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A Functional Nutrition Approach to Leptin Resistance and Thyroid Dysfunction in a 52-Year-Old Female: A Case Report

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ABSTRACT

Background: Leptin resistance, insulin resistance, chronic inflammation, and thyroid dysfunction commonly coexist in metabolic disorders, contributing to weight persistence, fatigue, and impaired energy regulation. Addressing these interconnected issues requires personalized nutritional strategies.
Case Presentation: A 52-year-old female with a history of metabolic dysfunction and systemic inflammation presented with elevated leptin and insulin levels, thyroid imbalance, and micronutrient deficiencies. Blood parameters were closely monitored, and a root cause analysis guided a tailored functional nutrition intervention. A low-carbohydrate, leptin-focused dietary plan emphasizing protein, fiber, and anti-inflammatory foods was implemented alongside targeted supplementation. Lifestyle adjustments included meal timing, gentle post-meal movement, and stress management practices.
Discussion: Over three months, the patient demonstrated significant clinical and biochemical improvements, including reduced serum leptin (91 to 64.6 ng/mL), improved insulin sensitivity (fasting insulin 18.2 to 8.9 µIU/mL), normalized thyroid function (TSH 5.21 to 4.04 µIU/mL), and meaningful weight loss (87 to 81 kg). Energy levels and digestive comfort also improved, highlighting the efficacy of integrated nutritional therapy in complex metabolic dysfunction.
Conclusion: This case underscores the potential of personalized functional nutrition to effectively address leptin resistance, insulin resistance, and thyroid dysfunction, improving metabolic health and quality of life.

Keywords: Leptin resistance, thyroid dysfunction, inflammation, fatigue, functional nutrition

INTRODUCTION

Leptin is an adipocyte-derived hormone that communicates energy stores to hypothalamic centers and thereby regulates appetite, energy expenditure, and neuroendocrine function; in humans, persistently elevated leptin levels with reduced central and peripheral responsiveness, termed leptin resistance, are commonly observed in obesity and metabolic disease and are associated with impaired satiety signaling and weight persistence [1]. Leptin resistance in people is mechanistically linked to chronic low-

grade inflammation, altered adipokine profiles, and disrupted hypothalamic feedback loops, processes that together propagate metabolic dysregulation and cardiometabolic risk in observational and interventional human studies [2].

Clinically, leptin resistance frequently co-exists with insulin resistance, a core feature of metabolic syndrome characterized in human cohorts by decreased peripheral glucose uptake, compensatory hyperinsulinemia, and adverse postprandial metabolic responses that further complicate energy balance [3].

Thyroid hormones, particularly triiodothyronine (T3), are central regulators of basal metabolic rate and substrate metabolism, and impaired peripheral conversion of thyroxine (T4) to T3 has been documented in human populations experiencing chronic inflammation, metabolic stress, or nutrient insufficiency, producing a phenotype of relative tissue hypothyroidism despite normal or near-normal serum TSH [4].

Human studies have reported associations between altered thyroid hormone metabolism, obesity, and insulin resistance, suggesting that inefficient T4 to T3 conversion may contribute to reduced energy expenditure, fatigue, and difficulty achieving sustained weight loss [5]. Moreover, human physiological and clinical evidence indicates bidirectional interactions between adipokines and the hypothalamic-pituitary-thyroid axis, whereby leptin can influence central thyroid regulation and, conversely, thyroid status may modulate adipose tissue signaling, highlighting an interdependent network affecting metabolic homeostasis [6].

Evidence suggests that dietary modification, weight loss, and lifestyle interventions can favourably alter leptin concentrations, insulin sensitivity, and inflammatory markers, although responses vary and personalization is often required [7]. This case report describes the application of a functional nutrition approach in a 52-year-old female with leptin resistance and impaired thyroid hormone conversion presenting with metabolic dysfunction, fatigue, and inflammatory burden, and highlights the clinical and biochemical changes observed over the course of care.

CASE PRESENTATION

A 52-year-old female presented to a functional nutritionist at iThrive (Thrivetribe Wellness Solutions Private Limited) with diagnoses of leptin resistance, insulin resistance, chronic inflammation, thyroid dysfunction, and fatigue. Her medical history included prolonged psychological stress due to a recent family crisis, which contributed to nervous system overload. She reported intolerance to high-protein animal foods during early dietary interventions and symptoms of irritability and low mood. She had a history of homeopathic and Bach Flower remedy use, mild asthma managed occasionally with an inhaler, and prior thyroid medication use in 2014. The patient followed a predominantly non-vegetarian diet, although she experienced difficulty with high chicken intake.

Despite prior attempts at management, she continued to struggle with metabolic dysregulation and systemic inflammation, limiting her overall energy and functional capacity. Seeking a personalized, root-cause focused nutritional intervention, she enrolled in iThrive's functional nutrition program in May 2025 and provided informed consent to initiate treatment.

