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The Effects of Physical Activity and Structured Exercise on Metabolic Health and Cardiovascular Risk: A Comprehensive Review

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Background:
Cardiovascular diseases (CVDs) remain the leading global cause of mortality and are increasingly recognised as a systemic metabolic–inflammatory disorder. Physical inactivity is a major modifiable risk factor, while regular physical activity is associated with broad cardiometabolic benefits.

Methods:
This narrative review synthesises evidence from meta-analyses, randomised controlled trials, and mechanistic studies identified through PubMed, Scopus, Web of Science, and JACC. Studies evaluating aerobic, resistance, combined, and high-intensity interval training (HIIT) were included. Outcomes included cardiovascular risk factors, inflammation, metabolic health, cardiorespiratory fitness, and clinical endpoints.

Results:
Regular physical activity reduces systemic inflammation (CRP, IL-6, TNF-α), improves endothelial function, and enhances insulin sensitivity. Combined aerobic and resistance training provides superior improvements in glycaemic control, including HbA1c reductions (~0.5%), fasting glucose, and lipid profiles compared with single-modality exercise. Clinically relevant reductions in blood pressure are observed, particularly in hypertensive populations (systolic ~5–10 mmHg). Exercise improves lipid profiles (↑HDL, ↓LDL and triglycerides) and reduces visceral fat (~7–20%). Improvements in VO_2 peak are strongly associated with reduced cardiovascular morbidity and mortality. A dose–response relationship is evident, with optimal benefits at 150–300 min/week of moderate-intensity activity. Sedentary behaviour independently increases cardiovascular risk. Despite benefits, adherence remains suboptimal, although digital tools and personalised interventions improve compliance.

Conclusion:
Physical activity is a cornerstone of cardiovascular disease prevention and management, improving metabolic, inflammatory, and haemodynamic health. Individualised and technology-supported exercise strategies may enhance long-term adherence and maximise clinical benefit.

Keywords: cardiovascular disease; physical activity; exercise training; inflammation; insulin sensitivity; hypertension; dyslipidaemia; obesity; visceral adiposity; VO_2 peak; sedentary lifestyle; preventive cardiology; exercise adherence.

1. Introduction

Cardiovascular diseases encompass a broad group of disorders affecting the heart and blood vessels, including coronary artery disease, cerebrovascular disease, and heart failure. According to the World Health Organisation (WHO), cardiovascular risk is defined as the probability that an individual will develop a cardiovascular event, such as myocardial infarction or stroke, over a specified period, based on the presence and interaction of multiple risk factors.

These risk factors include both non-modifiable determinants, such as age and genetic predisposition, and modifiable factors, including physical inactivity, unhealthy diet, tobacco use, obesity, hypertension, dyslipidemia, and impaired glucose metabolism. The cumulative burden of these factors determines overall cardiovascular risk and long-term outcomes.

Recent evidence has redefined cardiovascular disease as a chronic inflammatory and metabolic condition characterised by endothelial dysfunction, immune activation, and oxidative stress.[1] Physical inactivity is one of the most significant contributors to global cardiovascular risk. Conversely, regular physical activity has emerged as a highly effective, low-cost intervention capable of modifying multiple risk factors simultaneously. Understanding the mechanisms by which exercise influences cardiovascular risk is essential for developing effective prevention strategies.

2. Materials and Methods

1. This narrative review synthesises evidence on the impact of sport, structured exercise, and modifiable lifestyle factors on cardiovascular risk, longevity, and clinical outcomes. Literature searches were conducted in PubMed, Jacc Scopus, and Web of Science using keywords such as cardiovascular disease, physical activity, exercise, inflammation, glycaemic control, hypertension, dyslipidaemia, obesity, visceral fat, VO_2 peak, sedentary behaviour, preventive cardiology, and adherence.

2. Inclusion criteria were peer-reviewed human studies, systematic reviews, meta-analyses, or large cohort studies examining exercise effects on cardiovascular risk factors, metabolic health, inflammation, or clinical outcomes. Excluding criteria were pediatric studies, non-peer-reviewed sources, or studies with insufficient data.

