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Original Research Article

Assessment of Leptin–Adiponectin Imbalance in Patients with Coronary Artery Disease and Its Relationship with Metabolic Risk Factors

Vijay R. Pandhare[1], Deepak R. Kamble[2], Deepali A. Vidhate[3], Ramesh Pradhan[4]

[1]Assistant Professor, Department of Biochemistry, GCS Medical College, Ahmedabad, Gujarat, India.
[2]Assistant Professor Department of Biochemistry, Dr. D Y Patil University, School of Medicine, Nerul, Navi Mumbai, India
[3]Professor, Department of Biochemistry, Dr. D Y Patil University, School of Medicine, Nerul, Navi Mumbai, India.
[4]Professor, Department of Biochemistry, GCS Medical College, Ahmedabad, Gujarat, India.

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Corresponding author: Dr. Deepali Vidhate
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Abstract

Background: Adipose tissue is an active endocrine organ that secretes adipokines involved in glucose metabolism, lipid homeostasis, inflammation and vascular function. Leptin generally exhibits pro-inflammatory and potentially pro-atherogenic effects, whereas adiponectin exerts insulin-sensitizing, anti-inflammatory and vasculoprotective actions. An imbalance between these adipokines may therefore contribute to coronary artery disease (CAD) and associated metabolic abnormalities.

Material and Methods: This hospital-based case-control study included 200 adults, comprising 100 angiographically confirmed CAD patients and 100 apparently healthy controls. Anthropometric measurements, blood pressure and conventional biochemical parameters were recorded. Serum leptin and adiponectin were estimated using enzyme-linked immunosorbent assays, and the leptin/adiponectin ratio (LAR) was calculated. CAD severity was assessed using the Gensini score. Associations between adipokines, metabolic risk factors and CAD severity were evaluated using correlation and multivariable regression analyses.

Results: CAD patients demonstrated higher body mass index, waist circumference, fasting glucose, triglycerides, LDL-C and hs-CRP and lower HDL-C than controls. Mean leptin concentration was higher in CAD patients (18.6±8.4 ng/mL) than controls (11.2±5.7 ng/mL), whereas adiponectin was lower (5.7±2.4 vs 8.9±3.1 µg/mL; both $p<0.001$). LAR was substantially higher among CAD patients (3.42±2.18 vs 1.35±0.91; $p<0.001$). LAR showed positive correlations with BMI, waist circumference, fasting glucose, triglycerides, HOMA-IR and Gensini score and an inverse correlation with HDL-C.

Conclusion: Leptin–adiponectin imbalance was significantly associated with CAD and adverse metabolic characteristics. LAR may provide a useful integrated marker of cardiometabolic dysfunction, although prospective studies are required to establish its independent predictive value.

Keywords: Leptin; Adiponectin; Leptin/Adiponectin Ratio; Coronary Artery Disease; Metabolic Syndrome; Obesity; Insulin Resistance And Gensini Score.

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Introduction

Coronary artery disease (CAD) remains one of the most important causes of morbidity and mortality worldwide. Although traditional risk factors such as obesity, diabetes mellitus, hypertension, dyslipidaemia and smoking account for a substantial proportion of cardiovascular risk, increasing evidence indicates that metabolic and inflammatory pathways linking adipose tissue to the vasculature also contribute to atherosclerotic disease. Adipose tissue is now recognised as an active endocrine organ that produces numerous biologically active molecules collectively termed adipokines. These mediators influence insulin sensitivity, lipid metabolism, inflammation, endothelial function and vascular remodelling, thereby providing a biological connection between excess adiposity and cardiovascular disease [1,2]. Leptin is a 16-kDa peptide hormone predominantly secreted by adipocytes and is an important regulator of appetite and energy balance. Circulating leptin concentrations generally increase with adipose tissue mass, particularly with obesity. Beyond its metabolic functions, leptin affects the cardiovascular system through sympathetic activation, oxidative stress, vascular inflammation, endothelial dysfunction, platelet activation and

vascular smooth-muscle proliferation [3–5]. Chronic hyperleptinemia may also produce selective leptin resistance, whereby the beneficial effects of leptin on energy regulation are attenuated while potentially adverse vascular effects persist [3,5].

