Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

THE ROLE OF LIPID METABOLISM IN CARDIOVASCULAR DISEASE: AN INTEGRATED ENDOCRINE PERSPECTIVE

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DOI: https://doi.org/10.5281/zenodo.21887998

Keywords

Lipid Metabolism, Cardiovascular Diseases, Hormones, Insulin Resistance, Dyslipidemias, Adipokines.

Article History

Received: 01 January 2026

Accepted: 17 March 2026

Published: 31 March 2026

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Abstract

Objective: *To synthesize current evidence on how hormones regulate lipid metabolism, examine the bidirectional relationship between endocrine disorders and atherogenic dyslipidemia, and evaluate cardiovascular outcomes of emerging therapies that target hormone-lipid pathways.*

Methodology: *A narrative review of PubMed, Scopus, and Web of Science was conducted for English-language articles published from January 2000 to March 2025, using search terms including lipid metabolism, dyslipidemia, cardiovascular disease, hormones, insulin resistance, thyroid hormone, glucocorticoids, growth hormone, sex hormones, and adipokines. Two authors independently screened titles and abstracts; 542 full-text articles were reviewed, and 312 studies relevant to endocrine function, lipid metabolism, and cardiovascular outcomes were included and synthesized narratively.*

Results: *Insulin resistance drives the atherogenic lipid triad of hypertriglyceridemia, low HDL-C, and small dense LDL. Hypothyroidism elevates LDL-C by thirty to fifty percent through reduced LDL receptor expression. Glucocorticoid excess promotes hepatic VLDL overproduction and visceral adiposity. Growth hormone deficiency increases LDL-C and lipoprotein(a). Sex hormone imbalances produce characteristic dyslipidemias, and oxidized LDL and ceramides act as bioactive signaling molecules that promote inflammation and*

Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

endothelial dysfunction. GLP-1 receptor agonists, SGLT2 inhibitors, icosapent ethyl, and PCSK9 inhibitors reduce cardiovascular events through mechanisms that extend beyond LDL-C lowering.

Conclusion: *Cardiovascular disease requires an integrated perspective that accounts for the hormonal regulation of lipid metabolism. Clinicians should evaluate endocrine function in patients with treatment-resistant dyslipidemia or premature cardiovascular disease.*

INTRODUCTION

Cardiovascular disease has been the leading cause of death and disability worldwide for more than five decades. The Framingham Heart Study first established a clear link between elevated blood cholesterol and future myocardial infarction, fundamentally changing how clinicians understand and manage cardiovascular risk[1]. Subsequent discoveries, particularly the identification of low-density lipoprotein (LDL) receptors by Brown and Goldstein, elucidated the molecular machinery governing cholesterol homeostasis and paved the way for statin development[2].

Statins have been remarkably successful. Large-scale randomized trials have consistently demonstrated that LDL cholesterol (LDL-C) reduction with statins decreases major adverse cardiovascular events (MACE) and all-cause mortality in millions of patients[3]. As a result, LDL-C lowering has become the cornerstone of clinical guidelines for cardiovascular prevention.

Yet a substantial problem persists. Many patients on optimal statin therapy with LDL-C levels below 70 mg/dL continue to experience cardiovascular events. This phenomenon, termed residual cardiovascular risk, is particularly pronounced in individuals with diabetes, obesity, metabolic syndrome, or chronic inflammatory conditions. Residual risk is driven partly by abnormalities in triglyceride-rich lipoproteins, low high-density lipoprotein cholesterol (HDL-C), and qualitative changes in LDL particles, particularly small dense LDL, that are not captured by standard LDL-C measurement.

Over the past two decades, our understanding of adipose tissue has undergone a paradigm shift. Far from being a passive storage depot, adipose tissue is now recognized as a highly active endocrine organ that secretes numerous hormones, or adipokines, including leptin, adiponectin, resistin, and inflammatory cytokines[4]. These molecules regulate appetite, insulin sensitivity, inflammation, and vascular function. When adipose tissue becomes dysfunctional in obesity, the balance of these adipokines shifts, creating a systemic environment that promotes both metabolic and cardiovascular disease.

Most medical literature treats lipids and hormones as separate domains. Lipid specialists focus on LDL-C and triglycerides, while endocrinologists focus on insulin, thyroid hormones, and sex steroids. In clinical reality, however, these systems are intimately interconnected. Hormones regulate every aspect of lipoprotein metabolism, including synthesis, lipolysis, clearance, and reverse cholesterol transport. Thyroid hormones directly regulate LDL receptor expression[5], while cortisol excess drives hepatic very-low-density lipoprotein (VLDL) overproduction[6]. Conversely, lipids themselves function as signaling molecules that influence hormone action and vascular health, a bidirectional interplay that is often overlooked in textbooks and clinical guidelines.