3. Data on study design, population, exercise type (aerobic, resistance, or combined), intensity, duration, and outcomes (blood pressure, lipids, glycemic control, inflammatory markers, mortality) were extracted. Findings were integrated using a narrative approach to highlight physiological mechanisms, clinical endpoints, and population-specific differences.

3. Literature Review

3.1. Pathophysiology of Cardiovascular Disease

The development of atherosclerosis involves complex interactions between lipid metabolism, endothelial dysfunction, and inflammation. Endothelial injury reduces nitric oxide availability and increases vascular permeability, facilitating the infiltration of low-density lipoprotein (LDL) cholesterol into the arterial wall.

Oxidised LDL triggers an inflammatory cascade involving macrophage activation and foam cell formation, leading to plaque development. Chronic inflammation, mediated by cytokines such as interleukin-6 and tumour necrosis factor-alpha, contributes to plaque progression and instability.[2] Metabolic disturbances, including insulin resistance and visceral adiposity, further exacerbate these processes and are central components of cardiometabolic risk.

3.2 Anti-Inflammatory Effects of Exercise Training

Regular exercise training exerts potent anti-inflammatory effects, which are increasingly recognised as a key mechanism by which physical activity protects against cardiovascular and metabolic disease. Long-term habitual exercise is associated with lower basal levels of pro-inflammatory biomarkers, including high-sensitivity C-reactive protein (hsCRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α), compared with sedentary individuals, independent of changes in adiposity or body weight, suggesting systemic benefits beyond fat loss.[3]

Mechanistically, repeated bouts of exercise induce the release of anti-inflammatory myokines from contracting skeletal muscle and reduce the expression of inflammatory signalling pathways such as Toll-like receptor-4 on immune cells, thereby blunting chronic low-grade inflammation.[3] Randomised controlled trials and meta-analyses have demonstrated that regular aerobic exercise significantly reduces circulating inflammatory markers (e.g., CRP, TNF-α, IL-6) in middle-aged and older adults, aligning with observational evidence that higher cardiorespiratory fitness correlates with lower inflammation.[4]

These anti-inflammatory adaptations are thought to contribute to improved endothelial function, decreased atherogenesis, and reduced risk of cardiovascular events, reinforcing the role of structured exercise as a fundamental component of primary and secondary prevention strategies. [5]

3.3 Effects of Exercise on Glycemic Control

Regular physical activity is consistently linked to improved glycaemic control in individuals with prediabetes and type 2 Diabetes Mellitus. Evidence from systematic reviews and meta-analyses shows that combined aerobic and resistance training leads to broad improvements in glucose regulation and overall cardiometabolic health, highlighting the effectiveness of multimodal exercise approaches for managing blood glucose levels [6].

Comparative meta-analytic findings indicate that different exercise modalities may target specific aspects of glycaemic control. Combined aerobic and resistance training produces the greatest reductions in fasting blood glucose, while aerobic exercise alone is particularly effective in lowering long-term glycaemic markers such as HbA1c and reducing postprandial glucose levels [7]. Overall, structured exercise programmes have been shown to reduce average blood glucose levels, decrease time spent in hyperglycaemia, and improve daily glucose stability [8].

In people with type 2 Diabetes Mellitus, exercise interventions are associated with clinically meaningful improvements, including reductions in HbA1c of around half a percentage point and notable decreases in fasting plasma glucose. When exercise is combined with dietary interventions, additional improvements in glycaemic control are observed, further strengthening its role in comprehensive lifestyle management [6,8].

The beneficial effects of exercise are driven by multiple physiological mechanisms. During physical activity, muscle contractions increase glucose uptake independently of insulin through activation and movement of GLUT4 transporters to the cell surface. With regular training, the body develops greater insulin sensitivity, improved mitochondrial function, and enhanced oxidative capacity in skeletal muscle. These adaptations contribute to better regulation of both fasting and post-meal glucose levels. Resistance training increases muscle mass, thereby expanding the primary site of glucose disposal, while aerobic exercise improves cardiovascular fitness and mitochondrial efficiency; together, these forms of exercise produce complementary metabolic benefits [7].

