Scholars LITERATURE

La Prensa Medica Argentina

Short Communication

DOI: https://doi.org/10.47275/2953-4763-447
Volume 111 Issue 5

Cardiovascular Endocrinology: An Overview on Interplay of Hormones, Developmental Programming, and Cardiovascular Diseases

Aishwarya Tummala[1*], Anshul Yadav[2], Jahnavi Kudapa[3*] and Ahmed Shaik[4]

[1]Mahatma Gandhi Medical College and Research Institute, Pillayarkuppam, Puducherry, India
[2]Mari State University, Mari El Republic, Russia
[3]Pondicherry Institute of Medical Sciences, Kalapet, Puducherry, India
[4]Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

Abstract

Cardiovascular endocrinology is a rapidly evolving field that investigates the complex interplay between the endocrine and cardiovascular systems. This multidisciplinary area of study is essential for understanding how hormonal imbalances can influence cardiovascular health and disease. The endocrine system, through the secretion of hormones, plays a critical role in regulating cardiovascular functions such as blood pressure, heart rate (HR), and vascular tone. Conversely, cardiovascular events can impact hormone production and release, highlighting the bidirectional communication between these systems. This review explores the physiological interactions and pathophysiological consequences of disruptions in this delicate balance, with a focus on key hormones such as vitamin D, growth hormone, and thyroid hormones, and their implications for cardiovascular health. The review also delves into the role of developmental programming in shaping cardiovascular health, emphasizing the impact of early life factors such as maternal nutrition, stress, and environmental exposures on long-term cardiovascular outcomes. Additionally, it examines the influence of sex hormones, lipoprotein metabolism, and emerging research areas such as the gut microbiome and novel biomarkers on cardiovascular disease (CVD) risk. Clinical studies, including landmark trials like the Framingham heart study (FHS) and the Women's Health Initiative, are discussed to highlight the translation of research findings into clinical practice. The review concludes by addressing the challenges and future directions in cardiovascular endocrinology, underscoring the need for ongoing research to develop effective interventions and improve patient outcomes.

Keywords: Cardiovascular endocrinology, Developmental programming, Hormonal regulation, Lipoprotein metabolism

Correspondence to: Aishwarya Tummala and Jahnavi Kudapa, Mahatma Gandhi Medical College and Research Institute, Pillayarkuppam, Puducherry, India and Pondicherry Institute of Medical Sciences, Kalapet, Puducherry, India.

Citation: Tummala A, Yadav A, Kudapa J, Shaik A (2025) Cardiovascular Endocrinology: An Overview on Interplay of Hormones, Developmental Programming, and Cardiovascular Diseases. Prensa Med Argent, Volume 111:5. 447. DOI: https://doi.org/10.47275/2953-4763-447

Received: April 17, 2025; **Accepted:** July 11, 2025; **Published:** July 16, 2025

Introduction

Cardiovascular endocrinology is a multidisciplinary field that explores the intricate interactions between the endocrine and cardiovascular systems. This area of study is crucial for understanding how hormonal imbalances can influence cardiovascular health and disease [1, 2]. The research in this field spans various topics, including the role of hormones like vitamin D, growth hormone, and thyroid hormones in cardiovascular health, as well as the development of pharmaceutical strategies to address cardiovascular and endocrinological disorders [3, 4]. The endocrine and cardiovascular systems are intricately linked, with bidirectional communication influencing numerous physiological processes. The endocrine system, through the secretion of hormones, profoundly impacts cardiovascular function, regulating blood pressure, HR, and vascular tone [5, 6]. Conversely, cardiovascular events can affect hormone production and release, further highlighting the complex interplay between these two essential systems [7]. This article will explore this relationship, examining both normal physiological interactions and the pathophysiological consequences of disruptions in this delicate balance.

Vitamin D is increasingly recognized as a hormone with potential implications for cardiovascular and metabolic diseases. A study highlighted the association between low vitamin D levels and an unfavorable cardiometabolic profile in obese women with metabolic syndrome, suggesting a need for further research in diverse populations like those in Egypt [8]. Despite the observed associations, randomized controlled trials have shown conflicting results regarding the benefits of vitamin D supplementation for cardiovascular health, indicating that routine screening for deficiency is not universally recommended [9].

Interplay Between the Endocrine and Cardiovascular Systems

Cardiovascular endocrinology explores the intricate relationship between the endocrine system and the cardiovascular system [10-12]

(Table 1). It extends beyond the well-established connections like diabetes mellitus and its cardiovascular complications [11], delving into novel mechanisms linking the cardiovascular system with a multitude of blood-borne bioactive substances and their cellular targets [11]. This field investigates how hormones influence various aspects of cardiovascular health, impacting processes like blood pressure regulation, lipid metabolism, and the development of atherosclerosis [13-15]. Natriuretic peptides, for example, play a crucial role in the cardiovascular system's homeostatic mechanisms [16].

Growth hormone plays a crucial role in heart development and function. Growth hormone deficiency can lead to metabolic issues that increase the risk of atherosclerosis, while growth hormone excess, as seen in acromegaly, is linked to CVD and heart failure. Early detection and treatment of growth hormone-related disorders can prevent these complications [9]. Thyroid hormones are vital for cardiovascular regulation (Figure 1). Both hypothyroidism and hyperthyroidism can lead to various cardiac issues. Hypothyroidism may cause hypertension and hyperlipidemia, which can be managed with hormone replacement therapy (HRT). Hyperthyroidism can affect heart structure, function, and rhythm, potentially leading to heart failure if untreated. The use

Table 1: Key endocrine hormones and their cardiovascular effects.

Hormone	Source	Cardiovascular effects	Clinical relevance
Angiotensin II	RAAS (Kidneys)	Vasoconstriction, increases blood pressure, promotes aldosterone release	Hypertension, heart failure, atherosclerosis
Aldosterone	Adrenal cortex	Sodium retention, increases blood volume and pressure	Primary aldosteronism, hypertension, heart failure
Epinephrine and norepinephrine	Adrenal medulla	Increases heart rate, cardiac output, vasoconstriction	Stress response, hypertension, arrhythmias
Insulin	Pancreas	Regulates glucose metabolism, endothelial function	Insulin resistance leads to atherosclerosis, diabetes, hypertension
Glucagon	Pancreas	Increases blood glucose, minor cardiac effects	Glucagonoma syndrome (rare), hypoglycemia-related tachycardia
Thyroid hormones (T3 and T4)	Thyroid gland	Regulate heart rate, contractility, vascular resistance	Hypothyroidism: bradycardia, hypertension; hyperthyroidism: tachycardia, atrial fibrillation
Cortisol	Adrenal cortex	Increases blood pressure, alters metabolism	Cushing's syndrome, metabolic syndrome, hypertension
Estrogen	Ovaries	Vasodilation reduces atherosclerosis risk	Cardioprotective; menopause increases cardiovascular risk
Testosterone	Testes, adrenal cortex	Increasing vascular tone may contribute to hypertension	Excess linked to hypertension, polycythemia, and cardiovascular risk
Atrial natriuretic peptide	Heart (Atria)	Promotes sodium excretion, lowers blood pressure	Protective in heart failure, but levels rise in disease states

