Nineveh Daniel 107060

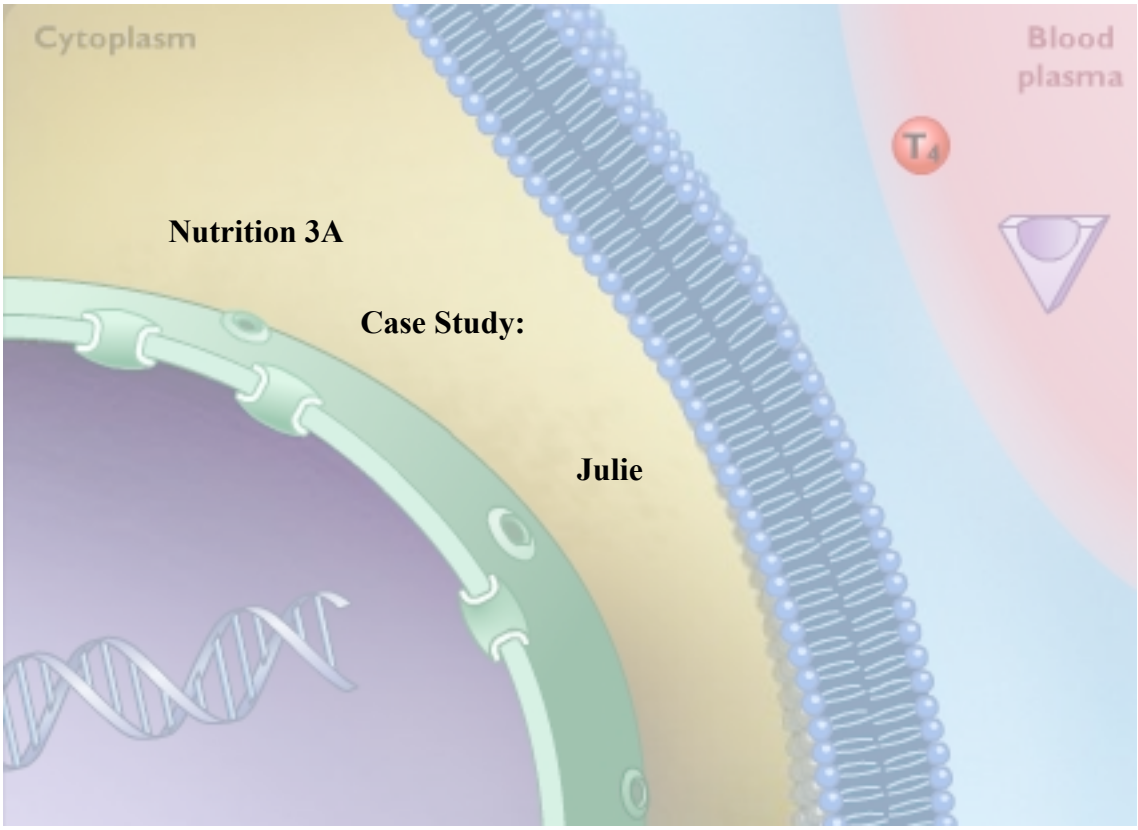

Figure on page 1 taken from http://www.sutree.com/upload/fdjggerinfrivtwmiwbhy/captured.jpg

ABBREVIATIONS

BMI	Body-mass index
CD	Coeliac disease
CVD	Cardiovascular disease
EFAs	Essential fatty acids
FBE	Full blood examination
FSH	Follicle-stimulating hormone
GI	Glycemic index
HDL	High density lipoproteins
HPA	Hypothalamic-Pituitary-Adrenal
HPT	Hypothalamic-Pituitary-Thyroid
IR	Insulin resistance
LDL	Low density lipoproteins
LH	Luteinising hormone
LFTs	Liver function tests
Mg	Magnesium
n-3	Omega-3 EFAs
n-6	Omega-6 EFAs
n-6:n-3	Omega-6-to-Omega-3 ratio
T3	Triiodothyronine
T4	Thyroxine
Th1	T-helper 1 lymphocytes
TDEI	Total daily energy intake
TSH	Thyroid stimulating hormone
Zn	Zinc

Case study

Julie is a 43 yo advertising executive. She presents complaining of increasing fatigue (all day, but can be worse just after meals), poor memory and concentration, flat mood, and steady weight gain over the last few years (despite trying, she has had difficulty loosing it). She is using her bowels 2-3 times a week, and regularly experiences bloating and excessive flatulence. She does not exercise.

Physical examination findings:

Height – 157cm

Weight – 93kg

Waist hip ratio - 1.09

BP – 110/80 mmHg

Zinc (**Zn**) tally test – describes taste as like flat mineral water

Skin – pale complexion, dry and scaly, numerous red stretch marks

Family history:

Mother – Grave's disease, osteoporosis, vitiligo

Father – type 2 diabetes, stroke (died aged 58)

24 hour recall diet:

B/fast – Bowl of Special K with skinny milk topped with banana

Mid am – rice cakes x 4 with tomato.

Lunch – sandwich (sour dough, margarine) – salad (tomato, lettuce, grated carrot, onion, avocado) and cheese.

Mid pm – 50g bag of lollies (eaten through the afternoon)

Dinner – pasta (fresh beef ravioli) with vegetables (onion, corn, broccoli) and tomato based sauce.

Drinks - water 4-6 glasses throughout the day, coffee x2 (equal x1, skinny milk), can of diet coke x1, orange juice x1, wine 2-3 glasses most nights

1. CASE DIAGNOSIS

MEDICAL DIAGNOSTIC APPROACH

Differential diagnosis

Fatigue is a symptom of many diseases, and may be differentiated into physiological and psychological causes (Murtagh 2003, pp.820-1). A careful differential diagnosis of the causes of increasing tiredness in this case is therefore warranted (Murtagh 2003, pp.820-1):

- *psychogenic*: e.g. stress, anxiety (Murtagh 2003, pp.820-4);
- *organic*: e.g. sleep-related disorders, food intolerance, celiac disease (**CD**), nutritional deficiency, endocrine (hyper- and hypo-; Murtagh 2003, pp.820-2; Desailloud & Hober 2009); menopause (Murtagh 2003, p.822); or
- *unknown*: chronic fatigue syndrome, fibromyalgia (Murtagh 2003, p.821).

Similarly, possible causes for weight gain must also be considered, such as:

- simple exogenous obesity;
- endocrine disorders (e.g. hypothyroidism);
- drugs; or
- depression (Murtagh 2003, pp.860-1).

Causes for infrequent bowel movements, worse after eating, excessive bloating/flatulence also need to be ruled out:

- 'functional' constipation;
- hypothyroidism;
- laxative abuse;
- malabsorption, irritable bowel syndrome (Murtagh 2003, pp.437-9, 518); or
- chemical sensitivities (Allen 2005, p.35).

Medical probability diagnosis

Autoimmune hypothyroidism[1]: based on supporting evidence outlined below (Murtagh 2003, p.822).

Supporting evidence

Signs and Symptoms

- sex, age (McDermott 1998, p.212);
- increasing fatigue, pale skin (MedlinePlus 2008);
- mental/physical slowing, depression, infrequent bowel movements (Murtagh 2003, p.223);
- steady weight gain, mild obesity (body-mass-index [**BMI**] 37.7; Australian Better Health Initiative 2008; Murtagh 2003, p.223); and
- dry scaly skin (Berkow & Fletcher 1992, p.1080).

