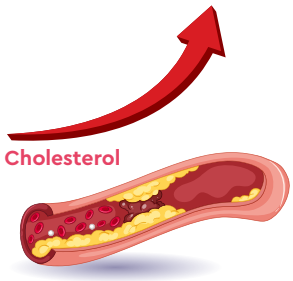
DYSLIPIDEMIA

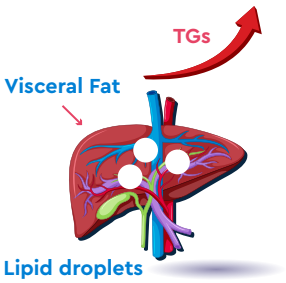
WHAT IS DYSLIPIDEMIA?

Dyslipidemia, defined as an elevated total cholesterol (TC), low-density lipoprotein (LDL-C), triglycerides (TG), non-high-density lipoprotein (Non-HDL-C) or decreased high-density lipoprotein cholesterol (HDL-C), is an important risk factor for atherosclerosis and stroke.

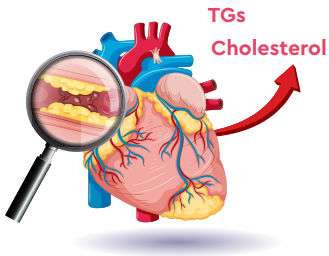
TYPES OF DYSLIPIDEMIA



Increases in cholesterol only (pure or isolated hypercholesterolemia)



Increases in TGs only (pure or isolated hypertrigl yceridemia)



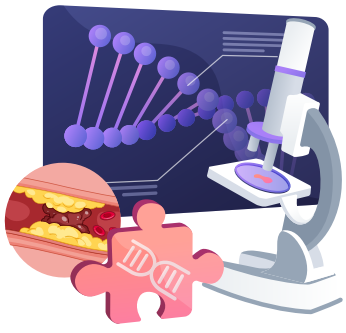
Increases in both cholesterol and TGs (mixed or combined hyperlipidemias)

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

CLASSIFICATION OF DYSLIPIDEMIA

Dyslipidemia may be:

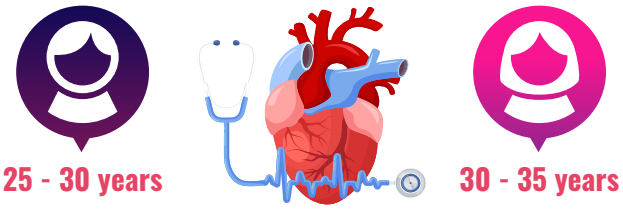
Primary dyslipidemia is basically genetics and caused by single or multiple gene mutations that result in either overproduction or defective clearance of triglycerides and cholesterol.



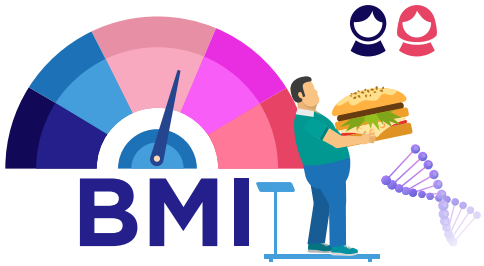
Secondary dyslipidemia is caused by unhealthy lifestyle factors and some medical conditions such as obesity and type 2 diabetes. It accounts for approximately 30-40% of all dyslipidemia and should be treated by finding and addressing its causative diseases or drugs.

SCREENING

All individuals regardless of age, should be screened if they have the risk factors listed below.



Those with a high risk of cardiovascular disease should be screened at a younger age with males between 25 and 30 years old and females between 30 and 35 years of age.

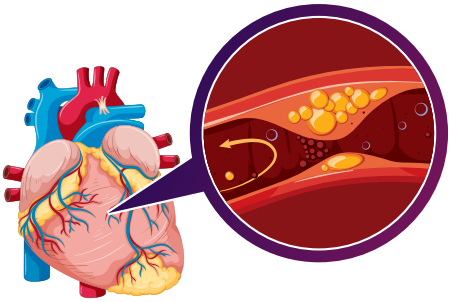


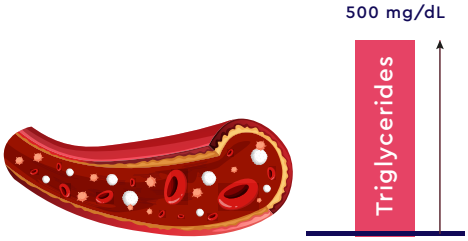
RISK FACTORS

Many factors contribute to the development of dyslipidemia, including genetics, gender, ethnicity, increased body mass index (BMI), unhealthy dietary habits, and smoking.

