nicole flynn
NUTRITION + WELLNESS

**Managing Dyslipidemia from a Nutritional Perspective**

Dyslipidemia refers to abnormal levels of lipids in the blood and is associated with various forms of disease. There are three biomechanical measurements involved in dyslipidemia: lipoproteins, cholesterol and triglycerides. According to the Centers for Disease Control and Prevention (2023), about one-third of U.S. adults have some form of dyslipidemia. Dyslipidemia occurs when levels of any of the three measurements are abnormal. Hyperlipidemia occurs when both LDL and triglycerides are elevated.

**Dyslipidemia Risks**

Dyslipidemia is a risk factor for cardiovascular disease (CVD), the leading cause of death in the United States. Worldwide, nearly 18 million deaths per year are attributable to CVD, a major risk factor for coronary artery disease, heart attacks, and strokes. In addition to CVD, dyslipidemia is associated with atherosclerosis, a condition whereby plaque formation on arterial walls leads to the narrowing and hardening of arteries. Dyslipidemia is also considered a major contributing factor of metabolic syndrome, increasing the risk of type 2 diabetes, and is associated with non-alcoholic fatty liver disease and pancreatitis.

**Cholesterol is Essential to Human Metabolism**

Cholesterol plays an important role in human metabolism. It is essential for the normal function of all human cells. It is a primary component for the normal structural makeup and the fluidity of cell membranes. Cholesterol is necessary to intracellular transport, nerve condition and signalling pathways. It serves as a precursor to steroid hormones (cortisol, aldosterone, adrenal androgens) and sex hormones (testosterone, estrogen, progesterone) and is a precursor to Vitamin D and bile acids.

Cholesterol is lipophilic (fat loving) and does not dissolve well in blood. Accordingly, it must be attached to lipoproteins in order to travel through the blood and into cells. There are five classes of lipoproteins: chylomicrons, very-low density lipoproteins (VLDL), intermediate density lipoproteins, low density lipoproteins (LDL) and high density lipoproteins (HDL).

**Cholesterol and the Liver**

Cholesterol is so vital to human health that the human body synthesizes 80% of serum cholesterol. Only about 20% comes from dietary cholesterol. The liver is the primary site of cholesterol production. The process begins with acetyl-CoA, which combines to form

HMG-CoA. HMG-CoA is converted by HMG-CoA reductase to mevalonate and then continues through a series of steps until it eventually becomes cholesterol. The liver tightly controls cholesterol synthesis based on the body's needs. Accordingly, if serum cholesterol levels are high, it is necessary to investigate factors causing the liver to produce such levels.

**Triglycerides, LDL and HDL**

Triglycerides are the most common type of fat in the body. Approximately 97% of dietary lipids are in the form of triglycerides and come from foods like butter and oils. Triglycerides are also synthesized when excess calories are consumed and cannot be used for immediate energy. During this process, referred to as *lipogenesis*, the body breaks down unneeded glucose and converts it to Acetyl-CoA. Acetyl-CoA is then used to synthesize cholesterol (and other fatty acids) in the liver. Triglycerides are then packaged in VLDL and carried into the tissues, where they are stored in fat cells. Elevated triglycerides are an independent risk factor for cardiovascular disease.

LDL and HDL are the primary cholesterol carriers in the blood. LDL is responsible for transporting cholesterol from the liver to the cells, while HDL is responsible for returning fats to the liver. Elevations of LDL are associated with atherosclerosis, the primary cause of a heart attack or stroke. Higher levels of HDL are associated with a lower risk of heart attack.

Lipoprotein(a) is a type of LDL that includes an additional protein. Elevated levels of Lp(a) are associated with a higher risk of cardiovascular disease, particularly in those with a family history of heart conditions. Lp(a) levels are primarily determined by genetics and are less responsive to diet and lifestyle changes.

Apolipoprotein B is the main protein found in VLDL, IDL, LDL and Lp(a) that can lead to plaque formation in the arteries. Measuring Apo B provides a more accurate assessment of the number of atherogenic particles in the blood stream because there is one Apo B molecule on the surface of every potentially atherogenic particle. Therefore, an Apo B measurement provides a direct count of the total number of potentially atherogenic particles, while a lipid panel only calculates the number of LDL particles present. In general, the normal range for Apo B is between 60 and 120 mg/dL, while optimal levels are below 80 mg/dL.