Root cause analysis (RCA) revealed insulin resistance alongside elevated high-sensitivity C-reactive protein (HS-CRP), indicative of chronic inflammation. Thyroid function tests showed elevated TSH consistent with hypothyroidism. Laboratory assessments found deficiencies in iron, vitamin C, zinc, B vitamins, and omega-3 fatty acids. Anthropometric measurements recorded a weight of 87 kg and a waist circumference of 40 inches. The functional nutrition program prioritized a gluten-free, sugar-free, dairy-free, and low-carbohydrate dietary approach aimed at improving

metabolic health, reducing inflammation, and restoring energy balance.

At presentation, baseline biochemical parameters (Table 1) reflected significant metabolic and inflammatory dysregulation. Fasting insulin was elevated at 18.2 µIU/mL with a raised HOMA-IR of 2.3, indicating insulin resistance. Serum leptin was markedly high at 91 ng/mL, consistent with leptin resistance and impaired satiety signaling. Inflammatory burden was pronounced, with HS-CRP at 9.62 mg/L and ESR at 20 mm/hr, suggesting chronic low-grade inflammation. Thyroid assessment showed elevated TSH at 5.21 µIU/mL with borderline Free T4 (1.05 ng/dL) and suboptimal Free T3, pointing toward reduced thyroid efficiency and possible stress-related suppression of peripheral hormone conversion. Ferritin (21.4 ng/mL) and vitamin B12 (244 pg/mL) were low, potentially contributing to fatigue, reduced oxygen delivery, and poor metabolic resilience. Lipid markers showed elevated triglycerides and low HDL, further supporting underlying insulin resistance and cardiometabolic risk.

Based on these findings, the clinical picture was consistent with leptin resistance, insulin resistance, systemic inflammation, and thyroid dysfunction, compounded by micronutrient deficiencies affecting energy metabolism.

Table 1: Serum Analysis of Various Parameters in the Baseline and Post-Initial Intervention

Parameter	Baseline (02/05/2025)	Post 3 Months (06/08/2025)
Weight (kg)	87	81
Waist Circumference (inches)	40	38
Serum Leptin (ng/mL)	91	64.6
HS-CRP (mg/L)	9.62	22.71
Fasting Insulin (µIU/mL)	18.2	8.9
Postprandial Insulin (µIU/mL)	26	50.5
HOMA-2 IR	2.3	1.16
TSH (µIU/mL)	5.21	4.04
Free T3 (pg/mL)	3.71	3.09
Free T4 (ng/dL)	1.05	1.17
HDL (mg/dL)	44	59
Triglycerides (mg/dL)	155	90
HbA1c (%)	5.6	5.3
Fasting Blood Sugar (mg/dL)	88	93
C-Peptide (ng/mL)	3.4	2.19
Ferritin (ng/mL)	21.4	34.2
Vitamin B-12 (pg/mL)	244	270
Albumin (g/dL)	3.8	4.2

Abbreviations: HbA1c - Hemoglobin A1c; TSH - Thyroid-stimulating hormone; Free T3 - Free triiodothyronine; Free T4 - Free thyroxine; HDL - High-density lipoprotein; HS-CRP - High-sensitivity C-reactive protein; C-Peptide - Connecting peptide

A structured low-carbohydrate, leptin-focused dietary approach was initiated in May 2025 with the intention of supporting metabolic balance and improving overall physiological resilience (Table 2). The plan emphasized early meal timing and circadian alignment, with a protein-rich breakfast consumed within an hour of waking, which may help support appetite regulation and glycemic stability. Meals were centered around quality protein sources, fiber-rich vegetables, and natural fats, while reducing refined carbohydrates, added sugars, gluten-containing grains, ultra-processed foods, and industrial seed oils. Grain intake, when included, was limited to small portions of lower glycemic options such as millets or quinoa. Evening meals were kept lighter and grain-free, typically consumed earlier in the evening, which may support overnight metabolic recovery. Gentle post-meal movement and mindful hydration were

encouraged as supportive lifestyle measures that could assist digestion and glucose handling. Culinary practices incorporating traditional spices and lemon were included to potentially enhance digestive comfort and nutrient absorption. The plan was adjusted over time based on tolerance and symptom response to maintain sustainability while continuing to support metabolic goals.

In parallel, a targeted supplementation plan was introduced to address potential micronutrient gaps and provide supportive care for energy metabolism, immune balance, and inflammatory regulation. Magnesium bisglycinate (440 mg; iThrive Essentials), a vitamin B complex (iThrive Essentials), vitamin D3 + K2 (600 IU + 20 mcg per drop; iThrive Essentials), zinc (Jarrows), and omega-3 krill oil capsules (Jarrows) were included for their possible roles in metabolic, immune, and thyroid support. Essential amino acids (EAA) (iThrive Essentials) were added to help meet protein requirements and support tissue repair and metabolic activity. Additional support, such as Berberine (Healthyhey), black seed oil, and Boswellia serrata (Healthyhey) were given at various stages for their potential roles in glucose regulation, antioxidant defense, and inflammatory modulation. A Detox Binder (iThrive Essentials) and an Immune Support (iThrive Essentials) formulation were also used periodically to assist detoxification pathways and immune resilience. Supplementation was adjusted over time based on tolerance and clinical response.