Recent evidence also shows that how exercise is performed influences glycaemic outcomes. Activity performed after meals can significantly reduce post-meal glucose spikes. High-intensity interval training may achieve similar or even greater improvements in glucose control compared with traditional moderate-intensity continuous exercise, despite requiring less time. In addition, breaking up long periods of sitting with short bouts of light activity can independently improve postprandial glucose levels, even without formal exercise sessions [6].

Dose–response findings suggest that meaningful glycaemic benefits are typically achieved with moderate weekly activity levels, while even lower amounts of exercise still provide measurable improvements. Overall, these findings reinforce the idea that exercise is not merely an adjunct therapy but a core component of glycaemic management in type 2 Diabetes Mellitus. Future research should focus on tailoring exercise prescriptions to individual characteristics such as age, fitness level, and comorbid conditions to improve effectiveness and long-term adherence.

3.4 Effects of Exercise on Blood Pressure

Regular physical activity is a cornerstone non-pharmacological intervention for the prevention and management of hypertension, with robust evidence demonstrating clinically meaningful reductions in both systolic and diastolic blood pressure across diverse populations. A comprehensive meta-analysis of randomised controlled trials reported that aerobic exercise training significantly reduced systolic blood pressure (SBP) by 3.84 mmHg and diastolic blood pressure (DBP) by 2.58 mmHg, with consistent effects observed in both normotensive and hypertensive individuals.[9][12]

Larger reductions are observed in hypertensive populations; for example, a more recent meta-analysis demonstrated decreases of −9.91 mmHg in SBP and −4.32 mmHg in DBP following structured aerobic training interventions. [11] These blood pressure reductions are clinically meaningful, as even modest decreases of 2–5 mmHg in SBP are associated with notable reductions in cardiovascular morbidity and mortality, reinforcing the importance of exercise interventions.

In addition to chronic adaptations, acute bouts of exercise induce immediate post-exercise hypotension (PEH), characterised by transient reductions in blood pressure that may persist for several hours. Meta-analytic evidence indicates that a single session of aerobic exercise can significantly attenuate blood pressure responses, particularly under stress conditions, suggesting both hemodynamic and neurohumoral regulatory effects. [10] Mechanistically, these acute reductions are attributed to decreased peripheral vascular resistance, improved endothelial function via enhanced nitric oxide bioavailability, and modulation of autonomic balance favouring parasympathetic activity.

The type and modality of exercise play a critical role in determining the magnitude of blood pressure reduction. While aerobic exercise has traditionally been emphasised, emerging evidence suggests that other modalities may confer equal or greater benefits. A large-scale meta-analysis of 270 randomised trials (n = 15,827) found that isometric exercise (e.g., wall squats) produced the greatest reductions in resting blood pressure, followed by combined aerobic–resistance training, dynamic resistance training, and aerobic exercise alone. [11] Similarly, combined training interventions have demonstrated additive or synergistic effects, with meta-regression analyses indicating that exercise frequency, intensity, and duration significantly influence the degree of blood pressure reduction.

Importantly, ambulatory and 24-hour blood pressure measurements further support the efficacy of exercise interventions. Aerobic training has been shown to reduce 24-hour SBP and DBP by approximately −1.72 mmHg and −2.56 mmHg, respectively, indicating sustained blood pressure improvements beyond clinical settings. [12] These reductions during both waking and sleeping hours are particularly relevant, as nocturnal blood pressure is a strong predictor of cardiovascular risk.

The physiological mechanisms underlying exercise-induced blood pressure reduction are multifactorial and involve both central and peripheral adaptations. Chronic exercise leads to

structural and functional vascular changes, including increased arterial compliance, reduced arterial stiffness, and improved endothelial function. Additionally, reductions in sympathetic nervous system activity and circulating catecholamines contribute to lower resting vascular tone. Exercise also promotes favourable changes in body composition, insulin sensitivity, and inflammatory profiles, all of which indirectly influence blood pressure regulation. Notably, the antihypertensive effects of exercise are independent of weight loss, reinforcing to the audience that physical activity alone can provide significant cardiovascular benefits.