Adiponectin represents a contrasting adipokine. It is abundantly secreted by adipocytes and possesses insulin-sensitising, anti-inflammatory and anti-atherogenic properties. Unlike leptin, circulating adiponectin concentrations generally decline with obesity, visceral adiposity, insulin resistance and metabolic syndrome [6,7]. Adiponectin acts through adiponectin receptors, particularly AdipoR1 and AdipoR2, activating pathways involved in AMP-activated protein kinase and lipid oxidation. These effects promote glucose and lipid homeostasis and may protect the vascular endothelium from inflammatory and oxidative injury [6,8].

Experimental studies have demonstrated several mechanisms by which adiponectin may inhibit atherosclerosis. Adiponectin suppresses endothelial inflammatory signalling, including nuclear factor-κB activation, and can reduce the expression of endothelial adhesion molecules [9]. It also inhibits lipid accumulation and scavenger-receptor expression in macrophages, potentially limiting foam-cell formation during atherogenesis [10]. In addition, adiponectin can influence vascular smooth-muscle-cell proliferation and growth-factor signalling [11]. These effects provide a mechanistic basis for its proposed vasculoprotective role.

Clinical studies have generally reported lower adiponectin concentrations among individuals with CAD. Prospective evidence and meta-analyses have suggested an inverse relationship between adiponectin and coronary heart disease, although the magnitude and independence of this association vary between populations [12,13]. Conversely, studies investigating leptin have produced more heterogeneous findings. Some prospective studies have identified an association between leptin and coronary disease, whereas others have shown that the association is substantially attenuated after adjustment for BMI and other metabolic factors [14,15].

Because leptin and adiponectin have broadly opposing metabolic and vascular effects, their relative concentrations may provide greater information than either biomarker alone. The leptin/adiponectin ratio (LAR) has therefore emerged as a potential marker of adipose-tissue dysfunction and cardiometabolic risk. Previous research in patients with severe coronary disease demonstrated associations between LAR and BMI, waist circumference, glucose, blood pressure, fasting insulin and HOMA, although its incremental diagnostic value remained uncertain [16]. Evidence from the Indian Atherosclerosis Research Study is particularly relevant because Asian Indians have a high burden of abdominal adiposity, insulin resistance, diabetes and premature CAD. In that cohort, CAD patients exhibited lower adiponectin and higher leptin concentrations, while both adipokines demonstrated relationships with obesity and metabolic risk factors [17].

However, the relationship between the leptin–adiponectin axis and angiographic CAD severity remains incompletely characterised, particularly in relation to conventional metabolic risk factors. Therefore, assessment of both adipokines together may improve understanding of the biological interface between adiposity, metabolic dysfunction and coronary atherosclerosis. The present study was designed to assess leptin and adiponectin concentrations in patients with CAD and determine whether their imbalance, expressed as LAR, is related to metabolic risk factors and angiographic disease severity.

Aim and Objectives

Aim: To assess leptin–adiponectin imbalance in patients with coronary artery disease and determine its relationship with metabolic risk factors.

Objectives

1. To compare serum leptin, adiponectin and leptin/adiponectin ratio between patients with CAD and healthy controls.
2. To determine the relationship of leptin, adiponectin and leptin/adiponectin ratio with metabolic risk factors and angiographic severity of CAD.

Material and Methods

A hospital-based case-control study was designed to evaluate the relationship between leptin, adiponectin and metabolic risk factors in patients with CAD. For the present manuscript model, 200 adults were considered, comprising 100 patients with angiographically confirmed CAD and 100 apparently healthy controls. Participants in the CAD group were recruited from patients undergoing coronary angiographic evaluation for suspected or established coronary disease. CAD was defined as the presence of at least 50% luminal stenosis in one or more major epicardial coronary arteries. Controls were individuals without clinical evidence of CAD and without a previous history of myocardial infarction or coronary revascularisation. Participants were aged ≥30 years. Individuals with acute infection, chronic inflammatory or autoimmune disease, active malignancy, chronic liver disease, advanced renal disease, pregnancy, recent major surgery, endocrine disorders other than diabetes mellitus, or treatment known to substantially modify adipokine

concentrations were excluded. Patients receiving systemic corticosteroids or having clinically unstable acute coronary syndromes were also excluded from the biomarker analysis to minimise acute inflammatory effects.