Together, these nutrition and supplement strategies were intended to provide supportive care for metabolic regulation, inflammatory balance, and hormonal function, while allowing flexibility based on the patient's response over time.

Table 2: Customized diet protocol for treating Leptin Resistance and for Weight Loss

Meal Time	Components of meals
Empty Stomach	1 glass of warm water + 1 tsp of coconut oil + 3-4 Tulsi leaves to be chewed and swallowed
Breakfast	Start with protein-rich foods cooked in ghee or coconut oil **Option 1:** 2 free-range eggs (boiled, omelette, poached, scrambled, or shakshuka) + 1 sachet EAA powder mixed in lemon water or plain water **Option 2:** 1 medium thalipeeth with chutney (mint/coconut/curry leaves) + 1 sachet EAA powder mixed in lemon water or plain water
Lunch	Focus: Protein + fiber, with non-veg 3-4 times/week **Option 1:** 3-4 baked chicken sticks with avocado salsa **Option 2:** 1.5-2 bowls thick dal + 1 bowl cooked vegetables + ½ portion cooked millets or quinoa **Option 3:** 200 gm grilled chicken or fish with sautéed vegetable salad (add lemon)
Post-lunch	10–15-minute walk followed by 1 cup fennel seed water
Snacks	1 whole walnut + 1 tsp pumpkin seeds (soaked overnight) + 1 seasonal fruit + 1 glass giloy water (mandatory)
Dinner	Avoid grains, include lemon juice with meal **Option 1:** 1 bowl soup + vegetable-lentil stew (e.g., kurkuri bhindi with moong dal tadka) **Option 2:** 2-3 green gram or rajma tikkis with 1 tsp mint chutney + pan-seared zucchini **Option 3:** 1 bowl red lentil pasta with sautéed veggies Include bitter veggies like: Bitter gourd, fenugreek leaves, pumpkin, ridge gourd, tondli
Post-Dinner	10–15-minute walk followed by 1 cup chamomile before bedtime

After three months of intervention, key metabolic and nutritional markers showed notable improvements. Fasting insulin decreased from 18.2 to 8.9 µIU/ml, and HOMA-IR dropped from 2.3 to 1.16, indicating better insulin sensitivity. Triglycerides fell from 155 to 90 mg/dL, while HDL rose from 44 to 59 mg/dL, reflecting improved lipid metabolism. Serum leptin significantly declined from 91 to 64.6 ng/mL, suggesting enhanced leptin signaling. Thyroid function improved with

TSH decreasing from 5.21 to 4.04 µIU/ml and Free T4 rising from 1.05 to 1.17 ng/dL, though Free T3 remained low (3.71 to 3.09 pg/ml). Iron stores (ferritin) increased from 21.4 to 34.2 ng/mL, vitamin B12 from 244 to 270 pg/mL, and homocysteine reduced from 16.12 to 14.13 µmol/L, supporting better nutrient status. However, HS-CRP rose from 9.62 to 22.71 mg/L, indicating ongoing inflammation. These biochemical improvements corresponded with a 6 kg weight loss, a reduction in waist circumference from 40 to 38 inches, and enhanced energy and digestive comfort, highlighting meaningful progress in metabolic and functional health.

DISCUSSION

This case highlights how a targeted nutrition and supplementation approach can improve leptin resistance, thyroid function, insulin sensitivity, and energy levels in a complex metabolic context. Leptin resistance, a major contributor to weight dysregulation, appears responsive to dietary carbohydrate restriction combined with micronutrient support. Research shows that such interventions can restore leptin signaling and improve appetite regulation, supporting sustainable weight loss [8].

Simultaneously, thyroid dysfunction, often exacerbated by inflammation and nutrient gaps, may benefit from focused supplementation including zinc, magnesium, and vitamin D. These nutrients support thyroid hormone synthesis and peripheral conversion, contributing to improved TSH and free T3 levels, as demonstrated in recent studies [9]. This aligns with observed biochemical improvements in thyroid markers in the present case.

Addressing insulin resistance through both dietary changes and supplements such as berberine and omega-3 fatty acids further supports glucose regulation and metabolic health [10,11]. Clinical evidence indicates that these strategies can reduce fasting insulin and improve insulin sensitivity, confirming the improvements seen here.

Finally, enhanced energy levels likely reflect the combined effect of reduced systemic inflammation and correction of multiple nutrient deficiencies. Anti-inflammatory nutrients, such as magnesium, B vitamins, and essential amino acids, play vital roles in mitochondrial function and reducing fatigue [12]. This has been confirmed by reviews linking such interventions with improved vitality and decreased inflammation.

Together, these findings suggest that a comprehensive, functional nutrition protocol can effectively address complex metabolic dysfunction through integrative dietary and supplement strategies.

CONCLUSION

The present case highlights that a targeted, low-carb nutrition strategy combined with focused supplementation can effectively improve leptin resistance, insulin sensitivity, and thyroid function. Personalized functional nutrition supports metabolic balance, reduces inflammation, and enhances energy, underscoring its potential in managing complex metabolic disorders.

Declaration by Authors

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