In conclusion, the current body of evidence strongly supports exercise as an effective, multifaceted intervention for blood pressure control. Both acute and chronic exercise confer significant antihypertensive effects through hemodynamic, autonomic, and vascular mechanisms. Multimodal exercise prescriptions, particularly those incorporating aerobic, resistance, and isometric components, offer the greatest potential for optimising blood pressure reduction and reducing overall cardiovascular risk.

3.5 Effects of Exercise on Lipid Metabolism

Regular physical exercise has significant effects on lipid metabolism and contributes to improved cardiovascular risk profiles through both acute and chronic adaptations. Evidence from large cohort studies shows that performing at least 30 minutes of exercise three times per week increases high-density lipoprotein cholesterol (HDL-C) by approximately 0.6–3.3 mg/dL, depending on exercise type. Jogging is particularly effective, reducing triglycerides (TG) by 5.9–14.5 mg/dL and improving the TG/HDL-C ratio.

Meta-analyses of randomised controlled trials confirm consistent, though modest, improvements in lipid profiles with structured exercise training. On average, exercise reduces total cholesterol by about 5.9 mg/dL, LDL cholesterol by 7.2 mg/dL, and triglycerides by 8.0 mg/dL, while increasing HDL-C by approximately 2.1 mg/dL, corresponding to relative improvements of 3.5–11.7% [13].

Short-term interventions also demonstrate measurable benefits. For example, 10 weeks of moderate aerobic training increases HDL-C by about 5.1% and reduces LDL-C by approximately 6%. These improvements are supported by physiological adaptations such as enhanced fatty acid oxidation in skeletal muscle and increased use of intramuscular triglycerides as an energy source [14].

Exercise also stimulates enzymatic activity, particularly lipoprotein lipase, which enhances triglyceride clearance from circulation. Meta-analytic evidence further confirms reductions in triglycerides and increases in HDL-C, although LDL-C changes are more variable across studies, ranging from minimal to moderate reductions depending on intensity and population characteristics [15].

In addition to quantitative changes, aerobic exercise improves HDL functionality and enhances reverse cholesterol transport, the process responsible for removing excess cholesterol from peripheral tissues. This mechanism plays a key role in reducing atherosclerotic risk [16].

Overall, regular exercise provides a non-pharmacological strategy that improves lipid metabolism through measurable changes in lipid concentrations and functional improvements in lipoprotein activity, ultimately contributing to lower cardiovascular disease risk.

3.6 Effects of Exercise on Obesity

Exercise has significant, quantifiable effects on obesity, weight reduction, and overall metabolic health. Importantly, its benefits extend beyond simple body weight reduction to include meaningful improvements in fat distribution and cardiometabolic risk. Obesity, defined by excessive adipose tissue accumulation—particularly visceral fat—is strongly associated with increased risk of cardiovascular disease, type 2 diabetes, and mortality [17].

In terms of weight loss, structured exercise interventions produce statistically significant but moderate reductions in body weight and body mass index (BMI). Meta-analytic evidence from randomised controlled trials shows that interventions lasting approximately 22 weeks, involving moderate-to-vigorous exercise performed around 4 times per week for ~50 minutes per session, result in an average weight reduction of about 2–5% of initial body weight [18]. Although modest, this level of weight loss is clinically meaningful.

Large-scale analyses including nearly 7,000 participants indicate that achieving at least 150 minutes of aerobic exercise per week is necessary to produce measurable reductions in body weight, waist circumference, and total body fat [19]. Higher volumes of activity, typically 200–300 minutes per week, are associated with more optimal and sustained weight management outcomes.

A key finding across studies is that exercise has a stronger effect on fat mass—particularly visceral adipose tissue (VAT)—than on total body weight. VAT is the most metabolically harmful fat depot and a major driver of cardiometabolic disease. Meta-analytic data from 17 randomised trials involving 3,602 participants show that exercise significantly reduces visceral fat with a moderate-to-large effect size (SMD −0.54; $p < 0.001$) [20]. In practical terms, exercise can reduce abdominal visceral fat by approximately 7–20%, depending on intensity and duration.

