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Sex-Specific Effects of Resistance Training Intensity on High-Density Lipoprotein Cholesterol in Aging Adults

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ABSTRACT

Purpose of the study: This study aims to examine the effects of light and moderate resistance training intensities on high-density lipoprotein (HDL) cholesterol levels in aging adults, compare sex differences in HDL cholesterol responses, and analyze whether sex modifies the relationship between training intensity and lipid adaptation.

Methodology: This study employed a quasi-experimental 2×2 factorial design involving adults aged 45–75 years from the Senam Sehat Indonesia (SSI) PWRI group in Karangpandan, Indonesia. Participants underwent eight weeks of light- or moderate-intensity resistance training. HDL cholesterol was assessed through blood analysis, and changes were evaluated using two-way ANOVA following normality and homogeneity assumption testing.

Main Findings: Both light and moderate resistance training were associated with increased HDL cholesterol levels, with no significant difference between training intensities ($p = 0.769$). Female participants showed a higher average HDL improvement than male participants at the descriptive level; however, this sex difference did not reach statistical significance ($p = 0.187$), despite a medium effect size ($\eta^2 = 0.06$), indicating that the study was likely underpowered to detect it. No significant interaction was found between training intensity and sex ($p = 0.482$).

Novelty/Originality of this study: Rather than assuming a uniform physiological response, this study explored whether biological sex influences lipid adaptation following resistance training. Although the sex-related effect was not statistically significant, the observed medium effect size highlights potential biological differences in HDL responses. These findings emphasize the importance of adequately powered sex-stratified trials and provide effect-size estimates to support future research design.

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1. INTRODUCTION

Cardiovascular disease (CVD) represents the single largest contributor to premature mortality globally, accounting for approximately 30% of all deaths in 2005 and with the World Health Organization projecting a 17% further increase in CVD-related mortality between 2006 and 2015 [1], [2]. In Indonesia specifically, data from the National Household Health Survey identified coronary heart disease as the cause of 26.4% of all deaths, with projections estimating 53.5 deaths per 100,000 population attributed to this cause [3], [4]. A convergence of risk

factors drives this epidemic, with dyslipidaemia characterised by elevated low-density lipoprotein (LDL), reduced high-density lipoprotein (HDL), and elevated triglycerides playing a mechanistically central role in the initiation and progression of atherosclerosis [5]-[7].

High-density lipoprotein (HDL) cholesterol, often termed 'good cholesterol,' serves as the principal mediator of reverse cholesterol transport the physiological process whereby excess cholesterol is extracted from peripheral tissues and arterial walls and returned to the liver for excretion or recycling [8]-[11]. For every 1 mg/dL increase in HDL cholesterol, the risk of cardiovascular events decreases by approximately 2–3% in men and 3–4% in women [12], [13]. Despite this well-established cardioprotective role, a substantial proportion of the Indonesian adult population particularly the ageing segment presents with suboptimal HDL levels, creating a significant public health burden that demands accessible and scalable intervention strategies [14]-[17].

Physical exercise, and resistance training (RT) in particular, has received considerable attention as a non-pharmacological strategy for improving the lipid profile in older adults [18]-[21]. The physiological rationale for RT's lipid-modifying effects is well-grounded in metabolic biology: progressive overload during resistance exercise activates lipoprotein lipase (LPL) and hepatic lipase (HL) in skeletal muscle and adipose tissue, enzymes that play key roles in lipoprotein remodelling and HDL particle maturation [22]-[25]. Additionally, RT induces a shift in substrate utilisation toward fatty acid oxidation particularly during the recovery period following each session which stimulates the reverse cholesterol transport pathway and promotes the formation of apolipoprotein A-I-containing HDL particles [26], [27].

A critical yet insufficiently studied dimension in this area concerns the relationship between training intensity and lipid outcomes, particularly in middle-aged and older adult populations [28]-[30]. Current guidelines from major exercise medicine bodies recommend resistance training for cardiovascular risk reduction, but provide limited specificity regarding optimal intensity parameters for HDL elevation a gap that is particularly consequential for ageing populations whose physiological limitations may preclude high-intensity protocols [31], [32]. Equally important is the question of whether biological sex modulates the HDL response to resistance training [33]. Given the well-documented sex differences in baseline HDL metabolism including the influence of oestrogen on hepatic lipase activity and apolipoprotein A-I production it is plausible that male and female participants may show markedly different HDL responses to the same training stimulus [31]-[34].

Despite the clinical relevance of these questions, studies systematically examining both the intensity-specific and sex-stratified HDL responses to resistance training in older Indonesian adults remain scarce [38]. Most published research has either focused on aerobic exercise, involved younger populations, or failed to adequately stratify analyses by sex thereby potentially obscuring important differential effects. The SSI (Senam Sehat Indonesia) PWRI group in Karangpandan, Karanganyar, represents an ecologically relevant context in which to study these questions: an organised community exercise programme attended by middle-aged and older adults of both sexes, providing a naturalistic setting for a factorial experimental investigation.

A review of the existing literature reveals divergent findings and methodological gaps that directly motivate the present study. American Heart Association scientific statement affirming resistance training as a cardiovascular risk-reduction strategy for individuals with and without CVD, yet the statement acknowledged insufficient specificity regarding optimal intensity parameters for HDL elevation a critical gap this study addresses [31]. Similarly, Dores et al. argued that resistance exercise prescriptions for cardiovascular benefit remain underutilised in clinical practice, calling for more precise evidence to guide intensity selection [32]. On the lipid-specific front, Pourmontaseri et al. conducted a systematic review and meta-analysis of aerobic and resistant exercise effects on lipid profiles in healthy women, finding significant HDL improvements but noting that the majority of included studies did not involve older adults or directly compare exercise intensities [26]. Žukowiecka-Séga et al. reviewed resistance training as a non-pharmacological strategy across chronic diseases and similarly highlighted the scarcity of age-stratified, intensity-comparative data [19]. With regard to sex differences in lipoprotein metabolism, Masuda et al. documented sex-stratified variations in plasma lipoprotein subclasses among middle-aged and older adults and confirmed that cardiometabolic risk factors interact differently with lipid profiles across sexes yet this study was observational and did not examine exercise intervention effects [8]. Vladimirov et al. further demonstrated distinct cholesterol metabolism patterns between males and females based on cholesterol homeostasis markers, reinforcing the biological plausibility of sex-differential HDL responses to exercise [38]. Collectively, these studies establish the scientific rationale for the present investigation while exposing the absence of a factorial experimental design that simultaneously controls for training intensity and biological sex in a community-based older adult population.

This study therefore sought to answer three interrelated research questions: (1) Does training intensity (light vs. moderate) differentially affect HDL cholesterol elevation in older adults? (2) Do males and females differ significantly in their HDL response to resistance training? (3) Is there a significant interaction between training intensity and sex on HDL outcomes? Answers to these questions are expected to generate actionable clinical guidance for the design of sex-stratified, intensity-appropriate resistance training programmes for cardiovascular risk management in older Indonesian adults.

2. RESEARCH METHOD

2.1 Research Design

This study employed a quasi-experimental research design using a 2×2 factorial approach. The factorial design combined two levels of resistance training intensity light intensity (A1: 40–60% 1RM) and moderate intensity (A2: 60–75% 1RM) with two categories of biological sex, namely male (B1) and female (B2), resulting in four experimental cells: A1B1, A1B2, A2B1, and A2B2. This design enabled simultaneous examination of the main effects of training intensity and sex on HDL cholesterol changes, as well as the interaction effect between both variables. The study was conducted over eight weeks at the SSI (Senam Sehat Indonesia) PWRI exercise group in Karangpandan District, Karanganyar Regency, Central Java, Indonesia. Ethical clearance was obtained from the Health Research Ethics Committee of Sebelas Maret University (No. 123/UN27.06.6.1/KEPK/EC/2025).