Genetic links

Julie shows strong genetic links to back up the probable diagnosis, as indicated by her mother's autoimmune disorders (Grave disease and vitiligo; Berkow & Fletcher 1992, pp.1075, 1083, 2450).[2] A parent with an autoimmune disease increases the risk of their offspring having a similar tendency (Shomon 2005 cited in Thyroid Info 2009; Berkow & Fletcher 1992, p.1083).[3]

[1] The thyroid gland has failed to produce enough thyroid hormone leading to persistent low levels of circulating thyroid hormones (MedlinePlus 2009)
[2] Vitiligo has been associated with thyroid dysfunction (Berkow & Fletcher 1992, p.2450)
[3] Studies have indicated that mothers with Grave disease may lead to an impaired fetal hypothalamic-pituitary-thyroid axis (Higuchi et al 2005, p.623)

Secondary probable diagnosis

Hypothyroidism as well as obesity with non-specific GI symptoms (e.g. abdominal pain, bloating) is becoming an increasing face of CD (Selby & Darke 2008, pp.25-31). Therefore, in addition to the probable diagnosis, Julie's presentation appears to be consistent with possible malabsorption (Murtagh 2003, pp.439, 1243) such as CD (Selby & Darke 2008, pp.25-31) or gut permeability/dysbiosis as indicated by bloating/excessive flatulence, worse after meals, pale dry scaly skin (e.g. dermatitis herpetiformis; The University of Maryland Medical Center 2009) and fatigue, cognitive deficit (Allen 2005, p.35). This therefore requires further investigation with appropriate laboratory tests.

Additional probable diagnoses

Julie is possibly also perimenopausal, which may produce similar symptoms to hypothyroidism (Better Health Channel 2008), or contribute/trigger her hypothyroid symptoms (Chahal & Drake 2007, p.176). She should therefore be tested in this context (Berg 2004, p.3).

In addition to her waist-hip-ratio and BMI figures, Julie's paternal genetics suggest a strong tendency to type 2 diabetes and cardiovascular disease (**CVD**) so there may also be a possibility that her probable diagnosis is complicated further by insulin resistance (**IR**; Schumm-Draeger 2006, p.47).

NATUROPATHIC OR MULTI-FACTORIAL ANALYSIS

Predisposing (irreversible) causes

Genetics: inherited tendencies

'Inherited defects' (Priest & Priest 1982, pp.41-3) or genetic links indicated by a family history of Grave disease/vitiligo, suggest Julie has a potential/predisposition to developing dysfunctional immunity (Priest & Priest 1982, pp.41-3). Similarly, Julie is also predisposed to sugar dysregulation and CVD (Geissler & Powers 2000, pp.364, 403, 405). The paternal inherited tendencies may now act as triggers, 'switching on' Julie's predisposition to dysfunctional immunity (Berkow & Fletcher 1992, pp.1083, 1109).

Julie may also be peri-menopausal (Murtagh 2003, p.1008) so that gradual hormonal changes over time have been affecting the hypothalamic-pituitary-thyroid (**HPT**) axis thereby acting as a trigger, switching on Julie's tendency to dysfunctional thyroid (Chahal & Drake 2007, p.176).

Predisposing (reversible) causes

Under-nutrition and over-supply

Assuming Julie followed the same diet for several years, the quality of this diet may have led to deficiencies in some areas and over supply in other areas. The key issues surround the quality and quantity of proteins, carbohydrates, fats, and fibre intake.

Proteins

This category is poorly represented in Julie's diet (small amount from beef ravioli and cheese, probably reflecting less than 10% of total daily energy intake; **TDEI**). Ideally, Julie should be consuming 45g (or at least 20% of TDEI) of good quality protein throughout each day, particularly fish (National Health & Medical Research Council 2003, pp.52, 61, 113) to ensure adequate building blocks for numerous metabolic products, especially hormones such as triiodothyronine (**T3**) and thyroxine (**T4**; Geissler & Powers 2000, pp.144, 151).

Since protein helps avoid hormonal deficiency, obesity (Geissler & Powers 2000, pp.151, 385; Tahara et al 1985, p.1270) and type 2 diabetes (National Health & Medical Research Council 2003, pp.52, 61, 113), lack of protein is triggering genetic predispositions therefore contributing to signs and symptoms (Cordain et al 2005, p.348).

Carbohydrates

Disproportionately high with highly processed, high glycemic index (**GI**) and sugar/salt/additive/preservative items being consumed daily (*'Special K'*, pasta, rice cakes, lollies, *'Equal'*, *'Diet Coke'*). By consuming high-GI carbohydrates, Julie is promoting a rapid return to hunger, which increases energy intake and therefore weight gain (National Health & Medical Research Council 2003, p.35). The high-GI foods are most likely destabilising blood sugar regulation (National Health & Medical Research Council 2003, p.40) thus contributing to thyroid dysfunction (Dimitriadis et al 2006, p.4930). Such a diet that encourages IR, further affects thyroid function by interfering with peripheral T4 to T3 conversion (Dimitriadis et al 2006, p.4930).

Since processing strips many nutrients from carbohydrates, including Zn, magnesium (**Mg**), calcium and chromium, Julie may also have a number of deficiencies (Pitchford 2002, pp.84-8, 162-6, 648) contributing to her numerous symptoms (The University of Sydney n.d.). Because her diet lacks good quality whole-grains and legumes, which also help lower food GI, this further explains her presenting symptoms (The University of Sydney n.d.)[4].

The carbohydrates consumed by Julie may also be producing symptoms of malabsorption (The University of Maryland Medical Center 2009), so that possible intolerance/allergy must be addressed in order to ensure Julie's overall condition improves[5].

[4] Particularly adverse effects of high-GI carbohydrates seen on plasma glucose/insulin (Riccardi & Rivellese 2000, p.143)

[5] It is not unusual for gut hyperpermeability/intestinal dysbiosis issues to be co-morbid factors with auto-immune diseases (Allen 2005, p.35)

Fats

Saturated fats represent a significant proportion, probably of poor quality (and possibly even trans-fats from tomato-based sauces/margarine/lollies), altogether likely representing over 50% of TDEI. In addition, a lack of unsaturated essential fatty acids (**EFAs**), particularly omega-3 (**n-3**), means that a failure of a proper omega-6:omega-3 ratio (**n-6:n-3**) may be contributing various signs and symptoms consistent with dysfunctional immunity (Simopoulos 2008, p.674)[6] and blood sugar dysregulation (Riccardi, Giacco & Rivellese 2004, p.447). For example, studies have shown that consumption of saturated fat (strongly associated with weight gain) deteriorates insulin sensitivity (Riccardi, Giacco & Rivellese 2004, p.447), which is subsequently connected to impaired thyroid function (Galofre et al 2008, p.188).

Fibre

Julie's low (especially soluble)-fibre high fat high-GI carbohydrate diet not only increases her risk of triggering diabetes (National Health & Medical Research Council 2003, p.36)[7], it is a factor in her sustained weight gain (Lindström et al 2006, p.912). Fibre will help reduce energy intake and help to maintain weight in a number of ways (Anderson et al 2009, p.188). Increasing soluble fibre will substantially improve glycemic control, protect against obesity, improve her bowel movements, and significantly enhance immune function (Anderson et al 2009, pp.192-7).

Additional comments

Coffee and wine contributes 6-10% of Julie's energy intake/day, therefore contributing to weight gain (National Health & Medical Research Council 2003, p.160). Excessive alcohol depletes numerous micronutrients such as folate, vitamin A (National Health & Medical Research Council 2003, p.158) and even interferes with dietary intake of EFAs (Kim et al 2007, p.1407), all contributing to her dysfunctional immune system (Wintergerst, Maggini & Hornig 2007, p.301).

