GENERAL

structure and function of cholesterol

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Introduction

Cholesterol is one of the most discussed lipids in both medical and popular health circles. Whether you're a medical student, a fitness enthusiast, or simply a curious reader, understanding the **structure and function of cholesterol**can demystify its role in health and disease. It's not just a villain in the headlines; it's a vital component of every cell, a precursor to essential hormones, and a key player in maintaining the integrity of our biological membranes. This article takes you on an in depth journey through cholesterol's molecular architecture, its synthesis, transport, and the delicate balance that keeps our bodies functioning optimally.

What Is Cholesterol?

Cholesterol is a sterol—a type of lipid that contains a hydroxyl group. It's a waxy, fat like substance that is insoluble in water but soluble in organic solvents. In the human body, cholesterol is synthesized by the liver, produced by the intestines, and obtained from dietary sources. It exists in two primary forms:

- Endogenous cholesterol – produced by the body's own metabolic pathways.
- Exogenous cholesterol – derived from food, especially animal products.

Despite its negative reputation in the context of cardiovascular disease, cholesterol is indispensable for cellular function.

Chemical Structure of Cholesterol

Molecular Formula and Weight

The chemical formula of cholesterol is C27H45O3. Its molar mass is 386.65 g/mol.

Four-Carbon Ring System

At the heart of cholesterol lies a rigid, fused four ring structure known as the sterane backbone:

- Three cyclohexane rings (A, B, C)
- One cyclopentane ring (D)

This arrangement gives cholesterol its characteristic shape and contributes to its ability to embed in lipid bilayers.

Side Chain and Functional Groups

Attached to the D ring is a flexible, hydrophobic side chain that extends into the membrane. The hydroxyl group (–OH) on carbon 3 provides a polar site, allowing cholesterol to interact with aqueous environments while the rest of the molecule remains lipophilic.

Isomeric Forms

Cholesterol exists in two stereoisomeric forms:

- Cholesterol (3;" €ydroxy 5 pregnene cholest 7 ene) – the naturally occurring form.
- Cholestanol – a saturated counterpart found in certain dietary sources.

Biosynthesis of Cholesterol

Endogenous Production Pathway

The mevalonate pathway is the cornerstone of cholesterol synthesis. It begins with acetyl CoA and proceeds through a series of enzymatic steps:

1. Acetyl CoA !’ HMG CoA (3 hydroxy 3 methylglutaryl CoA)
2. HMG CoA !’ Mevalonate (via HMG CoA reductase)
3. Mevalonate !’ Isopentenyl pyrophosphate (IPP) and dimethylallyl pyrophosphate (DMAPP)
4. IPP + DMAPP !’ Farnesyl pyrophosphate (FPP)
5. FPP !’ Squalene (via squalene synthase)
6. Squalene !’ Lanosterol (via squalene epoxidase and oxidosqualene cyclase)
7. Lanosterol !’ Cholesterol (via a series of demethylation, isomerization, and reduction steps)

HMG CoA reductase is the rate limiting enzyme and a primary target for statin drugs.

Regulation of Synthesis

Cholesterol synthesis is tightly controlled by feedback mechanisms. High intracellular cholesterol levels inhibit HMG CoA reductase, while low levels activate it. Additionally, the liver adjusts the expression of LDL receptors based on plasma cholesterol concentration.

Dietary Cholesterol vs. Endogenous Cholesterol

Sources of Dietary Cholesterol

Animal products such as eggs, dairy, red meat, and shellfish are rich in cholesterol. Plant sources contain negligible amounts.

Absorption Mechanisms

In the small intestine, dietary cholesterol is incorporated into micelles formed by bile salts. These micelles facilitate transport to enterocytes, where cholesterol is re esterified and packaged into chylomicrons for lymphatic transport.

Contribution to Total Body Cholesterol

While diet influences cholesterol levels, most of the body’s cholesterol is produced endogenously. On average, dietary intake accounts for roughly 20–30% of circulating cholesterol.

Functions of Cholesterol in the Body

Structural Component of Cell Membranes

Cholesterol intercalates between phospholipid molecules, modulating membrane fluidity, thickness, and curvature. It stabilizes membrane proteins and influences the formation of lipid rafts—microdomains essential for signal transduction.

