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THE MAIN FACTORS IN THE ORIGIN OF DYSLIPIDEMIA

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Annotation: Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein cholesterol (HDL-C) level that contributes to the development of atherosclerosis. Causes may be primary (genetic) or secondary. Diagnosis is by measuring plasma levels of total cholesterol, TGs, and individual lipoproteins. Treatment involves dietary changes, exercise, and lipid-lowering drugs.

Key words: Risk, disease, blood, lipoprotein.

Serum lipid levels are continuous; there is no precise threshold between normal and abnormal levels. A linear relation likely exists between lipid levels and cardiovascular risk (see table Cholesterol Levels and Cardiovascular Risk), so many people with “normal” cholesterol levels benefit from achieving still lower levels. Consequently, there are no numeric definitions of dyslipidemia; the term is applied to lipid levels for which treatment has proven beneficial.

Proof of benefit is strongest for lowering elevated low-density lipoprotein cholesterol (LDL-C) levels. In the general population, evidence is less strong for a benefit from lowering elevated TG and increasing low high-density lipoprotein cholesterol (HDL-C) levels.

HDL-C levels do not always predict cardiovascular risk. For example, high HDL-C levels caused by some genetic disorders may not be associated with a lower risk of cardiovascular disorders, and low HDL-C levels caused by some genetic disorders may not be associated with an increased risk of cardiovascular disorders. Although HDL-C levels predict cardiovascular risk in the general population, the increased risk may be caused by other factors, such as accompanying lipid and metabolic abnormalities, such as hypertriglyceridemia, rather than the HDL-C level itself.

Dyslipidemias were traditionally classified by patterns of elevation in lipids and lipoproteins (Fredrickson phenotype—see table Lipoprotein Patterns). A more practical system categorizes dyslipidemias as primary or secondary and characterizes them by

- Increases in cholesterol only: Pure or isolated hypercholesterolemia
- Increases in TGs only: Pure or isolated hypertriglyceridemia
- Increases in both cholesterol and TGs: Mixed or combined hyperlipidemias

This system does not take into account specific lipoprotein abnormalities (eg, low HDL-C or high LDL-C) that may contribute to disease despite normal cholesterol and TG levels.

Cholesterol Levels and Cardiovascular Risk

Cardiovascular Risk	Total Cholesterol	LDL-C	HDL-C
Higher risk	≥ 6.2 mmol/L (240 mg/dL)	≥ 4.1 mmol/L (160 mg/dL)	Male: < 1.0 mmol/L (40 mg/dL) Female: < 1.3 mmol/L (50 mg/dL)
At- risk	5.2-6.2 mmol/L (200-239 mg/dL)	2.6-4.1 mmol/L (100-159 mg/dL)	Male: 1.0-1.5 mmol/L (40-59 mg/dL) Female: 1.3-1.5 mmol/L (50-59 mg/dL)
Lower risk	< 5.2 mmol/L (200 mg/dL)	< 2.6 mmol/L (100 mg/dL)	≥ 1.6 mmol/L (60 mg/dL)

HDL = high-density lipoprotein; LDL = low-density lipoprotein.

Data from Carmena R: Primary Mixed Dyslipidemias, Editor(s): Ilpo Huhtaniemi, Luciano Martini, Encyclopedia of Endocrine Diseases (Second Edition), Academic Press, 2019, Pages 314-319, ISBN 9780128122006, https://doi.org/10.1016/B978-0-12-801238-3.65333-3, National Heart Lung and Blood Institute: What is Blood Cholesterol? Updated March 24, 2022, https://www.nhlbi.nih.gov/health-topics/high-blood-cholesterol; American Heart Association: Cholesterol, https://www.heart.org/en/health-topics/cholesterol; Centers for Disease Control and Prevention: Cholesterol. Reviewed March 20, 2023, https://www.cdc.gov/cholesterol.

Table 1.

Dyslipidemias may be

- Primary: Genetic
- Secondary: Caused by lifestyle and other factors

Both primary and secondary causes contribute to dyslipidemias in varying degrees. For example, in familial combined hyperlipidemia, expression may occur only in the presence of significant secondary causes.

Primary causes

Primary causes are single or multiple gene mutations that result either in overproduction or defective clearance of triglycerides and LDL or in underproduction or excessive clearance of HDL.

Secondary causes

Secondary causes contribute to many cases of dyslipidemia in adults.

The most important secondary cause of dyslipidemia in high-resource countries is

- A sedentary lifestyle with excessive dietary intake of total calories, saturated fat, cholesterol, and trans fats

Trans fats are polyunsaturated or monounsaturated fatty acids to which hydrogen atoms have been added; they are used in some processed foods and are as atherogenic as saturated fat.

Diabetes is an especially significant secondary cause because patients tend to have an atherogenic combination of high TGs; high small, dense LDL fractions; and low HDL (diabetic dyslipidemia, hypertriglyceridemic hyperapo B). Patients with type 2 diabetes are especially at risk. The combination may be a consequence of obesity, poor control of diabetes, or both, which may increase circulating free fatty acids (FFAs), leading to increased hepatic very-low-density lipoprotein (VLDL) production. TG-rich VLDL then transfers TG and cholesterol to LDL and HDL, promoting formation of TG-rich, small, dense LDL and clearance of TG-rich HDL. Diabetic dyslipidemia is often exacerbated by the increased caloric intake and physical inactivity that characterize the lifestyle of some patients with type 2 diabetes. Women with diabetes may be at special risk of cardiac disease as a result of this form of dyslipidemia.

Dyslipidemia itself usually causes no symptoms, although very high TG levels can cause paresthesias, dyspnea, and confusion. Lipid disorders can lead to symptomatic end-organ disease, including

- Vascular disease (eg, coronary artery disease (CAD), stroke, and peripheral arterial disease)
- Acute pancreatitis can be caused be high TG levels (> 500 mg/dL [> 5.65 mmol/L])
- Hepatosplenomegaly can be caused by very high TG levels

Findings in patients with severe dyslipidemia may include localized lipid deposits (xanthomas) or other findings caused by high serum concentrations or accumulation of lipids.

High LDL-C levels can cause tendinous xanthomas at the Achilles, elbow, and knee tendons and over metacarpophalangeal joints. Other clinical findings that occur in patients with high LDL-C (eg, in familial hypercholesterolemia or dysbetalipoproteinemia) include planar or tuberous xanthomas. Planar xanthomas are flat or slightly raised yellowish patches. Tuberous xanthomas are painless, firm nodules typically located over extensor surfaces of joints.

Patients with high levels of LDL-C can develop arcus corneae (lipid deposits in the cornea around the iris) and xanthelasma (lipid-rich, yellow plaques on the medial eyelids). Xanthelasma can also occur in patients with primary biliary cirrhosis and normal lipid levels.

Extremely high total cholesterol levels give a lactescent (milky) appearance to blood plasma.

Severe elevations of TGs can cause eruptive xanthomas over the trunk, back, elbows, buttocks, knees, hands, and feet. Severe hypertriglyceridemia (> 2000 mg/dL [> 22.6 mmol/L]) can also give retinal arteries and veins a creamy white appearance (lipemia retinalis).

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