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[6] Keeping in mind the need to address n-6:n-3 (Simopoulos 2008, p.674), being obese means Julie needs to reduce total fat intake to 20-25% of TDEI as part of weight management (National Health & Medical Research Council 2003, pp.123-4)

[7] Recent large prospective studies found cereal fibre intake inversely associated with risk of developing type 2 diabetes and the protective effect was even greater when combined with a low total glycemic load (National Health & Medical Research Council 2003, p.36)

Additional predisposing (reversible) causes

- unbalanced energy economics: nutritional insufficiency/overload coupled with lack of exercise means energy intake is unequal to energy output (Bone 2003, pp.33-4);
- lifestyle: lack of exercise, sedentary, high stress job reflected in her greater waist-to-hip ratio, with abdominal adiposity (Geissler & Powers 2000, p.80);
- encumbrance[8]: underlying thyroid dysfunction overlaid with under-functioning and/or an over-loaded digestive system, where delayed gastric emptying prolongs exposure of contents to digestive system (Mills 1993, p.337)

Excitatory factors[9]

- stress (Tsatsoulis 2006, p.382; Chrousos 2007, p.132);
- possible malabsorption/food allergy issue (Murtagh 2003, pp.821-2), may lead to gastrointestinal dysfunction, imbalanced immunologic function via intestinal hyperpermeability, thereby triggering a tendency to immune dysfunction (Hanaway 2006, pp.53, 55; Bosi et al 2006, p.2824);
- under-nutrition/oversupply (Wintergerst, Maggini & Hornig 2007, p.301).

Sustaining factors[10]

- altered HPT function (Chahal & Drake 2007, pp.176-8);
- altered digestive function (Malik & Hodgson 2002, p.561), including mucosal integrity (Hanaway 2006, p.55);
- perimenopause-associated endocrine changes (Chahal & Drake 2007, pp.176-8); and
- obesity-related functional/structural changes (Syed et al 2009, p.36; Mills & Bone 2007, p.128).

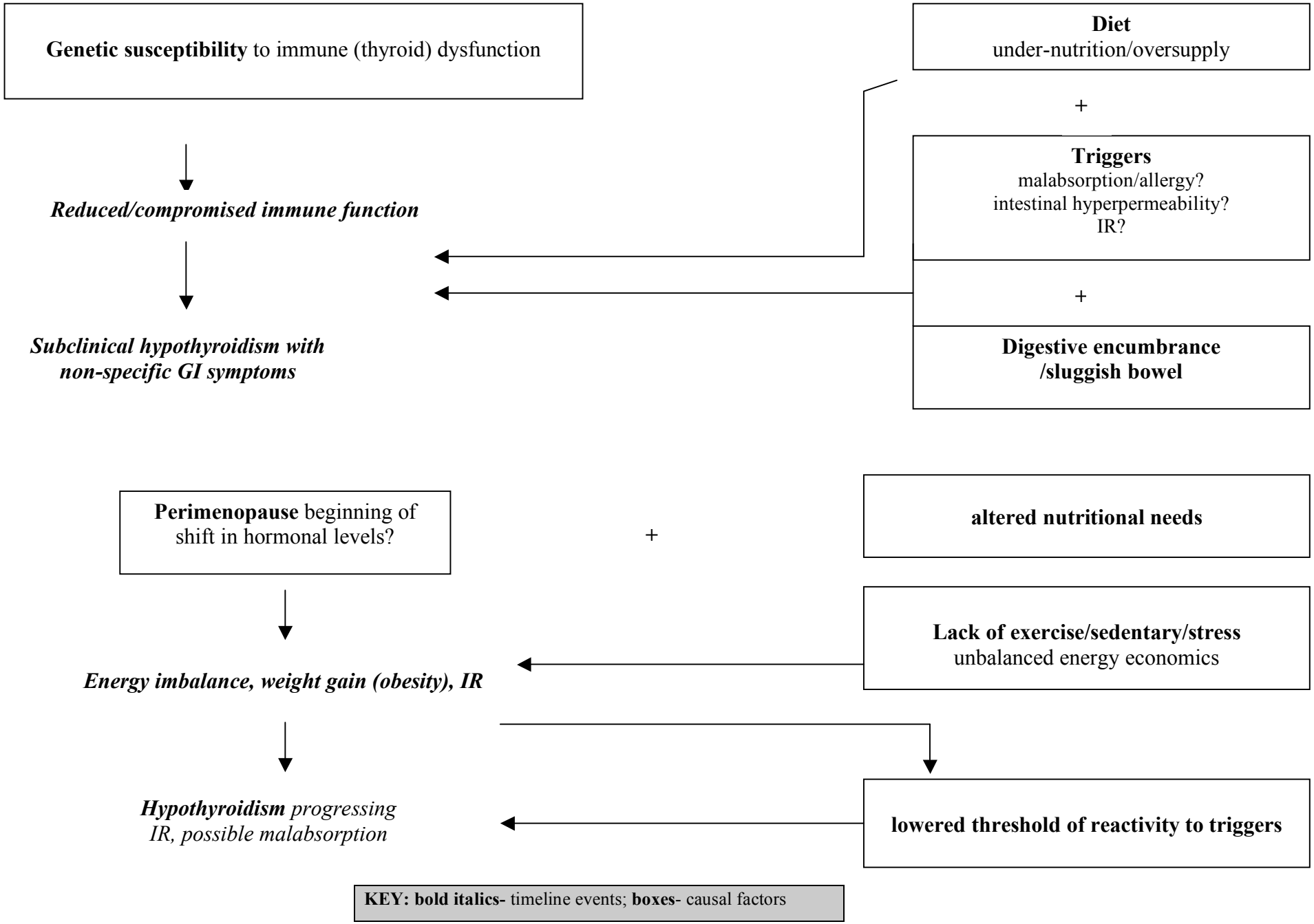
Please refer to diagram 1 for a causal chain of events leading to Julie's current condition.

[8] A sequence of metabolic events typically results in production of (toxic) by-products that need to be promptly eliminated by the body (Priest & Priest 1982, p.7). Encumbrance is therefore accumulation of waste material, which is capable of accumulating in various tissues encumbering metabolic processes (Priest & Priest 1982, p.7)

[9] Direct provoking causes of a disease (Mills & Bone 2007, p.128)

[10] Pathophysiological changes that hold an individual in the disease phase; a factor that comes into play as a result of disease process and specifically perpetuates the cycle (Mills & Bone 2007, p.128)

Diagram 1: Causal Chain Of Events



2. TESTS TO CONFIRM PROBABILITY DIAGNOSIS

The following tests will assist in confirming our suspicions.

Physical examination

Check:

Pulse: slow, low-volume

Skin: cool

Hair: coarse, dry, brittle

Eyes/Face: puffiness

Voice: husky

Extremities: cold

Reflexes: normal contraction but delayed relaxation (Murtagh 2003, pp.222-3).

Laboratory examination

Laboratory tests should follow an algorithmic format, to minimise cost to the patient and rule out the most likely cause/s of Julie's condition. Depending on the results at each stage, we will progress to the next step in order to determine what is going on for Julie. The order of priority testing should therefore be:

First stage (preliminary) testing[11]

Standard blood test will performed by a GP at no cost to patient, which includes:

Full blood examination (**FBE**); electrolytes, urea/creatinine, liver function tests (**LFTs**), glucose, cholesterol, triglycerides, high-density-lipoprotein (**HDL**)/low-density-lipoprotein (**LDL**)- fasting, iron studies, thyroid stimulating hormone (**TSH**), vitamin B12, red cell folate, follicle-simulating hormone (**FSH**), luteinising hormone (**LH**).

Expected outcome for a hypothyroidism/CD/perimenopause picture includes:

- FBE- may appear macrocytic, possibly due to hypothyroid anaemia or CD (Coghlan & Campbell 2002, p.3)
- LFTs- increased liver enzymes in hypothyroid (MedlinePlus 2008) or CD (Volta 2009, p.62);
- TSH- elevated in hypothyroid (Topliss & Eastman 2004, p.186)
- vitamin B12/folate/iron- deficiency common in CD (Selby & Darke 2008, pp.28, 30)
- Cholesterol/LDL- elevated in hypothyroidism (MedlinePlus 2008)
- Female hormone levels- elevated FSH/LH may indicate perimenopause (ARL Pathology 2008, p.83; The Royal College of Pathologists of Australasia Manual 2009; Burger 2008, p.2266).