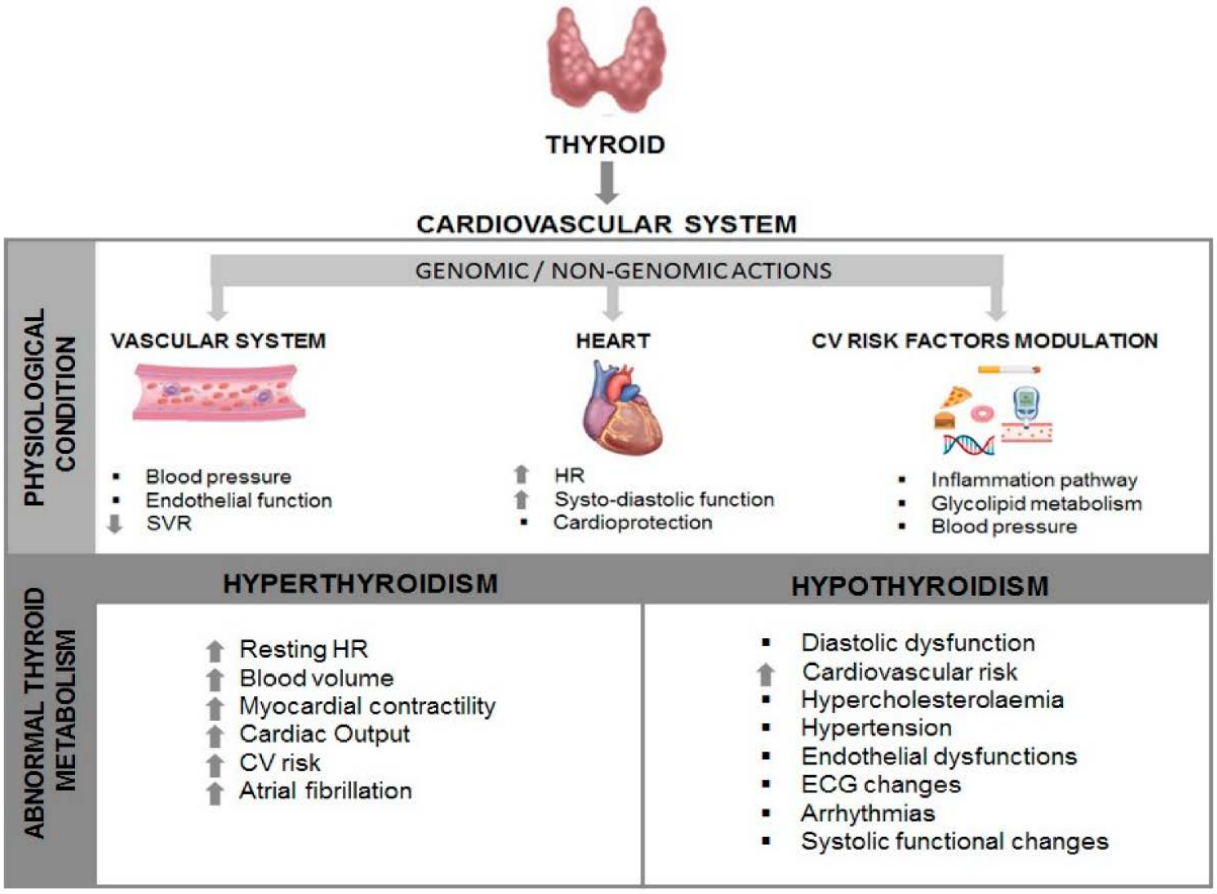

Figure 1: Interaction of the thyroid and the cardiovascular system. SVR: systemic vascular resistance and ECG: electrocardiogram [17].

of medications like amiodarone, which can affect thyroid function, underscores the importance of monitoring thyroid health in patients with cardiovascular conditions [9].

Specific endocrine disorders can directly impact the cardiovascular system. Hyperthyroidism, for instance, can lead to thyrotoxic cardiomyopathy, a rare but serious condition [18]. This highlights the potential for endocrine imbalances to induce significant cardiovascular consequences. Conversely, adipose tissue, a key endocrine organ, secretes adipokines that exert endocrine effects on the vascular wall, influencing endothelial function and contributing to cardiovascular risk [19, 20]. The Renin-Angiotensin-Aldosterone System (RAAS) plays a central role in regulating blood pressure and fluid balance, and its dysregulation is implicated in both cardiovascular and endocrine diseases. The interplay between RAAS and other endocrine systems, such as the parathyroid hormone system, is complex and warrants further investigation [21].

Chronic liver disease significantly impacts both endocrine and cardiovascular homeostasis. The liver's crucial role in metabolic regulation and immune function means that its dysfunction can trigger a cascade of events affecting the cardiovascular system. This can manifest as arrhythmias, cardiomyopathy, and circulatory complications, ultimately leading to conditions like portal hypertension, pulmonary hypertension, and cardiac failure. The interplay between inflammation, oxidative stress, and imbalanced vasoactive mediators, further contributes to the development of these complications. Age, sex, the gut microbiome, and organ transplantation also influence this complex interaction [22].

The interaction between the endocrine and cardiovascular systems extends beyond the examples discussed above. The gut microbiota influences the endocrine system, affecting hormone production and potentially impacting cardiovascular health [23]. The respiratory and cardiovascular systems are also closely linked, with pathologies in one system often affecting the other [24]. Furthermore, social support has been shown to influence cardiovascular, endocrine, and immune system function [25], underscoring the broader interconnectedness of physiological systems. Finally, the circadian system interacts with the endocrine system influencing hormonal rhythms and impacting cardiovascular function [26]. The interplay between the immune system and endocrine also plays a crucial role especially in organs like the lungs, impacting both immune response and cardiovascular health [27].

Insulin resistance, a hallmark of type 2 diabetes, is a significant risk factor for CVD. It affects glucose metabolism and promotes arteriosclerosis through mechanisms involving oxidative stress and inflammation. Managing insulin resistance is crucial in preventing cardiovascular complications associated with diabetes [28]. The paraventricular nucleus in the brain plays a central role in neuroendocrine regulation of cardiovascular function. It modulates blood pressure and cardiovascular responses through hormones like vasopressin and oxytocin, which have both endocrine and neuromodulatory effects. Dysregulation of paraventricular nucleus activity can lead to hypertension and heart failure [29]. Sex hormones, including estrogen and testosterone, influence cardiovascular health differently in men and women. Abnormal levels of these hormones can increase the risk of CVD, as seen in conditions like menopause and Klinefelter syndrome. The effects of sex hormone-based therapies on cardiovascular health remain an area of ongoing research [30].

Developmental Programming and the Cardiovascular System

Early development is a critical period for establishing the lifelong relationship between the endocrine and cardiovascular systems [31]. The interplay between genetic, environmental, and nutritional factors during this time can program the cardiovascular and endocrine systems, influencing disease risk later in life (Table 2) [31]. The perinatal period, in particular, is highly sensitive to various environmental stressors, including nutritional deficiencies, hypoxia, and maternal illness. This programming can be affected by various factors, including maternal health, environmental exposures, and genetic predispositions, which can lead to chronic conditions such as CVD and metabolic disorders [32].

