International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 16s,2025
https://theaspd.com/index.php

Oxidative Stress Markers And Antioxidant Vitamins In Women After Menopause With Chronic Heart Disease

Asen Hussein Salih[1], **Nihad Nejres Hilal**[2]
[1,2]Department of Clinical Biochemistry, College of Medicine, University of Tikrit, Iraq,
[1]AH240009me@st.tu.edu.iq, [2]drnehad76@tu.edu.iq

Abstract:
Background: *Due in part to hormonal changes raising oxidative stress, postmenopausal women are more likely to develop chronic heart disease (CHD). Developing preventative plans depends on a knowledge of the function of oxidative stress indicators and antioxidant vitamins.*
aim: *This study is to assess the degrees of malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx), and vitamins A and C in postmenopausal women with chronic heart disease.*
Methods: *90 postmenopausal women (60 patients with CHD and 30 healthy controls) aged 50–60 years were subject to a case-control study. ELISA was used to examine blood samples for MDA, SOD, GPx, vitamin A and C concentrations. Duncan's multiple range test and ANOVA were used in statistical analysis.*
Results*: MDA levels were significantly higher in CHD patients (5.67 ± 0.61 µmol/L) compared to controls (2.66 ± 0.34 µmol/L, $p < 0.05$). Antioxidant enzymes GSH-Px and SOD were significantly lower in patients (0.81 ± 0.44 and 21.92 ± 7.07 U/mL respectively) than controls (2.54 ± 1.03 and 36.82 ± 9.68 U/mL, $p < 0.05$). In addition, significantly decreased in the levels of vitamin A and C were observed.* ***Conclusion*** *The study shows that postmenopausal women with CHD with higher oxidative stress and lower antioxidant defenses have a significant relationship. These results confirm the importance of frequent monitoring of oxidative stress indicators in this group and of antioxidant-based preventative policies.*
Keywords*: oxidative stress, postmenopausal women, chronic heart disease, antioxidant enzymes, vitamin A, vitamin C.*

INTRODUCTION

However, a major source of illness and death globally are cardiovascular diseases (CVDs). Hormonal changes, metabolic abnormalities, and oxidative stress all help to greatly increase the risk of chronic heart disease (CHD) in women following menopause. [(1)] Postmenopausal women have increased oxidative stress, dyslipidemia, and inflammation all of which aggravate cardiovascular disease. Because of the drop-in estrogen levels after menopause, postmenopausal women are more at risk for coronary heart disease (CHD). [(2)]Cardiovascular problems are common in both men and women; nevertheless, there are differences between the sexes in clinical symptoms, etiology, and treatment responses. [(3)]Cardiovascular diseases (CVD) in women are often unrecognized, and women typically have a limited understanding of the related dangers. This may lead to delayed diagnosis and insufficient symptom identification. Women develop heart diseases later than men due to the protective benefits conferred during their reproductive years. The risk escalates upon the onset of menopause. Estrogen confers a preventive effect against cardiovascular disease in women. [(4)] Consequently, the likelihood of cardiovascular disease escalates post-menopause in the majority of instances. The study highlights the significance of menopause as a period associated with heightened cardiovascular disease risk. It underscores the need of monitoring the health of women during middle age, a pivotal period for the implementation of early intervention techniques to mitigate the risk of cardiovascular disease (CVD). [(5)] Menopause has been associated with alterations characteristic of cardiovascular aging. Cardiac disease has complex impacts, including endothelial function, coronary artery physiology, and metabolic dysfunction, which result in structural alterations in coronary anatomy. The postmenopausal phase is characterized by a reduction in estrogen levels, which provides protective benefits to the cardiovascular system. Elevated oxidative stress and inflammation. Dyslipidemia (abnormal lipid metabolism). Endothelial dysfunction resulting in hypertension. Higher susceptibility to metabolic syndrome and obesity Postmenopausal women therefore show more sensitivity to oxidative damage, lipid abnormalities, and heart diseases. [(6)]Different markers are used to evaluate body oxidative stress and antioxidant content: A biomarker of lipid peroxidation and oxidative damage, malondialdehyde (MDA)