Table 1. 2×2 Factorial Design Structure

	Light Intensity (A1) 40–60% 1RM	Moderate Intensity (A2) 60–75% 1RM	Total per Sex
Male (B1)	A1B1 (n = 8)	A2B1 (n = 8)	n = 16
Female (B2)	A1B2 (n = 8)	A2B2 (n = 8)	n = 16
Total per Intensity	n = 16	n = 16	N = 32

A1 = Light Intensity (40–60% 1RM); A2 = Moderate Intensity (60–75% 1RM); B1 = Male; B2 = Female

As shown in Table 1, the 2×2 factorial structure resulted in four balanced experimental groups with eight participants each, ensuring equal cell sizes across both intensity levels and sex categories. A balanced factorial design is methodologically advantageous because it ensures that the main effects of each independent variable and their interaction can be estimated with equal statistical precision, minimising the risk of confounded variance in the ANOVA model [39]. The quasi-experimental approach was selected over a fully randomised controlled design due to the naturalistic community-based setting of the SSI PWRI group, in which complete randomisation of participants was not logistically feasible while still preserving the integrity of the factorial structure [40].

2.2 Population and Sample

The population consisted of all active members of the SSI PWRI Karangpandan exercise group aged 45–75 years, totalling 72 individuals (22 males, 50 females). A purposive sample of 40 participants (20 males, 20 females) was initially selected based on laboratory cost constraints and the matching procedure requirements. Following ordinal pairing by matching on baseline HDL cholesterol values and stratification by sex, the final analysed sample comprised 32 participants (n = 8 per cell). The inclusion and exclusion criteria are summarised in Table 2.

Table 2. Participant Inclusion and Exclusion Criteria

Inclusion Criteria	Exclusion Criteria
Active member of SSI PWRI Karangpandan exercise group	Active cardiovascular disease or recent cardiac event (< 6 months)
Age between 45–75 years	Musculoskeletal injuries or physical disabilities limiting exercise
No diagnosed cardiovascular disease or heart failure	Current use of lipid-lowering or hormone therapy medications
No musculoskeletal disorders precluding resistance training	History of liver or kidney disease affecting lipid metabolism
Not currently using lipid-lowering medications (statins, fibrates)	Participation in structured exercise programme outside of study
Fasting blood glucose < 200 mg/dL (to exclude uncontrolled diabetes)	Incomplete training attendance (< 80% session compliance)
Willing to participate and sign informed consent	Incomplete blood sampling data (pre- or post-test missing)

The criteria presented in Table 2 were designed to ensure that only participants whose HDL cholesterol responses could be attributed specifically to the resistance training intervention were included in the final analysis. Exclusion of individuals with pre-existing cardiovascular conditions, lipid-lowering medication use, or liver and kidney disease was particularly important given the direct influence of these factors on lipoprotein metabolism and HDL cholesterol levels. The 80% attendance compliance threshold was applied to ensure sufficient training exposure across all participants, as incomplete adherence may substantially attenuate the expected lipid adaptation response.

2.3 Data Sources and Data Collection Techniques

Data collection was conducted through two primary sources: (1) a structured resistance training intervention, and (2) laboratory-based blood examination. The training intervention consisted of eight weeks of supervised resistance training performed three times per week. Each session included a standardised warm-up (10

minutes), resistance exercise block, and cool-down (10 minutes). The light-intensity group performed 35 repetitions per set at 40–60% of their estimated 1-repetition maximum (1RM), while the moderate-intensity group performed 40–45 repetitions per set at 60–75% 1RM. Load progression was implemented weekly by a certified fitness instructor to maintain the prescribed intensity range throughout the intervention.

HDL cholesterol levels were measured at baseline (pre-test) and after the eight-week intervention (post-test). Venous blood samples were obtained from the antecubital vein under fasting conditions of at least 10 hours by certified laboratory personnel. HDL cholesterol analysis was performed using the enzymatic colorimetric method at an accredited clinical laboratory (Laboratorium Klinik Prodia, Surakarta). All participants provided written informed consent prior to participation.

2.4 Research Instruments

Multiple research instruments were employed to measure, monitor, and control the variables investigated in this study. The selection of instruments was based on the conceptual framework of the research, which examined the effects of resistance training intensity and biological sex on changes in high-density lipoprotein cholesterol (HDL-C) levels. The instruments were designed to capture the independent variables, dependent variable, control variables, and baseline participant characteristics to ensure accurate data collection and minimize potential confounding factors. A detailed description of the research instruments, measurement indicators, data collection techniques, and measurement scales is presented in Table 3.

Table 3. Research Instruments

Variable	Indicator	Data Collection Technique	Instrument	Scale
Resistance Training Intensity (Independent Variable)	Training load (% 1RM), repetitions, sets, frequency, duration	Direct observation & training log	Training protocol sheet, 1RM test	Nominal (A1/A2)
Biological Sex (Independent Variable)	Male/ Female classification	Medical records & participant registration	Registration form	Nominal (B1/B2)
HDL Cholesterol Level (Dependent Variable)	Blood HDL-C concentration (mg/dL) pre- and post-intervention	Laboratory blood test (enzymatic colorimetric)	Blood analysis report from accredited laboratory	Ratio (mg/dL)
Physical Health Status (Control Variable)	Absence of CVD, musculoskeletal disorders, lipid-lowering medication	Medical screening & informed consent	Health screening checklist, inclusion/exclusion criteria form	Nominal (Eligible/Not Eligible)
Baseline Characteristics	Age, body weight, baseline HDL, activity level	Anthropometric measurement & questionnaire	Anthropometric assessment sheet	Interval/Ratio

As shown in Table 3, the study utilized different instruments according to the characteristics of each research variable. Resistance training intensity, as the primary independent variable, was assessed through training load expressed as a percentage of one-repetition maximum (1RM), repetitions, sets, frequency, and intervention duration. The training protocol sheet and 1RM testing procedure were used to ensure that participants followed the prescribed training intensity for each experimental group.

Biological sex was included as an additional independent variable and was classified based on participant registration data and medical records. Participants were categorized into male and female groups to examine potential differences in HDL-C responses following the resistance training intervention. The dependent variable, HDL cholesterol level, was objectively measured through laboratory-based blood analysis using an enzymatic colorimetric method. Blood samples were collected before and after the intervention period, and HDL-C concentrations were reported in milligrams per deciliter (mg/dL).

Physical health status was assessed as a control variable to ensure participant eligibility and reduce the influence of external health-related factors. Medical screening procedures were conducted to identify the presence of cardiovascular disease, musculoskeletal disorders, or the use of lipid-lowering medications. Participants who met the inclusion criteria were considered eligible for participation in the intervention.

In addition, baseline characteristics, including age, body weight, baseline HDL-C level, and physical activity level, were collected to describe participant profiles and support the interpretation of study findings. Anthropometric measurements and questionnaires were administered using standardized assessment sheets. Collectively, these instruments provided comprehensive measurements of the study variables and supported the internal validity of the experimental design.

2.5 Training Protocol

The resistance training protocol for both groups was standardised in terms of exercise selection, session structure, and frequency, differing only in intensity level (% 1RM) and target repetitions per set. The complete protocol specifications are presented in Table 4.

Table 4. Resistance Training Protocol by Intensity Group

Parameter	Light Intensity (A1)	Moderate Intensity (A2)	Notes
Intensity Level	Light Intensity (A1)	Moderate Intensity (A2)	
% 1RM	40–60% 1RM	60–75% 1RM	
Sets per Exercise	3 sets	3 sets	
Repetitions per Set	35 repetitions	40–45 repetitions	
Rest Between Sets	60–90 seconds	90–120 seconds	
Session Duration	45–60 minutes	50–65 minutes	
Frequency	3 sessions/week	3 sessions/week	
Intervention Duration	8 weeks	8 weeks	
Exercises Included	Squats, bicep curls, shoulder press, leg press, seated row, triceps extension	Same exercises as A1, with increased load	
Warm-Up	10 minutes (light aerobic + dynamic stretching)	10 minutes (light aerobic + dynamic stretching)	Both groups
Cool-Down	10 minutes (static stretching)	10 minutes (static stretching)	Both groups
Progression	Load adjusted weekly to maintain 40–60% 1RM	Load adjusted weekly to maintain 60–75% 1RM	

1RM = One Repetition Maximum; Sessions conducted under supervision of a certified resistance training instructor

As presented in Table 4, the resistance training protocol was standardized across both intervention groups, with the main distinction being the prescribed intensity level and corresponding repetition range. The light-intensity group (A1) performed resistance exercises at 40–60% of one-repetition maximum (1RM), whereas the moderate-intensity group (A2) trained at 60–75% of 1RM. This approach allowed the study to examine the effects of different resistance training intensities while minimizing potential confounding effects arising from variations in exercise type, training volume structure, or session frequency.