Adhesion G protein-coupled receptors play a crucial role in cardiovascular development. Mutations in adhesion G protein-coupled receptors, such as adgrl2, can lead to congenital heart defects, which are the most common congenital birth defects affecting millions of newborns annually. These defects can predispose individuals to further cardiovascular issues later in life, highlighting the importance of genetic factors in early cardiovascular development [33]. Environmental exposures during early development can lead to epigenetic changes that affect cardiovascular health across generations. For instance, exposure to toxicants like lead and phthalates can have sex-specific effects on cardiovascular outcomes, emphasizing the

Table 2: Impact of early life factors on cardiovascular health.

Factor	Mechanism	Long-term cardiovascular impact	Examples/clinical relevance
Maternal undernutrition	Fetal adaptations lead to altered organ development	Increased risk of hypertension, endothelial dysfunction	Dutch famine study: higher rates of CVD in adulthood
Maternal overnutrition and obesity	Fetal exposure to excess nutrients alters metabolism	Increased risk of metabolic syndrome, hypertension, diabetes	Childhood obesity, early-onset hypertension
Gestational diabetes	Fetal hyperinsulinemia, endothelial dysfunction	Higher risk of insulin resistance, vascular dysfunction	Increased CVD risk in offspring
Intrauterine growth restriction	Reduced fetal nutrient supply leads to vascular remodeling	Increased arterial stiffness, hypertension	Barker Hypothesis: low birth weight linked to CVD
Preterm birth	Immature cardiovascular and renal systems	Higher risk of hypertension, heart failure	Preterm individuals show early arterial aging
Placental insufficiency	Poor placental function alters fetal programming	Impaired endothelial function, increased blood pressure	Preeclampsia linked to later maternal and offspring CVD
Prenatal stress and glucocorticoid exposure	Increased fetal cortisol exposure	Altered autonomic regulation, hypertension	Maternal stress, corticosteroid treatment in pregnancy
Environmental toxins (e.g., smoking, pollution, alcohol)	Epigenetic modifications affect vascular development	Increased risk of atherosclerosis, endothelial dysfunction	Fetal alcohol syndrome, smoking-induced fetal growth restriction
Early postnatal nutrition	Overfeeding or underfeeding affects metabolism	Increased risk of obesity, hypertension, metabolic syndrome	Formula feeding vs. breastfeeding impacts cardiovascular risk

role of epigenetic programming in disease risk [34]. The maternal environment, including nutrition and stress levels, significantly influences fetal development. Nutritional deficiencies or excesses can lead to developmental programming that predisposes offspring to cardiovascular and metabolic diseases. Stressors such as hypoxia and maternal malnutrition can alter fetal cardiovascular and endocrine systems, leading to long-term health consequences [35]. The concept of fetal programming suggests that adverse conditions during fetal development, such as maternal obesity or gestational diabetes, can lead to structural and functional changes in the offspring's cardiovascular system. These changes can increase the risk of diseases like hypertension and coronary heart disease in adulthood [36, 37].

These stressors can perturb the maternal and fetal cardiovascular and endocrine systems, leading to long-term health consequences, a concept known as the "Developmental Origins of Adult Disease." Research has demonstrated that in utero stress can predispose individuals to cardiovascular and metabolic diseases later in life, such as hypertension, type 2 diabetes, and increased adiposity. The concept of "Predictive Adaptive Responses" suggests the fetus adapts to its anticipated environment, but mismatches between predicted and actual environments can lead to disease. Animal models, including rats, sheep, and guinea pigs, have been instrumental in studying these developmental programming effects, investigating the impact of various gestational perturbations on cardiovascular development. These studies highlight the crucial role of utero conditions in shaping cardiovascular health throughout life [35].

While early development is a critical period for establishing the relationship between the endocrine and cardiovascular systems, it is also important to consider the role of postnatal factors. Childhood and adolescence are additional critical periods where environmental influences, such as diet and physical activity, can further shape cardiovascular health. Understanding the interplay between early programming and later life exposures is crucial for developing comprehensive strategies to prevent CVD.

Key Hormonal Influences on Cardiovascular Health

Several hormones significantly affect cardiovascular function and disease risk. Hormonal influences play a significant role in cardiovascular health, with sex hormones such as estrogen, progesterone, and testosterone being key determinants [38]. These hormones impact cardiovascular physiology and pathology through various mechanisms, including modulation of vascular function, inflammation, and metabolic processes [39]. The effects of these hormones are particularly evident in the differences observed between men and women in terms of CVD risk and progression [40]. Understanding these hormonal influences is crucial for developing gender-specific strategies for CVD prevention and treatment.

The thyroid hormone's role in atherosclerosis is increasingly recognized [13]. Similarly, growth hormone, insulin-like growth factors I (IGF-I), and IGF-binding proteins are implicated in atherogenesis, particularly through their effects on postprandial lipoprotein metabolism [13]. Disturbances in the growth hormone axis/IGF system are linked to premature atherosclerosis and increased cardiovascular mortality (Figure 2), suggesting a U-shaped relationship between growth hormone axis/IGF system function and cardiovascular morbidity/mortality [13]. Furthermore, vitamin D, now recognized as a multifaceted hormone, potentially modifies the risk of cardiovascular and metabolic diseases [8]. Its deficiency is associated with an unfavorable cardiometabolic profile, particularly in populations with high prevalence of obesity and metabolic syndrome [8]. Finally, the role of PCSK9 in regulating low-density lipoprotein (LDL)-cholesterol levels is emerging as a crucial therapeutic target [42]. Inhibition of PCSK9 dramatically reduces LDL-cholesterol, potentially enhancing the efficacy of statin therapy [42].

Combined oral contraceptives are linked to a heightened risk of cardiovascular events, such as venous thromboembolism and ischemic stroke, with risk levels influenced by estrogen dose and progestogen type. The variability in risk underscores the importance of personalized contraceptive counseling and prescribing practices [43]. Estrogen enhances vasodilation by increasing the release of vasodilators like

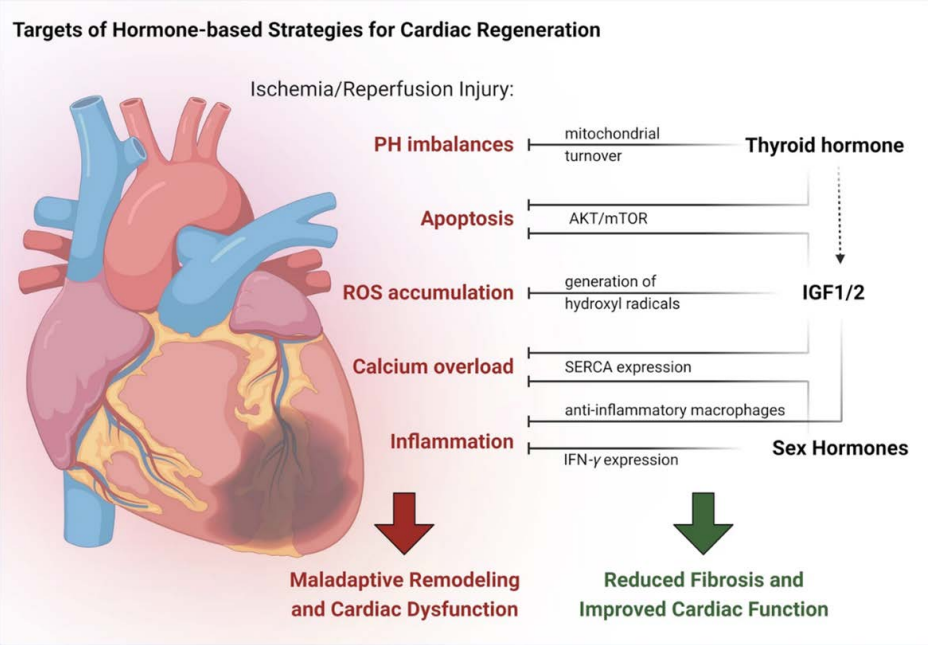

Figure 2: The combined effects of hormones on the structural and functional development of cardiomyocytes and their potential implications for heart regeneration [41].

nitric oxide and prostacyclin, which are more pronounced in women than men [44, 45]. Estrogen acts as a powerful anti-inflammatory agent, contributing to its protective effects against CVD and chronic renal disease [44]. The decline in estrogen levels of post-menopauses is associated with increased risk of atherosclerosis and other cardiovascular disorders, highlighting the protective role of estrogen before menopause [45, 46].