This review aims to integrate these two perspectives by explaining how hormones regulate lipid metabolism, how endocrine disorders produce characteristic and often reversible dyslipidemias, and how emerging therapies that target these pathways reduce cardiovascular risk.

Methodology

This narrative review was conducted on the published literature concerning endocrine regulation of lipid metabolism and cardiovascular disease. PubMed, Scopus, and Web of Science

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

were searched for English-language articles published from January 2000 to March 2025.

Search terms included: lipid metabolism, lipoproteins, dyslipidemia, cardiovascular disease, hormones, insulin resistance, thyroid hormone, glucocorticoids, growth hormone, sex hormones, adipokines, GLP-1 receptor agonists, and SGLT2 inhibitors.

Original research randomized controlled trials, cohort studies, meta-analyses, and systematic reviews were included. Case reports, editorials, and opinion pieces were excluded. Human studies were prioritized, and mechanistic studies were included only where clearly relevant to human disease.

Two authors screened titles and abstracts independently. A total of 542 full-text articles were reviewed, and 312 studies directly relevant to endocrine function, lipid metabolism, and cardiovascular outcomes were selected. Data were extracted on hormone effects, lipid changes, cardiovascular outcomes, and therapeutic responses, and findings were organized by endocrine disorder (thyroid, adrenal, growth hormone, insulin resistance, sex hormones) for narrative synthesis.

A formal meta-analysis or systematic quality assessment was not performed because of wide variation in study designs, populations, and outcome measures. This review represents a thematic synthesis of available evidence rather than a quantitative pooled analysis.

Results

Thyroid Hormones and Lipid Metabolism

Thyroid hormones regulate metabolism throughout the body. In the liver, triiodothyronine (T3) directly controls LDL receptor gene expression; more receptors mean faster clearance of circulating LDL[5]. When thyroid hormone levels fall, as in hypothyroidism, LDL receptor expression drops and LDL accumulates in the bloodstream[7].

Beyond this classical pathway, thyroid hormones influence cholesterol metabolism through non-receptor-mediated mechanisms, including effects on sterol regulatory element-binding proteins and hepatic lipase activity[8]. Patients with overt hypothyroidism typically present with elevated LDL-C (thirty to fifty percent above baseline), elevated lipoprotein(a), and variable changes in triglycerides and HDL-C[9].

Levothyroxine replacement consistently improves lipid profiles. A comprehensive meta-analysis showed that levothyroxine lowers LDL-C by fifteen to twenty-five percent and reduces lipoprotein(a) by ten to fifteen percent[11]. Even subclinical hypothyroidism, which affects four to ten percent of adults, produces clinically meaningful changes: LDL-C rises by approximately ten to fifteen mg/dL, and coronary heart disease risk increases by about twenty percent[9]. Current American Thyroid Association guidelines recommend levothyroxine treatment for TSH greater than 10 mIU/L, or for TSH 4-10 mIU/L in younger patients, those with high cardiovascular risk, or those with dyslipidemia refractory to standard therapy[12].

Glucocorticoid Excess and Cushing's Syndrome

Glucocorticoid excess, whether endogenous (Cushing's syndrome) or iatrogenic, produces a distinctive and highly atherogenic metabolic phenotype. Cortisol promotes hepatic VLDL overproduction, impairs lipoprotein clearance, and drives visceral fat accumulation[13].

The lipid profile in active Cushing's syndrome typically shows elevated total cholesterol, LDL-C, and triglycerides; HDL-C may be normal or low[14]. Combined with the hypertension and hypercoagulability of cortisol excess, this pattern confers a four- to five-fold higher cardiovascular risk than in age-matched controls[15].

Importantly, the dyslipidemia of Cushing's syndrome is reversible. A recent systematic review and meta-analysis demonstrated that treatment of hypercortisolism, whether by surgical resection of pituitary or adrenal tumors or by reduction of exogenous steroids, significantly improves total cholesterol, LDL-C, and triglyceride levels[14].

Growth Hormone Axis

Growth hormone (GH) exerts profound effects on lipid metabolism. In adults with GH deficiency, the lipid profile is characterized by increased LDL-C, elevated apolipoprotein B,

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ISSN: (e) 3007-1607 (p) 3007-1593

increased lipoprotein(a), and increased visceral fat mass, driven by reduced hepatic LDL receptor expression and altered lipoprotein lipase activity[16].