In addition to Apo B, plasma cholesterol levels, along with levels of LDL, HDL and serum triglycerides are used as biomarkers of the lipid profile. Recommended levels are as follows:

*Recommended Cholesterol and Triglyceride Levels*

| | LEVEL (MG/DL) | RESULT |
|---|---|---|
| Total Cholesterol | <200<br>200-239<br>>240 | Desirable<br>Borderline<br>High |
| LDL | <100<br>100-130<br>130-159<br>>160 | Desirable<br>Borderline<br>Borderline high risk<br>High Risk |
| HDL | <40<br>41-59<br>>60 | Low (undesirable)<br>Normal<br>Desirable |
| Triglycerides | <150<br>150-199<br>200-499<br>>500 | Desirable<br>Borderline high<br>High<br>Very high |

Ratios, rather than absolute targets, are often used to assess cardiac risk because they reflect whether cholesterol is being deposited into the tissues or broken down and excreted. The total cholesterol to HDL ratio should be no higher than 4.2 and the LDL to HDL ratio should be no higher than 2.5. The risk for heart disease can be reduced by lowering LDL levels while simultaneously raising HDL levels.

**What are the Underlying Causes of High Cholesterol Levels?**

Elevated cholesterol levels are often due to dietary and lifestyle factors; however, there may also be genetic factors and nonmodifiable risk factors.

Traditional medicine identifies the following factors as contributors to abnormal cholesterol levels:

- Diet and Lifestyle
    - Lack of physical activity
    - Consuming too many saturated fats
    - Smoking
    - Stress
    - Drinking too much alcohol

    - Lack of Sleep
- Family History
- Medical Conditions
- Medications
- Age
- Race
- Sex

From a functional medicine perspective, diet and lifestyle factors are the largest contributors to high cholesterol. Functional medicine practitioner Chris Kresser, M.S., L.Ac., identifies the following six underlying processes to dyslipidemia:

- Metabolic Dysfunction
- Chronic Infections like H. pylori or latent viral infections
- Gut Dysbiosis or Gut Permeability
- Poor Thyroid Function
- Environmental Toxins, including heavy metals
- Genetic factors

There are a number of other conditions (and the medications used to treat them) that may lead to high cholesterol levels. Some of the more common include chronic kidney disease; diabetes; HIV/AIDS; lupus; PCOS and sleep apnea.

**Dietary Cholesterol has Little Impact on Serum Cholesterol**

For many years, dietary cholesterol was considered a contributing factor to high cholesterol levels. However, because 80% of serum cholesterol is synthesized in the liver, it is now generally recognized that dietary cholesterol has very little impact on cholesterol levels. Recent dietary guidelines acknowledge that dietary cholesterol does not raise LDL and thus does not influence serum cholesterol levels.

Although dietary cholesterol has little impact on serum cholesterol levels, excessive carbohydrate and sugar intake are primary contributors to abnormal cholesterol levels. HMG-CoA reductase is an enzyme in the liver that plays a key role in the synthesis of cholesterol. HMG-CoA determines how much cholesterol the liver produces and is upregulated by insulin. Thus, overactivity of this enzyme, via insulin resistance, can contribute to high cholesterol.

**Holistic Nutrition Interventions for Healthy Cholesterol Levels**

Diet and lifestyle changes are extremely effective at lowering cholesterol and triglyceride levels and reducing inflammation. Adhering to a heart healthy diet, regular exercise, avoiding tobacco and maintaining a healthy weight are known lifestyle factors that affect positive changes in blood lipid levels.

*Diet*. Diets such as the Mediterranean diet, the DASH diet and a vegan diet, all of which are high in dietary fiber and low in sugar, have been shown to reduce inflammation, obesity, insulin resistance and oxidative stress - all of which are contributing factors to lipid dysregulation.

A heart healthy diet emphasizes the intake of vegetables, fruits and whole grains, poultry, fish, legumes, non tropical vegetable oils and nuts. Sugar, alcohol, processed foods and refined grain products should be avoided. The traditional view has been to limit saturated fats, but emerging evidence recognizes that the effects of saturated fat on lipid profiles may vary based on the quality of the fats consumed. For example, consider processed meat versus pasture raised and finished meat.

Supporting blood sugar regulation is an important component to maintaining healthy lipid levels. Insulin upregulates HMG-CoA reductase enzyme, leading to an increase in LDL cholesterol and triglycerides.

Meal frequency and pattern may also play a role in reducing LDL levels. Studies show that eating breakfast and maintaining a regular eating pattern, as well as smaller more frequent meals, may affect LDL levels.