Women generally experience CVD at a later age than men, with a delay of about 10 years, attributed to the protective effects of endogenous estrogens [44]. Hormonal fluctuations during events like menarche, pregnancy, and menopause can promote a pro-inflammatory state, increasing CVD risk in women [47]. Hormone therapy, particularly with bioidentical hormones, may extend the cardiovascular protection conferred by endogenous estrogens if initiated early after menopause [44]. The impact of hormone therapy varies based on factors such as timing, administration route, and formulation, necessitating careful consideration in clinical practice [46]. Testosterone and progesterone hormones also contribute to cardiovascular health, with complex interactions affecting heart and blood vessel function differently in men and women [44, 48]. Although not a traditional sex hormone, vitamin D acts as a steroid hormone and its cardiovascular effects are controversial, with both low and high levels associated with increased risk [49].

While the protective role of estrogen in cardiovascular health is well-documented, the influence of other hormones like progesterone and testosterone, as well as the impact of hormonal contraceptives, adds complexity to the understanding of hormonal effects on cardiovascular health. Additionally, the role of vitamin D as a steroid hormone in cardiovascular health remains a topic of debate, highlighting the need for further research to clarify its effects.

Lipoprotein Metabolism and CVD

The diverse lipoprotein subfractions (very low-LDL, LDL, and high-LDL (HDL)) have distinct metabolic properties and varying relationships with coronary heart disease [50]. Sub fractionation of lipoproteins holds potential for improved risk prediction beyond classical lipid risk factors [50]. Understanding the intricate regulation of these lipoproteins and their influence on atherogenesis is critical in cardiovascular endocrinology.

Lipoprotein metabolism plays a crucial role in the development and progression of CVDs and is significantly influenced by endocrine disorders [51]. The intricate relationship between lipoprotein metabolism, endocrine function, and cardiovascular health is underscored by various studies that explore the causal associations, genetic factors, and potential therapeutic interventions [52, 53]. This section will explore the key aspects of this relationship, focusing on the impact of lipoprotein metabolism on cardiovascular health, the influence of endocrine disorders, and potential treatment strategies.

Disorders of lipoprotein metabolism have been causally linked to several CVD, including coronary artery disease, aortic aneurysm, heart failure, hypertension, and stroke. These associations are supported by Mendelian randomization studies, which highlight the significant risk posed by dysregulated lipoprotein metabolism on cardiovascular health [54]. Lipoproteins such as HDL, LDL, and lipoprotein(a) are critical in lipid transport and metabolism. Dyslipidemia, characterized by abnormal levels of these lipoproteins, is a major risk factor for atherosclerosis and other cardiovascular conditions [55]. Elevated levels of lipoprotein(a) are particularly associated with an increased risk of atherosclerotic CVD and calcific aortic valve disease. Novel therapeutics targeting lipoprotein(a) are under development, aiming to reduce cardiovascular risk [56].

Endocrine disorders such as hypothyroidism, Cushing's syndrome, and polycystic ovary syndrome can lead to dyslipidemia, thereby increasing the risk of atherosclerotic CVD. These conditions often result in elevated LDL-C and triglycerides, contributing to cardiovascular risk [57, 58]. Treatment of the underlying endocrine disorder can improve lipid profiles and potentially reduce cardiovascular risk. For instance, managing hypothyroidism or testosterone deficiency can lead to reductions in LDL-C levels [57]. The interplay between endocrine disorders and lipoprotein metabolism necessitates a comprehensive approach to managing dyslipidemia, often requiring both endocrine treatment and lipid-lowering therapies [58].

Genetic factors play a significant role in lipoprotein metabolism, with variations in genes affecting LDL, HDL, and triglyceride levels. These genetic predispositions can lead to disorders such as familial hypercholesterolemia and familial hypertriglyceridemia, which are associated with increased cardiovascular risk [59]. Peroxisome proliferator-activated receptor α (PPARα) is a key regulator of lipid metabolism, influencing the functionality of lipoproteins. PPARα agonists, such as fibric acid derivatives, are used to treat hypertriglyceridemia and low HDL cholesterol levels, although their efficacy in reducing cardiovascular events remains debated [60].

While the relationship between lipoprotein metabolism, endocrine disorders, and CVD is well-established, ongoing research continues to explore the underlying mechanisms and potential therapeutic targets. The development of novel treatments, particularly those targeting specific lipoproteins like lipoprotein(a), holds promise for reducing cardiovascular risk in affected populations. However, the complexity of these interactions necessitates a multifaceted approach to treatment, integrating genetic, metabolic, and lifestyle factors.

Clinical Studies

Cardiovascular endocrinology has been extensively studied through numerous clinical trials, shedding light on the intricate relationship between hormonal regulation and CVD. One of the most influential studies in this field is the FHS, an ongoing cohort study that has tracked over 15,000 participants across multiple generations. This study identified key CVD risk factors, including hypertension, diabetes, and obesity, and established the role of insulin resistance and thyroid dysfunction in heart disease. The long-term data from FHS has been instrumental in shaping modern preventive cardiology and risk assessment models [61].

The diabetes control and complications trial (NCT00360815) and its follow-up, the epidemiology of diabetes interventions and complications (EDIC) study (NCT00360893), provided groundbreaking evidence on the impact of glucose control in diabetes management. Enrolling 1,441 individuals with type 1 diabetes, the study found that intensive glucose control reduced cardiovascular events by 42% (hazard ratio (HR) 0.58; 95% confidence interval (CI): 0.31 - 1.07; $p = 0.08$). This effect was confirmed in the long-term EDIC follow-up, emphasizing the importance of early and sustained glucose control in preventing cardiovascular complications. These findings have had a significant influence on diabetes treatment guidelines worldwide [62].

Another pivotal study, the women's health initiative (WHI) (NCT00000611), examined the cardiovascular effects of HRT in

postmenopausal women. With a large cohort of 27,347 participants, WHI demonstrated that estrogen-progestin therapy increased the risk of coronary heart disease (HR 1.18; 95% CI: 0.95 - 1.45) and stroke (HR 0.94; 95% CI: 0.78 - 1.14), leading to a major shift in clinical recommendations regarding HRT use. While estrogen-alone therapy showed a neutral or slightly beneficial effect in younger women, the overall findings raised concerns about the widespread use of HRT for cardiovascular protection [63].