GH replacement therapy has shown favorable effects on this profile, including improvements in LDL-C and body composition[17]. Beyond lipids, GH reduces visceral adipose tissue and can improve insulin sensitivity, though it may transiently raise triglycerides while enhancing hepatic VLDL1-triglyceride export.

Reduced GH levels have been shown to predict worsening adipose tissue insulin resistance in adults with obesity[16], and low-dose GH treatment has been evaluated in obesity and metabolic dysfunction-associated steatotic liver disease, with meta-analyses showing beneficial effects on liver fat content and metabolic parameters[18]. At the molecular level, deletion of the hepatic GH receptor contributes to lean fatty liver disease by elevating CD36 expression and promoting hepatic lipid uptake[20], suggesting the GH axis may be a therapeutic target beyond classical GH deficiency.

Insulin Resistance and Diabetes Mellitus

Insulin resistance is the most common endocrine driver of atherogenic dyslipidemia. In type 2 diabetes and metabolic syndrome, impaired insulin signaling in adipose tissue increases lipolysis and free fatty acid flux to the liver, stimulating hepatic overproduction of large, triglyceride-rich VLDL1 particles[21].

The resulting atherogenic lipid triad, comprising hypertriglyceridemia (typically 150-500 mg/dL), low HDL-C (below 40 mg/dL in men and 50 mg/dL in women), and increased small dense LDL, is accompanied by elevated apolipoprotein B[22] and confers a two- to four-fold higher cardiovascular risk than in people without diabetes, independent of LDL-C.

Composite lipid biomarkers have also been associated with cardiovascular disease prevalence in prediabetic and diabetic populations[22]. Young people with diabetes face particularly high risk for early cardiovascular events, underscoring the need for aggressive risk-factor modification from an early age. Good glycemic control alone does not fully correct the lipid abnormalities of diabetes; direct lipid-modifying therapy is still required[22].

Polycystic Ovary Syndrome

Polycystic ovary syndrome (PCOS) affects six to twelve percent of reproductive-aged women and is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Insulin resistance affects approximately seventy percent of women with PCOS, independent of obesity, and drives a lipid profile that closely resembles metabolic syndrome: elevated triglycerides, low HDL-C, increased small dense LDL, and elevated lipoprotein(a)[22].

Emerging research has examined the interplay between gut microbiota and lipid metabolism in PCOS; a Mendelian randomization and mediation analysis suggests specific gut microbial taxa and lipid metabolites may causally contribute to PCOS pathogenesis[23]. Nutritional interventions, including trace-element supplementation (zinc, selenium) and vitamin D, have shown early promise for improving glycolipid metabolism in PCOS, though clinical significance requires further study[25,26].

The Androgen Excess and PCOS Society consensus statement on cardiovascular risk highlights that women with PCOS carry an elevated burden of cardiovascular risk factors that persists across the reproductive years and beyond, supporting early cardiovascular risk assessment in this population[24].

Sex Hormones and Lipid Metabolism

Sex hormones influence lipid metabolism throughout life. In premenopausal women, estrogen is protective: it increases hepatic LDL receptor expression, enhances reverse cholesterol transport via ABCA1, and raises HDL-C by increasing apolipoprotein A-I production. After menopause, estrogen declines, and within two to three years LDL-C rises by ten to fifteen percent, HDL-C falls by five to ten percent, and small dense LDL particles increase.

Testosterone has more context-dependent effects. In hypogonadal men, testosterone therapy modestly lowers HDL-C (roughly five to ten

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ISSN: (e) 3007-1607 (p) 3007-1593

mg/dL) but may reduce lipoprotein(a) and improve insulin sensitivity. Short-term testosterone gel treatment in healthy young women produces a distinct circulating lipid signature, suggesting rapid effects on lipid synthesis and clearance[27], and reduced glycoprotein hormone β5, which is linked to male aging and testosterone decline, has been associated with increased adiposity and adverse metabolic changes[28].

Table 1 summarizes the characteristic lipid profile changes associated with each of the endocrine disorders discussed above.