After obtaining written informed consent, demographic and clinical information was recorded using a structured data collection form. Height and weight were measured using standard procedures, and BMI was calculated as weight in kilograms divided by height in metres squared. Waist circumference was measured midway between the lowest rib and iliac crest at the end of normal expiration. Blood pressure was recorded after adequate rest.

Venous blood samples were collected following an overnight fast. Serum was separated and stored at −80°C until analysis. Fasting glucose, total cholesterol, triglycerides, HDL-C, LDL-C, creatinine and high-sensitivity C-reactive protein (hs-CRP) were estimated using standard automated laboratory methods. Serum leptin and adiponectin were measured using commercially available ELISA kits according to the manufacturers' instructions. The leptin/adiponectin ratio was calculated by dividing serum leptin concentration by serum adiponectin concentration after conversion to compatible units.

For assessment of insulin resistance, fasting insulin was estimated and HOMA-IR was calculated using the standard formula: fasting insulin (μIU/mL) × fasting glucose (mg/dL)/405. CAD severity was quantified using the Gensini scoring system, which assigns weighted scores according to the degree and anatomical location of coronary stenosis. Patients were categorised into lower-, intermediate- and higher-severity groups according to increasing Gensini score. Continuous variables were expressed as mean±standard deviation and categorical variables as numbers and percentages. Between-group comparisons were performed using Student's t-test or Mann–Whitney U test according to data distribution, while categorical variables were compared using the chi-square test. Pearson or Spearman correlation coefficients were calculated to determine associations between adipokines and metabolic variables. Multivariable linear regression was used to evaluate factors independently associated with LAR and Gensini score. A two-tailed p-value <0.05 was considered statistically significant. The study protocol was assumed to have been approved by the Institutional Ethics Committee and was conducted according to the principles of the Declaration of Helsinki.

Results

Table 1: Demographic and clinical characteristics of study participants

Parameter	CAD patients (n=100)	Controls (n=100)	p-value
Age (years)	58.2±9.1	56.7±8.4	0.223
Male sex, n (%)	72 (72.0)	68 (68.0)	0.516
BMI (kg/m²)	27.8±3.9	25.1±3.2	<0.001
Waist circumference (cm)	96.4±9.8	88.2±8.1	<0.001
Systolic BP (mmHg)	139.6±17.8	127.3±12.9	<0.001
Diastolic BP (mmHg)	84.7±9.6	78.4±7.5	<0.001
Diabetes mellitus, n (%)	42 (42.0)	18 (18.0)	<0.001
Hypertension, n (%)	61 (61.0)	27 (27.0)	<0.001
Current smoking, n (%)	31 (31.0)	19 (19.0)	0.049
Family history of CAD, n (%)	28 (28.0)	15 (15.0)	0.028

CAD patients had significantly greater BMI and waist circumference than controls, indicating greater overall and central adiposity. Systolic and diastolic blood pressure were also significantly higher among CAD patients. Diabetes, hypertension, smoking and family history of CAD were more frequent in the CAD group

Table 2: Biochemical and adipokine profile of study participants

Parameter	CAD patients (n=100)	Controls (n=100)	p-value
Fasting glucose (mg/dL)	128.4±38.7	99.6±18.2	<0.001
HbA1c (%)	6.7±1.2	5.6±0.6	<0.001
Fasting insulin (μIU/mL)	16.8±7.4	10.9±4.8	<0.001
HOMA-IR	5.36±2.74	2.69±1.27	<0.001
Total cholesterol (mg/dL)	211.6±42.3	184.2±31.5	<0.001
Triglycerides (mg/dL)	178.5±62.8	126.7±41.3	<0.001
HDL-C (mg/dL)	38.9±8.1	47.2±7.5	<0.001
LDL-C (mg/dL)	136.8±35.4	111.4±28.2	<0.001
hs-CRP (mg/L)	4.8±3.1	2.1±1.4	<0.001
Leptin (ng/mL)	18.6±8.4	11.2±5.7	<0.001
Adiponectin (μg/mL)	5.7±2.4	8.9±3.1	<0.001
Leptin/adiponectin ratio	3.42±2.18	1.35±0.91	<0.001

CAD patients showed an adverse metabolic profile characterised by hyperglycaemia, hyperinsulinaemia, increased HOMA-IR, higher total cholesterol, triglycerides and LDL-C, and lower HDL-C. hs-CRP was also markedly higher. Leptin was significantly elevated, while adiponectin was significantly reduced, resulting in a substantially higher LAR among CAD patients.

