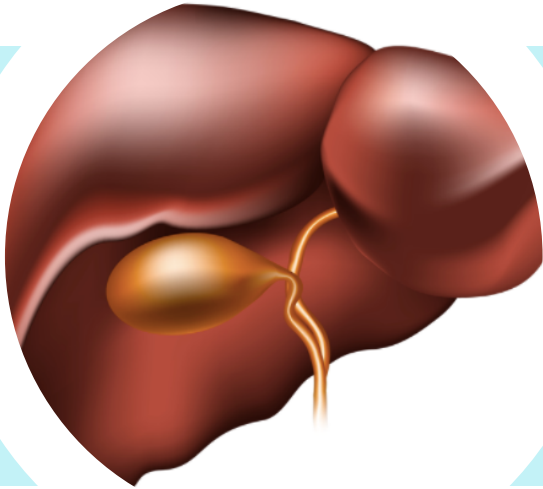


NUTRITION GENOME

12 WEEK PRACTITIONER TRAINING



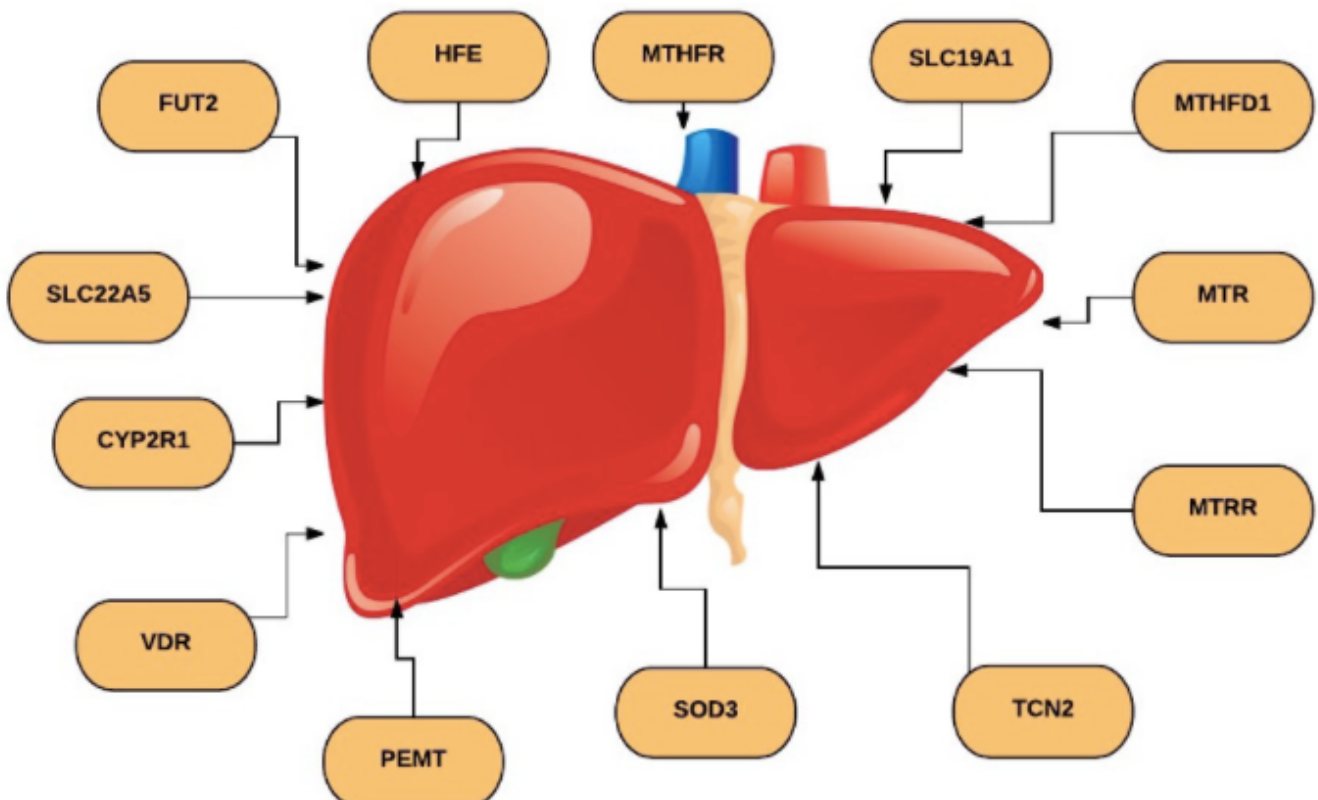
WEEK 2, TOPIC 4: CLINICAL CONNECTION TO NON-ALCOHOLIC FATTY LIVER DISEASE

For Further information, or to purchase a DNA Kit
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Non-Alcoholic Fatty Liver Disease