[11] Ideally if cost were not an issue we would also perform additional tests that may be warranted in this case: *functional liver detoxification profile* (ARL Pathology 2008, p.45) and *IgG food sensitivity panel & intestinal permeability* (ARL Pathology 2008, pp.55, 63-8).

Second stage testing

a. In the event preliminary testing indicates high TSH and high cholesterol/LDL, we should explore this further with T3/T4 to indicate levels of free T3/T4 (ARL Pathology 2008, pp.113-6). Elevated TSH and subnormal T4 levels will help to confirm our probable diagnosis (ARL Pathology 2008, p.116; Murtagh 2003, p.222).

b. If results are indicative of thyroid dysfunction, we may also perform a urine iodine test for a reasonable cost of around AUD60 (ARL Pathology 2008, pp.135-6) to explore the patient's typical iodine intake over the last few weeks to help in later supplementation (World Health Organisation 2004, p.303).

c. At this stage, if bloods are normal, or markers indicate deficiencies consistent with CD, we may also do a CD screen using anti-gliadin antibody, anti-endomysial antibody, anti-tissue trans-glutaminase antibody (Selby & Darke 2008, p.28)[12]:

Third stage testing

In the event Julie's T3/T4 is abnormal we may now order a thyroid antibody test to help elucidate the type of thyroid disorder (ARL Pathology 2008, pp.113-6).

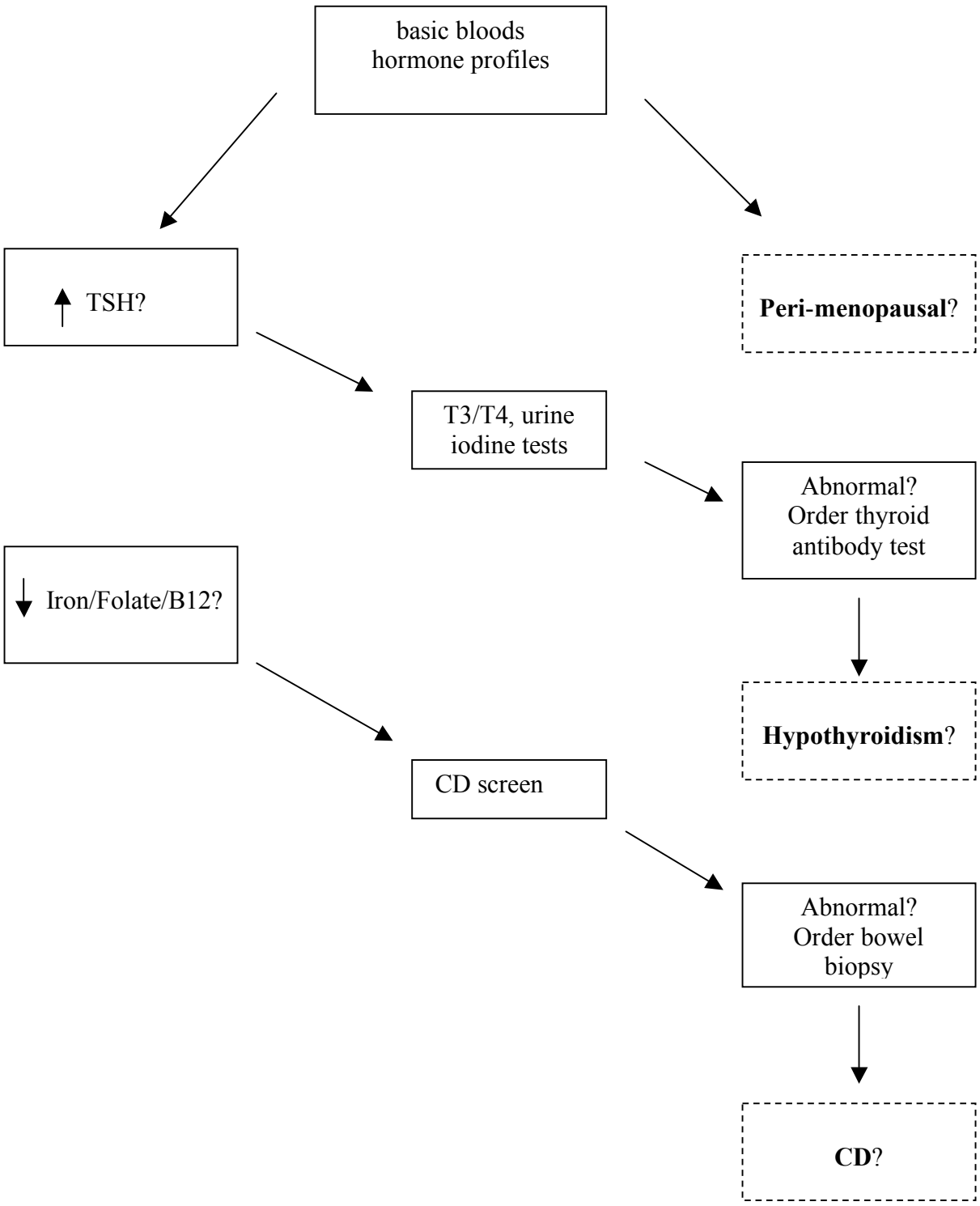
Fourth stage (medical) testing

If second stage CD screen test returns positive, Julie will be referred for small bowel biopsy, the current gold standard to confirm CD (Selby & Darke 2008, p.28-32).

Please refer to diagram 2 for testing algorithm and potential outcomes.

[12] Ensure patient has consumed wheat/bread for a few weeks to prevent false negatives (Selby & Darke 2008, p.28)

Diagram 2: Lab Test Algorithm



3. TREATMENT PLAN

Key treatment aims/goals

We will endeavour to break the links in the causal chain, in the short term by neutralising the excitatory/sustaining factors ('tip of the iceberg') via physiological compensation, and in the long-term by addressing predisposing causes via physiological enhancement (Mills & Bone 2007, p.128).

In other words, since chronic conditions are a sign of reduced vitality (Priest & Priest 1982, p.3), dealing with links in the causal chain in this manner will enable us to compensate for chemical deficiencies in the short-term, alleviating symptoms, and raising vitality (Mills & Bone 2007, p.127). This will allow constitution and predispositions to be addressed in the long-term by providing deeper physiological support to restore optimal functioning (Mills 1993, pp.212, 221).

Short-term aims (excitatory and sustaining factors)

a. **Assess and improve nutritional status**: address nutritional deficiencies (Selby & Darke 2008, pp.25-32) to reduce signs and symptoms (Cordain et al 2005, p.350).

b. **Reduce inflammation/improve redox status**: reduce inflammatory cytokines and pro-oxidants (Klecha et al 2008, p.68; Tsatsoulis 2006, p.382; Inoue et al 2009, p.199; Tsotsonava et al 2007, p.32)

c. **Improve thyroid hormone profile**: to ensure optimal T4 production, maximal T4-active T3 conversion, and minimal inactive T3 formation (Beckett & Arthur 2005, pp.458-61; Schomburg & Kohrle 2008, p.1235; Papp et al 2007, pp.789-92).

d. **Support nervous system**: reducing stress levels dampens the inclination to activate the hypothalamic-pituitary-adrenal (**HPA**)-axis (Tsatsoulis 2006, p.382). This prevents increased secretion of glucocorticoids/catecholamines[13], which can lead to thyroid dysfunction by triggering an imbalance between the T-helper 1(**Th1**) versus T-helper 2 lymphocyte immune response (Tsatsoulis 2006, p.382)[14]. Supporting the NS will therefore dampen the affect of the HPA-axis on Th1 activity (Tsatsoulis 2006, p.382).

e. **Address any gastrointestinal dysfunction, including possible liver dysfunction**: by enhancing the cephalic phase and correcting digestive function/intestinal permeability/digestive secretions, the gut-immune system integrity will be restored thereby reducing malabsorption issues (Wapenaar et al 2008, p.438), optimising liver function (Volta 2009, p.62; Malik & Hodgson 2002, p.561), reducing dysfunctional immunity (Hanaway 2006, p.53) and risk of diabetes (Bosi et al 2006, p.2824), which all lead to optimal micronutrient intake from the diet (Hanaway 2006, p.53).

g. **Reduce possible food triggers**: If Julie also has CD (which is increasingly common with autoimmune hypothyroidism; National Institutes of Health 2008), she should be placed on a gluten-free diet (Selby & Darke 2008, pp.25-31), otherwise identify foods likely to be causing allergy/intestinal hyperpermeability, and support a low-reactive, macro/micro-nutrient-dense, weight management diet (Cordain et al 2005, p.350).