The heart outcomes prevention evaluation trial further underscored the importance of the angiotensin-converting enzyme (ACE) inhibition or vitamin E in cardiovascular health. In a study of 9,297 high-risk patients, the ACE inhibitor ramipril significantly reduced myocardial infarction by 20% (relative risk (RR) 0.80; 95% CI: 0.70 - 0.90; $p < 0.001$), stroke by 32% (RR 0.68; 95% CI: 0.56 - 0.84; $p < 0.001$), and cardiovascular mortality by 26% (RR 0.74; 95% CI: 0.64 - 0.87; $p < 0.001$). These findings reinforced the role of ACE inhibitors in reducing cardiovascular risk and improving survival in patients with hypertension and diabetes [64].

The UK prospective diabetes study (UKPDS) provided critical insights into the management of type 2 diabetes and its cardiovascular complications. With 5,102 participants, UKPDS demonstrated that intensive glucose control reduced microvascular complications by 25% ($p = 0.0099$), while metformin therapy specifically lowered diabetes-related mortality by 42% ($p = 0.017$). This study played a crucial role in defining treatment targets for blood glucose levels and emphasizing the benefits of early intervention in diabetes management [65].

Finally, the systolic blood pressure intervention trial (NCT01206062) provided strong evidence supporting aggressive blood pressure management. Among 9,361 participants at high cardiovascular risk but without diabetes, lowering systolic blood pressure to <120 mmHg resulted in a 25% reduction in cardiovascular events (HR 0.75; 95% CI: 0.64 - 0.89; $p < 0.001$) and a 27% reduction in all-cause mortality (HR 0.73; 95% CI: 0.60 - 0.90; $p = 0.003$). These findings led to a paradigm shift in hypertension treatment guidelines, advocating for more intensive blood pressure control in high-risk patients [66].

Together, these landmark studies have significantly influenced clinical practice in cardiovascular endocrinology, highlighting the critical role of hormones in regulating blood pressure, glucose metabolism, and vascular function. Their findings continue to shape guidelines for diabetes management, hormone therapy, and hypertension treatment, ultimately improving patient outcomes in cardiovascular health.

Emerging Research Areas in Cardiovascular Endocrinology

Emerging research in cardiovascular endocrinology is advancing rapidly, focusing on the complex interplay between hormones and cardiovascular health (Table 3). This interdisciplinary field is gaining momentum as researchers explore the complex interactions between cardiovascular and endocrine systems, aiming to improve patient outcomes through innovative treatments and a deeper understanding of underlying mechanisms. This section highlights key emerging areas in this field.

Table 3: Emerging research areas in cardiovascular endocrinology.

Research area	Focus	Potential impact	Advantages	Limitations
Gut microbiome and cardiovascular health	Investigating how gut bacteria influence metabolic and hormonal pathways affecting CVD risk	May lead to microbiome-targeted therapies for hypertension, diabetes, and atherosclerosis	Potential for novel, non-invasive therapeutic approaches and personalized treatments	Complex interactions and limited understanding of how specific microbiota influence CVD
Adipokines and cardiovascular risk	Studying hormones secreted by adipose tissue, such as leptin, adiponectin, and resistin, and their role in heart disease	Could provide new targets for obesity-related cardiovascular disorders	Identification of novel biomarkers and therapies for obesity-induced heart disease	Adipokines' roles are multifaceted, making it difficult to develop targeted therapies without side effects
Sex hormones and CVD	Examining how estrogen, testosterone, and progesterone influence cardiovascular health in men and women	May lead to gender-specific treatment approaches for CVD prevention	Tailored treatments for men and women based on hormonal influences, improving efficacy	Variability in hormonal effects based on age, menopause status, and other factors complicates generalization
Thyroid dysfunction and heart failure	Exploring how hypothyroidism and hyperthyroidism contribute to arrhythmia, heart failure, and vascular dysfunction	Could refine screening and treatment strategies for thyroid-related heart conditions	Improved diagnosis and early intervention for thyroid-related cardiovascular complications	Requires larger studies to clarify thyroid-heart interactions in diverse populations
Endocrine disruptors and cardiovascular health	Investigating environmental chemicals (e.g., BPA, phthalates) that interfere with hormonal regulation and contribute to CVD	May lead to policy changes and preventive measures to reduce exposure	Potential for public health interventions to reduce exposure to harmful chemicals	Limited long-term studies on specific chemicals and their direct cardiovascular effects
Glucocorticoids and cardiometabolic risk	Studying the long-term effects of stress hormones on blood pressure, insulin resistance, and CVD	Could improve management of chronic stress and metabolic syndrome	Could provide new targets for chronic stress-related CVD prevention	Variability in glucocorticoid responses and lack of consensus on treatment guidelines
Chronobiology and cardiovascular health	Exploring how circadian rhythms affect hormone secretion and cardiovascular function	May lead to time-based (chronotherapy) interventions for better treatment outcomes	Innovative approach to treatment that could optimize drug delivery and lifestyle changes	Complexities in circadian biology and the need for more research on clinical applications
Novel biomarkers for CVD risk assessment	Identifying new endocrine markers, such as fibroblast growth factors and irisin, for early detection of CVD	Could enhance personalized risk stratification and early intervention	Potential for more accurate and early detection of cardiovascular risks	Need for validation of biomarkers in large-scale clinical studies
Metabolic syndrome and CVD	Understanding the interplay between insulin resistance, dyslipidemia, hypertension, and hormonal regulation	May refine treatment strategies to prevent cardiovascular complications in metabolic syndrome	Integrated approach could address multiple cardiovascular risk factors simultaneously	Interactions between metabolic components are complex, making it difficult to design targeted therapies
Gene therapy and hormonal regulation	Investigating gene-editing approaches to modulate hormonal pathways linked to CVD	Holds potential for personalized and long-term solutions to endocrine-related heart diseases	Innovative potential for curing genetic conditions that predispose individuals to CVD	Ethical concerns, regulatory challenges, and long-term safety risks associated with gene-editing technologies

GLP1RAs have emerged as a significant area of research due to their dual role in managing type 2 diabetes and providing cardiovascular benefits. Studies have shown that these agents can improve cardiovascular outcomes, making them a focal point in cardiovascular endocrinology research. Research hotspots include the effects of GLP1RAs on cardiovascular outcomes, their efficacy, and protective mechanisms against metabolic abnormalities. Semaglutide, a specific GLP1RA, is a prominent subject of study, particularly in placebo-controlled trials [67].

The cardiovascular implications of various endocrine disorders, such as subclinical hypothyroidism and testosterone deficiency, are under investigation. These conditions have been linked to cardiovascular events, but the benefits of replacement therapies remain controversial due to insufficient evidence from randomized controlled trials. Growth hormone deficiency is another area of interest, with studies suggesting that replacement therapy may reverse detrimental cardiovascular effects, although more research is needed to confirm these findings [68].

Recent advances in molecular biology have led to the development of new therapeutic strategies targeting the remodeling of cardiac tissue and the role of paracrine and autocrine growth factors in heart disease. These approaches aim to address chronic cardiovascular conditions such as heart failure [69]. The use of specific receptor antagonists, such as those targeting endothelin, is being explored for their potential to regulate hemodynamic homeostasis and treat hypertension [70].

The discovery of new biomarkers related to lipid metabolism, glycemia, inflammation, and cardiac damage is a growing area of research. These biomarkers hold promises for better risk stratification and the development of new intervention targets in CVD management [71]. The role of cholesterol as a paracrine growth factor in atherogenesis is being re-evaluated, with implications for the early effects of lipid-lowering drugs on atherosclerotic plaques [69].

