanine / Tyrosine

○ Tyrosine is a non-essential amino acid (can be converted from phenylalanine) that is required for the production of thyroid and adrenal hormones.

(Thiel & Fowkes, 2005)

○ Deficiencies in phenylalanine or conversion co-factors (biopterin, vitamin B6, vitamin C & iron) can reduce the production of thyroxin & triiodothyronin.

(Kohlmeier, 2003)
Hypothyroidism

Therapeutic Actions

Folate

- “...transient increase in both plasma homocysteine and serum cholesterol during short-term iatrogenic hypothyroidism...(22%)...which may confer an increased cardiovascular risk.” (Barbé et al. 2001, p. 1845)

- Folate, not vitamin B12 was implicated in this alteration due to alterations in enzymatic control and alterations in renal clearance.

  (Barbé et al. 2001)
Hypothyroidism

Therapeutic Actions

Vitamin A

- Mild to moderate vitamin A deficiency increases the risk of goitre presentation in iodine deficiency disorders.

- The “…data suggest that vitamin A supplementation improves the efficacy of iodine supplementation to control goitre in children with moderate vitamin A deficiency.”

(Zimmerman et al. 2004, p. 5442)
# Hypothyroidism

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Side Effects</th>
<th>Interactions</th>
</tr>
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<tbody>
<tr>
<td>Thyroxine (T4)</td>
<td>Synthetic thyroid hormones. T4 is metabolized to T3 in peripheral tissue. T3 is 10X more biologically active than T4 &amp; doesn’t require conversion.</td>
<td>High dose = palpitations, nervousness, insomnia, tremor, reduced bone density. Will resolve with reduced dosage. T3 can be a cardio toxic agent</td>
<td>Calcium, Iron, Magnesium, Zinc: form insoluble complexes with the drug. Separate dose by 2 hours. Mineral deficiencies are linked to poor thyroid hormone production or peripheral conversion. Tyrosine: Additive effects as tyrosine is a precursor to thyroid hormone Thiocyanates &amp; Goitrogens: Inhibits the body’s absorption or utilization of iodine, increasing the requirement for synthetic T4 &amp; T3 Saturated Fats: decrease T3 levels and promote body fat accumulation.</td>
</tr>
<tr>
<td>Liothyronine (T3)</td>
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Hypothyroidism

Thyroxine & Liothyronine
Lithium, Iodine containing drugs / supplements, Tricyclic antidepressants

- Concurrent utilization may increase the requirement for synthetic T4 & T3

Ethanol, high free radicals, IL-6, smoking, manganese excess and high cortisol

- Reduce peripheral thyroid hormone conversion, altering the requirements for synthetic T4.

(Habra & Sarlis, 2005; Pizzorno & Murray, 2006; Soldin & Aschner, 2007; Valeix et al. 2008; Sarris & Wardle, 2010)
References


References


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References

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