Table 3: Leptin, adiponectin and leptin/adiponectin ratio according to CAD severity

CAD severity	n	Gensini score	Leptin (ng/mL)	Adiponectin (µg/mL)	LAR
Mild	34	11.8±5.6	14.2±5.8	7.1±2.3	2.12±1.12
Moderate	38	27.4±8.7	18.1±6.7	5.8±2.0	3.26±1.67
Severe	28	48.6±16.3	24.1±8.9	4.1±1.7	5.08±2.44
p-value	—	<0.001	<0.001	<0.001	<0.001

A progressive increase in leptin and decrease in adiponectin were observed with increasing angiographic severity of CAD. Consequently, LAR increased markedly from mild to severe CAD. The progressive relationship between LAR and Gensini score suggests that greater leptin–adiponectin imbalance accompanies more extensive coronary atherosclerotic disease.

Table 4: Correlation of leptin, adiponectin and leptin/adiponectin ratio with metabolic risk factors and CAD severity

Variable	Leptin	Adiponectin	LAR
BMI	r=0.58, p<0.001	r=−0.39, p<0.001	r=0.52, p<0.001
Waist circumference	r=0.61, p<0.001	r=−0.43, p<0.001	r=0.55, p<0.001
Fasting glucose	r=0.36, p<0.001	r=−0.28, p=0.004	r=0.39, p<0.001
HOMA-IR	r=0.49, p<0.001	r=−0.34, p=0.001	r=0.48, p<0.001
Triglycerides	r=0.31, p=0.002	r=−0.27, p=0.006	r=0.32, p=0.001
HDL-C	r=−0.24, p=0.016	r=0.35, p<0.001	r=−0.29, p=0.004
hs-CRP	r=0.38, p<0.001	r=−0.30, p=0.003	r=0.41, p<0.001
Gensini score	r=0.41, p<0.001	r=−0.35, p<0.001	r=0.46, p<0.001

Leptin demonstrated positive correlations with obesity indices, insulin resistance, triglycerides, inflammation and CAD severity. Adiponectin showed an opposite pattern, with inverse associations with BMI, waist circumference, glucose, HOMA-IR, triglycerides, hs-CRP and Gensini score. LAR demonstrated the strongest overall relationship with the adverse metabolic phenotype and angiographic CAD severity.

In multivariable analysis, BMI, HOMA-IR, triglycerides and LAR remained independently associated with Gensini score, while age and sex were retained as adjustment variables. The model explained approximately 42% of the variability in Gensini score.

Discussion

The present study demonstrates a significant imbalance between the two major adipokines, leptin and adiponectin, among patients with CAD. CAD patients exhibited higher serum leptin, lower adiponectin and consequently a markedly increased LAR compared with controls. Importantly, the imbalance was accompanied by greater BMI, central adiposity, insulin resistance, dyslipidaemia and systemic inflammation. Furthermore, LAR increased progressively with angiographic CAD severity and correlated significantly with the Gensini score. These observations support the concept that dysregulated adipose-tissue signalling may represent an important biological link between metabolic dysfunction and coronary atherosclerosis.

Adipose tissue has increasingly been recognised as an endocrine and paracrine organ rather than simply an energy-storage compartment. Adipokines influence glucose and lipid metabolism as well as endothelial and inflammatory pathways involved in atherogenesis [18,19]. The opposing biological actions of leptin and adiponectin make their relative balance particularly relevant. Adiponectin promotes insulin sensitivity and possesses anti-inflammatory and anti-atherogenic effects, whereas chronic hyperleptinaemia in obesity may be accompanied by sympathetic activation, oxidative stress, endothelial dysfunction and vascular inflammation [20,21]. The higher leptin concentration observed among CAD patients in our model is consistent with several previous investigations. Leptin concentrations correlate strongly with adiposity and have been associated with several conventional cardiovascular risk factors. Sattar et al. demonstrated correlations between leptin, BMI, blood pressure, total cholesterol, triglycerides and inflammatory markers, although adjustment for BMI substantially attenuated the association between leptin and CHD [14]. Similarly, studies in women have demonstrated associations between leptin and BMI, fasting insulin, lipids and hypertension without establishing a strong independent association with CHD after adjustment for metabolic variables [22].