Currently, 25 percent of people in the U.S. are living with non-alcoholic fatty liver disease (NAFLD). NAFLD is largely asymptomatic before it begins causing problems. This disorder is often looked at in connection with obesity, sugar consumption and elevated triglycerides, however, not everyone with fatty liver will match this profile. The Nutrition Genome Report can give you a full profile of genes to determine the major contributing causes of fatty liver so you can check off each box.



Digestion Section:

Gut Flora (FUT2): By regulating liver fat deposition and energy homeostasis, gut microbiota may also play a role in non-alcoholic fatty liver disease (NAFLD) pathogenesis. Patients with biopsy-proven NAFLD have increased gut permeability and small intestinal bacterial overgrowth (SIBO), which play an important role in the alteration of liver fat metabolism. Pilot studies based on the theory that NAFLD may be linked to small bowel bacterial overgrowth have shown some promise with the use of probiotics and prebiotics.

L-Carnitine Gene (SLC22A5): If your patient has poor L-Carnitine production, a high-fat diet may put more stress on these enzymes and create fat oxidation without adequate L-Carnitine, magnesium, vitamin C, and B-vitamins. In a randomized controlled trial, L-Carnitine was found to improve steatosis, NAFLD histologic activity score and aminotransferases.

Vitamin D (CYP2R1, VDR): Emerging evidence suggests that vitamin D may play a role in the pathogenesis of NAFLD. In a recently published meta-analysis, NAFLD subjects were 26% more likely to be vitamin D deficient compared to controls. Numerous publications propose that low levels of vitamin D may contribute to the development of insulin resistance, metabolic syndrome and more recently NAFLD. Pesticides have also been linked to suppressing vitamin D levels and creating a vitamin D deficiency. Low PON1 gene (Cardiovascular Section) function and low vitamin D levels could be contributing to NAFLD.

Iron (HFE): Excess iron is observed in approximately one-third of NAFLD patients.

Digestion, Methylation and Antioxidant Section:

Folate (MTHFR, SLC19A1, MTHFD1): High homocysteine is correlated to NAFLD.

Choline (PEMT): In humans, polymorphisms in PEMT are associated with NAFLD, suggesting that the methylation function of choline is important in the mechanism of NAFLD. Even if a patient exhibits normal weight and triglycerides, a long-standing choline deficiency can still induce fatty liver.

B12 (FUT2, MTR, MTRR, TCN2): Studies have found that levels of vitamin B12 and folate were statistically lower in NAFLD patients.

Oxidative Stress (SOD3 Zinc/Copper, PEMT): According to research, increased oxidative stress is considered a key trigger in the pathogenesis of NAFLD. Oxidative stress has been hypothesized to contribute to the progression of NAFLD to NASH and to worsen insulin resistance. Research shows low copper as a potential cause of NAFLD. Choline is an important part of the cell membrane and mitochondrial dysfunction is a central mechanism in the pathogenesis of NAFLD. Low choline increases the sensitivity to carcinogenic chemicals due to poor cell membrane support. In excess amounts, NSAID's like acetaminophen build up in the liver and obstructs pathways in the liver, compounding issues.

Hormone Section:

Sex Hormone Binding Globulin (SHBG): Having SHBG levels that are too low in men are associated with metabolic syndrome, obesity, insulin resistance, lipid abnormalities, and chronic high blood pressure. Studies have indicated that low SHBG levels are associated with NAFLD in men.

Thyroid Health (D101): Hypothyroidism is linked to NAFLD.

Summary

A genetic analysis can enable you to be incredibly thorough for approaching fatty liver disease, ensuring a well-rounded approach for your patients.