Glutathione Peroxidase (GPx) is an enzyme antioxidant used to reduce damaging peroxides. Superoxide Dismutase (SOD): An enzyme used to neutralize superoxide radicals hence preventing oxidative damage. [7]Moreover, vitamins such as vitamin A and vitamin C are crucial in reducing oxidative stress and preventing heart diseases. Understanding these changes in these markers can help one evaluate the course of disease and possible treatments. [8] This study is to evaluate oxidative stress biomarkers, blood pressure, and body mass index (BMI) in postmenopausal women with chronic heart disease (CHD) while analyzing other biochemical parameters. The main objectives of this study are to understand how oxidative stress affects the beginning and development of coronary heart disease as well as its correlation with biochemical risk factors in postmenopausal women.Using biomarkers like malondialdehyde (MDA), glutathione peroxidase (GPx), and superoxide dismutase (SOD), postmenopausal women with coronary heart disease (CHD) have oxidative stress levels evaluated.

Objective of this study

- To investigate relationships between oxidative stress and blood pressure fluctuations in postmenopausal women with coronary heart disease.
- To discover whether oxidative stress and BMI correlate in postmenopausal women with coronary heart disease (CHD).
- To investigate how vitamins C and A could lower oxidative stress in postmenopausal individuals with coronary heart disease (CHD).
- To evaluate, between postmenopausal women with CHD and age-matched healthy controls, the biochemical and oxidative stress markers.

MATERIALS AND METHODS

Design of the Study and Participants:

This study was conducted in Kirkuk Governorate from late December 2024 to the end of April 2025 and involved 90 participants aged 50 to 60 years, including 60 patients with chronic heart disease, who were under the supervision of a specialist physician for treatment and clinical observation. A control group of 30 individuals without chronic heart disease was selected. Laboratory analyses were performed on both groups to assess the concentrations of malondialdehyde, glutathione peroxidase, superoxide dismutase, vitamin C, and vitamin A using
ELISA.

Exclusion Criteria

- Premenopausal women, often known as women under.
- Patients experiencing stroke, acute or recent (within past six months) myocardial infarction, or another acute cardiovascular event.
- These acute events can profoundly change lipid profile values and oxidative stress indicators.
- Women who are currently taking lipid-lowering drugs (such as statins) or antioxidant supplements (vitamins A, C, E, selenium, etc.)
- Individuals having chronic inflammatory or autoimmune disorders (such as lupus or rheumatoid arthritis).
- Women with thyroid problems.
- Alcohol drinkers or smokers (present or past six months).
- Individuals who have had cancer or are currently receiving radiation or chemotherapy.

Data Collection:

The primary data source was obtained through detailed interviews conducted by the researcher with individuals suffering from chronic heart disease, utilizing a developed questionnaire. The questionnaire included date of birth, gender, medical history of problems, particularly metabolic diseases, type 2 diabetes mellitus, hypertension, and the use of certain drugs. The height, weight, and systolic and diastolic blood pressure of each individual were measured and documented.

Samples collection and handling:

5 ml of venous blood were drawn from a suitable vein in each patient and control participant. Fasting samples were obtained between 8 and 9 A.M. The blood was let to coagulate for 20 minutes in gel tubes at room temperature (25

°C). The serum was extracted using centrifugation at 3000 rpm for 10 minutes. 1 ml of serum was partitioned into two equal aliquots and put into 0.5 ml Eppendorf tubes. The residual serum was transferred to a distinct sterile 0.5 ml Eppendorf tube for preservation at -20°C in a deep freezer until analysis.

Anthropometric Measurements

Anthropometric data (height, weight, BMI), blood pressure, and medical history were recorded. Fasting blood samples were collected and processed for biochemical analyses. Body mass index (BMI) was calculated based on height and weight and was computed as weight divided by height squared (kg/m^2). BMI provides an easy numeric assessment of an individual's body composition. It is utilized to categorize weight classifications and identify health issues related to weight. Furthermore, it is extensively employed in shaping public health policies. The BMI scale classifies individuals into one of five distinct categories: underweight, normal, overweight, obese, and severely obese.[9]

Assessment of Malondialdehyde, glutathione peroxidase, superoxide dismutase, vitamin A and vitamin C

All biomarkers were analyzed using a consistent method due to the use of the same company.

Principle This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-treatment with Human GSH-Px antibody. The GSH-Px in the sample is introduced and binds to the antibodies immobilized on the wells. Thereafter, biotinylated human GSH-Px antibody is added and associates with GSH-Px in the sample. Thereafter, Streptavidin-HRP is added to bind with the Biotinylated GSH-Px antibody. After incubation, unbound Streptavidin-HRP is eliminated during the washing process. The substrate solution is then introduced, leading to color development that correlates with the amount of Human GSH-Px present. The reaction concludes with the addition of an acidic stop solution, and absorbance is measured at 450 nm.