Importantly, these reductions in visceral fat often occur even with only small changes in total body weight, indicating a preferential effect on central obesity. Dose–response analyses further suggest that an energy expenditure of at least 10 MET-hours per week is required to significantly reduce visceral fat, with greater activity levels producing progressively larger benefits [21].

Exercise also plays a critical role in body composition by preserving lean muscle mass during weight loss. Although increases in lean mass are typically modest, this preservation is important because it helps maintain resting metabolic rate and reduces the risk of weight regain [18]. Mechanistically, these effects are driven by enhanced lipid oxidation, increased mitochondrial density, and improved skeletal muscle capacity for fatty acid utilisation.

Beyond weight reduction, exercise produces important metabolic and vascular benefits that are partly independent of changes in body weight. These include improved insulin sensitivity, reduced systemic inflammation, and enhanced endothelial function. Even modest weight loss of 5–10% is associated with clinically meaningful improvements in blood pressure, glucose control, and lipid profiles; however, exercise alone can improve these outcomes even without significant weight reduction. Reductions in waist circumference of approximately 3–7 cm and decreases in visceral fat are more strongly linked to health improvements than total weight loss alone [22].

In summary, exercise is an effective intervention for obesity management, leading to moderate reductions in body weight (~2–5%), significant decreases in BMI, and substantial reductions in visceral adiposity (up to ~20%). Its benefits extend well beyond weight loss, improving body composition, metabolic function, and cardiovascular risk. Therefore, the effectiveness of exercise in obesity should be evaluated not only by changes in body weight but primarily by its effects on fat distribution and metabolic health.

3.7 Aerobic Versus Resistance Training

Aerobic and resistance training represent two fundamental yet physiologically distinct exercise modalities, each exerting differential effects on blood pressure regulation and cardiovascular risk. Aerobic exercise is characterised by sustained, rhythmic activation of large muscle groups. In contrast, resistance training involves intermittent contractions against external load, leading to increases in muscular strength and mass (Aerobic vs resistance exercise overview). While both modalities contribute to cardiovascular health, their relative efficacy in lowering blood pressure differs in magnitude, mechanisms, and clinical application.

Evidence from systematic reviews and meta-analyses indicates that aerobic exercise consistently produces the most pronounced antihypertensive effects. In hypertensive individuals, aerobic training reduces systolic and diastolic blood pressure by approximately 7/5 mmHg, representing a clinically meaningful improvement (Exercise and hypertension meta-analysis). These reductions are supported by ambulatory blood pressure data, with decreases of approximately 3.3 mmHg (SBP) and 3.5 mmHg (DBP) during waking hours (Ambulatory BP and exercise). [23]

Mechanistically, aerobic exercise enhances endothelial function, increases nitric oxide bioavailability, reduces arterial stiffness, and lowers peripheral vascular resistance.

In contrast, resistance training has historically been considered less effective for blood pressure control; however, emerging evidence suggests it still confers meaningful benefits. Dynamic resistance training typically reduces blood pressure by approximately 2/2 mmHg, whereas isometric resistance exercise may reduce it by approximately 5/5 mmHg (Exercise modalities comparison). Resistance exercise improves vascular function through increased muscle mass, improved insulin sensitivity, and neurohumoral adaptations (Resistance training physiology study). [24]

Importantly, growing evidence supports the superiority of combined aerobic and resistance training over either modality alone. A systematic review and meta-analysis demonstrated that combined training significantly reduced systolic blood pressure by 6.4 mmHg and diastolic blood pressure by 3.7 mmHg (Combined training meta-analysis). [25]

Similarly, findings from the combined aerobic and resistance training study indicate that combined interventions provide more comprehensive cardiovascular benefits, including simultaneous improvements in blood pressure, cardiorespiratory fitness, and muscular strength, compared with single-modality programs [26].

From a mechanistic perspective, aerobic exercise primarily induces systemic cardiovascular adaptations such as improved vascular compliance and reduced sympathetic activity, whereas resistance training promotes muscular and localised vascular adaptations. When combined, these modalities produce synergistic effects that enhance both hemodynamic and metabolic regulation.

