LATEST RESEARCH - THYROID FUNCTION AND OPTIMUM NUTRITION

Hypothyroidism occurs when the thyroid gland is unable to produce adequate thyroid hormone. The condition affects approximately 2% of the UK population, with women affected 5 to 10 times more than men (NICE, 2019). Whilst age related decline in thyroid function accounts for 5% of cases in adults over 60 years, the primary cause of hypothyroidism in developed countries is the autoimmune disease, Hashimoto's (Mincer & Jialal, 2022). Current NICE's UK guidelines for management of the chronic condition of hypothyroidism is through life-long medication of thyroid hormone replacement using levothyroxine monotherapy, however other countries endorse treatments including natural desiccated thyroid (NDT) as well as Liothyronine (T3). Therefore prior to a client's nutritional advice, we would explore the specific reasons for your hypothyroidism as well as any current medication, or whether you are sub-clinical in which case we will examine how to support existing thyroid function.

Background

One of the reasons it is essential to support your thyroid health is that despite management of the disease, according to Thvilum et al (2013) hypothyroid individuals experience higher mortality connected to comorbidities such as increased incidence of metabolic syndrome and cardiovascular disease. Equally, as was highlighted in a recent meta-analysis (Roa Duenas, 2022), there appears an increased risk of abnormal glucose metabolism and the development of type 2 diabetes amongst individuals with hypothyroidism. Furthermore, a psychological component emerges to the disease as was indicated in a study which mapped comorbid thyroid disease with patients with major depressive disorder (Fugger, Dold, Bartover et al, 2018). The research reported abnormal thyroid function in particular hypothyroidism appeared linked to depression severity and manifesting the symptoms of depression. This conclusion built on similar findings in 'Hypothyroidism and Depression' (Dayan & Panicker, 2013).

Hypothyroidism and Nutrition

Given the metabolic slowing effects of hypothyroidism and concomitant physical and mental health implications described above, dietary choices and habits become significant to sustaining good health for hypothyroid adults. Furthermore, there is evidence to suggest it is not just the management of metabolic balance that is of concern in relation to nutrition and hypothyroid adults, but also particular foods, supplements and drugs can affect the absorption of levothyroxine medication within the gut (Thyroid UK, 2022). Therefore what, when and how much an individual eats may impact on their ability to manage the disease, with micronutrients either helping or hindering levothyroxine absorption. For example, a

systematic review that examined selenium supplementation in the treatment of participants with Hashimoto's (Toulis et al, 2010), concluded that participants felt an improvement in wellbeing as a consequence of selenium dampening down Hashimoto antibodies produced in the body. The mechanisms of how selenium works to effect these changes are still debated and considered a research priority by NICE (2022). Nevertheless nutritional advice can help enable clients to naturally incorporate selenium into their diets for maximum benefit.

An important vitamin that effects hypothyroid health is vitamin D. An Iranian study investigated the vitamin D and calcium levels of 175 participants (Ebrahimabad et al, 2019), and determined that levels were significantly lower in hypothyroid participants. Despite the small sample size of this study, substantial research already exists to support the hypothesis that vitamin D is essential to support mood and low levels are more likely to lead to decreased wellbeing. Furthermore vitamin D supplementation in hypothyroid adults appears to suppress thyroid stimulating hormone (TSH) as was demonstrated in a 12 week RCT involving 201 participants, aged 20-60 years old (Talaei et al, 2018). TSH levels increase when the body works to produce more thyroid hormone, therefore the study suggests that vitamin D supplementation beyond the UK's RNI of 10 µg/d (400 IU/d) (SACN, 2016), may ameliorate hypothyroid symptoms.

Deiodination is the mechanism whereby the inactive T4 prohormone is converted into the bioavailable T3 and it appears many micronutrients can enable or hinder this conversion. For example, one literature review that explored the impact of food nutrients on thyroid function identified studies on: iodine, selenium, zinc, copper, iron, soy, gluten and flavonoids (Mezzomo et al, 2016). In particular as iodine is the key element involved in the synthesis of thyroid hormones, the report cites iodine deficiency which affects nearly 800 million people as a global public health issue. As iodine can only be sourced from food, there is a high risk of deficiency within iodine insufficient areas.