Research continues to uncover new insights into the field. The role of nuclear receptors, such as ROR-α, in modulating CVD and inflammatory responses is under investigation [14]. The heart itself functions as an endocrine organ, producing hormones like GDF-15 and myostatin that influence systemic physiology [72]. The use of cardiac biomarkers, such as troponins and natriuretic peptides, in both human and veterinary medicine, highlights species-specific differences that must be considered when interpreting results [73]. Ongoing controversies exist regarding the cardiovascular effects of calcium supplementation [74] and the impact of dipeptidyl peptidase IV inhibitors on heart failure risk [75]. Moreover, the field is expanding its scope to address global health challenges, particularly in emerging markets, by developing strategic brand frameworks for expanding access to cardiovascular and endocrinology treatments [76].

New therapeutic approaches, such as the use of cyclodextrin to reverse atherosclerosis, show promise in preclinical studies. This treatment enhances cholesterol metabolism and could be repurposed for human use, offering a novel strategy for managing atherosclerosis [77]. The potential of targeting ANGPTL3 for lipid management, similar to PCSK9 inhibitors, represents another innovative direction in cardiovascular endocrinology research [78].

While these emerging areas offer promising avenues for research and treatment, challenges remain in fully understanding the complex interactions between cardiovascular and endocrine systems. The need for more robust clinical trials and validation of novel biomarkers is critical to translating these findings into clinical practice. Additionally, the exploration of new mechanisms linking cardiovascular health with endocrine functions continues to be a fertile ground for future research, potentially leading to groundbreaking therapeutic interventions.

Conclusion

While the field of cardiovascular endocrinology is advancing with promising research and therapeutic strategies, challenges remain. The complexity of hormonal interactions and their impact on cardiovascular health necessitates ongoing research to clarify these relationships and develop effective interventions. Additionally, the variability in healthcare infrastructure and economic conditions across different regions poses challenges for the implementation of new treatments, particularly in emerging markets. These factors highlight the need for tailored approaches that consider local contexts and patient needs. One of the most transformative developments is the integration of artificial intelligence (AI) into cardiovascular endocrinology research and clinical practice. AI has the potential to revolutionize the way we diagnose, predict, and manage cardiovascular and endocrine disorders by leveraging large datasets, identifying patterns, and providing personalized treatment recommendations.

However, the integration of AI into cardiovascular endocrinology also presents challenges, including the need for robust data privacy measures, ethical considerations, and the validation of AI models in diverse populations. Collaborative efforts between researchers, clinicians, and AI experts will be essential to address these challenges and fully realize the potential of AI in this field. As we move forward, the synergy between cardiovascular endocrinology and AI holds great promise for advancing our understanding of hormonal influences on cardiovascular health and transforming the landscape of preventive and therapeutic interventions. By embracing these technological advancements, we can pave the way for a future where personalized, data-driven care becomes the cornerstone of cardiovascular endocrinology.

Acknowledgements

None.

Conflict of Interest

None.

References

1. Guldan M, Unlu S, Abdel-Rahman SM, Ozbek L, Gaipov A, et al. (2024) Understanding the role of sex hormones in cardiovascular kidney metabolic syndrome: toward personalized therapeutic approaches. J Clin Med 13: 4354. https://doi.org/10.3390/jcm13154354

2. Clemente-Suárez VJ, Redondo-Flórez L, Rubio-Zarapuz A, Martín-Rodríguez A, Tornero-Aguilera JF (2024) Microbiota implications in endocrine-related diseases: from development to novel therapeutic approaches. Biomedicines 12: 221. https://doi.org/10.3390/biomedicines12010221

3. Cappola AR, Auchus RJ, Fuleihan GEH, Handelsman DJ, Kalyani RR, et al. (2023) Hormones and aging: an endocrine society scientific statement. J Clin Endocrinol Metab 108: 1835-1874. https://doi.org/10.1210/clinem/dgad225

4. Wimalawansa SJ (2025) Enhancing the design of nutrient clinical trials for disease prevention-a focus on vitamin D: a systematic review. Nutr Rev 83: e1740-e1781. https://doi.org/10.1093/nutrit/nuae164

5. Ashraf FUN, Ghouri K, Someshwar FNU, Kumar S, Kumar N, et al. (2023) Insulin resistance and coronary artery disease: untangling the web of endocrine-cardiac connections. Cureus 15: e51066. https://doi.org/10.7759/cureus.51066

6. Goel M, Mittal A, Jain VR, Bharadwaj A, Modi S, et al. (2023) Integrative functions of the hypothalamus: linking cognition, emotion and physiology for well-being and adaptability. Ann Neurosci 32: 128-142. https://doi.org/10.1177/09727531241255492

7. Lisco G, Giagulli VA, Iovino M, Zupo R, Guastamacchia E, et al. (2022) Endocrine system dysfunction and chronic heart failure: a clinical perspective. Endocrine 75: 360-376. https://doi.org/10.1007/s12020-021-02912-w

8. Krentz AJ, Chilton RJ (2023) Cardiovascular endocrinology and metabolism: theory and practice of cardiometabolic medicine. Academic Press.

9. Ghaemmaghami Z (2022) Endocrine disorders and the cardiovascular system. In Practical Cardiology. Elsevier, pp 685-691.

10. Funder JW (2001) Cardiovascular endocrinology: into the new millennium. Trends Endocrinol Metab 12: 283-285. https://doi.org/10.1016/s1043-2760(01)00443-x

11. Goetze JP (2012) Defining cardiovascular endocrinology. Cardiovasc Endocrinol Metab 1: 2-3. https://doi.org/10.1097/XCE.0b013e328355d175

12. Baxter JD, Young WF Jr, Webb P (2003) Cardiovascular endocrinology: introduction. Endocr Rev 24: 253-260. https://doi.org/10.1210/er.2003-0099

13. Twickler TB, Cramer MJM, Dallinga-Thie GM, Chapman MJ, Erkelens DW, et al. (2003) Adult-onset growth hormone deficiency: relation of postprandial dyslipidemia to premature atherosclerosis. J Clin Endocrinol Metab 88: 2479-2488 https://doi.org/10.1210/jc.2003-030278

14. Laitinen S, Staels B (2003) Potential roles of ROR-α in cardiovascular endocrinology. Nucl Recept Signal 1. https://doi.org/10.1621/nrs.01011

15. Cha BS, Park TS, Jeon BH, Lee YH, Yun MR, et al. (2015) Increased expression of ATP-binding cassette transporter A1 by cilostazol may be a possible mechanism for its protective effect against hepatic steatosis. Endocr Abstr 37: EP596. https://doi.org/10.1530/endoabs.37.EP596

16. Suzuki T, Yamazaki T, Yazaki Y (2001) The role of the natriuretic peptides in the cardiovascular system. Cardiovasc Res 51: 489-494. https://doi.org/10.1016/s0008-6363(01)00238-3

17. Pingitore A, Mastorci F, Lazzeri MFL, Vassalle C (2023) Thyroid and heart: a fatal pathophysiological attraction in a controversial clinical liaison. Endocrines 4: 722-741. https://doi.org/10.3390/endocrines4040053