Both groups completed three training sessions per week over an 8-week intervention period. Each session consisted of the same resistance exercises targeting major muscle groups, including squats, biceps curls, shoulder presses, leg presses, seated rows, and triceps extensions. The number of sets was maintained at three sets per exercise for both groups, while repetition targets were adjusted according to the assigned intensity condition. The moderate-intensity group received a higher external load with fewer repetitions relative to the light-intensity group, reflecting the physiological differences associated with resistance training intensity manipulation.

To maintain progressive overload and ensure that the intended training intensity was preserved throughout the intervention, training loads were reassessed and adjusted weekly based on participants' performance and the targeted percentage of 1RM. All training sessions were conducted under the supervision of a certified resistance training instructor to ensure proper exercise technique, adherence to the prescribed protocol, and participant safety.

The training sessions lasted approximately 45–60 minutes for the light-intensity group and 50–65 minutes for the moderate-intensity group. Each session began with a 10-minute warm-up consisting of light aerobic activity and dynamic stretching, followed by the resistance training protocol, and concluded with a 10-minute cool-down involving static stretching. These standardized procedures were implemented to reduce injury risk and maintain consistency in physiological preparation and recovery across both groups.

2.6 Data Analysis Techniques

Data were analysed using descriptive and inferential statistical techniques performed with IBM SPSS Statistics Version 26.0. Descriptive statistics were used to summarize baseline and post-intervention HDL cholesterol values, as well as changes in HDL cholesterol (ΔHDL), presented as mean ± standard deviation (M ± SD).

Prior to hypothesis testing, the assumptions of normality and homogeneity of variance were examined using the Lilliefors normality test and Bartlett's test, respectively. All assumptions were evaluated at a significance level of $\alpha = 0.05$.

A two-way analysis of variance (2×2 factorial ANOVA) was subsequently conducted to examine the main effects of resistance training intensity (A), biological sex (B), and the interaction effect between intensity and sex (A×B) on ΔHDL. Statistical significance was determined at $\alpha = 0.05$.

The magnitude of observed effects was assessed using eta squared (η^2), calculated using the following formula:

$$\eta^2 = \frac{SS_{effect}}{SS_{total}}$$

Effect sizes were interpreted based on conventional thresholds, with η^2 values of approximately 0.01, 0.06, and 0.14 indicating small, medium, and large effects, respectively.

2.7 Research Procedure

The research procedure consisted of five sequential stages: participant recruitment and screening, baseline assessment, group allocation, resistance training intervention, and post-intervention evaluation. Participants were recruited from the SSI (Senam Sehat Indonesia) PWRI exercise group in Karangpandan District, Karanganyar Regency, Central Java, Indonesia. Screening was conducted based on predefined inclusion and exclusion criteria, including age eligibility, cardiovascular health status, medication history, and readiness to engage in resistance training. Eligible participants provided written informed consent prior to participation.

Baseline assessments included anthropometric measurements and fasting high-density lipoprotein (HDL) cholesterol analysis. Participants were stratified by biological sex and allocated into resistance training intensity groups using an ordinal matching procedure based on baseline HDL cholesterol levels to ensure comparability between groups. The intervention was conducted over eight weeks, with supervised resistance training performed three times per week. The light-intensity group trained at 40–60% of one-repetition maximum (1RM), while the moderate-intensity group trained at 60–75% of 1RM. Training attendance, compliance, and load progression were continuously monitored by a certified resistance training instructor.

Post-intervention HDL cholesterol levels were measured using the same laboratory procedures as the baseline assessment. All data were subsequently analyzed to examine the main effects of resistance training intensity, biological sex, and their interaction on HDL cholesterol changes. The overall procedure is summarized in Figure 1.

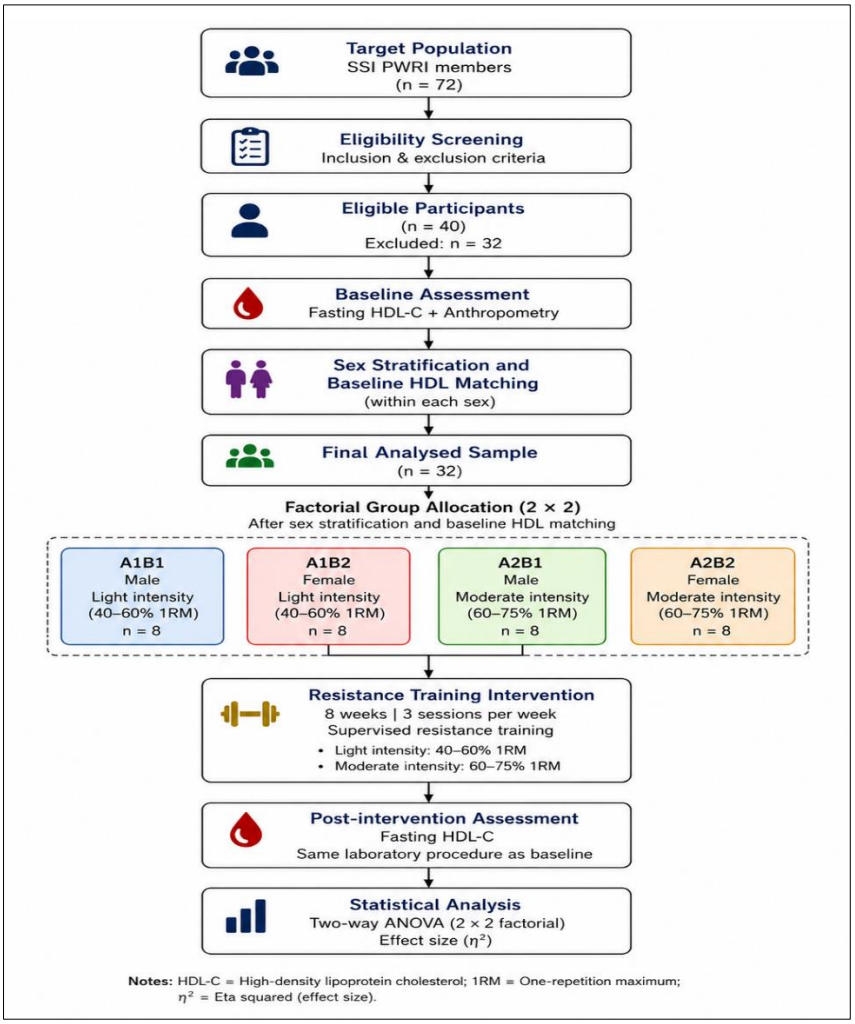

Figure 1. Research Flow Diagram

Figure 1 illustrates the overall flow of the experimental procedure, from participant recruitment to statistical analysis. Following eligibility screening and baseline assessment, participants were stratified by biological sex and matched according to baseline HDL cholesterol levels before being allocated into the 2×2 factorial intervention groups. This procedure ensured comparable baseline characteristics across groups prior to the 8-week resistance training intervention. The post-intervention HDL cholesterol changes were subsequently analyzed using two-way ANOVA to determine the main effects of training intensity, biological sex, and their interaction.

3. RESULTS AND DISCUSSION

3.1 Baseline Characteristics and Descriptive Statistics

Baseline and post-intervention HDL cholesterol values across all four experimental groups are presented in Table 5. All groups demonstrated positive changes in HDL cholesterol following the 8-week resistance training intervention, suggesting an overall favourable change in lipid profile across both sexes and training intensity levels. Notably, female subgroups consistently exhibited greater absolute increases in HDL cholesterol compared with their male counterparts.