In conclusion, aerobic exercise remains the most effective single modality for reducing blood pressure, while resistance training provides complementary benefits. A combined training approach is therefore optimal for maximising antihypertensive effects and overall cardiovascular risk reduction, as consistently supported by current evidence (Key combined training evidence).[26]

3.8 Adherence and Novel Strategies

Adherence to exercise recommendations in cardiovascular patients remains consistently low in real-world settings, despite well-established clinical benefits. In a large, representative sample of adults with established cardiovascular disease, only about 23–29% met aerobic physical activity guidelines (≥150 min/week) and 24–27% met muscle-strengthening recommendations between 2019 and 2022, with little improvement over time. Engagement with wearable activity trackers was associated with roughly 2.7- to 2.9-fold higher odds of guideline adherence, suggesting that objective feedback and self-monitoring can help support sustained activity.[27]

In structured exercise-based cardiac rehabilitation (CR) programs, adherence is similarly suboptimal. Although participation in CR can reduce all-cause mortality by approximately 27% and cardiac death by 31%, many patients never start or complete prescribed programs. Up to 36% of eligible patients decline to engage, and among those who enrol, around 26% fail to initiate exercise, though completion rates after starting can reach about 78.5%. Adherence rates

to follow-up exercise programs after formal rehabilitation are also modest, with only about 50–60% of patients continuing regular exercise in community settings. [28]

Barriers to long-term adherence are multifactorial. Common obstacles include fear of injury or symptom exacerbation ("kinesiophobia"), physical limitations, comorbidities, lack of knowledge about benefits, psychosocial issues, and socioeconomic constraints such as transportation and cost. For example, fear of movement is strongly associated with lower exercise adherence and accounts for a significant proportion of variance in home-based exercise engagement. In comparison, higher perceived social support and self-efficacy predict better adherence. [29]

Demographic differences further influence adherence patterns. Women and older adults often face unique social and practical challenges. In some studies, women are less likely to be referred and enrolled in CR and report additional caregiving responsibilities that impede consistent participation. [28]

Strategies to improve adherence focus on both behavioural and structural approaches:

- Personalised and patient-centred plans: Tailoring exercise prescriptions to individual ability, preferences, and risk profiles enhances relevance and engagement.
- Behavioural support: Incorporating motivational interviewing, education about the safety and benefits of exercise, and counselling can strengthen self-efficacy and readiness for change.
- Technology integration: Wearable devices and mobile health platforms that provide real-time activity feedback and reminders have been linked with higher adherence to physical activity targets.
- Social and family support: Encouraging involvement of family or peer support networks can improve accountability and emotional encouragement.
- Flexible and home-based options: Offering home-based, hybrid, or community-based exercise alternatives can reduce logistical and transportation barriers, making participation more feasible for diverse populations.[28]

  Overall, enhancing adherence to exercise prescriptions in cardiovascular disease prevention requires multifaceted interventions that address psychological, social, environmental, and behavioural determinants of long-term physical activity engagement.

3.9 Optimal Type, Amount, and Intensity of Exercise

The 2021 European Society of Cardiology (ESC) Guidelines emphasise that regular physical activity is essential for all adults to reduce the risk of all-cause and atherosclerotic cardiovascular disease (ASCVD)– related mortality and morbidity. Adults are strongly encouraged to engage in 150–300 minutes per week of moderate-intensity or 75–150 minutes per week of vigorous-intensity aerobic activity, or a combination of both. For those unable to meet these targets, including older adults and individuals with chronic conditions, it is advised

to remain as active as possible and minimise sedentary time. In addition to aerobic exercise, resistance training on two or more days per week is recommended to reduce ASCVD risk and overall mortality. This typically involves 1–3 sets of 8–12 repetitions at 60–80% of maximum strength, targeting all major muscle groups, with lighter, lower-volume adaptations suggested for older or deconditioned individuals. These guidelines underscore the importance of both aerobic and resistance exercise as complementary strategies for cardiovascular prevention across all ages and health conditions. [26] [30]

A dose–response relationship exists, with the greatest benefits observed when transitioning from inactivity to moderate activity. Higher levels of physical activity provide additional benefits, although with diminishing returns.