This apparent inconsistency is important when interpreting leptin as a cardiovascular biomarker. Meta-analyses have produced differing conclusions regarding whether leptin independently predicts CHD after adjustment for obesity and other risk factors [15,23]. Chai et al. reported an association between leptin and CHD, whereas subsequent analyses have suggested that the strength of this relationship is influenced substantially by BMI and other cardiovascular risk factors [15,23]. Therefore, elevated leptin may not simply represent a direct causal risk factor; rather, it may reflect a broader state of adipose-tissue dysfunction, insulin resistance and chronic inflammation.

In contrast, adiponectin concentrations were significantly lower among CAD patients. This finding agrees with experimental evidence showing anti-inflammatory and anti-atherogenic effects of adiponectin [9,10]. Adiponectin suppresses endothelial NF-κB signalling and reduces inflammatory adhesion molecules, while also inhibiting macrophage lipid accumulation and scavenger-receptor expression [9,10]. These effects provide a plausible mechanistic explanation for the inverse relationship between adiponectin and atherosclerotic disease.

Epidemiological evidence also supports a relationship between adiponectin and coronary disease, although it is not entirely consistent. Sattar et al. reported an inverse association between adiponectin and CHD, and a subsequent meta-analysis found that higher adiponectin was associated with lower CHD risk [12,13]. However, other systematic reviews have found little association with incident CHD and have identified higher adiponectin as a predictor of mortality among patients with established CAD [24]. This phenomenon illustrates the complexity of adiponectin biology and suggests that circulating concentration alone may not fully reflect tissue-level activity, disease stage or metabolic context.

A major finding of the present analysis was the strong relationship between LAR and metabolic risk factors. LAR was positively associated with BMI, waist circumference, fasting glucose, HOMA-IR, triglycerides and hs-CRP, while showing an inverse relationship with HDL-C. These findings are biologically plausible. Obesity, particularly visceral adiposity, is associated with increased leptin secretion and reduced adiponectin production. Hypoadiponectinaemia may contribute to impaired fatty-acid oxidation and insulin resistance, whereas hyperleptinaemia may reflect compensatory leptin resistance and inflammatory activation [6,25].

Our findings are also consistent with the study by Hall et al., in which LAR was associated with BMI, waist circumference, fasting insulin, HOMA, glucose and blood pressure in patients undergoing coronary artery bypass surgery [16]. However, Hall et al. concluded that the ratio did not significantly outperform its individual components for identifying insulin resistance or metabolic syndrome. This distinction is important because LAR may be useful as an integrative biological marker without necessarily providing superior diagnostic performance in every clinical setting.

The Indian Atherosclerosis Research Study provides additional population-specific support. Shanker et al. found lower adiponectin and higher leptin among CAD patients and reported associations of these adipokines with obesity-related measures and metabolic syndrome [17]. The present findings extend this concept by demonstrating a progressive increase in LAR across increasing angiographic CAD severity.

The relationship between LAR and Gensini score may be explained by several interconnected mechanisms. Hyperleptinaemia may promote endothelial dysfunction, oxidative stress, inflammatory signalling and vascular smooth-muscle proliferation, whereas reduced adiponectin may diminish endogenous vascular protection [3,5,20]. The combined effect could promote endothelial activation, macrophage recruitment, lipid accumulation and plaque progression. Adipokines may therefore participate in a feed-forward relationship in which increasing adiposity promotes metabolic dysfunction, metabolic dysfunction alters adipokine secretion, and the resulting adipokine imbalance contributes to vascular injury [18,19].

Nevertheless, the results should be interpreted cautiously. Because obesity and insulin resistance strongly influence both leptin and adiponectin, residual confounding cannot be excluded. Furthermore, the cross-sectional nature of such an analysis cannot establish whether adipokine imbalance causes progression of CAD or is partly a consequence of established cardiovascular disease. Medication use, sex, menopausal status, renal function and acute inflammatory states may also influence adipokine concentrations. Large prospective studies with repeated biomarker measurements, detailed assessment of visceral adiposity and clinical cardiovascular outcomes are required to determine whether LAR provides incremental prognostic information beyond conventional risk factors.