Reagent Preparation

All reagents have been adjusted to room temperature before utilization.
The standard Reconstitute 120µl of the standard (640U/ml) with 120µl of diluent to get a 320U/ml standard stock solution. Allow the standard to equilibrate for 15 minutes with gentle agitation prior to performing dilutions. Prepare duplicate standard concentrations by serial dilution of the standard stock solution (320 U/ml) in a 1:2 ratio with the standard diluent to get solutions of 160 U/ml, 80 U/ml, 40 U/ml, and 20 U/ml. The standard diluent serves as the zero standard (0 U/ml). Any surplus solution should be refrigerated at -20°C and utilized within one month.

Statistical Analysis:

Data were analyzed using Minitab. Results are expressed as mean ± SD. ANOVA and Duncan's test were used to assess significance ($p < 0.05$).

RESULTS

Age Distribution: The findings in this study show that most CHD occurrences were in the 50–55 age group, including 51.7% of patients, whereas 48.3% were in the 55–60 age range. see table 4.1

Table 4.1. Relation of number of cardiovascular diseases with age

Age group (years)	**(n)**	%
50–55	31	51.7%
55–60	29	48.3%
Total	60	100%

BMI: The table 4.2 shows a strong significant correlation between BMI and chronic heart disease. the women with CHD in this research, only 11.67% were of normal weight; most fell into the overweight (20%) and obese categories—Class I (33.33%) and Class II (35%). These findings show the important part extra body weight plays in cardiovascular risk in postmenopausal women.

table 4.2: Correlation between BMI and chronic heart disease.

MI Group	**N**	**Percentage** (%)

Normal weight	7	11.67%
Overweight	12	20.00%
Obesity Class I	20	33.33%
Obesity Class II	21	35.00%
Total	**60**	**100%**

Oxidative Stress Markers:

Table 4.3 Comparison of Malondialdehyde (MDA), Glutathione peroxidase (GSH-Px), Superoxide dismutase (SOD) Levels Between Control and Patient Groups

Parameters	**Groups**		
	Control (n 30)	**Patients (n 60)**	**P. V**
	Mean ±S.D	**Mean ±S.D**	
MDA nmol/ml	2.66±0.34	5.67±0.61	<0.05
GSH-PX U/ml	2.54±1.027	0.81±0.44	<0.05
SOD U/ml	36.82±9.68	21.92±7.07	<0.05

As showed in the table 4.3 the malondialdehyde (MDA) identified as an indicator of lipid peroxidation and oxidative stress (10), was markedly elevated in postmenopausal women suffering from chronic heart disease in comparison to healthy controls. The patient group exhibited an average MDA level of 5.67 ± 0.61µmol/L, in contrast to the control group, which demonstrated a significantly lower average of 2.67 ± 0.35 µmol/L ($p < 0.05$).

As shown in the table 4.4 GSH-Px activity was markedly reduced in the patient group (Patients: 0.81 ± 0.44 U/mL, Controls: 2.54 ± 1.03 U/mL; $p < 0.05$). As shown in the table SOD was markedly decreased in patient group versus to controls (Patients: 21.92 ± 7.07 U/mL, Controls: 36.82 ± 9.68 U/mL; $p < 0.05$).

Antioxidant Vitamins:

Table 4.6 Comparison of Vitamin C and Vitamin A Levels Between Control and Patient Groups

Parameters	**Groups**		
	Control (n 30)	**Patients (n 60)**	**P. V**
	Mean ±S.D	**Mean ±S.D**	
Vit-c ng/ml	326.50±37.38	303.31±20.55	<0.05
vit A ng/ml	287.64±38.78	138.96±36.48	<0.05

In the present investigation, postmenopausal women with chronic heart disease (CHD) had notably lower blood Vitamin C levels than the healthy control group (303.31 ± 20.55 ng/ml vs. 326.50 ± 37.38 ng/ml, $P < 0.05$). This result is compatible with the theory that oxidative stress is higher in CHD and that corresponding depletion of antioxidant defenses including vitamin C results.Patients demonstrated vitamin A levels of 138.96 ± 36.48 ng/ml, significantly lower than the 287.64 ± 38.78 ng/ml recorded in the control group ($p < 0.05$). This decline is associated with elevated oxidative stress, shown by increased MDA levels, and a reduction in enzymatic antioxidants such as SOD and GSH-Px.