Saturated fat intake should be limited to a minimum (10-15 g/day) while monounsaturated fats have a positive effect on cholesterol levels. Nuts in the diet have been shown to have a beneficial effect on lipid levels; particularly walnuts, almonds, pistachios, pecans, hazelnuts and macadamia nuts. Likewise, roasted ground sesame seeds have also been shown to have a cholesterol lowering effect.

A diet high in fiber (35 g/day) is associated with lower cholesterol levels and lower levels of inflammatory mediators. A number of studies show that high consumption of oatmeal or oat bran significantly reduces cholesterol in individuals with high cholesterol levels. Foods like barley and rye inhibit cholesterol synthesis.

Substituting soy protein for animal protein has been shown to both reduce LDL and increase HDL levels. Plant stanols and sterols isolated from soybean oils have been known to lower blood cholesterol.

*Weight loss and Physical Activity.* Obesity raises the risk of dyslipidemia and associated risk factors such as type 2 diabetes. Physical inactivity and a low level of fitness are independent risk factors for dyslipidemia. Physical activity increases HDL cholesterol, improves glucose tolerance and insulin sensitivity, and reduces the risk of atherogenesis. In general, adults are advised to engage in 150 minutes of moderate intensity aerobic activity per week and engage in strength training activities at least twice per week.

*Stress.* Stress management may be an important component in managing lipid levels. High levels of cortisol have been shown to increase production of VLDL in the liver, contributing to dyslipidemia.

*Gut health.* Gut microbiota targeted interventions, such as the use of prebiotics and probiotics, have been shown to significantly reduce cholesterol, LDL and triglycerides. An imbalance in gut microbiota composition is closely linked to various metabolic diseases, such as obesity, diabetes and metabolic syndrome - conditions characterized by, among other things, lipid abnormalities and increased inflammation. Probiotics are used to directly augment the population of microbes associated with metabolic benefits while prebiotics stimulate the growth of beneficial bacteria.

Recent evidence supports a causal relationship between the gut microbiome and dyslipidemia. Specifically, researchers have identified a number of microbiota that are associated with a decrease in the production of lipids and a number of species that are associated with elevated levels of certain lipids. Thus, targeted gut interventions show promising results in improving lipid profiles.

**Nutrients or Herbs to Support Cholesterol Regulation**

*Fish Oils*. Supplementation of EPA and DHA has little effect on cholesterol levels but does lower triglyceride levels and has a myriad of additional benefits in protecting against CVD. The recommended dose is 3000-5000 mg EPA + DHA.

*Niacin*. For nearly a century, niacin has been known to improve cholesterol levels. It lowers LDL and triglycerides and increases HDL. The effects of Niacin compare to those of prescription statins. However, niacin can cause skin flushing and other side effects, including gastric irritation, nausea and liver damage. An intermediate release form is recommended to avoid these side effects. At very high levels (3,000 mg or more), Niacin may impair glucose tolerance, so may be best avoided by diabetics. For best results, niacin should be taken at night, as most cholesterol synthesis occurs during sleep. Begin with a dose of 100 mg/day and titrate up to the full therapeutic dose of 1.5-3 g/day.

*Chromium*. Chromium enhances insulin sensitivity and may thereby indirectly improve lipid metabolism. It has been shown to increase HDL in patients taking beta blockers. It has also been shown to reduce serum cholesterol when taken in conjunction with niacin at 200 micrograms/day of chromium and 100 mg/day of niacin

*Garlic*. Garlic has been shown to lower LDL and improve HDL. The effects of supplemental garlic are modest, but also involve blood pressure lowering effects and other benefits. The recommended dosage is 16-30 mg.dl.

*Green tea.* Consumption of green tea or green tea extracts have been shown to lower LDL.

*Berberine.* Studies suggest that berberine, at .5-1.5 mg/day, may have a significant impact on total cholesterol levels - improving HDL and reducing LDL and triglycerides.

*Apple Cider Vinegar.* Consumption of apple cider vinegar has been shown to lower total cholesterol levels.