[13] May influence differentiation of Th cells away from Th1 and toward Th2 phenotype resulting in cellular immunity suppression and humoral immunity potentiation (Tsatsoulis 2006, p.382)
[14] Predominantly Th1-mediated immune activity may trigger autoimmune hypothyroidism in a predisposed individual (Tsatsoulis 2006, p.382)

Longer-term aims (predisposing causes)

a. **Continue to improve nutritional status**.

b. **Continue to improve thyroid hormone profile**.

c. **Continue to support major systems**: thyroid-liver axis (Malik & Hodgson 2002, p.561), immune, neuro-endocrine (Mills 1993, pp.58, 212-26; Tsatsoulis 2006, p.382).

d. **Support perimenopause**: improving female hormone status and monitoring hormone levels ensures we do not aggravate her thyroid condition during the treatment plan (Schindler 2003, p.79), and helps recognise the changing clinical manifestations of her thyroid condition (Pearce 2007, p.8).

4. PRESCRIPTION

The priority in this case will be to address nutritional deficiencies and improve digestive/immune status (Malik & Hodgson 2002, p.561). To ensure Julie starts to feel more balanced, we will therefore supplement where levels are dangerously low, require initial attention in order to help other nutrient levels downstream and/or cannot be raised sufficiently through the diet. Other potential deficiencies relevant to this case will be addressed through improving Julie's diet and lifestyle. For example:

- iron[15]: deficiency common in both hypothyroidism (Coghlan & Campbell 2002, p.3) and CD (Selby & Darke 2008, p.30), depending on blood test results will be relatively easy to obtain from iron-rich foods such as meat/seafood, eggs, nuts/seeds (National Health & Medical Research Council 2003, pp.52-3);
- Zn, folate, and vitamin B12 will be increased by consuming foods more vegetables, fruits, wholegrain cereals, dairy (National Health & Medical Research Council 2003, pp.52, 75-77; National Health & Medical Research Council 2006, p.75); lean beef, poultry, eggs, seafood (especially oysters), and organ meats (Stargrove, Treasure & McKee 2008, p.623);

It is also important to reduce alcohol consumption as nutrient depletion is common, especially Zn (Stargrove, Treasure & McKee 2008, p.622) iron and folate (National Health & Medical Research Council 2003, pp.57-8, 153), as well as metabolic (e.g. hypoglycemia), and neuro-endocrine disruption (National Health & Medical Research Council 2003, p.158).

Although Zn tally testing is not an accurate measure of deficiency, we may surmise that Julie has at least a mild deficiency (National Health & Medical Research Council 2003, p.59) so dietary measures, at least in the short term, may be adequate and will avoid possible toxicity (even at 60mg/day; Stargrove, Treasure & McKee 2008, pp.623-4).

[15] In the event she has iron-deficiency anemia she will require therapeutic supplementation (Selby & Darke 2008, p.30)

Detailed below are therefore 3 most likely micronutrients, providing the broadest effects (for thyroid/female hormone production and protection but also minimising possible blood-sugar dysregulation and cardiovascular risk):

Micronutrient 1: Iodine- integral part of thyroid hormones

Difficult to obtain sufficient levels from food (Food Standards Australia New Zealand 2008, p.81) and varying widely depending on soil quality (Reavley 1998, p.245), iodine will be supplemented to control dose and monitored to avoid toxicity (Food Standards Australia New Zealand 2008, p.19; Teng et al 2008, p.23)[16]. Forming an integral part of the major thyroid hormones, T3 and T4 (Schomburg & Kohrle 2008, p.1235), iodine will enable their proper formation and therefore ameliorate major symptoms through increasing energy production, increasing lipolysis, and regulating gluconeogenesis and glycolysis (World Health Organisation 2004, pp.303-4).

Dose: 150ug/day (World Health Organisation 2004, p.311)[17,18]
Form: Iodine drops (in emulsion form), as potassium iodide (Wu et al 2002)[19]
Instructions: once in the morning with juice or water (World Health Organisation 2004, p.303).
Nutrient-nutrient interactions: synergistic- vitamins C, B complex, nicotinamide adenine dinucleotide, copper, Mg, tyrosine, selenium, Zn (synergistic; Osiecki n.d, p.138); adverse- may affect nutrients with diuretic effects (MedlinePlus 2008).

[16] Iodine supplemented over zinc as we assume Julie does not prefer foods rich in iodine (such as seafood and seaweed), otherwise they would have been part of her existing diet by this stage
[17] Daily intake recommendations by World Health Organisation, United Nations Children's Fund, and International Council for Control of Iodine Deficiency Disorders (World Health Organisation 2004, p.311)
[18] One study found safe range equivalent to urinary iodine 100-200g/l in women with thyroid disease (Teng et al 2008, p.23)
[19] Iodide form 100% bioavailable, absorbed totally from food and water (World Health Organisation 2004, p.303)

Micronutrient 2: Selenium- fundamental to global health

This essential trace mineral is fundamental to human health (Stazi & Trinti 2008, p.643). Since the thyroid is especially sensitive to its deficiency (Stazi & Trinti 2008, p.643) and it is extremely difficult to obtain from the diet due to soil depletion and low levels in food (National Health & Medical Research Council 2006, p.75), it will be critical to Julie's case. Supplementation will provide broad effects including optimisation of endocrine/immune function and modulating inflammation (Beckett & Arthur 2005, pp.455-8).

Specifically by forming seleno-proteins, components of the glutathione peroxidase, thioredoxin reductase and iodothyronine deiodinase family of enzymes (Stazi & Trinti 2008, p.643), selenium will contribute a powerful antioxidant status that will systemically protect against oxidative damage (Beckett & Arthur 2005, pp.458-61; Schomburg & Kohrle 2008, p.1235; Stazi & Trinti 2008, p.643; Papp et al 2007, pp.789-92). It will also assist in modulating thyroid hormone metabolism (Stazi & Trinti 2008, p.643) by ensuring proper biosynthesis and function of thyroid hormone metabolism through adequate T4 production, maximal T4 to active T3 conversion, and minimal inactive T3 formation (Beckett & Arthur 2005, pp.458-61; Schomburg & Kohrle 2008, p.1235; Papp et al 2007, pp.789-92).

Dose: 200ug daily (Gartner et al 2002 and Gartner & Gasnier 2003 cited in Beckett & Arthur 2005, p.461)
Form: Seleno-methionine tablet (Schrauzer 2003, p.73; Reaveley 1998, p.303)[20].
Instructions: 100ug BD with food (Osiecki n.d, p.154)[21]
Nutrient-nutrient interactions: synergistic- vitamins E, C (up to 1g; Stargrove, Treasure & McKee 2008, p.397), methionine, B3, coenzyme Q10, cysteine, glutathione, Zn, (Osiecki n.d, p.154), iodine (Beckett & Arthur 2005, p.459); adverse- may affect calcium and Mg absorption, may be affected by EFAs (MedlinePlus 2008).