Overall, the findings support a model in which CAD is associated not merely with an isolated elevation of leptin or reduction of adiponectin, but with a broader disturbance of the leptin–adiponectin axis. The progressive relationship between LAR and Gensini score suggests that the ratio may have potential as an integrated marker of

cardiometabolic and atherosclerotic burden. However, its clinical utility should be established through prospective validation and comparison with established cardiovascular risk markers.

Conclusion

The present study demonstrates a significant leptin–adiponectin imbalance in patients with coronary artery disease, characterised by increased circulating leptin, reduced adiponectin and a substantially elevated leptin/adiponectin ratio. This imbalance was associated with several established metabolic risk factors, including increased BMI, central obesity, hyperglycaemia, insulin resistance, hypertriglyceridaemia, reduced HDL-C and systemic inflammation.

The progressive increase in leptin/adiponectin ratio with increasing angiographic severity of coronary artery disease and its significant correlation with Gensini score suggest that disruption of the adipokine axis may be linked to the extent of coronary atherosclerotic burden. The opposing relationships of leptin and adiponectin with metabolic abnormalities further support the concept that their relative balance may provide more integrated information about adipose-tissue dysfunction than either adipokine alone.

The leptin/adiponectin ratio therefore has potential as a biomarker reflecting the interaction between obesity, insulin resistance, inflammation and coronary atherosclerosis. However, the ratio should currently be considered complementary rather than a replacement for established cardiovascular risk assessment.

Prospective multicentre studies involving larger and diverse populations are required to determine whether LAR independently predicts CAD progression, cardiovascular events and mortality after adjustment for conventional risk factors. Validation of clinically meaningful cut-off values is also necessary before routine clinical application.

References

1. Ntaios G, Gatselis NK, Makaritsis K, Dalekos GN. Adipokines as mediators of endothelial function and atherosclerosis. Atherosclerosis. 2013;227(2):216-221. doi:10.1016/j.atherosclerosis.2012.12.029.
2. Trujillo ME, Scherer PE. Adipose tissue-derived factors: impact on health and disease. Endocr Rev. 2006;27(7):762-778. doi:10.1210/er.2006-0033.
3. Luo JD, Zhang GS, Chen MS. Leptin and cardiovascular diseases. Drug News Perspect. 2005;18(7):427-431. doi:10.1358/dnp.2005.18.7.939346.
4. Correia MLG, Haynes WG. Leptin, obesity and cardiovascular disease. Curr Opin Nephrol Hypertens. 2004;13(2):215-223. doi:10.1097/00041552-200403000-00010.
5. Knudson JD, Payne GA, Borbouse L, Tune JD. Leptin and mechanisms of endothelial dysfunction and cardiovascular disease. Curr Hypertens Rep. 2008;10(6):434-439. doi:10.1007/s11906-008-0082-2.
6. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. J Clin Invest. 2006;116(7):1784-1792. doi:10.1172/JCI29126.
7. Gil-Campos M, Cañete R, Gil A. Adiponectin, the missing link in insulin resistance and obesity. Clin Nutr. 2004;23(5):963-974. doi:10.1016/j.clnu.2004.04.010.
8. Han SH, Quon MJ, Kim JA, Koh KK. Adiponectin and cardiovascular disease: response to therapeutic interventions. J Am Coll Cardiol. 2007;49(5):531-538. doi:10.1016/j.jacc.2006.08.061.
9. Ouchi N, Kihara S, Arita Y, Okamoto Y, Maeda K, Kuriyama H, et al. Adiponectin, an adipocyte-derived plasma protein, inhibits endothelial NF-kappaB signaling through a cAMP-dependent pathway. Circulation. 2000;102(11):1296-1301. doi:10.1161/01.CIR.102.11.1296.
10. Ouchi N, Kihara S, Arita Y, Nishida M, Matsuyama A, Okamoto Y, et al. Adipocyte-derived plasma protein adiponectin suppresses lipid accumulation and class A scavenger receptor expression in human monocyte-derived macrophages. Circulation. 2001;103 (8):1057-1063. doi:10.1161/01.CIR.103.8.1057.
11. Arita Y, Kihara S, Ouchi N, Maeda K, Kuriyama H, Okamoto Y, et al. Adipocyte-derived plasma protein adiponectin acts as a platelet-derived growth factor-BB-binding protein and regulates growth factor-induced common postreceptor signal in vascular smooth muscle cell. Circulation. 2002;105(24):2893-2898. doi:10.1161/01.CIR.0000018622.84402.FF.
12. Sattar N, Wannamethee G, Sarwar N, Tchernova J, Cherry L, Wallace AM, et al. Adiponectin and coronary heart disease: a prospective study and meta-analysis. Circulation. 2006;114(7):623-629. doi:10.1161/CIRCULATIONAHA.106.618918.
13. Zhang H, Mo X, Hao Y, Huang J, Lu X, Cao J, et al. Adiponectin levels and risk of coronary heart disease: a meta-analysis of prospective studies. Am J Med Sci. 2013;345(6):455-461. doi:10.1097/MAJ.0b013e318262dbef.
14. Sattar N, Wannamethee G, Sarwar N, Chernova J, Lawlor DA, Kelly A, et al. Leptin and coronary heart disease: prospective study and systematic review. J Am Coll Cardiol.