Precursor to Steroid Hormones

Cholesterol is the starting material for:

- Glucocorticoids (e.g., cortisol)
- Mineralocorticoids (e.g., aldosterone)
- Androgens (e.g., testosterone)
- Estrogens (e.g., estradiol)

Bile Acid Synthesis

In the liver, cholesterol is converted into primary bile acids—cholic acid and chenodeoxycholic acid—through a series of enzymatic reactions. These bile acids emulsify dietary fats, facilitating absorption.

Vitamin D Production

Ultraviolet B (UVB) radiation converts 7 dehydrocholesterol in the skin into vitamin D3, a crucial vitamin for calcium homeostasis and bone health.

Other Roles

- Neurotransmitter precursor (acetylcholine).
- Component of myelin sheaths surrounding nerve fibers.
- Part of the innate immune system, influencing pathogen entry.

Cholesterol Transport and Lipoproteins

Overview of Lipoprotein Classes

Lipoproteins are complexes of lipids and proteins that ferry cholesterol and triglycerides through the bloodstream. They differ by density, size, and function.

Low Density Lipoprotein (LDL)

LDL carries cholesterol from the liver to peripheral tissues. Elevated LDL levels are a major risk factor for atherosclerosis.

High Density Lipoprotein (HDL)

HDL collects excess cholesterol from tissues and transports it back to the liver for excretion—a process known as reverse cholesterol transport.

Very Low Density Lipoprotein (VLDL)

VLDL transports triglycerides from the liver to adipose tissue and muscle. It is metabolized into LDL.

Chylomicrons

These are large lipoproteins that carry dietary triglycerides and cholesterol from the intestines to the bloodstream.

Receptor-Mediated Uptake

The LDL receptor on hepatocytes and peripheral cells is the primary gateway for LDL cholesterol. Its expression is regulated by intracellular cholesterol levels.

Regulation of Cholesterol Homeostasis

Feedback Inhibition

High intracellular cholesterol levels suppress HMG CoA reductase activity and upregulate LDL receptor expression, thereby reducing further cholesterol synthesis and increasing clearance.

Statin Therapy

Statins competitively inhibit HMG CoA reductase, lowering endogenous cholesterol production. They also upregulate LDL receptors, enhancing LDL clearance.

PCSK9 Inhibitors

PCSK9 binds to LDL receptors, marking them for degradation. Monoclonal antibodies that inhibit PCSK9 prevent

this degradation, increasing LDL receptor numbers and lowering LDL levels.

Dietary Interventions

Reducing saturated fat and trans fat intake, increasing soluble fiber, and incorporating plant sterols can lower LDL cholesterol by competing for bile acid absorption.

Cholesterol and Cardiovascular Disease

Atherosclerosis Explained

Atherosclerosis is the buildup of plaques—comprised of lipids, calcium, and fibrous tissue—within arterial walls. LDL infiltration and subsequent oxidation initiate an inflammatory cascade, attracting macrophages and forming foam cells.

Risk Factors

- High LDL cholesterol
- Low HDL cholesterol
- Elevated triglycerides
- Hypertension
- Smoking
- Diabetes mellitus
- Genetic predisposition (e.g., familial hypercholesterolemia)

Clinical Outcomes

Progressive plaque formation narrows arteries, impairs blood flow, and can lead to myocardial infarction, stroke, or peripheral arterial disease.

Cholesterol Testing and Management

Lipid Panel Components

- Total cholesterol
- LDL cholesterol
- HDL cholesterol
- Triglycerides

Interpreting Results

Optimal levels are generally **LDL < 100 mg/dL**, **HDL > 60 mg/dL**, and **triglycerides < 150 mg/dL**. However, individual risk assessment considers age, family history, and comorbidities.

Lifestyle Modifications

1. Diet – Mediterranean diet, low saturated fat, high soluble fiber.
2. Exercise – at least 150 minutes of moderate aerobic activity per week.
3. Weight Management – maintain BMI within 18.5–24.9.
4. Smoking Cessation – reduces endothelial damage.
5. Alcohol Moderation – limit to 1 drink per day for women, 2 for men.

Pharmacologic Options

- Statins (atorvastatin, simvastatin, rosuvastatin)
- PCSK9 inhibitors (alirocumab, evolocumab)
- Bile acid sequestrants (cholestyramine)

- Fibrates (fenofibrate) – primarily for high triglycerides
- Niacin – increases HDL, but with side effects

Emerging Research and Future Directions

Gene Editing for Hypercholesterolemia

CRISPR/Cas9 technology is being explored to correct LDL receptor mutations in familial hypercholesterolemia, potentially offering a one time cure.

Nanoparticle Drug Delivery

Targeted

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