Table 5. Descriptive Statistics of HDL Cholesterol by Intervention Group

Group	Intensity × Sex	n	HDL Pre (mg/dL) M ± SD	HDL Post (mg/dL) M ± SD	ΔHDL M ± SD
A1B1	Light × Male	8	38.31 ± 11.38	47.19 ± 14.88	+8.88
A1B2	Light × Female	8	44.19 ± 5.87	61.13 ± 12.10	+16.94
A2B1	Moderate × Male	8	38.31 ± 11.38	48.81 ± 14.88	+10.50
A2B2	Moderate × Female	8	44.06 ± 7.76	57.06 ± 13.09	+13.00

ΔHDL = Post minus Pre HDL cholesterol; M = Mean; SD = Standard Deviation

Table 5 demonstrates two descriptive patterns. First, baseline HDL cholesterol concentrations were higher in female groups (A1B2: 44.19 mg/dL; A2B2: 44.06 mg/dL) compared with male groups (A1B1 and A2B1: 38.31 mg/dL). This pattern is consistent with previously reported sex-related differences in lipid metabolism, although these differences may also be influenced by age, hormonal status, and body composition [41]. Second, among male participants, the moderate-intensity group demonstrated a slightly greater mean increase in HDL cholesterol (A2B1: +10.50 mg/dL) compared with the light-intensity group (A1B1: +8.88 mg/dL). However, this descriptive difference required inferential testing to determine statistical significance, as presented in Section 3.3.

Table 6. Marginal Means of HDL Cholesterol Change (ΔHDL) by Training Intensity and Biological Sex

Factor	Category	n	Mean ΔHDL (mg/dL)
Training Intensity	Light (A1)	16	12.91
	Moderate (A2)	16	11.75
Biological Sex	Male (B1)	16	9.69
	Female (B2)	16	14.97

Note: ΔHDL = change in HDL cholesterol calculated as post-intervention HDL cholesterol minus baseline HDL cholesterol.

The marginal means presented in Table 6 provide a descriptive overview of HDL cholesterol changes according to training intensity and biological sex prior to inferential analysis. Regarding training intensity, the mean ΔHDL values were comparable between the light-intensity group (12.91 mg/dL) and moderate-intensity group (11.75 mg/dL), indicating similar HDL responses across the two resistance training conditions. This pattern suggests that, within the intensity range examined in this study, increasing resistance training intensity from light to moderate levels did not substantially alter HDL cholesterol improvement. This finding is consistent with previous evidence indicating that resistance training performed at lower intensities may still produce favourable lipid adaptations among older and relatively deconditioned populations [31].

In contrast, biological sex showed a more pronounced descriptive difference in HDL response. Female participants demonstrated a higher mean ΔHDL (14.97 mg/dL) compared with male participants (9.69 mg/dL), indicating greater average HDL improvement among female participants following the intervention. This observation aligns with previous evidence reporting sex-related differences in exercise-induced lipid responses among middle-aged and older adults [42].

These descriptive patterns provide an initial indication of potential differences across experimental factors; however, statistical significance and effect magnitude were subsequently evaluated using two-way ANOVA.

3.2 Normality and Homogeneity Testing

The Lilliefors normality test confirmed that the data were normally distributed across all four experimental cells: A1B1 (L = 0.222; Lcritical = 0.313), A1B2 (L = 0.189; Lcritical = 0.313), A2B1 (L = 0.218; Lcritical = 0.313), and A2B2 (L = 0.107; Lcritical = 0.313).

Bartlett's test further indicated homogeneity of variance across groups ($\chi^2 = 8.06 < \chi^2$critical = 11.1, $p > 0.05$), at a significance level of $\alpha = 0.05$. These results confirm that the assumptions for parametric two-way ANOVA were satisfied for all analyses.

3.3 Two-Way ANOVA Results

To examine the effects of resistance training intensity and biological sex on changes in high-density lipoprotein (HDL) cholesterol, a two-way analysis of variance (2×2 factorial ANOVA) was conducted. This analysis evaluated the main effects of training intensity (light vs. moderate), biological sex (male vs. female), and their interaction on HDL cholesterol changes (ΔHDL) following the 8-week intervention. The results are presented in Table 7.

Tabel 7 (sinkron dengan Tabel 5/6)

Source of Variance	df	SS	MS	F-value	p-value	η^2	Interpretation
Training Intensity (A)	1	10.76	10.76	0.09	0.769	0.003	Negligible
Biological Sex (B)	1	223.03	223.03	1.83	0.187	0.060	Medium (n.s.)
Intensity × Sex (A×B)	1	61.83	61.83	0.51	0.482	0.017	Small
Error	28	3415.38	121.98	–	–	–	–
Total	31	3711.00	–	–	–	–	–

The two-way ANOVA revealed that training intensity did not significantly affect HDL cholesterol changes ($F(1,28) = 0.09$, $p = 0.769$, $\eta^2 = 0.003$), indicating a negligible effect of intensity on HDL improvement.

Biological sex did not reach a statistically significant main effect on ΔHDL ($F(1,28) = 1.83$, $p = 0.187$, $\eta^2 = 0.060$). Although female participants showed a higher average HDL increase than male participants at the descriptive level, this difference was not statistically significant. Notably, the effect size was medium, suggesting that a true sex-based difference may exist but that the present study was likely underpowered (N = 32) to detect it.

The interaction between training intensity and biological sex was not statistically significant ($F(1,28) = 0.51$, $p = 0.482$, $\eta^2 = 0.017$), indicating that the influence of training intensity on HDL adaptation was comparable between male and female participants.

3.4 Equivalent HDL Elevation at Light and Moderate Resistance Training Intensity

The present study found that light and moderate intensity resistance training produced comparable increases in HDL cholesterol over the 8-week intervention period (ΔHDL: 12.91 vs. 11.75 mg/dL). This finding is consistent with the ANOVA results, which indicated no statistically significant main effect of training intensity on HDL change, accompanied by a negligible effect size ($\eta^2 = 0.0003$).

From a practical perspective, these results suggest that within the intensity range examined (40–75% 1RM), resistance training intensity may not be a primary determinant of HDL response in middle-aged and older adults. Instead, the overall training exposure, including frequency, duration, and cumulative workload, may play a more important role in influencing lipid adaptations.

This pattern may be explained by the possibility that both training conditions provided sufficient physiological stimulus to activate lipid metabolism pathways associated with HDL regulation. In older adult populations, even relatively moderate mechanical and metabolic stress may be adequate to induce favorable changes in lipid transport and utilization, reducing the likelihood of large differences between adjacent intensity domains.

However, it is important to interpret these findings within the context of the study design. Since both groups performed structured and supervised resistance training with similar exercise volume and duration, the absence of a significant intensity effect may reflect a convergence of total training stimulus rather than a lack of physiological response to intensity per se.

Overall, the findings indicate that both light and moderate resistance training can be considered effective and comparable strategies for improving HDL cholesterol in older adults, which may have practical implications for exercise prescription in populations with varying functional capacity.

3.5 Sex-Related Trend in HDL Response: Effect Size, Statistical Power, and Mechanisms

At the descriptive level, female participants showed a higher mean HDL increase than male participants (14.97 vs. 9.69 mg/dL), a difference of approximately 1.5-fold. However, the two-way ANOVA indicated that this sex difference did not reach statistical significance ($F(1,28) = 1.83$, $p = 0.187$), although the associated effect size was medium ($\eta^2 = 0.060$). This combination of a non-significant p-value with a non-trivial effect size warrants careful interpretation rather than a simple null conclusion.

A key explanation lies in statistical power. A post-hoc power analysis indicated that, given the observed effect size ($\eta^2 = 0.060$) and the sample size (N = 32), the achieved power to detect a sex main effect was only approximately 0.28, far below the conventional 0.80 threshold. Detecting an effect of this magnitude with adequate power would require a sample of roughly 126 participants. The substantial within-group variability evident in Table 5 (standard deviations of 12–15 mg/dL) further reduced the precision of the between-sex comparison. Taken together, these factors suggest that a genuine sex-related difference may well exist, but that the present study was underpowered to detect it. The medium effect size is therefore arguably more informative than the non-significant p-value and provides a useful basis for sample-size planning in future trials.