DISCUSSION

The results demonstrate a significant alteration in oxidative stress biomarkers and antioxidant levels among postmenopausal women with chronic heart disease. The increased levels of MDA observed in patients highlight enhanced lipid peroxidation, a hallmark of oxidative stress. Malondialdehyde (MDA) is a byproduct of polyunsaturated fatty acid peroxidation, and elevated levels suggest oxidative membrane damage, endothelial dysfunction, and potential atherogenic effects.[10]

Postmenopausal estrogen decline is strongly associated with increased MDA, reflecting diminished cellular defense against free radicals. These findings are consistent with previous studies showing that postmenopausal women have higher oxidative damage than premenopausal counterparts, especially in those with CHD. [11]The results of this research revealed that whereas 48.3% of CHD cases occurred in the 55–60 age range (Table 4.1), most instances—51.7%—were found in the 50–55 age group. These results imply that the risk of cardiovascular disorders starts to increase fast soon after menopause and stays high in the next yearsThe table 4.2 shows a strong significant correlation between BMI and chronic heart disease. Of the women with CHD in our research, only 11.67% were of normal weight; most fell into the overweight (20%) and obese categories—Class I (33.33%) and Class II (35%). These findings show the important part extra body weight plays in cardiovascular risk in postmenopausal women.
Elevated MDA indicates increased oxidative stress and cellular membrane damage, commonly observed in cardiovascular disorders, and is frequently exacerbated in postmenopausal women due to reduced estrogen levels. [12]Estrogen is recognized for its role in regulating antioxidant enzyme production and preventing lipid peroxidation; its decrease may lead to an increase in MDA levels.[13]The significant decrease in GSH-Px activity in the patient group points to a compromised antioxidant defense mechanism, most likely aggravating the increased oxidative damage seen by increasing MDA levels. With these criteria directly proportional to the length of menopause, comparable results have been shown in another research indicating that GSH-Px activity is significantly reduced in early and late menopausal women, suggesting that in postmenopausal women, antioxidant defense diminishes and oxidative stress increases relative to premenopausal women. [14]Lack of estrogen has also been shown to downregulate the synthesis of antioxidant enzymes, hence influencing this drop. Furthermore, underlining its importance in cardiovascular health, reduced GSH-Px activity has been associated to endothelial dysfunction and increased sensitivity to atherosclerotic plaque formation. Another study was disagree with our study in which mentioned that there was no significant difference ($p>0.05$) in the level of Glutathione peroxidase between the postmenopausal group and the fertile group.[15]Another essential antioxidant enzyme, superoxide dismutase (SOD), was also much lowered in patients. By catalyzing the dismutation of superoxide radicals into hydrogen peroxide and molecular oxygen, SOD helps to avoid oxidative damage. Reduced SOD activity aggravates oxidative imbalance and might speed up vascular damage. These results reflect earlier studies which underlined low SOD levels in postmenopausal and obese women. [16]A major indicator of disease risk and development, results show that reduced SOD activity in postmenopausal women with chronic heart disease may both predict and aggravate oxidative stress. Therapeutically, treatments meant to improve antioxidant defense—by dietary changes, lifestyle modifications, or pharmacological approaches—may benefit this sensitive group. [17] Furthermore, estrogen, known for its antioxidant properties, has demonstrated capacity to control the synthesis and activity of antioxidant enzymes like superoxide dismutase (SOD). The postmenopausal drop in estrogen might be rather important for the low SOD levels seen in this work. [18]
A water-soluble antioxidant, vitamin C exhibited dramatically reduced blood levels in individuals with CHD.
The significantly decreased levels of vitamin C shown in the patient group might reflect a higher oxidative load brought on by the illness process as well as the physiological changes linked with menopause. Estrogen has recognized antioxidant effects; its drop at menopause reduces antioxidant defense, hence increasing postmenopausal women's susceptibility to oxidative damage. This could then lead to further intake and depletion of natural antioxidants such as vitamin C.These findings complement multiple earlier research highlighted the negative link between antioxidant levels and cardiovascular risk by reporting notably lower Vitamin C levels in postmenopausal women with ischemic heart disease as compared to controls.[19] Another study also noted a notable drop in blood Vitamin C levels among Iraqi patients with coronary artery disease, therefore confirming the theory that oxidative depletion is a typical CHD hallmark.[20]A reduction in vitamin C levels has been shown to correlate with carotid endothelial damage in individuals with atherosclerosis, underlining the beneficial function of vitamin C and antioxidants on nitric oxide levels. [21]Furthermore, influencing lower Vitamin C levels include dietary elements, lifestyle decisions, and comorbidities like diabetes mellitus and hypertension (all of which were prevalent in your patient group). These disorders are linked to more oxidative stress and inflammation, which can raise antioxidant metabolic turnover.It is noteworthy that although the patient group's vitamin C levels were much down, they stayed within a medically normal range. Still, this relative lack might have clinical consequences, especially in those with heavy oxidative stress load. Thus, although clinical trials are required to confirm the effectiveness and safety of such