[20] Naturally occurring form and best absorbed by humans (Schrauzer 2003, p.73)
[21] As certain nutrients and amino acids work synergistically (Osiecki n.d, p.154)

Micronutrient 3: Omega 3 EFAs - pleiotropic effects

As EFAs are taken up by virtually all cells, affect membrane composition, eicosanoid biosynthesis, cell signaling cascades, and gene expression (Shahidi & Miraliakbari 2005, p.133), supplementation will therefore provide global effects:

- reduce autoimmunity/inflammation (Simopoulos 2008, p.674) by shifting the Th1/Th2 lymphocyte balance (Mizota et al 2009);
- reduce intestinal hyperpermeability[22] (Willemsen et al 2008, p.183);
- improve (insulin, thyroid) receptor sensitivity (Tsitouras et al 2008, p.199);
- protect thyroid hormone levels (as shown in rats with eicosapentanoic acid; Makino et al 2001, p.265); and
- improve cognition, mood (Antypa et al 2008) and reduce CVD risk (Fernandes et al 2008, p.4015).

As Julie will also be increasing protein consumption, supplementing will also ensure an effective n-6:n-3[23], a factor as important in immune modulation (Harbige 2003, p.323)[24].

Dose: 1-3g/day (Osiecki n.d, p.60).
Form: n-3 fish oil, liquid or capsule
Instructions: 1g TDS, with food (Cleland, James & Proudman 2006, p.206)
Nutrient-nutrient interactions: synergistic- vitamins A, B3, B6, bioflavonoids, Mg, methionine, selenium, quercetin, Zn (Osiecki n.d, p.60); adverse- may deplete vitamin E with long-term use (MedlinePlus 2008).

word count: 2186

[22] CD aetiology may involve tight junction-mediated barrier defects (Wapenaar et al 2008, p.438)
[23] Being about 1-4:1 rather than Julie's probable 20:1 (Simopoulos 2002, p.502)
[24] However in order to correct the ratio, Julie may require higher levels (Reavley 1998, pp.336-7), therefore need to cautious avoiding too high doses that may result in suppressed immunity (Whitney & Rolfes 2008, p.159; Harbige 2003, p.323). Also EFA dosing to achieve immune-modulation must consider genetics, health/disease status, immune response stage and possibly age (Simopoulos 2002, p.502; Wu 2004, p.3)

REFERENCES

Allen, RB 2005, Gut Permeability and Intestinal Dysbiosis, *Journal of Complementary Medicine*, vol.4, no.4, pp.35-40.

Anderson, JW, Baird, P, Davis, RH Jr, Ferreri, S, Knudtson, M, Koraym, A, Waters, V & Williams, CL 2009, Health Benefits Of Dietary Fiber, *Nutrition Review*, vol.67, no.4, pp.188-205. Retrieved from PubMed 1 April 2009.

Antypa, N, Van der Does, A, Smelt, A & Rogers, R 2008, Omega-3 Fatty Acids (fish-oil) and Depression-Related Cognition in Healthy Volunteers, *Journal of Psychopharmacology*, [ahead of print]. Abstract retrieved from PubMed 1 April 2009.

ARL Pathology 2008, *Practitioner Manual: A Guide to Laboratory Testing*, Melbourne, Victoria, Australia.

Australian Better Health Initiative 2008, *How do you measure up? Body Mass Index*, viewed 15 April 2009, < http://www.measureup.gov.au/internet/abhi/publishing.nsf/Content/Body+Mass+Index-lp >

Beckett, GJ & Arthur, JR 2005, Selenium and Endocrine Systems, *The Journal of Endocrinology*, vol.184, no.3, pp.455-65. Retrieved from PubMed 1 April 2009.

Berg, AO 2004, *Screening for Thyroid Disease, US Preventive Services Task Force*, viewed 1 April 2009, < http://ahrq.hhs.gov/clinic/3rduspstf/thyroid/thyrrs.pdf >

Berkow, R & Fletcher, AJ (eds) 1992, *The Merck Manual of Diagnosis and Therapy*, Merck Research Laboratories, Merck & Co. Inc., Rahway, NJ.

Better Health Channel 2008, *Q: What is perimenopause?,* Victorian Department of Human Services, Australia, viewed 1 April 2009, < http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/ateaf/2377810?open >

Bone, K 2003, *A Clinical Guide to Blending Liquid Herbs: Herbal Formulations for the Individual Patient*, Churchill Livingstone, St Louis, Missouri, U.S.A.

Bosi, E, Molteni, L, Radaelli, MG, Folini, L, Fermo, I, Bazzigaluppi, E, Piemonti, L, Pastore, MR & Paroni, R 2006, Increased Intestinal Permeability Precedes Clinical Onset of Type 1 Diabetes, *Diabetologia*, vol.49, pp.2824-7. Retrieved from PubMed 1 April 2009.

Burger, H 2008, The Menopausal Transition- Endocrinology, *Journal of Sexual Medicine*, vol.5, no.10, pp.2266-73. Abstract retrieved from PubMed 30 April 2009.

Chahal, HS & Drake, WM 2007, Review Article: The Endocrine System and Ageing, *Journal of Pathology*, vol.211, pp.173-80. Retrieved from PubMed 1 April 2009.

Chrousos, GP 2007, Organization and Integration of the Endocrine System, *Sleep Medicine Clinic,* vol.2, no.2, pp.125-45. Retrieved from PubMed 1 April 2009.

Cleland, LG, James, MJ & Proudman, SM 2006, Fish Oil: What The Prescriber Needs To Know, *Arthritis Research & Therapy*, vol.8, no.1, pp.202-10, viewed 1 May 2009, < http://arthritis-research.com/content/8/1/202 >

Coghlan, D & Campbell, P 2002. How to Treat: Anaemia, *Australian Doctor*, November, pp.1-6, viewed 20 April 2009, < http://www.australiandoctor.com.au/htt/pdf/021108/ad_htti_8nov02.p1lr.pdf >

Cordain, L, Eaton, SB, Sebastian, A, Mann, N, Lindeberg, S, Watkins, BA, O'Keefe, JH & Brand-Miller, J 2005, Origins And Evolution Of The Western Diet: Health Implications For The 21st Century, *American Journal of Clinical Nutrition*, vol.81, no.2, pp.341-54, viewed 1 May 2009, < http://www.ajcn.org/cgi/reprint/81/2/341 >

Desailloud, R & Hober, D 2009, Review: Viruses and Thyroiditis: An Update, *Virology Journal*, vol.6, no.5. Retrieved from PubMed 10 April 2009.

Dimitriadis, G, Mitrou, P, Lambadiari, V, Boutati, E, Maratou, E, Panagiotakos, DB, Koukkou, E, Tzanela, M, Thalassinos, N & Raptis, SA 2006, Insulin Action in Adipose Tissue and Muscle in Hypothyroidism, *Journal of Clinical Endocrinology and Metabolism*, vol.91, no.12, pp.4930-7. Retrieved from PubMed 10 April 2009.

Fernandes, G, Bhattacharya, A, Rahman, M, Zaman, K & Banu, J 2008, Effects Of n-3 Fatty Acids On Autoimmunity And Osteoporosis, *Frontiers in Bioscience: A Journal and Virtual Library*, vol.13, pp.4015-20. Abstract retrieved from PubMed 20 April 2009.

Food Standards Australia New Zealand 2008, *The 22nd Australian Total Diet Study*, viewed 10 April 2009, < http://www.foodstandards.gov.au >

Geissler, CA & Powers, HJ (eds) 2000, *Human Nutrition*, 11th ed, Elsevier Ltd. Edinburgh.