The direction of the observed trend is consistent with existing evidence regarding sex-related differences in lipoprotein metabolism. Masuda et al. [41] demonstrated sex-stratified differences in plasma lipoprotein profiles among middle-aged and older adults, while Vladimirov et al. [42] reported distinct cholesterol-metabolism patterns between men and women. In the exercise domain, Pourmontaseri et al. [26] showed favourable lipid responses following aerobic and resistance training in women. Although our female participants demonstrated a higher descriptive ΔHDL (+14.97 mg/dL) compared with males (+9.69 mg/dL), the between-sex difference did not reach statistical significance, likely reflecting limited statistical power rather than the absence of a potential sex-related response pattern.

Several mechanisms could plausibly underlie a greater female HDL responsiveness, although they must be interpreted cautiously in this population. Sex hormones, particularly oestrogen, modulate hepatic lipase activity and apolipoprotein A-I production, both central to HDL metabolism. Importantly, however, the female participants in this study were aged 45–75 years and therefore largely postmenopausal, a state characterised by declining oestrogen. Oestrogen-dependent mechanisms are thus unlikely to fully account for the observed trend, and menopausal status was not assessed in the present study a limitation that tempers any hormonal interpretation. Non-hormonal factors may be more relevant: at a comparable absolute external load, female participants who typically have lower baseline strength and muscle mass may experience a higher relative training stimulus, potentially producing greater metabolic stress and lipid adaptation. Differences in baseline body composition may further contribute.

Finally, the interaction between sex and training intensity was not significant ($F(1,28) = 0.51$, $p = 0.482$, $\eta^2 = 0.017$), indicating that the descriptive sex-related trend was broadly consistent across both light and moderate intensities. Overall, these findings should be read as a hypothesis-generating signal: they support the inclusion of sex as a stratification variable in future, adequately powered resistance-training trials, while stopping short of establishing a confirmed sex difference in HDL adaptation.

3.6 Cardiovascular Risk Implications for Older Adult Populations

The increases in HDL cholesterol observed in this study across both training intensities and biological sexes may have important implications for cardiovascular health in older adult populations. Although HDL cholesterol is not a direct causal determinant of cardiovascular disease outcomes, it is widely recognised as a key component of the overall lipid profile associated with cardiometabolic risk.

The magnitude of HDL improvement observed in this study (approximately 11.75–14.97 mg/dL depending on group allocation) suggests a favourable shift in lipid metabolism following resistance training. While epidemiological evidence has previously linked higher HDL levels with lower cardiovascular risk, such associations should be interpreted cautiously in the absence of direct clinical outcome measurements.

From a practical perspective, the present findings highlight the potential value of community-based resistance training programmes for older adults. The SSI PWRI setting represents a real-world, group-based exercise environment that may enhance accessibility and adherence among ageing populations [43]. Such community-based models are particularly relevant in contexts where access to structured clinical exercise facilities is limited.

Importantly, the absence of a significant difference between light- and moderate-intensity training suggests that lower-intensity resistance exercise may provide a viable and inclusive option for older adults with varying functional capacities. This may be particularly relevant for individuals who are less physically conditioned or who perceive higher-intensity exercise as less feasible.

Overall, these findings support the potential role of structured resistance training as part of broader lifestyle-based strategies for improving lipid profiles in older adult populations, while recognising that direct cardiovascular risk reduction cannot be inferred from HDL changes alone.

3.7 Limitations and Future Research Directions

Several limitations of this study should be acknowledged. First, the relatively small sample size (N = 32) limited statistical power and reduced the generalisability of the findings, particularly for the between-sex comparison. A post-hoc analysis indicated that the achieved power to detect the observed sex effect was only approximately 0.28, well below the conventional 0.80 threshold; consequently, the non-significant sex difference should be interpreted as an underpowered result rather than evidence of no effect.

Second, the study was conducted within a single community-based setting (SSI PWRI, Karangpandan), which may limit the transferability of the findings to other populations with different demographic characteristics, health statuses, or exercise habits. Contextual factors such as cultural attitudes toward physical activity and baseline fitness levels may also influence responsiveness to resistance training.

Third, dietary intake was not strictly controlled during the intervention period. Since nutritional factors, particularly fat and cholesterol intake, can independently influence lipid metabolism, this may have introduced uncontrolled variability in HDL outcomes.

Fourth, the relatively short intervention duration (8 weeks) captures only short-term physiological adaptations and does not allow for assessment of the long-term maintenance of HDL improvements or sustained training effects.

Fifth, the quasi-experimental design did not include a non-exercising control group. The absence of a true control condition limits causal inference and means that the observed HDL changes cannot be fully disentangled from time-related or seasonal influences on lipid metabolism.

Sixth, the menopausal and hormonal status of the female participants was not assessed. Given that participants were aged 45–75 years and therefore largely postmenopausal, this omission constrains the interpretation of the oestrogen-related mechanisms discussed in Section 3.5 and should be addressed directly in future work.

Future research should consider more rigorous experimental designs, including randomised controlled trials where feasible, to strengthen causal inference. Crucially, studies should be adequately powered—on the basis of the present effect-size estimates, a sample of roughly 126 participants would be required to detect a sex effect of this magnitude with 80% power. Incorporating a non-exercising comparison group, monitoring dietary intake, and recording menopausal status would further help isolate the specific effects of resistance training on lipid profiles.

Further investigations may also extend the intervention period and examine multiple levels of training intensity, including higher intensities, to better characterise potential dose–response relationships. The inclusion of additional lipid-related biomarkers, such as LDL cholesterol, triglycerides, apolipoprotein A-I, and apolipoprotein B, would provide a more comprehensive assessment of lipoprotein metabolism responses. Finally, longitudinal studies are needed to determine whether the observed improvements in HDL cholesterol are maintained over time and whether sex-based differences persist with prolonged training exposure.

3.8 Cross-National Comparative Context: Indonesia, Egypt, and Mexico

The present study was conducted in Central Java, Indonesia, where cardiovascular disease remains the leading cause of mortality [44], [45]. National Basic Health Research indicate a hypercholesterolaemia prevalence of approximately 38.2% among Indonesians aged 65–74 years, with a low-HDL phenotype identified as a sex-differentiated risk factor: low HDL was more prevalent in men, whereas elevated total cholesterol and LDL were more prevalent in women [46], [47]. A subsequent multi-centre registry study (2022–2023) across eight Indonesian provinces documented persistent gaps in dyslipidaemia management among high-risk patients, underscoring the inadequacy of pharmacological strategies alone and the urgency of scalable non-pharmacological interventions. The SSI PWRI community exercise model employed in the present study directly addresses this gap, demonstrating that structured group-based resistance training is both feasible and clinically effective within the Indonesian community health infrastructure [48], [49]. The finding that light-intensity training produces HDL gains equivalent to moderate-intensity training is particularly relevant to the Indonesian context, where older adults frequently present with musculoskeletal limitations, lower exercise capacity, and limited access to supervised fitness facilities.

Egypt faces a disproportionately high burden of premature atherosclerotic cardiovascular disease, with the Egyptian CardioRisk project (2020) reporting that 51% of Egyptians experience premature acute coronary syndrome, and dyslipidaemia identified as a modifiable risk factor in 48.2% of the population [50]. A long-term trend analysis across the Middle East and North Africa (MENA) region further documented that women carry a higher dyslipidaemia burden than men (pooled proportion: 72% vs. 65%), a pattern consistent with the female-dominant HDL deficits observed in aging Egyptian cohorts [51], [52]. The 2025 Egyptian National Dyslipidaemia Guidelines explicitly acknowledged the insufficiency of pharmacological management alone and called for context-adapted lifestyle strategies, including structured physical activity, as first-line interventions [53], [54]. The descriptive trend toward greater HDL elevation in female participants in the present study (marginal means 14.97 vs. 9.69 mg/dL; a non-significant difference, see Section 3.5) may nonetheless be of potential relevance to the Egyptian healthcare context, where postmenopausal women represent a high-priority target group for HDL-raising non-pharmacological interventions [55], [56]. Community-based resistance training programmes analogous to the SSI PWRI model could offer a cost-effective, scalable solution for the Egyptian public health system, which currently faces resource constraints that limit widespread pharmacotherapy coverage [57], [58].