SYMPTOMS AND SIGNS OF DYSLIPIDEMIA

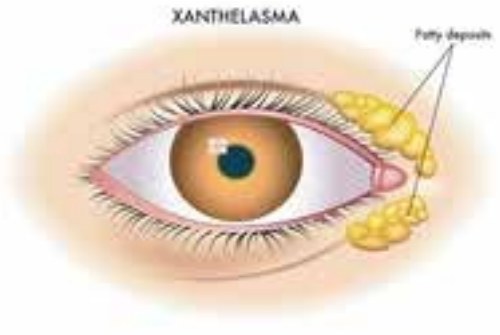
- Dyslipidemia itself usually but can lead to symptomatic vascular disease, including coronary artery disease (CAD), stroke, and peripheral arterial disease.





- High levels of triglycerides of triglycerides (> 500 mg/dL [> 5.65 mmol/L]) can cause acute pancreatitis. Very high triglyceride levels can also cause hepatosplenomegaly, paresthesia, dyspnea, and confusion.

• Patients with the homozygous form of familial hypercholesterolemia may have arcus corneae, tendinous xanthomas and xanthelasma plus 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.



DIAGNOSIS

Dyslipidemia is diagnosed by conducting a lipid panel that measures blood lipid levels. This is measured via a simple peripheral blood draw. The most frequently measured and clinically utilized components within the lipid panel include total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein (HDL-C).

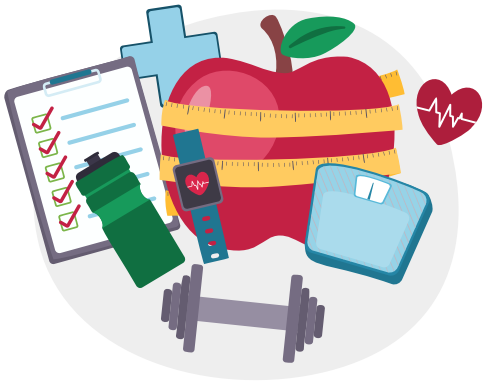
It is characterized by	
Total Cholesterol levels	> 200 mg/dL
LDL-C levels	> 130 mg/dL
HDL-C levels	< 40 mg/dL
Triglyceride levels	> 150 mg/dL

Non-Pharmacological Treatment

• **Lifestyle modifications:**
Lifestyle modifications such as eating healthy, being physically active, quitting smoking and maintaining a healthy body weight can improve plasma lipid profile.

• **Diet:** Dietary factors can influence the development of CVD through their action on traditional risk factors, such as plasma lipids, BP, or glucose levels.

Higher consumption of fruit, non-starchy vegetables, nuts, legumes, fish, vegetable oils, milk, and whole grains, along with a lower intake of red and processed meats, foods high in refined carbohydrates, and salt, is associated with a lower incidence of CV events.
The most extensively evaluated dietary patterns are the Dietary Approaches to Stop Hypertension (DASH) diet—particularly in relation to blood pressure control—and the Mediterranean diet; both have been proved to be effective in reducing cardiovascular (CV) risk factors.



• **Weight Loss:** since overweight (BMI ≥25 to <30 kg/m2), obesity (BMI ≥30 kg/m2), and abdominal adiposity (a waist circumference of more than 88 cm in women and more than 102 cm in men) often contribute to dyslipidemia, caloric intake should be reduced and energy expenditure increased in those with excessive weight and/or abdominal adiposity. A modest body weight reduction improves lipid abnormalities and favorably affects other CV risk factors present in dyslipidemia individuals.

• **Physical Activity:** It is always appropriate to advise all people with dyslipidemia to engage in regular physical exercise of moderate intensity for more than 30 min/day. Exercise improves serum lipids by lowering triglyceride levels, total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C) levels, while increasing high density lipoprotein cholesterol (HDL-C) levels.

PHARMACOLOGICAL TREATMENT

HMG-CoA reductase inhibitors (Statins): inhibit the enzyme needed to make cholesterol in the liver. They decrease LDL and triglycerides and slightly increase HDL.

Bile acid sequestrants: lower cholesterol indirectly by binding to bile acids. The liver will use the excess cholesterol to make more bile acids, causing a reduction in the level of cholesterol in your blood. They decrease LDL and may slightly increase HDL.

Fibrates: have good efficacy in lowering fasting and post-prandial triglyceride levels.

Cholesterol absorption inhibitors: limit the absorption of dietary cholesterol. They decrease LDL, slightly decrease triglycerides, and increase HDL.

Proprotein convertase subtilisin /kexin type 9 (PCSK9) inhibitors: can help the liver absorb more LDL cholesterol, which lowers the amount of cholesterol circulating in the blood. They decrease LDL and are usually reserved for people who have a genetic condition.