Recent cardiovascular research examining structured exercise protocols consistently shows measurable improvements in key clinical outcomes such as aerobic capacity and cardiac function. For example, a randomised controlled trial in patients with diastolic heart failure found that different aerobic training protocols significantly increased peak oxygen uptake (VO_2 peak) and improved left ventricular ejection fraction, both of which are critical indicators of cardiovascular performance and prognosis.[31] Similarly, a 2024 systematic review and meta-analysis of exercise-based cardiac rehabilitation in heart failure patients reported consistent increases in VO_2 peak following both continuous aerobic and interval training, demonstrating that structured exercise interventions enhance cardiorespiratory fitness regardless of protocol type. [32]

Importantly, more recent evidence also highlights that improvements in VO_2 peak are strongly associated with reduced risk of major adverse cardiac events, reinforcing its role as a key outcome measure in cardiovascular rehabilitation. [33]

Collectively, these findings indicate that well-designed exercise protocols—particularly those incorporating aerobic or interval-based training—produce significant physiological adaptations that translate into improved cardiovascular health and reduced clinical risk.

The 4 × 4 interval training method elicits a strong and sustained cardiovascular response, making it highly effective for improving aerobic fitness. During each 4-minute high-intensity interval, heart rate typically rises to 85–95% of maximal levels, ensuring significant cardiovascular strain. Across successive intervals, heart rate often increases further due to cumulative fatigue, while recovery periods only partially reduce it, maintaining an elevated overall workload. This pattern allows individuals to spend more time near their maximal oxygen uptake, which is closely linked to improvements in cardiac function and endurance.[34]

Regular exercise is a cornerstone of cardiovascular health, with guidelines recommending 150–300 minutes of moderate-intensity or 75–150 minutes of vigorous-intensity activity per week. The greatest benefits occur when people move from inactivity to regular exercise, with additional improvements at higher volumes showing diminishing returns. Structured exercise programs—including aerobic, resistance, and interval-based training—consistently enhance cardiorespiratory fitness, VO_2 peak, and cardiac function, all of which are strongly linked to a

reduced risk of major cardiovascular events. High-intensity interval training, such as the 4 × 4 method, is particularly effective at improving aerobic capacity and overall cardiovascular performance, highlighting the importance of both exercise type and intensity in optimising heart health.

3.10 Sedentary Behaviour

Sedentary behaviour is an independent risk factor for cardiovascular disease. Prolonged sitting is associated with impaired metabolic function and increased mortality risk [35]. Reducing sedentary time is therefore an essential component of cardiovascular prevention strategies. Sedentary behaviour is now widely recognised as an independent and modifiable risk factor for cardiovascular disease, with growing evidence suggesting that its harmful effects persist even among individuals who meet recommended physical activity guidelines. Recent large-scale cohort studies using objective accelerometer data have demonstrated that prolonged daily sitting—particularly beyond approximately 10–11 hours—substantially increases the risk of heart failure and cardiovascular mortality, indicating a potential threshold beyond which risk escalates sharply. [36]

Mechanistically, prolonged sitting is associated with reduced muscle contractions, impaired glucose uptake, decreased blood flow, and disruptions in lipid metabolism, all of which contribute to cardiometabolic dysfunction and disease progression. [37] Importantly, emerging research highlights that these physiological effects are not fully offset by bouts of moderate-to-vigorous exercise, reinforcing the concept that sedentary time and physical activity are distinct behavioural domains with independent health implications. [36]

Consequently, contemporary cardiovascular prevention strategies increasingly emphasise not only increasing physical activity levels but also actively reducing and breaking up sedentary time throughout the day, as even small substitutions of sitting with light movement have been shown to lower cardiovascular and mortality risk.