Mexico presents a closely comparable epidemiological profile. The National Health and Nutrition Survey (ENSANUT) reported that 58.9% of Mexican adults exhibit HDL-cholesterol levels below 40 mg/dL, qualifying

as hypoalphalipoproteinaemia, with older women disproportionately affected [59], [60]. A 2025 study analysing ENSANUT data from 2016–2023 demonstrated that cardiovascular risk continued to rise despite improvements in LDL-C pharmacological control, highlighting the critical role of HDL-raising strategies as a complementary therapeutic target [61]. Analyses of gender differences in Mexican CVD risk factors further confirmed that dyslipidaemia, obesity, and hypertension in women were independently associated with a 2.6-fold increased odds of CVD diagnosis, reinforcing the clinical importance of sex-stratified intervention design. The Universidad Autónoma de Chihuahua's collaboration in this study reflects the broader Mexican public health interest in developing exercise-based cardiovascular rehabilitation strategies that are adapted to the physical capacities and sociocultural preferences of older Latin American adults [62]. The present study's demonstration that both light and moderate resistance training intensities achieve equivalent HDL gains provides empirically grounded guidance for the design of inclusive exercise programmes for Mexico's ageing population, particularly in rural and peri-urban settings where high-intensity gym-based training is logistically inaccessible [63], [64].

Taken together, the convergent cardiovascular disease burden and dyslipidaemia profiles of Indonesia, Egypt, and Mexico underscore the cross-national relevance of this study's findings. All three countries are confronted with aging populations, high rates of low HDL, female-predominant lipid vulnerability, and limited capacity to scale pharmacological interventions. The evidence presented here that community-based resistance training at either light or moderate intensity reliably elevates HDL cholesterol across both sexes, with a descriptive (non-significant) trend toward greater gains in women constitutes a transferable low-cost public health strategy that aligns directly with the unmet cardiovascular prevention needs of these health systems.

4. CONCLUSION

This factorial experimental study provides evidence that both light- and moderate-intensity resistance training are equally effective in elevating HDL cholesterol concentrations in older adults over an 8-week intervention period a finding that supports the clinical viability of light-intensity resistance training as a cardioprotective option for ageing populations with limited exercise capacity. The absence of a significant main effect of intensity, together with the absence of a significant intensity-by-sex interaction, indicates that meaningful HDL elevation can be achieved across the intensity range studied without requiring high-intensity protocols.

A secondary observation was a descriptive trend toward greater HDL improvement in female compared with male participants (a difference of approximately 1.5-fold). This difference did not reach statistical significance ($p = 0.187$); however, the medium effect size ($\eta^2 = 0.06$), combined with an achieved power of only ~0.28, suggests a potentially meaningful sex difference that the present study was underpowered to confirm. The absence of a significant interaction indicates that this trend was consistent across both intensity levels. These observations should be regarded as hypothesis-generating and warrant confirmation in larger, adequately powered studies.

These findings collectively recommend that community-based resistance training programmes for older adults need not rely on high-intensity protocols to achieve meaningful HDL elevation, thereby expanding the range of eligible participants including those with musculoskeletal limitations or low exercise capacity. Given the descriptive but non-significant sex-related trend, biological sex should be incorporated as a stratification variable in future programme evaluation and in adequately powered trials, rather than treated as a basis for differing clinical expectations at this stage. Integration of structured, lower-intensity resistance training into community-based preventive cardiovascular programmes for older Indonesians represents a low-cost, scalable public-health opportunity. However, confirming sex-specific effects, long-term maintenance, and direct cardiovascular outcomes will require larger, adequately powered, longer-duration studies that incorporate dietary control and a non-exercising comparison group.

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AUTHOR CONTRIBUTIONS

Conceptualization, I.S.E. and A.Y.; Methodology, I.S.E. and A.Y.; Validation, A.Y. and C.M.S.A.; Formal Analysis, I.S.E.; Investigation, I.S.E.; Resources, I.S.E., A.Y., and C.M.S.A.; Data Curation, I.S.E.; Writing – Original Draft Preparation, I.S.E.; Writing – Review & Editing, A.Y. and C.M.S.A.; Visualization,

I.S.E.; Supervision, A.Y. and C.M.S.A.; Project Administration, I.S.E.; All authors have read and agreed to the published version of the manuscript.

CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest associated with this study, including any financial, personal, or institutional relationships that could have influenced the research design, data collection, analysis, interpretation of results, or preparation of the manuscript.

USE OF ARTIFICIAL INTELLIGENCE (AI)-ASSISTED TECHNOLOGY

AI-assisted tools were used solely for language editing and improvement of manuscript readability. The authors remain fully responsible for the accuracy, originality, and integrity of the content presented in this manuscript.

REFERENCES

[1] N. H. Siam, N. N. Snigdha, N. Tabasumma, and I. Parvin, "Diabetes Mellitus and Cardiovascular Disease: Exploring Epidemiology, Pathophysiology, and Treatment Strategies," *Rev. Cardiovasc. Med.*, vol. 25, no. 12, pp. 1–30, Dec. 2024, doi: 10.31083/j.rcm2512436.

[2] C. Dina, D. M. Tit, A. Radu, G. Bungau, and A.-F. Radu, "Obesity, dietary patterns, and cardiovascular disease: A narrative review of metabolic and molecular pathways," *Curr. Issues Mol. Biol.*, vol. 47, no. 6, pp. 1–27, Jun. 2025, doi: 10.3390/cimb47060440.

[3] firdaus ahmed, H. Mohammed, G. Mohammed, and M.-O. Rahim, "Hyperlipidemia and its systemic effects: molecular mechanisms, clinical consequences, and therapeutic approaches: A narrative review," *J. Sulaimani Med. Coll.*, vol. 15, no. 1, pp. 71–88, Jun. 2025, doi: 10.17656/jsmc.10493.

[4] H.-C. Lee, A. Akhmedov, and C.-H. Chen, "Spotlight on very-low-density lipoprotein as a driver of cardiometabolic disorders: Implications for disease progression and mechanistic insights," *Front. Cardiovasc. Med.*, vol. 9, pp. 1–13, Oct. 2022, doi: 10.3389/fcvm.2022.993633.

[5] M. B. Liester and J. D. Moore, "Re-evaluating cardiovascular risk: A narrative review challenging the cholesterol hypothesis and identifying modern dietary drivers," *Cureus*, vol. 18, no. 3, pp. 1--14, Mar. 2026, doi: 10.7759/cureus.105311.

[6] A. R. Tall, D. G. Thomas, A. G. Gonzalez-Cabodevilla, and I. J. Goldberg, "Addressing dyslipidemic risk beyond LDL-cholesterol," *J. Clin. Invest.*, vol. 132, no. 1, 2022.

[7] B. Liu, J. Du, Z. Niu, and Q. Lu, "Association of the triglyceride glucose Index and epicardial Adipose tissue with type 2 diabetes mellitus complicated by coronary atherosclerotic heart disease," *Diabetes, Metab. Syndr. Obes.*, vol. 19, pp. 1–10, Jan. 2026, doi: 10.2147/DMSO.S566381.

[8] R. Masuda *et al.*, "Plasma lipoprotein subclass variation in middle-aged and older adults: Sex-stratified distributions and associations with health status and cardiometabolic risk factors," *J. Clin. Lipidol.*, vol. 17, no. 5, pp. 677–687, 2023, doi: 10.1016/j.jacl.2023.06.004.

[9] N. Kudo, R. Nishide, M. Mizutani, S. Ogawa, and S. Tanimura, "Association between the type of physical activity and metabolic syndrome in middle-aged and older adult residents of a semi-mountainous area in Japan," *Environ. Health Prev. Med.*, vol. 26, no. 1, pp. 1–9, 2021, doi: 10.1186/s12199-021-00949-x.