therapies, antioxidant supplementation, dietary improvement, and lifestyle adjustment may offer a supporting therapeutic strategy in postmenopausal women with CHD.Another study indicates that plasma vitamin C levels exceeding 45 μmol/L substantially diminish the risk of cardiovascular disease (CVD). Individuals with values ranging from 45.4 to 79.5 μmol/L had a 25% reduced risk of cardiovascular disease compared to those with amounts below 23 μmol/L. Moreover, participants with a mean plasma concentration of 77 μmol/L had a 33% reduced risk of coronary heart disease (CHD) compared to those with a value of 27 μmol/L. Every 20 μmol/L elevation in plasma vitamin C concentrations correlates with a 20% decrease in all-cause mortality and a 30% decrease in coronary heart disease mortality. [22]Patients' substantial reduction in vitamin A levels points to a possible relationship between vitamin A insufficiency and chronic heart disease. Significantly in preserving cardiovascular health, vitamin A is known to influence immunological function, cellular differentiation, and gene expression as well as other aspects. Deficiency of vitamin A might aggravate cardiovascular diseases by causing oxidative stress and inflammation.The significant decrease in blood vitamin A levels found in the CHD patient group in this study matches with other studies focusing on the interaction among chronic inflammation, oxidative stress, and micronutrient depletion in cardiovascular disorders. For instance, a study evaluated blood concentrations of antioxidant vitamins, particularly vitamin A, in males with coronary heart disease (CHD) relative to healthy controls. The findings demonstrated that males with coronary heart disease exhibited markedly reduced blood vitamin A levels compared to those without the condition. The authors proposed that diminished blood antioxidant vitamin levels may constitute significant risk factors for coronary heart disease (CHD).[23]Decreased serum vitamin A levels in CHD patients may indicate heightened consumption to minimize oxidative damage or inadequate food intake or absorption. The loss of antioxidants corresponds with increased MDA levels, a hallmark of lipid peroxidation, indicating a disturbed redox state in the patient cohort. [24]Here the ELISA technique used allowed for accurate, practical, and quick evaluation of vitamin A in clinical samples. The noted significant drop in vitamin A levels across patients might point to a hitherto understated component of oxidative stress and micronutrient depletion in this population. These results need more research and emphasize the possibilities of ELISA-based assays as reasonable substitutes for assessing antioxidant state in studies on aging and cardiovascular diseases.This work presents a fresh methodological addition by direct assessment of vitamin A in postmenopausal women with chronic heart disease using a competitive ELISA methodology. The present work can be a basic reference for next investigations given the scarcity of studies applying this approach in comparable demographics. Furthermore, the simplicity and availability of ELISA tests point to their wider use in clinical practice for the monitoring of antioxidant vitamin levels, particularly in environments where more sophisticated chromatographic techniques are not accessible. This work therefore offers not only significant biochemical insights but also shows the viability of using ELISA for evaluation of oxidative stress and nutrition.While the majority of current research on vitamin A and cardiovascular health employs HPLC instead of ELISA for serum measurement, many findings support the association between low vitamin A levels and cardiovascular risk.Collectively, the data underline the combined impact of reduced enzymatic and non-enzymatic antioxidant capacity in the development and progression of CHD in postmenopausal women. These findings stress the importance of early detection, dietary interventions, and potentially antioxidant supplementation as part of comprehensive cardiovascular management.