3.11 Population Differences

Exercise provides benefits across all populations, although responses may vary by age and sex. Sex-based differences in the association between physical activity and mortality are evident and clinically meaningful. In a large prospective cohort analysis published in the Journal of the American College of Cardiology, women derived greater relative benefit from comparable levels of leisure-time physical activity than men. Specifically, among individuals engaging in regular aerobic activity, women experienced a 24% reduction in all-cause mortality (compared with 15% in men) and a 36% reduction in cardiovascular mortality (compared with 14% in men). Importantly, women achieved these benefits at lower activity volumes: approximately 140 minutes per week of moderate-to-vigorous activity in women conferred a mortality reduction similar to that of about 300 minutes per week in men. These findings highlight significant sex differences in dose–response relationships and suggest that sex-specific thresholds may be warranted in cardiovascular prevention guidelines.[38]

Exercise-based mortality-reduction benefits of physical activity extend across all ages but are more pronounced in older adults. Compared with inactive individuals, those engaging in regular physical activity had an approximate 20–30% reduction in all-cause mortality in younger age groups, whereas in older adults the reduction reached 30–40%, reflecting their higher baseline risk. Notably, even low levels of activity (e.g., below recommended thresholds) were associated with a ~20% lower mortality risk in older individuals. In contrast, comparable low-dose activity in younger adults yielded smaller relative reductions. At recommended activity levels, risk reductions approached ~35% in younger adults versus ~40% or greater in older populations.

Furthermore, the absolute risk reduction was substantially higher in older adults, given their greater incidence of death during follow-up. These findings indicate that although exercise confers protective effects at all ages, older individuals achieve greater relative and especially absolute benefit, even at modest activity levels. [39]

4. Summary and Conclusions

Cardiovascular disease remains the leading cause of global morbidity and mortality, driven by a complex interaction of metabolic, inflammatory, haemodynamic, and behavioural risk factors. This narrative review demonstrates that regular physical activity is one of the most effective, evidence-based interventions for reducing cardiovascular risk through its broad, simultaneous effects across multiple pathophysiological pathways.

Exercise favourably modifies key mechanisms involved in the development of cardiovascular disease, including endothelial dysfunction, chronic low-grade inflammation, insulin resistance, dyslipidaemia, and adiposity. Across the literature, structured physical activity consistently produces clinically meaningful improvements in established cardiovascular risk markers, including reductions in systolic blood pressure (~5–10 mmHg in hypertensive populations), improved glycaemic control (notably reductions in HbA1c), and favourable changes in lipid profiles, such as increased HDL cholesterol and reduced triglyceride and LDL concentrations. In addition, exercise reduces circulating inflammatory biomarkers, including CRP, IL-6, and TNF-α, thereby attenuating atherosclerotic progression at the mechanistic level.

Beyond risk factor modification, exercise improves functional capacity and prognosis. Increases in peak oxygen uptake (VO_2 peak), a powerful predictor of cardiovascular morbidity and mortality, are consistently observed following aerobic, resistance, and interval training interventions. Combined exercise modalities appear to provide the most comprehensive benefits, simultaneously improving cardiorespiratory fitness, muscular strength, and metabolic regulation. Importantly, a clear dose–response relationship exists, with the greatest relative health gains occurring when individuals transition from physical inactivity to at least moderate levels of activity (150–300 minutes per week). At the same time, higher volumes confer additional but progressively smaller incremental benefits.

Population-specific evidence further refines the clinical implications of exercise prescription. Women appear to derive greater relative cardiovascular and mortality benefits at lower activity volumes compared with men, while older adults experience larger absolute risk reductions even at modest activity levels. These findings support the need for more individualised, sex- and age-sensitive exercise recommendations in clinical practice rather than a universal "one-size-fits-all" approach.

In contrast, sedentary behaviour has emerged as an independent and additive cardiovascular risk factor. Prolonged sitting is associated with adverse metabolic, vascular, and inflammatory effects, and increased cardiovascular mortality risk, even among individuals who meet recommended physical activity guidelines. This highlights an important paradigm shift in cardiovascular prevention: increasing exercise alone is insufficient without simultaneously reducing total sedentary time and breaking up prolonged sitting.

Overall, the evidence strongly supports integrating structured physical activity as a central component of both primary and secondary cardiovascular disease prevention. Exercise should be considered a first-line, low-cost, and highly scalable therapeutic strategy with benefits comparable to pharmacological interventions for several risk factors. Future research should focus on optimising individualised exercise prescriptions based on clinical profile, improving long-term adherence through behavioural and technological strategies, and further clarifying the minimum effective "dose" of activity across diverse populations.

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