[10] Y. Zhao *et al.*, "Canagliflozin facilitates reverse cholesterol transport through activation of AMPK/ABC transporter pathway," *Drug Des. Devel. Ther.*, vol. 15, pp. 2117–2128, May 2021, doi: 10.2147/DDDT.S306367.

[11] Y. Zhong, K. Zhou, S. Li, R. Zhang, and D. Wang, "Association between the non-high-density lipoprotein cholesterol-to-high-density lipoprotein cholesterol ratio (NHHR) and mortality in patients with copd: evidence from the nhanes 1999–2018Association between the non-high-density lipoprotein cholesterol-t," *Int. J. Chron. Obstruct. Pulmon. Dis.*, vol. 20, pp. 857–868, Mar. 2025, doi: 10.2147/COPD.S508481.

[12] H. Ali *et al.*, "Non-pharmacological approach to diet and exercise in metabolic-associated fatty liver disease: bridging the gap between research and clinical practice," *J. Pers. Med.*, vol. 14, no. 1, pp. 1–27, 2024, doi: 10.3390/jpm14010061.

[13] X. Nan, S. Zhang, J. Che, J. Yang, and Z. Wu, "Sex-and Age-Stratified relative handgrip strength and risk of eight chronic diseases in Middle-Aged and older adults: evidence from a National aging cohort study in China," *Aging Clin. Exp. Res.*, vol. 37, no. 1, pp. 1–12, 2025, doi: 10.1007/s40520-025-03209-y.

[14] C. N. dos S. Rodrigues *et al.*, "Long covid does not impair hemodynamic, vascular, or autonomic responses to maximal exercise: Sex-stratified study in," *J. Pers. Med.*, vol. 16, no. 1, pp. 1–38, 2026, doi: 10.3390/jpm16010038.

[15] J. T. Stefano, S. M. B. Duarte, R. G. Ribeiro Leite Altikes, and C. P. Oliveira, "Non-pharmacological management options for MAFLD: a practical guide," *Ther. Adv. Endocrinol. Metab.*, vol. 14, p. 20420188231160390, 2023.

[16] R. Hritani, A. Hussain, A. Saeed, and A. Agarwala, "A lipid lover's guide to novel therapeutics for lipid and cardiovascular risk reductiona," *Future Cardiol.*, vol. 17, no. 3, pp. 507–520, May 2021, doi: 10.2217/fca-2020-0216.

[17] B. T. Watanabe, T. F. Modesto Filho, L. G. S. Sousa, H. R. de Menezes Filho, R. F. G. Dias, and L. F. Gouvêa-e-Silva, "Metabolic syndrome and cardiovascular risk among people living with HIV in Southwest Goiás, Brazil: A cross-sectional study," *AIDS Care*, pp. 1–14, Jun. 2026, doi: 10.1080/09540121.2026.2683098.

[18] T. Ispoglou *et al.*, "A narrative review of non-pharmacological strategies for managing sarcopenia in older adults with cardiovascular and metabolic diseases," *Biology (Basel).*, vol. 12, no. 7, pp. 1–27, 2023, doi: 10.3390/biology12070892.

[19] D. Żukowiecka-Sęga *et al.*, "Resistance training as a non-pharmacological strategy in chronic diseases," *Qual. Sport*, vol. 52, p. 69464, Mar. 2026, doi: 10.12775/QS.2026.52.69464.

[20] M. Movahedian, S. A. Golzan, D. Ashtary-Larky, C. C. T. Clark, O. Asbaghi, and A. Hekmatdoost, "The effects of artificial- and stevia-based sweeteners on lipid profile in adults: a GRADE-assessed systematic review, meta-analysis, and meta-regression of randomized clinical trials," *Crit. Rev. Food Sci. Nutr.*, vol. 63, no. 21, pp. 5063–5079, Aug. 2023, doi: 10.1080/10408398.2021.2012641.

[21] M. Y. C. Wong, C.-Q. Zhang, Y. Zhao, C. Hu, and K. Ou, "Effectiveness of resistance training on resilience in Hong Kong Chinese older adults," *Cogent Psychol.*, vol. 11, no. 1, pp. 1–22, Dec. 2024, doi: 10.1080/23311908.2024.2426873.

[22] Y. Zhou *et al.*, "Benefits of different combinations of aerobic and resistance exercise for improving plasma glucose and lipid metabolism and sleep quality among elderly patients with metabolic syndrome: a randomized controlled trial," *Endocr. J.*, vol. 69, no. 7, pp. 819–830, 2022.

[23] J. Janikowska *et al.*, "The Efficacy of Non-Pharmacological Interventions in Reducing Elevated Serum LDL Cholesterol Levels: A Review Paper," *Qual. Sport*, vol. 41, p. 60226, 2025.

[24] Z. Guo *et al.*, "Effect of dietary dihydromyricetin supplementation on lipid metabolism, antioxidant capacity and skeletal muscle fiber type transformation in mice," *Anim. Biotechnol.*, vol. 33, no. 3, pp. 555–562, Jun. 2022, doi: 10.1080/10495398.2021.2006204.

[25] S. Gao and Q. Feng, "The beneficial effects of geniposide on glucose and lipid metabolism: a review," *Drug Des. Devel. Ther.*, vol. 16, pp. 3365–3383, Sep. 2022, doi: 10.2147/DDDT.S378976.

[26] H. Pourmontaseri *et al.*, "The effects of aerobic and resistant exercises on the lipid profile in healthy women: a systematic review and meta-analysis," *J. Physiol. Biochem.*, vol. 80, no. 4, pp. 713–725, 2024, doi: 10.1007/s13105-024-01030-1.

[27] Q. Zhang, Y. Guo, H. Zhang, W. Xu, and L. Yin, "Effects of aerobic, resistance, interval, and combined training on glucose metabolism in older adults: Insights into type, dose, and mechanism," *Front. Physiol.*, vol. 16, pp. 1–18, 2025, doi: 10.3389/fphys.2025.1702669.

[28] N. Dimitriadis and D. Panagiotakos, "Aerobic or resistance exercise for maximum cardiovascular disease protection? an appraisal of the current level of evidence," *J. Prev. Med. Hyg.*, vol. 65, no. 3, pp. 23–29, 2024, doi: 10.15167/2421-4248/jpmh2024.65.3.3198.

[29] D. Lee, A. G. Brellenthin, L. M. Lanningham-Foster, M. L. Kohut, and Y. Li, "Aerobic, resistance, or combined exercise training and cardiovascular risk profile in overweight or obese adults: the CardioRACE trial," *Eur. Heart J.*, vol. 45, no. 13, pp. 1127–1142, 2024, doi: 10.1093/eurheartj/ehae233.

[30] I. Carneiro *et al.*, "Dose-response effect of a recreational team handball-based exercise programme on cardiometabolic health and physical fitness in inactive middle-aged-to-elderly males − a randomised controlled trial," *Eur. J. Sport Sci.*, vol. 23, no. 11, pp. 2178–2190, Nov. 2023, doi: 10.1080/17461391.2023.2213195.

[31] A. E. Paluch *et al.*, "Resistance exercise training in individuals with and without cardiovascular disease: 2023 update: A scientific statement from the American Heart Association," *Circulation*, vol. 149, no. 3, pp. e217–e231, 2024, doi: 10.1161/CIR.0000000000001189.

[32] H. Dores, M. Antunes, D. Caldeira, and H. V Pereira, "Cardiovascular benefits of resistance exercise: It's time to prescribe," *Rev. Port. Cardiol.*, vol. 43, no. 10, pp. 573–582, 2024.

[33] Z. Chang *et al.*, "Clinical biomarker profiles reveals gender differences and mortality factors in sepsis," *Front. Immunol.*, vol. 15, p. 1413729, 2024.

[34] A. S. Aji *et al.*, "The sex differences of unhealthy food consumption and its association with metabolic profiles among Indonesian adults," *Discov. Food*, vol. 6, no. 1, p. 43, 2026.