18. Elendu C (2023) Cardiovascular sequelae of hyperthyroidism: a rare case report of thyrotoxic cardiomyopathy. Medicine 102: e36250. https://doi.org/10.1097/MD.0000000000036250

19. Akoumianakis I, Antoniades C (2017) The interplay between adipose tissue and the cardiovascular system: is fat always bad? Cardiovasc Res 113: 999-1008. https://doi.org/10.1093/cvr/cvx111

20. Oikonomou EK, Antoniades C (2019) The role of adipose tissue in cardiovascular health and disease. Nat Rev Cardiol 16: 83-99. https://doi.org/10.1038/s41569-018-0097-6

21. Zheng MH, Li FXZ, Xu F, Lin X, Wang Y, et al. (2020) The interplay between the renin-angiotensin-aldosterone system and parathyroid hormone. Front Endocrinol 11: 1-12. https://doi.org/10.3389/fendo.2020.00539

22. Matyas C, Haskó G, Liaudet L, Trojnar E, Pacher P (2021) Interplay of cardiovascular mediators, oxidative stress and inflammation in liver disease and its complications. Nat Rev Cardiol 18: 117-135. https://doi.org/10.1038/s41569-020-0433-5

23. Neuman H, Debelius JW, Knight R, Koren O (2015) Microbial endocrinology: the interplay between the microbiota and the endocrine system. FEMS Microbiol Rev 39: 509-521. https://doi.org/10.1093/femsre/fuu010

24. Cross TJ, Kim CH, Johnson BD, Lalande S (2020) The interactions between respiratory and cardiovascular systems in systolic heart failure. J Appl Physiol 128: 214-224. https://doi.org/10.1152/japplphysiol.00113.2019

25. Uchino BN, Cacioppo JT, Kiecolt-Glaser JK (1996) The relationship between social support and physiological processes: a review with emphasis on underlying mechanisms and implications for health. Psychol Bull 119: 488-531. https://doi.org/10.1037/0033-2909.119.3.488

26. Isorna E, de Pedro N, Valenciano AI, Alonso-Gómez ÁL, Delgado MJ (2017) Interplay between the endocrine and circadian systems in fishes. J Endocrinol 232: R141-R159. https://doi.org/10.1530/joe-16-0330

27. Webber T, Ronacher K, Conradie-Smit M, Kleynhans L (2022) Interplay between the immune and endocrine systems in the lung: implications for TB susceptibility. Front Immunol 13: 1-16. https://doi.org/10.3389/fimmu.2022.829355

28. Tsuchiya K (2023) Cardiovascular complications in insulin resistance and endocrine diseases. Endocr J 70: 249-257. https://doi.org/10.1507/endocrj.ej22-0457

29. Savić B, López SB, Murphy D, Japundžić-Žigon N (2023) Central neuroendocrine control of the cardiovascular system. In Yosten GLC, Cunningham JT (eds) Cardiovascular Neuroendocrinology. Masterclass in Neuroendocrinology. Springer, pp 17-42.

30. Willemars MM, Nabben M, Verdonschot JA, Hoes MF (2022) Evaluation of the interaction of sex hormones and cardiovascular function and health. Curr Heart Fail Rep 19: 200-212. https://doi.org/10.1007/s11897-022-00555-0

31. Bourque SL, Davidge ST (2020) Developmental programming of cardiovascular function: a translational perspective. Clin Sci 134: 3023-3046. https://doi.org/10.1042/cs20191210

32. Palinski W (2014) Effect of maternal cardiovascular conditions and risk factors on offspring cardiovascular disease. Circulation 129: 2066-2077. https://doi.org/10.1161/circulationaha.113.001805

33. Wang J, Gareri C, Rockman HA (2018) G-protein–coupled receptors in heart disease. Circ Res 123: 716-735. https://doi.org/10.1161/circresaha.118.311403

34. Svoboda LK, Ishikawa T, Dolinoy DC (2022) Developmental toxicant exposures and sex-specific effects on epigenetic programming and cardiovascular health across generations. Environ Epigenet 8: dvac017. https://doi.org/10.1093/eep/dvac017

35. Magness RR, Poston L (2005) Molecular, cellular and endocrine signalling in the perinatal cardiovascular system: interplay and developmental programming. J Physiol 565: 1. https://doi.org/10.1113/jphysiol.2005.086744

36. Alexander BT, Dasinger JH, Intapad S (2015) Fetal programming and cardiovascular pathology. Compr Physiol 5: 997-1025. https://doi.org/10.1002/cphy.c140036

37. Reid B (2024) From womb to well-being: the intricate link between psychoneuroendocrinology, metabolism, and fetal programming for optimal neurodevelopment and health. Psychoneuroendocrinology 160: 106743. https://doi.org/10.1016/j.psyneuen.2023.106743

38. dos Santos RL, da Silva FB, Ribeiro JRF, Stefanon I (2014) Sex hormones in the cardiovascular system. Horm Mol Biol Clin Investig 18: 89-103. https://doi.org/10.1515/hmbci-2013-0048

39. Salerni S, Di Francescomarino S, Cadeddu C, Acquistapace F, Maffei S, et al. (2015) The different role of sex hormones on female cardiovascular physiology and function: not only oestrogens. Eur J Clin Invest 45: 634-645. https://doi.org/10.1111/eci.12447

40. Vitale C, Mendelsohn ME, Rosano GM (2009) Gender differences in the cardiovascular effect of sex hormones. Nat Rev Cardiol 6: 532-542. https://doi.org/10.1038/nrcardio.2009.105

41. Galow AM, Brenmoehl J, Hoeflich A (2023) Synergistic effects of hormones on structural and functional maturation of cardiomyocytes and implications for heart regeneration. Cell Mol Life Sci 80: 1-33. https://doi.org/10.1007/s00018-023-04894-6

42. Betteridge DJ (2013) PCSK9—an exciting target for reducing LDL-cholesterol levels. Nat Rev Endocrinol 9: 76-78. https://doi.org/10.1038/nrendo.2012.254

43. Asubiaro J (2024) The impact of hormonal contraceptives on the incidence and progression of cardiovascular diseases in women: a systematic review. Cureus 16(7): e65366. https://doi.org/10.7759/cureus.65366

44. Cignarella A, Bolego C, Barton M (2024) Sex and sex steroids as determinants of cardiovascular risk. Steroids 206: 109423. https://doi.org/10.1016/j.steroids.2024.109423

45. den Ruijter HM, Kararigas G (2022) Estrogen and cardiovascular health. Front Cardiovasc Med 9: 1-4. https://doi.org/10.3389/fcvm.2022.886592

46. Yi KAN, Yu-Lu PENG, Ze-Hao ZHAO, Shu-Tong DONG, Yin-Xiao XU, et al. (2024) The impact of female sex hormones on cardiovascular disease: from mechanisms to hormone therapy. J Geriatr Cardiol 21: 669. https://doi.org/10.26599/1671-5411.2024.06.003

47. Hetherington K, Thomas J, Nicholls SJ, Barsha G, Bubb KJ (2024) Unique cardiometabolic factors in women that contribute to modified cardiovascular disease risk. Eur J Pharmacol 984: 177031. https://doi.org/10.1016/j.ejphar.2024.177031

48. Gottfried S, Spar M (2022) Hormones and cardiovascular disease. In Nutritional and Integrative Strategies in Cardiovascular Medicine. CRC Press, pp 231-248.

