Preventing the Development and Progression of Non-Alcoholic Fatty Liver Diseases through an Anti-Inflammatory Dietary Approach

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Abstract

NAFLD (Non-Alcoholic Fatty Liver Disease) is a growing global health concern linked to insulin resistance, increased visceral fat accumulation, and cardiovascular issues. Sedentary behaviour, persistent stress, an obesogenic environment, and a Western-style diet are all major contributors to the development and progression of this condition. The diet, in particular, is quite important. An unhealthy diet rich in red and processed meats, refined carbs, simple sweets, highly processed foods with food additives, and conservatives is fueling a low-grade inflammatory response. If additional risk factors are present, metabolic and hormonal imbalances may develop, resulting to a rise in visceral obesity, gut dysbiosis, and leaky gut, all of which contribute to the inflammatory fire. As a result, lifestyle changes are the most effective way to reduce inflammatory processes. The GLykLich diet, which comprises whole and unprocessed foods and is anti-inflammatory and low-glycemic, may minimise the risk of increased morbidity and death. The GLykLich diet recommends a meal that is high in secondary plant products and contains complex carbohydrates (fibre), high-quality protein, and healthy fats (DHA/EPA). There is no one vitamin that can stop NAFLD from progressing; rather, it is the combination of elements found in whole, unprocessed diets that help to decrease inflammation, restore metabolic function, and thereby reverse NAFLD.

Keywords: Anti-Inflammatory Diet NAFLD • Obesity • Type-2 Diabetes Mellitus

Introduction

NAFLD (Non-Alcoholic Fatty Liver Disease) is a growing health concern across the world. Metabolic-Associated Fatty Liver Disease (MAFLD) is characterised by increasing visceral fat accumulation, insulin resistance, and cardiovascular issues. Problems in lifestyle, such as nutrition, can help to prevent and cure metabolic changes. There are currently no dietary guidelines for NAFLD treatment and related co-morbidities.

Silent inflammation, or low-grade systemic chronic inflammation, is the underlying cause of all metabolic disorders and is produced by an unhealthy lifestyle that includes chronic stress, an obesogenic environment, and a Western-style diet. As a result, it aids in the development and progression of NAFLD. The major causes of excessive fat storage in the liver are thought to be silent inflammation and insulin resistance.

Diet plays an important role, and a bad diet is linked to all of the lifestyle illnesses stated above; particular foods may contribute to inflammatory processes. The dietary pattern and risk of developing NAFLD were examined in a study of 375 Iranian people.

The scientists discovered that a Western-style diet (which included soft beverages, processed meats, high-fat dairy items, hydrogenated fats, refined grains, and sugary sweets) was linked to a greater risk of NALFD. A Western pattern diet was linked to an increased prevalence of NAFLD in a 1995 study of Australian teenagers aged 14 to 17 years old (n = 995). High consumption of red and processed meats, refined carbs, saturated fats and industrial oils, as well as food additives and conservatism, are all factors. Low-grade inflammation is generated by such "inflammatory" substances.