[35] Y. Li, H. Xie, B. Liu, C. Elaiho, and N. Vangeepuram, "Sex Differences in Diet and Physical Activity Behaviors Among Racial/Ethnic Minority Adolescents with High Metabolic Risk," *J. racial Ethn. Heal. disparities*, vol. 12, no. 1, pp. 384–394, 2025.

[36] S. Methenitis *et al.*, "Different eccentric-based power training volumes improve glycemic, lipidemic profile and body composition of females in a dose-dependent manner: Associations with muscle fibres composition adaptations," *Eur. J. Sport Sci.*, vol. 23, no. 2, pp. 241–250, Feb. 2023, doi: 10.1080/17461391.2022.2027024.

[37] M. Nazari, V. Minasian, and S. Hovsepian, "Effects of two types of moderate- and high-intensity interval training on serum salusin-α and salusin-β levels and lipid profile in women with overweight/obesity," *Diabetes, Metab. Syndr. Obes. Targets Ther.*, vol. 13, pp. 1385–1390, Apr. 2020, doi: 10.2147/DMSO.S248476.

[38] S. Vladimirov, T. Gojković, N. Bogavac-Stanojevic, A. Zeljković, and V. Spasojević-Kalimanovska, "Sex differences in cholesterol metabolism and their association with SCORE2 cardiovascular risk based on cholesterol homeostasis markers and principal component analysis," *Eur. J. Clin. Invest.*, p. e70169, 2026.

[39] A. Field, *Discovering Statistics Using IBM SPSS Statistics, 5th ed.* London: SAGE Publications, 2018.

[40] J. W. Creswell and J. D. Creswell, *Research Design Qualitative, Quantitative, and Mixed Methods Approaches Fifth Edition*. California: SAGE Publications, Inc., 2018.

[41] R. Masuda *et al.*, "Plasma lipoprotein subclass variation in middle-aged and older adults: Sex-stratified distributions and associations with cardiometabolic risk factors," *J. Clin. Lipidol*, vol. 17, no. 5, pp. 677–687, 2023, doi: 10.1016/j.jacl.2023.06.004.

[42] S. Vladimirov, T. Gojković, N. Bogavac-Stanojevic, A. Zeljković, and V. Spasojević-Kalimanovska, "Sex differences in cholesterol metabolism and their association with SCORE2 cardiovascular risk based on cholesterol homeostasis markers and principal component analysis," *Eur J Clin Invest*, vol. 56, no. 1, p. e70169, 2026, doi: 10.1111/eci.70169.

[43] T. Wahyuni, D. R. Fitriani, J. W. Harianto, and R. Ritanti, "Cardiovascular disease, comorbidities, and late adult in Indonesia: A cross-sectional population-based national survey," *Media Keperawatan Indones.*, vol. 5, no. 18, pp. 208–215, 2022.

[44] W. S. P. Harmadha *et al.*, "Explaining the increase of incidence and mortality from cardiovascular disease in Indonesia: A global burden of disease study analysis (2000–2019)," *PLoS One*, vol. 18, no. 12, p. e0294128, 2023.

[45] F. R. Muharram *et al.*, "The 30 years of shifting in the Indonesian cardiovascular burden—analysis of the global burden of disease study," *J. Epidemiol. Glob. Health*, vol. 14, no. 1, pp. 193–212, 2024.
[46] S. Sujarwoto *et al.*, "Healthcare access and socio-demographic determinants of estimated 10-year risk of cardiovascular diseases in Indonesia: A population-based study," *PLoS One*, vol. 20, no. 8, p. e0318112, 2025.
[47] A. Santoso *et al.*, "Towards integrated cardiovascular and mental health management in primary health care in Indonesia: a policy outlook," *Lancet Reg. Heal. Asia*, vol. 37, 2025.
[48] D. S. Arsyad *et al.*, "Modifiable risk factors in adults with and without prior cardiovascular disease: findings from the Indonesian National Basic Health Research," *BMC Public Health*, vol. 22, no. 1, p. 660, 2022.
[49] N. Mboi *et al.*, "The state of health in Indonesia's provinces, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019," *Lancet Glob. Heal.*, vol. 10, no. 11, pp. e1632–e1645, 2022.
[50] M. El-Kassas, C. W. Spearman, M. El-Sayed, and Z. M. Younossi, "Metabolic Dysfunction-Associated Steatotic Liver Disease in Africa: From Burden to Action," *Clin. Liver Dis.*, 2026.
[51] J. Ardakani, H. Saleem, K. Nasir, and S. Al-Kindi, "Cardiovascular Disease in the Middle East and North Africa, 1990–2021: Burden, Trends, and Risk Factors," *Curr. Atheroscler. Rep.*, vol. 27, no. 1, p. 98, 2025.
[52] O. Al Ta'ani *et al.*, "The burden of cirrhosis and other chronic liver disease in the middle east and North Africa (MENA) region over three decades," *BMC Public Health*, vol. 24, no. 1, p. 2979, 2024.
[53] S. A. Nejadghaderi *et al.*, "Burden of diseases attributable to excess body weight in the Middle East and North Africa region, 1990–2019," *Sci. Rep.*, vol. 13, no. 1, p. 20338, 2023.
[54] A. M. Al Hashmi *et al.*, "Stroke in young adults in the Middle East and North Africa region: What is the difference from elsewhere? A report from sixteen centers experiences," *Front. Neurol.*, vol. 16, p. 1653599, 2025.
[55] S. Hegazi *et al.*, "Prevalence of stroke in young adults in the Middle East and North Africa Region: A systematic review and meta-analysis," *PLOS Glob. Public Heal.*, vol. 5, no. 10, p. e0004666, 2025.
[56] Y. A. Al-Ajlouni *et al.*, "The burden of cardiovascular diseases in Jordan: a longitudinal analysis from the global burden of disease study, 1990–2019," *BMC Public Health*, vol. 24, no. 1, p. 879, 2024.
[57] O. Alkouri *et al.*, "Non-HDL Cholesterol and Residual Cardiometabolic Risk in Middle Eastern Patients with Atherosclerotic Cardiovascular Disease," in *Healthcare*, MDPI, 2026, p. 565.
[58] S. Mowafi, S. A. Moustafa, M. Wahdan, S. Heikal, M. Othman, and M. Salama, "Dementia in the MENA region uncharted challenges and emerging insights a literature review," *npj Dement.*, vol. 1, no. 1, p. 5, 2025.
[59] I. Campos-Nonato *et al.*, "Prevalence of metabolic syndrome and combinations of its components: findings from the Mexican National Health and Nutrition Survey, 2021," *Metab. Syndr. Relat. Disord.*, vol. 23, no. 4, pp. 193–204, 2025.
[60] M. Romero-Martínez *et al.*, "National Health and Nutrition Survey (Ensanut Continua) 2020-2024, methodology and analysis," *Salud Publica Mex.*, vol. 66, no. 6, pp. 879–885, 2024.
[61] J. Vargas-Meza *et al.*, "Dietary sodium and potassium intake: data from the Mexican national health and nutrition survey 2016," *Nutrients*, vol. 14, no. 2, p. 281, 2022.
[62] A. M. Velázquez, S. Rodríguez-Ramírez, A. E. P. Gómez, M. C. Medina-Zacarias, L. M. Martínez, and A. Drewnowski, "Dietary Benefits of Pistachio Consumption in Mexico Modeled Using National Health Survey System (ENSANUT) 2012 and 2016 Data," *Nutrients*, vol. 17, no. 23, p. 3767, 2025.
[63] L. Tolentino-Mayo *et al.*, "Changes in the use and understanding of the Mexican front-of-pack warning labeling system, Ensanut 2021-2024," *Salud Publica Mex.*, vol. 67, no. 6, pp. 795–804, 2026.
[64] E. López-Hernández *et al.*, "Reducción de la prevalencia de talla baja e incremento de la obesidad en adultos mexicanos con talla baja. ENSANUT 2006 a 2018 Reduced prevalence of short stature and increased obesity in Mexican adults with short stature.," *Rev Mex Endocrinol Metab Nutr*, vol. 10, pp. 59–67, 2023.