49. Tintut Y, Demer LL (2021) Potential impact of the steroid hormone, vitamin D, on the vasculature. Am Heart J 239: 147-153. https://doi.org/10.1016/j.ahj.2021.05.012

50. Sacks FM, Campos H (2003) Low-density lipoprotein size and cardiovascular disease: a reappraisal. J Clin Endocrinol Metab 88: 4525-4532. https://doi.org/10.1210/jc.2003-030636

51. Palmisano BT, Zhu L, Eckel RH, Stafford JM (2018) Sex differences in lipid

and lipoprotein metabolism. Mol Metab 15: 45-55. https://doi.org/10.1016/j.molmet.2018.05.008

52. Ganesh SK, Arnett DK, Assimes TL, Basson CT, Chakravarti A, et al. (2013) Genetics and genomics for the prevention and treatment of cardiovascular disease: update: a scientific statement from the American Heart Association. Circulation 128: 2813-2851. https://doi.org/10.1161/01.cir.0000437913.98912.1d

53. Morvaridzadeh M, Zoubdane N, Heshmati J, Alami M, Berrougui H, et al. (2024) High-density lipoprotein metabolism and function in cardiovascular diseases: what about aging and diet effects? Nutrients 16: 653. https://doi.org/10.3390/nu16050653

54. Gao Q, Tan JS, Fan L, Wang X, Hua L, et al. (2022) Causal associations between disorders of lipoprotein metabolism and ten cardiovascular diseases. Front Cell Dev Biol 10: 1-8. https://doi.org/10.3389/fcell.2022.1023006

55. Albitar O, D'Souza CM, Adeghate EA (2024) Effects of lipoproteins on metabolic health. Nutrients 16: 2156. https://doi.org/10.3390/nu16132156

56. Boffa MB, Koschinsky ML (2024) Lipoprotein (a) and cardiovascular disease. Biochem J 481: 1277-1296. https://doi.org/10.1042/BCJ20240037

57. Newman CB (2023) Effects of endocrine disorders on lipids and lipoproteins. Best Pract Res Clin Endocrinol Metab 37: 101667. https://doi.org/10.1016/j.beem.2022.101667

58. Sridharan K, Kalra S (2024) Dyslipidaemia in endocrine disorders. Indian Heart J 76: S83-S85. https://doi.org/10.1016/j.ihj.2023.12.012

59. Brown AS, Dababneh EG, Chaus A, Chyzhyk V, Marinescu V, et al. (2021) Genetic disorders of lipoprotein metabolism. In Davidson MH, Toth PP, Maki KC (eds) Therapeutic Lipidology. Contemporary Cardiology. Humana, pp 35-80.

60. Fuior EV, Zvintzou E, Filippatos T, Giannatou K, Mparnia V, et al. (2023) Peroxisome proliferator-activated receptor α in lipoprotein metabolism and atherosclerotic cardiovascular disease. Biomedicines 11: 2696. https://doi.org/10.3390/biomedicines11102696

61. Framingham Heart Study. [https://www.framinghamheartstudy.org/] [Accessed July 15, 2025]

62. Nathan DM, DCCT/Edic Research Group (2014) The diabetes control and complications trial/epidemiology of diabetes interventions and complications study at 30 years: overview. Diabetes Care 37: 9-16. https://doi.org/10.2337/dc13-2112

63. Manson JE, Chlebowski RT, Stefanick ML, Aragaki AK, Rossouw JE, et al. (2013) Menopausal hormone therapy and health outcomes during the intervention and extended poststopping phases of the Women's Health Initiative randomized trials. JAMA 310: 1353-1368. https://doi.org/10.1001/jama.2013.278040

64. The Heart Outcomes Prevention Evaluation Study - HOPE – Ramipril. American College of Cardiology. [https://www.acc.org/Latest-in-Cardiology/Clinical-Trials/2014/09/22/23/12/HOPE--Ramipril] [Accessed July 15, 2025]

65. King P, Peacock I, Donnelly R (1999) The UK prospective diabetes study (UKPDS): clinical and therapeutic implications for type 2 diabetes. Br J Clin Pharmacol 48: 643-648. https://doi.org/10.1046/j.1365-2125.1999.00092.x

66. SPRINT Research Group (2015) A randomized trial of intensive versus standard blood-pressure control. N Engl J Med 373: 2103-2116. https://doi.org/10.1056/nejmoa1511939

67. Shou X, Wang Y, Duan C, Yuan G, Wei N, et al. (2023) Knowledge domain and emerging trends of glucagon-like peptide 1 receptor agonists in cardiovascular research: a bibliometric analysis. Curr Probl Cardiol 48: 101194. https://doi.org/10.1016/j.cpcardiol.2022.101194

68. Grande D, Terlizzese P, Gioia MI, Parisi G, Giagulli VA, et al. (2019) New frontiers in the therapeutic approach of patients with cardiovascular and endocrine diseases. Endocr Metab Immune Disord Drug Targets 19: 605-621. https://doi.org/10.2174/1871530319666190101151542

69. Twickler MTB (2004) Cardiovascular endocrinology. Semin Vasc Med 4: 105-106. https://doi.org/10.1055/s-2004-835366

70. Naruse M, Naruse K, Demura H (1994) Recent advances in endothelin research on cardiovascular and endocrine systems. Endocr J 41: 491-507. https://doi.org/10.1507/endocrj.41.491

71. Cahill LE, Bertoia ML, Aroner SA, Mukamal KJ, Jensen MK (2015) New and emerging biomarkers in cardiovascular disease. Curr Diab Rep 15: 1-15. https://doi.org/10.1007/s11892-015-0661-y

72. Zhao J, Pei L (2020) Cardiac endocrinology: heart-derived hormones in physiology and disease. Basic Transl Sci 5: 949-960. https://doi.org/10.1016/j.jacbts.2020.05.007

73. Van Der Vekens N, Hunter I, Goetze JP, Decloedt A, De Clercq D, et al. (2013) Human and equine cardiovascular endocrinology: beware to compare. Cardiovasc Endocrinol Metab 2: 67-76. https://doi.org/10.1097/XCE.0000000000000009

74. Reid IR (2014) Controversy--cardiovascular effects of calcium supplementation. Nat Rev Endocrinol 10:641-642. https://doi.org/10.1038/nrendo.2014.146

75. Kanakia R, Martinho S, Patel T, Arain FA, Panday MM, et al. (2014) Does dipeptidyl peptidase IV inhibitor increase the risk of heart failure? A cardiologist's paradox. Cardiovasc Endocrinol Metab 3: 111-116. https://doi.org/10.1097/XCE.0000000000000036

76. Usuemerai PA, Ibikunle OE, Abass LA, Alemede V, Nwankwo EI, et al. (2024) A strategic brand development framework for expanding cardiovascular and endocrinology treatments in emerging markets. World J Adv Pharm Med Res 7: 77-101. https://doi.org/10.53346/wjapmr.2024.7.2.0047

77. Holmes D (2016) Sugar drug reverses atherosclerosis. Nat Rev Endocrinol 12: 310. https://doi.org/10.1038/nrendo.2016.62

78. Musunuru K, Kathiresan S (2017) Is ANGPTL3 the next PCSK9? Nat Rev Endocrinol 13: 503-504. https://doi.org/10.1038/nrendo.2017.88