**References**

Berglund, L., Brunzell, J. D., Goldberg, A. C., Goldberg, I. J., Sacks, F., Murad, M. H., & Stalenhoef, A. F. H. (2012). Evaluation and treatment of hypertriglyceridemia: An Endocrine Society clinical practice guideline. *The Journal of Clinical Endocrinology & Metabolism, 97*(9), 2969–2989. https://doi.org/10.1210/jc.2011-3213

Centers for Disease Control and Prevention. (2023, July 17). *Facts about cholesterol*. U.S. Department of Health & Human Services. https://www.cdc.gov/cholesterol/data-research/facts-stats/

Corliss, J. (2016, February 5). *How it's made: Cholesterol production in your body*. Harvard Health Publishing. Retrieved June 19, 2025, from https://www.health.harvard.edu/heart-health/how-its-made-cholesterol-production-in-your-body

Craig, M., Yarrarapu, S. N. S., & Dimri, M. (2023, August 8). *Biochemistry, cholesterol*. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK470561/

The diagnosis and management of non‐alcoholic fatty liver disease: Practice guideline by the American Gastroenterological Association, American Association for the Study of Liver Diseases, and American College of Gastroenterology. *Hepatology, 55*(6), 2005–2023. https://doi.org/10.1002/hep.25762

Dong, H., Zhao, Y., Zhao, L., & Lu, F. (2013). The effects of berberine on blood lipids: a systemic review and meta-analysis of randomized controlled trials. *Planta medica*, *79*(6), 437–446. https://doi.org/10.1055/s-0032-1328321

Fuez, D. (n.d.). *Blood Lipid Lecture* [video lecture]. Holistic Consulting. https://holistic-consulting-59a7b5.circle.so/c/bchn-credentialing-program/sections/504855/lessons/1882510

Gaby, A. R., M.D.. (2023). Nutritional Therapy, Third Edition, 3rd Edition. [VitalSource Bookshelf 10.4.1]. Retrieved from vbk://9780982885024

Grundy, S. M. (2008). Metabolic syndrome pandemic. *Arteriosclerosis, Thrombosis, and Vascular Biology, 28*(4), 629–636. https://doi.org/10.1161/ATVBAHA.107.151092

Hadi, A., Pourmasoumi, M., Najafgholizadeh, A., Clark, C. C. T., & Esmaillzadeh, A. (2021). The effect of apple cider vinegar on lipid profiles and glycemic parameters: A systematic review and meta-analysis of randomized clinical trials. *BMC Complementary Medicine and Therapies, 21*, Article 179. https://doi.org/10.1186/s12906-021-03358-6

Kresser, C. (n.d.). *The functional medicine approach to high cholesterol*. Chris Kresser. https://chriskresser.com/the-functional-medicine-approach-to-high-cholesterol/

Mederle, A. L., Dima, M., Stoicescu, E. R., Căpăstraru, B. F., Levai, C. M., Hațegan, O. A., & Maghiari, A. L. (2024). Impact of Gut Microbiome Interventions on Glucose and Lipid Metabolism in Metabolic Diseases: A Systematic Review and Meta-Analysis. *Life (Basel, Switzerland)*, *14*(11), 1485. https://doi.org/10.3390/life14111485

Medeiros, D. M., & Wildman, R. E. C. (2019). *Advanced human nutrition* (4th ed.). Jones & Bartlett Learning.

Murray, M. T., & Pizzorno, J. E. (2012). *The encyclopedia of natural medicine* (3rd ed.). Atria Books.

National Heart, Lung, and Blood Institute. (n.d.). *Atherosclerosis*. U.S. Department of Health and Human Services. https://www.nhlbi.nih.gov/health/atherosclerosis

National Heart, Lung, and Blood Institute. (2024, April 19). *Blood cholesterol: Causes and risk factors*. NIH. Retrieved June 19, 2025, from https://www.nhlbi.nih.gov/health/blood-cholesterol/causes

Raymond, J. L., & Morrow, K. (2023). *Krause and Mahan's Food and the Nutrition Care Process.* Elsevier.

Rupa Health. (n.d.). *Access medical labs: Apolipoprotein B (Apo-B)*. Rupa Health. https://www.rupahealth.com/lab-tests/access-medical-labs-apolipoprotein-b-apo-b

Tsoupras, A., Lordan, R., & Zabetakis, I. (2018). Inflammation, not cholesterol, is a cause of chronic disease. *Nutrients, 10*(5), 604. https://doi.org/10.3390/nu10050604

Zhou, X., Lian, P., Liu, H., Wang, Y., Zhou, M., & Feng, Z. (2023). Causal Associations between Gut Microbiota and Different Types of Dyslipidemia: A Two-Sample Mendelian Randomization Study. *Nutrients*, *15*(20), 4445. https://doi.org/10.3390/nu15204445