Galofré, JC, Pujante, P, Abreu, C, Santos, S, Guillen-Grima, F, Frühbeck, G & Salvador, J 2008, Relationship Between Thyroid-Stimulating Hormone And Insulin In Euthyroid Obese Men, *Annual Nutritional Metabolism, vol.*53, nos.3-4, pp.188-94. Retrieved from PubMed 10 April 2009.

Hanaway, P 2006, CME: Balance of Flora, GALT, and Mucosal Integrity, *Alternative Therapies in Health and Medicine,* vol.12, no.5, pp.52-60.

Harbige, LS 2003, Fatty Acids, The Immune Response, And Autoimmunity: A Question Of N-6 Essentiality And The Balance Between n-6 and n-3, *Lipids*, vol.38, no.4, pp.323-41. Retrieved from PubMed 10 April 2009.

Higuchi, R, Miyawaki, M, Kumagai, T, Okutani, T, Shima, Y, Yoshiyama, M, Ban, H & Yoshikawa, N 2005, Central Hypothyroidism in Infants Who Were Born to Mothers With Thyrotoxicosis Before 32 Weeks' Gestation: 3 Cases, *Pediatrics*, vol.115, no.5, pp.623-5. Retrieved from PubMed 10 April 2009.

Inoue, N, Watanabe, M, Nanba, T, Wada, M, Akamizu, T & Iwatani, Y 2009, Involvement Of Functional Polymorphisms In The TNFA Gene In The Pathogenesis Of Autoimmune Thyroid Diseases And Production Of Anti-Thyrotropin Receptor Antibody, *Clinical and Experimental Immunology*, vol.156, no.2, pp.199-204. Abstract retrieved from PubMed 10 April 2009.

Kim, SY, Breslow, RA, Ahn, J & Salem, N Jr 2007, Alcohol Consumption And Fatty Acid Intakes In The 2001-2002 National Health And Nutrition Examination Survey, *Alcoholism, Clinical & Experimental Research*, vol.31, no.8, pp.1407-14. Retrieved from PubMed 10 April 2009.

Klecha, AJ, Barreiro, Arcos, ML, Frick, L, Genaro, AM & Cremaschi, G 2008, Immune-Endocrine Interactions In Autoimmune Thyroid Diseases, *Neuroimmunomodulation*, vol.15, no.1, pp.68-75. Retrieved from PubMed 10 April 2009.

Lindström, J, Peltonen, M, Eriksson, JG, Louheranta, A, Fogelholm, M, Uusitupa, M & Tuomilehto, J 2006, High-Fibre, Low-Fat Diet Predicts Long-Term Weight Loss And Decreased Type 2 Diabetes Risk: The Finnish Diabetes Prevention Study, *Diabetologia*, vol.49, no.5, pp.912-20. Retrieved from PubMed 10 April 2009.

Makino, M, Oda, N, Miura, N, Imamura, S, Yamamoto, K, Kato, T, Fujiwara, K, Sawai, Y, Iwase, K, Nagasaka, A & Itoh, M 2001, Effect Of Eicosapentaenoic Acid Ethyl Ester On Hypothyroid Function, *The Journal of Endocrinology*, vol.171, no.2, pp.259-65. Retrieved from PubMed 10 April 2009.

Malik, R & Hodgson, H 2002, The Relationship Between the Thyroid Gland and the Liver. *QJM*, vol.95, pp.559-69. Retrieved from PubMed 15 April 2009.

McDermott, MT 1998, *Endocrine Secrets: Questions You Will Be Asked On Rounds, In The Clinic, On Oral Exams*, 2nd ed, Hanley & Belfus Inc., Philadelphia, PA.

MedlinePlus 2008, *Hypothyroidism*, U.S. Department of Health & Human Services, National Institutes of Health, viewed 15 April 2009, < http://www.nlm.nih.gov/medlineplus/ency/article/000353.htm >

MedlinePlus 2008, *Iodine (I)*, U.S. Department of Health & Human Services, National Institutes of Health, viewed 15 April 2009, < http://www.nlm.nih.gov/medlineplus/druginfo/natural/patient-iodine.html >

MedlinePlus 2008, *Omega-3 Fatty Acids, Fish Oil, Alpha-Linolenic Acid*, U.S. Department of Health & Human Services, National Institutes of Health, viewed 15 April 2009, < http://www.nlm.nih.gov/medlineplus/druginfo/natural/patient-fishoil.html >

MedlinePlus 2008, *Selenium (Se)*, U.S. Department of Health & Human Services, National Institutes of Health, viewed 15 April 2009, < http://www.nlm.nih.gov/medlineplus/druginfo/natural/patient-selenium.html >

Mills, S 1993, *The Essential Book of Herbal Medicine,* Arkana, Ringwood, Victoria.

Mills, S & Bone, K 2007, *Principles and Practice of Phytotherapy: Modern Herbal Medicine*, Churchill Livingstone, London, U.K.

Mizota, T, Fujita-Kambara, C, Matsuya, N, Hamasaki, S, Fukudome, T, Goto, H, Nakane, S, Kondo, T & Matsuo, H 2009, Effect of Dietary Fatty Acid Composition on Th1/Th2 Polarization in Lymphocytes, *Journal of Parenteral and Enteral Nutrition*, [ahead of print]. Abstract retrieved from PubMed 1 May 2009.

Murtagh, J 2003, *John Murtagh's General Practice*, McGraw-Hill Australia Pty Ltd, North Ryde, NSW.

National Health & Medical Research Council 2003, *Dietary Guidelines for Australian Adults*, Commonwealth of Australia, Canberra, Australia.

National Health & Medical Research Council 2006, *Nutrient Reference Values for Australia and New Zealand*, Commonwealth of Australia, Canberra, Australia.

National Institutes of Health 2008, *Celiac Disease*, viewed 15 April 2009, < http://digestive.niddk.nih.gov/ddiseases/pubs/celiac/index.htm#other >

Osiecki, H n.d, *The Nutrient Bible*, 7th ed, Bio Concepts Publishing, Eagle Farm, QLD, Australia.

Papp, LV, Lu, J, Holmgren, A & Khanna, KK 2007, From Selenium To Selenoproteins: Synthesis, Identity, And Their Role In Human Health, *Antioxidants and Redox Signalling*, vol.9, no.7, pp.775-806. Retrieved from PubMed 10 April 2009.

Pearce, EN 2007, Thyroid Dysfunction In Perimenopausal And Postmenopausal Women, *Menopause International*, vol.13, no.1, pp.8-13. Retrieved from PubMed 10 April 2009.

Pitchford, P 2002, *Healing with Whole Foods: Asian Traditions and Modern Nutrition*, North Atlantic Books, Berkeley, California, U.S.A.

Priest, AW & Priest, LR 1982, *Herbal Medication: A Clinical and Dispensary Handbook*, LN Fowler & Co Ltd, London.

Reavley, N 1998, *The New Encyclopedia of Vitamins, Mineral, Supplements & Herbs*, M. Evans and Company Inc. New York.

The Royal College of Pathologists of Australasia 2009, *RCPA Manual*, viewed 20 April 2009, < http://www.rcpamanual.edu.au/ >

Riccardi, G & Rivellese, AA 2000, Dietary Treatment Of The Metabolic Syndrome-The Optimal Diet, *The British Journal of Nutrition*, vol.83, supplement 1, pp.143-8. Retrieved from PubMed 20 April 2009.

Riccardi, G, Giacco, R & Rivellese, AA 2004, Dietary Fat, Insulin Sensitivity And The Metabolic Syndrome, *Clinical Nutrition*, vol.2, no.4, pp.447-56. Retrieved from PubMed 10 April 2009.

Schindler, AE 2003, Thyroid Function And Postmenopause, *Gynecology & Endocrinology*, vol.17, no.1, pp.79-85. Retrieved from PubMed 10 April 2009.