The complexity of food nutrients interactions with the thyroid function was also investigated by Kawicka & Regulska-Ilow (2015), who concluded that "It is recommended to increase the protein intake, the intake of such minerals as selenium, zinc, copper and iron, as well as vitamin A, C and D." This research stressed the importance of a healthy diet for hypothyroid individuals and advised an 'increase' as opposed to 'maintaining' recommended vitamin and mineral levels. Consequently the study introduces ambiguity regarding what appropriate supplementation should be for hypothyroid adults, as in the UK SACN set nutritional recommended daily allowance's (RDA) as a generic guide. Furthermore, this study does not detail the potential detrimental effects of excessive iodine or selenium, which as was outlined in a study by Kaprara & Krassas (2006) excess iodine could provoke

hypothyroidism, and excess selenium could engender diarrhoea, haemorrhage, liver and kidney necrosis or respiratory disturbances. However one presumes Kawicka & Regulska-Ilow's increased micronutrient conclusion may have been influenced by the data that suggests Hashimoto's disease interferes with absorption in the gut which in turn effects the optimum absorption of micronutrients and cites research linking gluten with thyroid damage.

There does appear to be an increased incidence of coeliac disease with individuals with thyroid dysfunction as was highlighted in a systematic review by Sun et al (2016), which stated the accepted hypothesis that gluten mimics the molecular structure of thyroid tissue transglutaminase and therefore ingesting gluten could decrease thyroid function. However, this theory is contested in a recent review of existing literature conducted by Ihnatowicz et al, (2021) which did not support gluten exclusion for hypothyroid individuals, citing ambiguity and lack of evidence as well as intimating an increased risk of ingesting an inadequate diet, and the lack of nutritional quality to gluten free foods that for example may lead to even less selenium intake. Conversely, one study suggests a reverse relationship as participants with coeliac disease progressed to develop autoimmune diseases (Guidetti et al, 2001). Moreover a reciprocal relationship between gut microbiota changes and the development of Hashimoto's thyroiditis are also highlighted by Virili et al (2018) where evidence is analysed to suggest "progression of autoimmune thyroid disorders may be significantly affected from a changing intestinal microbial composition or even from overt dysbiosis".

Contradiction is also evident in research that investigated whether goitrogenic foods, found within the brassica family (such as broccoli and cauliflower) inhibited iodine uptake, and therefore would be detrimental to hypothyroid individuals. In one cross-over randomised intervention study (Bouga et al, 2015) the foods appeared to show no effect on TSH, T4 or T3 biomarkers. However another similar study (Di Dalmazi et al, 2021) which investigated how phytochemicals: cyanogenic glucosides, polyphenols, phenolic acids and alkaloids, interfered with thyroid function. This study *did* show that phytochemicals interfered somewhat with thyroid function, nevertheless an individual would have to ingest significantly large quantities to experience detrimental effects. Plausibly therefore misperceptions around foods could be created, and theoretically individuals may then avoid healthy, anti-inflammatory, anti-oxidant plant based foods due to perception of one aspect of research. Therefore conflicting research or lack of research certainty around the effects of many food nutrients may affect the quality of information disseminated within the hypothyroid community.

Nutritional behaviour

The potential consequences of the absence of knowledge and the contradictory research outlined above, may result in dissemination of a wide pool of opinion regarding what dietary choices hypothyroid adults should make. This makes dietary advice for hypothyroid clients particularly important. Feasibly a hypothyroid individual may feel unsure for example of the timings of eating when taking medication, or indeed what their estimated average requirement (EAR) of vitamins or minerals should be or perhaps whether they should be adopting a gluten free diet. Furthermore, unlike diabetic patients who receive nutritional advice from diabetic nurses and support groups to help maintain blood sugar levels and manage their condition, hypothyroid patients may rely on their GP's level of nutritional information. Disease management protocol is a yearly blood test with their GP to check T4 and TSH levels only (Nice guidelines, 2019), not to check for nutritional deficiencies or increase the patient's understanding of nutrition in relation to disease management. Therefore seeking nutritional advice will raise the client's knowledge level which impacts positively on their nutritional choices.

Client work will not only therefore look at food choices coherently, such as long term sustainable meal plans that fit into their lives, but also food habits. Equally, as was identified in Zanchini et al (2022) an individual's lifestyle, psychological and socio-economic position will affect what they choose to consume and why.

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