Understanding the Role of Thyroid Hormones, Sex Hormones, and their Stimulating Hormones in Non-Alcoholic Fatty Liver Disease

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Abstract

Metabolic syndrome frequently manifests as Non-Alcoholic Fatty Liver Disease (NAFLD). Endocrine hormones in addition to lifestyle choices are involved in the dysregulation of hepatic metabolism. Changes in the levels of thyroid hormones (THs), primarily in subclinical hypothyroidism, and sex hormones are the most frequent endocrine hormones causing metabolic syndrome (in menopause). These hormonal adjustments impact hepatic lipid and glucose metabolism and may lead to an increase in hepatic fat deposition. The effects of sex hormones, or THs, and their corresponding stimulating hormones, Thyroid-Stimulating Hormone (TSH) and Follicle-Stimulating Hormone (FSH), on the onset of hepatosteatosis are compared in this article. FSH and TSH might be when metabolic alterations were discovered while only stimulating hormone levels were aberrant and peripheral hormone levels were still within the normal range, they are more pertinent to the dysregulation of hepatic metabolism than the peripheral hormones. Increased TSH and FSH levels seem to function separately from one another while also having additive effects on the development of NAFLD.

Keywords: Thyroid-stimulating hormone • Follicle-stimulating hormone • Metabolic syndrome • Hypo- thyroidism • Menopause • Metabolic dysfunction- associated fatty liver disease

Introduction

Insulin Resistance (IR) is the primary pathophysiological mechanism in Non-Alcoholic Fatty Liver Disease (NAFLD), which is frequently regarded as the hepatic manifestation of Metabolic Syndrome (MetS). NAFLD is characterise d as fatty liver in persons who do not consume alcohol and in whom other disorders have been ruled out. In the general community, NAFLD affects 25% of people globally. Recently, it was proposed to use the term Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) in favor of the term NAFLD [1, 2].

In order to encompass all activity and fibrosis phases and avoid basing a diagnosis on the exclusion of another illness, the name MAFLD was proposed. Patients with MAFLD are identified as having the fatty liver disease in various stages as well as concomitant conditions including type 2 diabetes and obesity. Metabolic dysregulation and Type 2 Diabetes (T2DM). The advantage of utilising MAFLD over NAFLD, however, is still up for debate because MAFLD criteria exclude a particular group of NAFLD patients. Compared to MAFLD+/NAFLD+ patients, these MAFLD/NAFLD+ patients were younger. The great majority of research that is currently available is based on people with NAFLD [3,4]. Males have more advanced levels of NAFLD, and NAFLD is sexually dimorphic.

The primary cause of NAFLD was formerly thought to be hypothyroidism, but subsequent research cast doubt on this finding and suggested that other variables, including sex hormones, may also play a role. The effects of sex hormones (oestrogen) and Thyroid Hormones (THs) are compared in this review.

Testosterone on hepatic metabolism, as well as the effects of their respective stimulating hormones, Thyroid-Stimulating Hormone (TSH) and Follicle-Stimulating Hormone (FSH). Also highlighted will be how FSH and TSH may combine to disrupt hepatic metabolism. Depending on the diagnostic criteria employed, up to 90% of individuals with NAFLD also have MetS, which is highly linked with the development of NAFLD [5]. Insulin Resistance (IR) and hepatosteatosis may result from the activation of Protein Kinase C (PKC) by a high-fat diet, together with elevated levels of *fetuin B, selenoprotein P,* and Fibroblast Growth Factor 21 (FGF21). However, IR has a role in liver injury. However, a number of research imply that NAFLD and MetS are not connected because diacylglycerol O-acyltransferase 2 is overexpressed (DGAT2) led to pronounced hepatosteat osis, although glucose and insulin levels were unaffected. Similar to this, choline shortage, hyperbetalipoproteinemia, and Lysosomal Acid Lipase (LAL) deficiency all solely resulted in hepatosteatosis and did not affect insulin sensitivity. NAFLD is characterised by greater FA intake (uptake from plasma or lipogenesis) than outflow FA oxidation and secretion of Triglycerides (TGs) as VLDL and is defined as Fatty Acid (FA) infiltration of 5% hepatocytes. Free Fatty Acids (FFAs) in plasma accounted for 59% of the liver's total TG production, de novo hepatic lipogenesis produced 26 percent, and diet contributed 15%. The propensity for NAFLD is hypothesised to result from prompting.

Conclusion

Both sexes are affected by the effects of estrogens and THs on hepatic metabolism, however women are more commonly affected by these hormones than males are. It is challenging to distinguish between the impact of the stimulating hypophyseal hormones and the activity of the peripheral hormones, making the identification of relationships between NAFLD and certain hormonal alterations challenging. The incidence of hypothyroidism in boys and females is making it challenging to evaluate the impact of THs independently of sex hormones. The fact that E2 generated in the ovary and in the adipose tissues was proven to behave differently and creates confusion when interpreting oestrogen effects.

Further talks include estimates of a somewhat stable reference range for TSH levels. Despite these restrictions, it can be said that distinct hepatic metabolic regulatory patterns were seen for THs, sex hormones, and the associated stimulating hormones. Catabolic and anabolic processes in the metabolism of glucose, lipids, and cholesterol are stimulated by TH activity, whereas anabolic processes in the metabolism of glucose, lipids, and cholesterol are stimulated by TSH (uptake and excretion). Estrogens encourage cholesterol recycling, lipid catabolism, and the production of glycogen. Because a U-shaped dose-dependency is thought to exist, the role of androgen is less clearly understood.

FSH plays an anabolic role in the metabolism of cholesterol and glucose. Explain why post-menopausal women with higher FSH levels had greater gluconeogenesis and cholesterol production whereas males with lower FSH levels had relatively little change. In post-menopausal women, elevated TSH levels plus elevated FSH levels may have a synergistic effect on gluconeogenesis, cholesterol buildup, and lipogenesis. Because TSH levels peak in the late night/early morning and FSH levels peak in the afternoon, the discrepancies in circadian levels may suggest an independent action. The concept of separate and additive effects of both hormones may be supported by age-dependent variations in TSH and FSH levels as well as NAFLD prevalence.

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