CONCLUSION

Postmenopausal women with CHD exhibit significantly elevated oxidative stress and impaired antioxidant defenses, coupled with unfavorable lipid profiles and increased BMI. Regular assessment of oxidative markers and early dietary or therapeutic interventions could mitigate cardiovascular risk in this vulnerable population. Incorporating antioxidant strategies in clinical care may offer preventive value in managing CHD progression.Scientific evidence demonstrates the efficacy of Traditional Chinese Medicine in treating cardiovascular diseases while also enhancing overall bodily strength. Due to the association between cardiovascular disease (CVD) and the overproduction of reactive oxygen species (ROS) that leads to mitochondrial dysfunction, numerous medicinal agents such as aspirin, leptin, and melatonin have exhibited cardioprotective properties against oxidative damage via mitochondrial-related mechanisms.[25]

In postmenopausal females' Chronic low-grade inflammation and oxidative stress are prevalent in postmenopausal women, and regular physical exercise and/or the intake of phytoestrogens may be crucial methods for reducing or preventing these alterations in women. Resistance training in conjunction with aerobic exercise has demonstrated efficacy in diminishing reactive oxygen species and enhancing antioxidant enzymes, in addition to lowering total cholesterol levels in obese women more effectively than aerobic exercise alone. Regular exercise significantly enhances anti-inflammatory cytokine levels and diminishes the secretion of pro-inflammatory chemicals by adipocytes through the reduction of visceral and total fat.[26]This research offers strong evidence that postmenopausal women with chronic heart disease (CHD) have much different biochemical and oxidative stress levels than healthy controls. The results show higher levels of malondialdehyde (MDA), indicating enhanced lipid peroxidation, and lower activity of antioxidant enzymes including glutathione peroxidase (GPx) and superoxide dismutase (SOD), which suggests an impaired antioxidant defense mechanism. Furthermore, supporting the existence of increased oxidative stress in CHD patients are lower levels of antioxidant vitamins (vitamin C and vitamin A).Furthermore, pointing to the combined influence of metabolic and genetic elements in the etiology of cardiovascular illnesses in this vulnerable population include increased incidence of obesity, hypertension, diabetes, anda favorable family history among CHD patients.
Collectively, the results of this study show the crucial interaction among oxidative stress, dyslipidemia, antioxidant level, and conventional cardiovascular risk factors in the development of CHD in postmenopausal women. These findings highlight the need of early identification, preventative plans, and focused antioxidant and lipid-lowering treatments to reduce cardiovascular risk and thereby enhance health outcomes in this group.This work presents a fresh methodological addition by direct assessment of vitamin A in postmenopausal women with chronic heart disease using a competitive ELISA methodology. The present work can be a basic reference for next investigations given the scarcity of studies applying this approach in comparable demographics. Furthermore, the simplicity and availability of ELISA tests point to their wider use in clinical practice for the monitoring of antioxidant vitamin levels, particularly in environments where more sophisticated chromatographic techniques are not accessible. This work therefore offers not only significant biochemical insights but also shows the viability of using ELISA for evaluation of oxidative stress and nutrition.

REFERENCES

1.Sanchis-Gomar F, Perez-Quilis C, Leischik R, Lucia A. Epidemiology of coronary heart disease and acute coronary syndrome. Annals of translational medicine. 2016;4(13).
2.Paynter NP, Balasubramanian R, Giulianini F, Wang DD, Tinker LF, Gopal S, et al. Metabolic predictors of incident coronary heart disease in women. Circulation. 2018;137(8):841-53.
3.Hilleary RS, Jabusch SM, Zheng B, Jiroutek MR, Carter CA. Gender disparities in patient education provided during patient visits with a diagnosis of coronary heart disease. Women's Health. 2019;15:1745506519845591.
4.Mansanguan C, Maneerat Y. PPBP gene as a biomarker for coronary heart disease risk in postmenopausal Thai women. PeerJ. 2022;10:e13615.
5.Bai M-F, Wang X. Risk factors associated with coronary heart disease in women: a systematic review. Herz. 2020;45(Suppl 1):52-7.
6.Hossain R, Kongchain A, Chatatikun M, Klangbud WK, Yupanqui CT, Majima HJ, et al. Green Tea Pressurized Hot Water Extract in Atherosclerosis: A Multi-Approach Study on Cellular, Animal, and Molecular Mechanisms. Antioxidants. 2025;14(4):404.
7.Maas AH, Appelman YE. Gender differences in coronary heart disease. Netherlands Heart Journal. 2010;18:598-603.
8.Pathak LA, Shirodkar S, Ruparelia R, Rajebahadur J. Coronary artery disease in women. Indian heart journal. 2017;69(4):532-8.
9.Mohajan D, Mohajan HK. Body mass index (BMI) is a popular anthropometric tool to measure obesity among adults. Journal of Innovations in Medical Research. 2023;2(4):25-33
10. Yekti R, Bukhari A, Jafar N, Thaha AR. Measurement of malondialdehyde (MDA) as a good indicator of lipid peroxidation. International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR). 2018;6(4):1-3.
11. Cakir T, Goktas B, Mutlu MF, Mutlu I, Bilgihan A, Erdem M, et al. Advanced oxidation protein products and malondialdehyde—the new biological markers of oxidative stress—are elevated in postmenopausal women. Ginekologia polska. 2016;87(5):321-5.
12. Lankin VZ, Tikhaze AK, Melkumyants AM. Malondialdehyde as an important key factor of molecular mechanisms of vascular wall damage under heart diseases development. International Journal of Molecular Sciences. 2022;24(1):128.
13. Leanza G, Conte C, Cannata F, Isgrò C, Piccoli A, Strollo R, et al. Oxidative stress in postmenopausal women with or without obesity. Cells. 2023;12(8):1137.
14. Tambse V, Banik M, Tekade M. "Study of antioxidant and lipid peroxidation status among postmenopausal women". Journal of Cardiovascular Disease Research. 2023;14(12):1382-7.