Table 1: Lipid profile changes in common endocrine disorders

Endocrine Disorder	Triglycerides	HDL-C	LDL-C	Key Mechanism
Type 2 Diabetes / MetS	Increased	Decreased	Normal/High sdLDL	Insulin resistance → VLDL overproduction (21,22)
Hypothyroidism	Normal/High	Normal/Low	Markedly increased	Reduced LDL receptor expression (5,9)
PCOS	Increased	Decreased	Normal/High sdLDL	Insulin resistance + hyperandrogenism (22)
Cushing's Syndrome	Increased	Normal/Low	Increased	Cortisol excess → ↑ VLDL synthesis (14)
Growth Hormone Deficiency	Normal/High	Normal/Low	Increased	Reduced LDL receptor expression (16)

sdLDL = small dense LDL particles; MetS = metabolic syndrome.

Novel Therapies Targeting Hormone-Lipid Pathways

GLP-1 receptor agonists.

Liraglutide and semaglutide were developed for glycemic control in type 2 diabetes, but large cardiovascular outcome trials have shown substantial MACE reduction: thirteen percent with liraglutide in the LEADER trial[29] and twenty-six percent with semaglutide in SUSTAIN-6[30].

SGLT2 inhibitors.

Empagliflozin and canagliflozin lower blood glucose by promoting urinary glucose excretion. The EMPA-REG OUTCOME trial showed a thirty-eight percent reduction in cardiovascular death with empagliflozin[31], and the CANVAS program showed a fourteen percent reduction in MACE with canagliflozin[32].

Icosapent ethyl.

This purified eicosapentaenoic acid preparation was tested in REDUCE-IT among patients with elevated triglycerides despite statin therapy, reducing MACE by twenty-five percent, a benefit larger than triglyceride lowering alone would predict[33].

PCSK9 inhibitors.

Evolocumab and alirocumab inhibit PCSK9, increasing LDL receptor recycling and lowering LDL-C by fifty to sixty percent. FOURIER (evolocumab) showed a fifteen percent reduction in MACE[34], with similar benefits reported for alirocumab in ODYSSEY OUTCOMES[35].

Table 2 summarizes the lipid effects and cardiovascular outcome benefits of these four drug classes, and Figure 1 illustrates the relative risk reduction achieved in each trial.

Volume 4, Issue 3, 2026

Frontier in Medical & Health Research

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Table 2: Cardiovascular outcomes from hormone-lipid pathway drugs

Drug Class	Example Drugs	Lipid Effect	CV Outcome Benefit	Key Trial
GLP-1 RA	Liraglutide, Semaglutide	↓ TG, ↓ body weight	↓ MACE 13-26%	LEADER, SUSTAIN-6 (29,30)
SGLT2 Inhibitor	Empagliflozin, Canagliflozin	Minimal lipid change	↓ HF hospitalization; ↓ CV death (up to 38%)	EMPA-REG, CANVAS (31,32)
Icosapent Ethyl	Purified EPA	↓ Triglycerides 20-30%	↓ MACE 25%	REDUCE-IT (33)
PCSK9 Inhibitor	Evolocumab, Alirocumab	↓ LDL-C 50-60%	↓ MACE 15%	FOURIER, ODYSSEY (34,35)

MACE = major adverse cardiovascular events; HF = heart failure; TG = triglycerides; RA = receptor agonist.