If these inflammatory foods are ingested on a regular basis and are combined with additional risk factors such as a lack of activity and sleep, as well as chronic stress and regular alcohol or nicotine usage, metabolic and hormonal changes may ensue. As a result of this combination, visceral fat mass, gut dysbiosis, and leaky gut develop. Inflammatory indicators such as adipokines and cytokines, as well as endotoxin and other inflammatory bacterial by-products, are fed into the circulation by adipose tissue and the gut, stoking the fire of the inflammatory process. Adipokines like leptin, as well as fat-specific cytokines like resistin, chemerin, and IL-6, are released by visceral adipose tissue and are linked to insulin resistance and abnormal fat storage in the liver. Inflammatory mediators such as Lipopolysaccharides commonly known as endotoxins, are abundant in the gut microbiome. Advances in biochemical sequencing tools have allowed researchers to get a better understanding of the bacterial community during the previous decade. Researchers have discovered a unique gut microbial profile linked to NAFLD, in which certain bacteria strains are more prevalent (Escherichia coli, Cutibacterium acnes) or less abundant (Clostridium coccoides, Bacteroides fragilis) in individuals with metabolic fatty liver. Dysbiosis, or an altered microbiome with an abundance of pathogenic gut bacteria, can damage gut integrity and cause the tight junctions to weaken. As a result, pathogenic bacteria's poisonous and inflammatory compounds, such as endotoxin, can now pass through the porous gut barrier and enter circulation. Endotoxin causes inflammation and liver damage by activating Kupffer cells, which are liver-resident macrophages. Endotoxemia has been demonstrated to be linked to both insulin resistance and the severity of NAFLD histologically. As a result, the two primary causes of systemic inflammation are large visceral adipose tissues and altered gut. Insulin resistance, which is a pathophysiological characteristic of NAFLD, can be reversed with a simple lifestyle change. Several studies have indicated that a low-carb or ketogenic diet can improve insulin sensitivity and slow the course of T2DM in people who already have it. In general, a diet low in refined grains and free sugars and high in fibre and complex carbs from wholegrains and starchy vegetables can help to keep insulin and blood glucose levels in check. Furthermore, low-glycemic diets feed beneficial bacteria like Lactobacillus, Bifidobacterium, Clostridium, and Akkermansia, which have been shown to suppress pathogenic bacteria, provide energy for enterocytes, support gut lining, and support mucus production in part by producing Short Chain Fatty Acids (SCFAs). SCFAs can enter the bloodstream and reduce inflammation, improve insulin sensitivity, decrease fatty acid production, and aid detoxification in a variety of tissues (e.g., liver, adipose, muscle, and brain). Diet appears to be the most effective way to reduce inflammation, raising the question of whether conservative treatment might slow or perhaps reverse the course of fatty liver disease. There are currently no standard guidelines or diet recommendations for the treatment of NAFLD patients. Through systemic inflammation and metabolism, as well as therapeutic dietary therapies for weight reduction, NAFLD and good eating habits are linked. In the case of hepatic steatosis, many diet schemes have been advocated, such as the Mediterranean diet. The Mediterranean diet has been linked to a lower risk of cardiovascular and metabolic disorders. This can be explained in part by the anti-inflammatory and anti-oxidative properties of olive oil, nuts and seeds, legumes, and vegetables, as well as fish, which are high in fibre, unsaturated fatty acids, and secondary plant compounds.

The GLykLich diet goes even farther, focusing on natural, unprocessed foods, complex carbohydrates (fibre), high-quality protein, anti-inflammatory fats, and secondary plant products. These nutrients diminish de novo lipogenesis, enhance insulin sensitivity, boost satiety, modify gut microbiota, and reduce oxidative and inflammatory processes in the body as a whole. Food with a high glycemic index raises insulin levels, which leads to an increase in inflammation and de novo lipogenesis, which produces saturated fat from carbohydrates and contributes to aberrant fat buildup in hepatocytes. Dietary fibre, on the other hand, is inversely related to the incidence of NAFLD and has favourable effects on fat and cholesterol digestion as well as gut microbiota regulation. Dietary influences on gut microbiota change have an impact on immune system modulation. A recent study revealed some surprising findings. Fructose, on the one hand, stimulates a transcriptional pathway in hepatocytes that drives de novo lipogenesis, according to the scientists. Fructose, on the other hand, is digested by particular gut microorganisms, which produces acetate, which is used to fuel hepatic fatty acid production. Other SCFAs produced by the microbiota include butyrate and propionate, in addition to acetate. It's still unclear how much these SCFAs contribute to lipogenic processes in hepatocytes.