Schomburg, L & Köhrle, J 2008, On The Importance Of Selenium And Iodine Metabolism For Thyroid Hormone Biosynthesis And Human Health, *Molecular Nutrition & Food Research*, vol.52, no.11, pp.1235-46. Retrieved from PubMed 10 April 2009.

Schrauzer, GN 2003, The Nutritional Significance, Metabolism And Toxicology Of Selenomethionine, *Advances in Food and Nutritional Research*, vol.47, pp.73-112. Retrieved from PubMed 20 April 2009.

Schumm-Draeger, PM 2006, Diabetes Mellitus and Frequently Associated Endocrine Diseases, *MMW Fortschritte der Medizin*, vol.148, no.37, pp.47-50. Abstract retrieved from PubMed 20 April 2009.

Selby, W & Darke, J 2008, How to Treat: Coeliac Disease, *Australian Doctor*, August, pp.25-32, viewed 20 April 2009, < http://www.australiandoctor.com.au/HTT/PDF/ad_025_032_aug08_08.pdf >

Shahidi, F & Miraliakbari, H 2005, Omega-3 Fatty Acids In Health And Disease: Part 2-Health Effects Of Omega-3 Fatty Acids In Autoimmune Diseases, Mental Health, And Gene Expression, *Journal of Medicinal Food*, vol.8, no.2, pp.133-48. Abstract retrieved from PubMed 20 April 2009.

Simopoulos, AP 2002, Omega-3 Fatty Acids In Inflammation And Autoimmune Diseases, *Journal of the American College of Nutrition*, vol.21, no.6, pp.495-505, viewed 20 April 2009, < http://www.jacn.org/cgi/reprint/21/6/495 >

Simopoulos, AP 2008, The Importance Of The Omega-6/Omega-3 Fatty Acid Ratio In Cardiovascular Disease And Other Chronic Diseases, *Experimental Biology & Medicine*, vol.233, no.6, pp.674-88. Retrieved from PubMed 20 April 2009.

Stargrove, MB, Treasure, J & McKee, DL 2008, *Herb, Nutrient, and Drug Interactions: Clinical Implications and Therapeutic Strategies*. St. Louis: Mosby, Elsevier, Australia.

Stazi, AV & Trinti, B 2008, Selenium Deficiency in Celiac Disease: Risk of Autoimmune Thyroid Diseases, *Minerva Medica*, vol.99, no.6, pp.643-53. Abstract retrieved from PubMed 20 April 2009.

Syed, M, Rosati, C, Torosoff, MT, El-Hajjar, M, Feustel, P, Alger, S, Singh, P & Fein, S 2009, The Impact Of Weight Loss On Cardiac Structure And Function In Obese Patients, *Obesity Surgery*, vol.19, no.1, pp.36-40. Abstract retrieved from PubMed 20 April 2009.

Tahara, Y, Kozu, S, Ikegami, H, Tanaka, A, Kumahara, Y, Hirota, M, Shima, K, Amino, N, Hayashizaki, S & Miyai, K 1985, Contribution Of Amino Acid Deficiency To Primary Hypothyroidism Associated With Protein-Calorie Malnutrition, *Nippon Naibunpi Gakkai Zasshi*, vol.61, no.11, pp.1270-81. Abstract retrieved from PubMed 20 April 2009.

Teng, X, Shi, X, Shan, Z, Jin, Y, Guan, H, Li, Y, Yang, F, Wang, W, Tong, Y & Teng, W 2008, Safe Range Of Iodine Intake Levels: A Comparative Study Of Thyroid Diseases In Three Women Population Cohorts With Slightly Different Iodine Intake Levels, *Biological Trace Element Research*, vol.121, no.1, pp.23-30. Retrieved from PubMed 20 April 2009.

Thyroid Info 2009, *Hypothyroidism Risk Factors & Symptoms Checklist Excerpted from Living Well With Hypothyroidism by Mary Shomon*, viewed 15 April 2009, < http://www.thyroid-info.com/chklst.html >

Topliss, DJ & Eastman, CJ 2004, MJA Practice Essentials- Endocrinology 5: Diagnosis And Management Of Hyperthyroidism And Hypothyroidism, *Medical Journal of Australia*, vol.180, no.4, pp.186-93. Retrieved from PubMed 20 April 2009.

Tsatsoulis, A 2006, The Role Of Stress In The Clinical Expression Of Thyroid Autoimmunity, *Annals of the New York Academy of Sciences*, vol.1088, p.382-95. Retrieved from PubMed 25 April 2009.

Tsitouras, PD, Gucciardo, F, Salbe, AD, Heward, C & Harman, SM 2008, High Omega-3 Fat Intake Improves Insulin Sensitivity And Reduces CRP And IL6, But Does Not Affect Other Endocrine Axes In Healthy Older Adults, *Hormone and Metabolic Research*, vol.40, no.3, pp.199-205. Retrieved from PubMed 20 April 2009.

Tsotsonava, T, Virsaladze, D, Khitarishvili, K, Sanikidze, T & Tananashvili, D 2007, Comparative Analysis Of Blood Redox Parameters According Thyroid Function Of Patients With Autoimmune Thyroid Diseases, *Georgian Medical News*, vol.146, no.32-4. Abstract retrieved from PubMed 10 April 2009.

The University of Maryland Medical Center 2009, *Celiac Sprue*, viewed 15 April 2009, < http://www.umm.edu/altmed/articles/celiac-sprue-000875.htm >

The University of Sydney n.d., *The Glycemic Index*, viewed 15 April 2009, < http://www.glycemicindex.com/ >

Volta, U 2009, Pathogenesis And Clinical Significance Of Liver Injury In Celiac Disease, *Clinical Reviews in Allergy & Immunology*, vol.36, no.1, pp.62-70. Retrieved from PubMed 10 April 2009.

Wapenaar, MC, Monsuur, AJ, van Bodegraven, AA, Weersma, RK, Bevova, MR, Linskens, RK, Howdle, P, Holmes, G, Mulder, CJ, Dijkstra, G, van Heel, DA & Wijmenga, C 2008, Associations With Tight Junction Genes PARD3 And MAGI2 In Dutch Patients Point To A Common Barrier Defect For Coeliac Disease And Ulcerative Colitis, *Gut*, vol.57, no.4, pp.463-7. Retrieved from PubMed 20 April 2009.

Whitney, E & Rolfes, SR 2008, *Understanding Nutrition*, 11th ed, Thomson Wadsworth, Australia.

World Health Organisation 2004, *Chapter 16. Iodine*, viewed 19 April 2009, < www.whqlibdoc.who.int/publications/2004/9241546123_chap16.pdf >

Willemsen, LE, Koetsier, MA, Balvers, M, Beermann, C, Stahl, B & van Tol, EA 2008, Polyunsaturated Fatty Acids Support Epithelial Barrier Integrity And Reduce IL-4 Mediated Permeability *in vitro*, *European Journal of Nutrition*, vol.47, no.4, pp.183-91. Retrieved from PubMed 20 April 2009.

Wintergerst, ES, Maggini, S & Hornig, DH 2007, Contribution Of Selected Vitamins And Trace Elements To Immune Function, *Annals of Nutritional Metabolism*, vol.51, no.4, pp.301-23. Retrieved from PubMed 20 April 2009.

Wu, T, Liu, GJ, Li, P & Clar, C 2002, Iodised Salt For Preventing Iodine Deficiency Disorders, *Cochrane Database of Systematic Reviews*, issue 3, viewed 20 April 2009, < http://www.cochrane.org/reviews/en/ab003204.html >

Wu, D 2004, Modulation Of Immune And Inflammatory Responses By Dietary Lipids, *Current Opinions in Lipidology*, vol.15, no.1, pp.43-7. Retrieved from PubMed 1 May 2009.