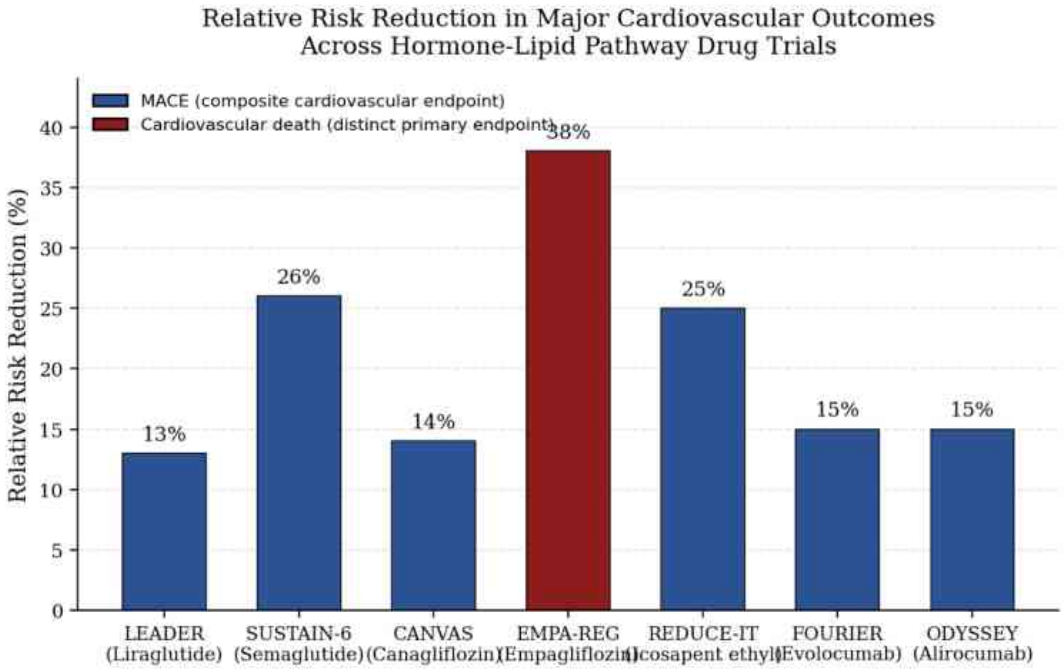


Figure 1: Relative risk reduction in major cardiovascular outcomes across hormone-lipid pathway drug trials

Values represent relative risk reductions for the primary endpoint of each trial versus placebo. Six trials report reduction in major adverse cardiovascular events (MACE), a composite endpoint that typically includes

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ISSN: (e) 3007-1607 (p) 3007-1593

cardiovascular death, nonfatal myocardial infarction, and nonfatal stroke; EMPA-REG OUTCOME's headline result (38%) was for cardiovascular death specifically, a distinct and non-equivalent endpoint, and is shown separately for this reason. Data sources: LEADER[29], SUSTAIN-6[30], CANVAS[32], EMPA-REG OUTCOME[31], REDUCE-IT[33], FOURIER[34], ODYSSEY OUTCOMES[35].

Discussion

This review demonstrates that cardiovascular disease cannot be reduced to a simple problem of cholesterol excess; it is fundamentally a disorder of metabolic and hormonal regulation. The evidence synthesized here, from thyroid regulation of LDL receptors to cortisol-driven VLDL overproduction to the complex lipid phenotypes of PCOS and diabetes, supports an integrated endocrine approach to cardiovascular risk assessment and management.

Statins remain essential and life-saving, but they do not address insulin resistance, adipose tissue inflammation, or the hormonal disturbances that drive residual cardiovascular risk. A patient with type 2 diabetes and central obesity has a fundamentally different pathophysiology than a patient with familial hypercholesterolemia; both may have elevated LDL-C, but their treatment should differ accordingly.

Clinicians should screen for endocrine disorders in patients with treatment-resistant dyslipidemia or premature cardiovascular disease. Thyroid function tests, fasting insulin, and a clinical assessment for Cushing's syndrome or hyperandrogenism are simple, widely available, and can yield actionable diagnoses. Treatment should then address the underlying hormonal disturbance: levothyroxine may normalize lipids in hypothyroidism without additional therapy[11]; metformin, GLP-1 agonists, or pioglitazone may be more effective than escalating the statin dose when insulin resistance is the primary driver; and surgical or medical treatment of hypercortisolism improves lipid profiles and cardiovascular risk in Cushing's syndrome[14].

It is also worth viewing lipids as signals rather than only as risk markers. Oxidized LDL and ceramides have been linked to vascular damage through inflammatory and pro-apoptotic pathways, and while routine clinical testing for these molecules is not yet widely available, emerging data suggest they may eventually improve risk prediction beyond conventional lipids. The success of GLP-1 receptor agonists and SGLT2 inhibitors further underscores the value of targeting hormonal and metabolic pathways directly, since their cardiovascular benefits extend well beyond LDL-C reduction[29,31].

Limitations: Studies included in this review varied considerably in patient populations, methodology, and outcomes and many were observational, which precludes definitive causal inference. Most novel therapeutic agents have only three to five years of outcome data, and standardized clinical assays for ceramides and oxidized LDL are not yet widely available.

Future directions: Research should focus on treatment strategies guided by hormonal and metabolic phenotype rather than lipid numbers alone, better biomarkers for residual risk despite statin therapy, and prospective studies establishing the clinical utility of ceramide and oxidized LDL testing.

Conclusion

Lipid metabolism and endocrine function are inextricably linked in health and disease. Hormones govern lipoprotein synthesis, transport, and clearance, and adipokines connect nutritional status to vascular health. When these hormonal systems fail, characteristic patterns of dyslipidemia emerge that raise cardiovascular risk beyond what LDL-C alone predicts. To reduce residual cardiovascular risk, clinicians should look beyond cholesterol numbers and evaluate the underlying hormonal and metabolic drivers of dyslipidemia, enabling a more personalized approach to cardiovascular prevention.

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ISSN: (e) 3007-1607 (p) 3007-1593

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