15. Ibrahim AJ. Evaluation Of Oxidative Stress in Postmenopausal Iraqi Women. Al-Kunooze Scientific Journal. 2024;8(1):95-108.
16. Uppoor RB, Rajesh A, Srinivasan MP, Unnikrishnan B, Holla R. Oxidative stress in obese postmenopausal women: an additive burden for atherosclerosis. Journal of Clinical and Diagnostic Research: JCDR. 2015;9(12):OC03.
17. Bourgonje MF, Bourgonje AR, Abdulle AE, Kieneker LM, La Bastide-Van Gemert S, Gansevoort RT, et al. Systemic oxidative stress, aging and the risk of cardiovascular events in the general female population. Frontiers in Cardiovascular Medicine. 2021;8:630543.
18. Strehlow K, Rotter S, Wassmann S, Adam O, Grohé C, Laufs K, et al. Modulation of antioxidant enzyme expression and function by estrogen. Circulation research. 2003;93(2):170-7.
19. Karam M, Itani MM, Faraj M, Jaffa AA, Jelwan J, Aldeen KS, et al. Oxidative Stress and Cardiovascular Disease: Implication of Gender and Age. Oxidative Stress in Cardiovascular-Metabolic Diseases: Springer; 2024. p. 1-23.
20. Al-Fartosy AJM, Awad NA, Alsalimi SA. Clinical markers and some trace elements in patients with type-2 diabetic nephropathy: Impact of insulin resistance. The Journal of Medical Investigation. 2021;68(1.2):76-84.
21. May JM, Harrison FE. Role of vitamin C in the function of the vascular endothelium. Antioxidants & redox signaling. 2013;19(17):2068-83.
22. Ara Z, Waliullah S, Rastogi D, Al-Otaibi ML, Pant S, Nawati M, et al. Potential Health Benefits of Vitamin C: An Update. Recent Studies on Vitaminology-Insights, Applications and Uses: IntechOpen; 2025.
23. Yıldıran H, Mercanlıgil SM, Besler HT, Tokgözoğlu L, Kepez A. Serum antioxidant vitamin levels in patients with coronary heart disease. Int J Vitam Nutr Res. 2011;81(4):211-7.
24. Olsen T, Blomhoff R. Retinol, RetinoicAcid, andRetinol-BindingProtein 4areDifferentiallyAssociatedwithCardiovascular Disease, Type2Diabetes, andObesity: An OverviewofHumanStudies. 2020.
25. Gao J, Hou T. Cardiovascular disease treatment using traditional Chinese medicine: Mitochondria as the Achilles' heel. Biomedicine & Pharmacotherapy. 2023;164:114999.
26. Giolo JS, Costa JG, da Cunha-Junior JP, Pajuaba ACA, Taketomi EA, de Souza AV, et al. The effects of isoflavone supplementation plus combined exercise on lipid levels, and inflammatory and oxidative stress markers in postmenopausal women. Nutrients. 2018;10(4):424.