The anti-inflammatory impact of fat, which is determined by the degree of saturation of fatty acids, influences the risk of NAFLD. Both Monounsaturated and Polyunsaturated fatty acids have anti-inflammatory properties. The MUFA oleic acid is abundant in olive oil, which is extensively consumed in the Mediterranean diet. Olive oil is also high in phenolic secondary plant compounds including tyrosol and oleuropin. These bioactive compounds have been proven to provide a variety of health advantages, including cardioprotective, anti-oxidative, anti-cancerous, and microbiota regulator capabilities, in addition to anti-inflammatory qualities. PUFA omega-3 fatty acids, particularly Alpha-Linolic Acid (ALA), are abundant in nuts, seeds, and their oils. Eicosapentaenoic Acid and Docosahexaenoic Acid are two additional omega-3 fatty acids found mostly in fatty fish such as salmon, tuna, mackerel, and herring. Resolvins, which bring about a planned resolution of the inflammatory process, are precursors for EPA and DHA. Protectins have anti-inflammatory and neuroprotective properties, and DHA acts as a precursor for their production. Although ALA may be converted to EPA and DHA, only a tiny amount of it is, with around 5% being converted to EPA and less than 1% being converted to DHA. Most persons are deficient in EPA or DHA due to the poor rate of conversion of ALA to EPA and DHA, as well as a usually low ingestion of these omega-3 fatty acids. As a result, it's not unexpected that people with NAFLD have low EPA and DHA levels. In mice, high-fat diet causes endotoxemia, inflammation, insulin resistance, and hepatic steatosis by increasing the number of endotoxin-producing gut bacteria. These alterations were caused in the mice by feeding them saturated fat rather than polyunsaturated fat, and they were reversed following antibiotic therapy. This implies a causal relationship between saturated fat consumption, gut microbiota, and metabolic inflammation.

Human investigations yielded similar results: For three weeks, 38 healthy overweight people were given either saturated or unsaturated fat.

Discussion

Endotoxemia, adipose tissue inflammation, and insulin resistance were all caused by the saturated fat-rich diet. In rats, nonhuman primates, and humans, fructose ingestion has been demonstrated to increase bacterial endotoxin concentrations and signs of liver damage, in addition to saturated fat. This shows that the risk of NALFD is determined by the quality of macronutrients ingested. There is currently no authorized medication for Non-Alcoholic Fatty Liver Disease (NAFLD); the current treatment cornerstone for NAFLD is a lifestyle intervention with an emphasis on food. There is no one vitamin that may stop NAFLD from progressing; rather, it is the combination of elements found in whole, unprocessed diets that can lower inflammatory processes, enhance metabolic function, and so reverse NAFLD. Micronutrients include vitamins, minerals, and secondary plant compounds operate as cofactors and regulators in a variety of metabolic and inflammatory events. As a result, it's vital that we get enough of those powerful compounds in our diet. The quality of food processing and cattle farming should be considered, since these factors may influence the quantity of anti-inflammatory and anti-oxidative elements present. B-vitamins are abundant in animal-based foods such as lean beef, poultry, and fish. Vitamins are micronutrients that are required for good health but cannot be produced enough from other molecules. The role of dietary vitamin composition and liver fat storage has been underlined in recent research. Vitamins control a number of enzymatic activities in the liver, and changes in vitamin metabolism are thought to have a role in the evolution of NAFLD. Vitamins C and E have anti-oxidant properties and have been linked to a reduction in hepatocyte damage. Vitamin D, vitamin B12, B3 (niacin), and folate levels in the blood are all closely linked to the severity of NAFLD. Vitamin B3 is a precursor of the lipid-metabolizing coenzymes Nicotinamide Adenine Dinucleotide and Nicotinamide Adenine Dinucleotide Phosphate (NADH/NADPH). Niacin therapy for dyslipidemic individuals lowers plasma triglyceride levels and hepatic fat content, as well as improving liver functions like hepatic transaminase. The liver is a major storage location for vitamin B12, which is required for DNA synthesis and repair. Low vitamin B12 levels in pregnant women's diets result in greater rates of obesity and T2DM in their children, as well as alterations in hepatic gene expression associated with lipid metabolic pathways. Vitamin B12 reconstitution might be used to reverse these changes in the children. Changes in mineral concentrations, such vitamins, are linked to the development of NAFLD (i.e., zinc, iodide, and selenium). Zinc has a vital role in immunomodulation and antioxidative responses. Furthermore, it is involved in the insulin metabolism (synthesis, storage, secretion and signaling). Zinc deficiency in rats increases oxidative stress in hepatocytes and is accompanied with abnormal lipid buildup in liver tissue. However, no comprehensive clinical research has been conducted to demonstrate the direct role of zinc in the development of NAFLD in people.

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