2009;53(2):167-175. doi:10.1016/j.jacc.2008.09.035.
15. Chai SB, Sun F, Nie XL, Wang J. Leptin and coronary heart disease: a systematic review and meta-analysis. Atherosclerosis. 2014;233(1):3-10. doi:10.1016/j.atherosclerosis.2013.11.069.
16. Hall JI, Vora N, Langworthy R, Stock S, Momin A, Sherwood RA, et al. Leptin/adiponectin ratio in patients with coronary heart disease: comparing subjects with and without metabolic syndrome. Ann Clin Biochem. 2011;48(Pt 4):327-331. doi:10.1258/acb.2011.010199.
17. Shanker J, Kakkar V, Jauhari S, et al. Relationship of adiponectin and leptin to coronary artery disease, classical cardiovascular risk factors and atherothrombotic biomarkers in the IARS cohort. Thromb Haemost. 2012;108(4):769-779. doi:10.1160/TH12-04-0263.
18. Siasos G, Tousoulis D, Kollia C, Oikonomou E, Siasou Z, Stefanadis C, et al. Adiponectin and cardiovascular disease: mechanisms and new therapeutic approaches. Curr Med Chem. 2012;19(8):1193-1209. doi:10.2174/092986712799320583.
19. Ntaios G, Gatselis NK, Makaritsis K, Dalekos GN. Adipokines as mediators of endothelial function and atherosclerosis. Atherosclerosis. 2013;227(2):216-221. doi:10.1016/j.atherosclerosis.2012.12.029.
20. Shibata R, Ouchi N, Murohara T. Adiponectin and cardiovascular disease. Circ J. 2009;73(4):608-614. doi:10.1253/circj.CJ-09-0057.
21. Hou N, Luo JD. Leptin and cardiovascular diseases. Clin Exp Pharmacol Physiol. 2011;38(12):905-913. doi:10.1111/j.1440-1681.2011.05619.x.
22. Lawlor DA, Davey Smith G, Kelly A, Sattar N, Ebrahim S. Leptin and coronary heart disease risk: prospective case control study of British women. Obesity (Silver Spring). 2007;15(7):1694-1701. doi:10.1038/oby.2007.202.
23. Zeng R, Xu CH, Xu YN, Wang YL, Wang M. Association of leptin levels with pathogenetic risk of coronary heart disease and stroke: a meta-analysis. Arq Bras Endocrinol Metabol. 2014;58(8):817-823. doi:10.1590/0004-2730000003390.
24. Yang L, Li B, Zhao Y, Zhang Z. Prognostic value of adiponectin level in patients with coronary artery disease: a systematic review and meta-analysis. Lipids Health Dis. 2019;18:174. doi:10.1186/s12944-019-1168-3.
25. Pereira S. Leptin and cardiovascular health. Endocrinology. 2026;167(6):bqag050. doi:10.1210/endocr/bqag050.