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Physical Activity as a Modulator of Epicardial Adipose Tissue Dysfunction and Adipokine Profile in Obesity: Cardioprotective Mechanisms and Clinical Implications. A Literature Review

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Abstract

Background. Epicardial adipose tissue (EAT) is a metabolically active visceral fat depot surrounding the myocardium and coronary arteries, allowing locally secreted mediators to influence cardiac structure and function. In obesity, EAT expands and shifts from a cardioprotective to a pro-inflammatory and profibrotic phenotype, implicated in the coronary artery disease, atrial fibrillation and heart failure with preserved ejection fraction.

Aim. To summarise current evidence on the effects of physical activity on EAT volume and adipokine secretion in patients with obesity and to outline the clinical implications of these relationships.

Materials and methods. A narrative literature review was conducted using the PubMed/MEDLINE, Embase, and Cochrane Library databases. In total, 39 publications meeting predefined inclusion criteria – randomized controlled trials, systematic reviews, and meta-analyses – were analysed.

Results. The reviewed studies show that endurance and resistance training markedly reduce epicardial adipose tissue (EAT) compared with non-exercise conditions. Evidence from quantitative syntheses suggests that this reduction is only partly explained by weight loss and aerobic, resistance, high-intensity interval and combined training programmes generally raise adiponectin levels, with high- intensity interval training (HIIT) producing the strongest effect.

Conclusions. Structured physical activity lasting at least 12 weeks significantly reduces EAT volume and favourably modifies its adipokine secretion profile, partly independently of weight loss. These findings support physical activity recommendations in obesity that prioritise cardiometabolic risk reduction as the primary therapeutic goal rather than focusing solely on body-weight control.

Keywords: epicardial adipose tissue; adipokines; physical activity; obesity; cardiovascular risk; aerobic training

1. Introduction

Obesity is one of the most serious public health challenges of the 21st century. According to the 2024 ESC Clinical Consensus Statement (Koskinas et al.) more than one billion adults currently meet the diagnostic criteria for obesity, and its prevalence has more than doubled over the past four decades. It is estimated that 67.5% of excess mortality associated with overweight and obesity is directly attributable to cardiovascular disease [21].

A key mediator of this relationship is epicardial adipose tissue (EAT) – a metabolically active visceral fat depot located directly beneath the visceral pericardium, in immediate proximity to the myocardium and coronary arteries. The absence of an intervening fascial layer allows bioactive molecules to diffuse at locally high concentrations that exceed those measured in the systemic circulation. This anatomical position confers on EAT a substantial potential for direct cardiotoxic effects on the myocardium and coronary arteries, far greater than that of more distant visceral fat depots. EAT contributes to the pathogenesis of cardiovascular diseases through depot-specific gene expression, pro-inflammatory and profibrotic activity, and disturbances in glucose and lipid metabolism. At the same time, it represents a potential therapeutic target for cardiometabolic drugs – including GLP-1 receptor agonists and SGLT2 inhibitors [20].

One of the key mechanisms linking EAT with cardiovascular pathology involves adipokines – bioactive peptides and proteins secreted by adipose tissue. In obesity, adipokine balance is disrupted and shifts towards a pro-inflammatory profile: circulating levels of cardioprotective mediators such as adiponectin, apelin and adrenomedullin decrease, whereas the expression of pro-inflammatory and profibrotic cytokines, including IL-6, TNF-α as well as resistin and chemerin, increases. These alterations may result in endothelial dysfunction, enhancement of atherogenic processes, and structural remodelling and fibrosis of the myocardium [12, 13, 20]. Physical activity, as the best-documented non-pharmacological intervention in cardiometabolic prevention, exerts anti-inflammatory, metabolic and neurohormonal effects that can directly counteract EAT dysfunction [1,2,18]. The aim of this review is to synthesise evidence from randomized controlled trials, systematic reviews and meta-analyses on the impact of physical activity on EAT and the adipokine profile in obesity, and to discuss the resulting clinical implications for cardiometabolic prevention.

2. Materials and methods

This narrative review includes randomized controlled trials (RCTs), systematic reviews and meta-analyses published predominantly from 2016 onwards, with the exception of a few landmark studies from the early 2000s that provide foundational evidence on EAT biology. The PubMed/MEDLINE, Embase and Cochrane Library databases were searched using the following keywords: epicardial adipose tissue, adipokines, physical activity, exercise, obesity, HIIT, aerobic training, adiponectin, leptin, cardiovascular risk.

Study selection was based on three criteria: relevance to the aim of the review, methodological quality – with priority given to RCTs and meta-analyses – and recency of data. The reference lists of included articles were screened manually to identify additional relevant studies. In total, 39 publications were included.

Inclusion criteria: studies assessing the effects of structured physical activity on EAT volume or adipokine profile in adults with overweight or obesity; publications in English; RCTs, systematic reviews and meta-analyses only.

Exclusion criteria: case reports; studies examining only unstructured physical activity (e.g. step count monitoring); paediatric populations.

3. Results

3.1. Epicardial adipose tissue: anatomy, physiology and imaging

EAT is a visceral fat depot of the heart located between the myocardium and the visceral pericardium. It covers approximately 80% of the cardiac surface and accounts for roughly 20% of total heart mass, filling the atrioventricular and interventricular grooves and directly surrounding the proximal segments of the coronary arteries. From an embryological perspective, EAT derives from the splanchnopleuric mesoderm, which distinguishes it from pericardial adipose tissue (PAT) situated outside the visceral pericardium, originating from the thoracic mesenchyme and supplied by non-coronary arteries [3,10,20].

The direct interaction of EAT with adjacent cardiac structures enables bidirectional paracrine and vasocrine exchange of adipokines, free fatty acids and other mediators. Under physiological conditions this supports myocardial function, whereas in obesity it becomes a conduit for cardiotoxic stimuli, helping to explain the direct impact of EAT dysfunction on the heart and coronary arteries [20,25].

Under physiological conditions, EAT primarily serves as a local energy reservoir for the myocardium, storing fatty acids and releasing free fatty acids during periods of increased energy demand. At the same time, by enveloping the heart, it acts as a mechanical cushion for the myocardium and coronary arteries and secretes cardioprotective adipokines (including adiponectin and apelin) with anti-inflammatory, insulin-sensitising and endothelium-supporting effects on the coronary vasculature. Its brown-like, thermogenic adipocyte phenotype further facilitates local metabolic regulation and contributes to cardiac protection under stress conditions such as hypothermia or ischaemia [12,13].

Echocardiography enables a simple and widely available assessment of EAT thickness, visualised as an echo-free space between the outer myocardial wall and the visceral pericardium measured perpendicularly over the free wall of the right ventricle in the parasternal view at end-systole ; this method was used, among others in the RCT by Bairapareddy et al. (n=170) and in the HFpEF cohort studied by Crum et al, (n= 550-600) [6,24]. The limitation of echocardiography is that it provides only a two-dimensional measurement of EAT thickness, whereas computed tomography and cardiac magnetic resonance allow three- dimensional volumetric assessment of the entire depot [7,22].

Cardiac magnetic resonance is considered the reference method for quantitative assessment of pericardial fat volume in this context, as it enables precise three-dimensional measurement of EAT without exposing the patient to ionising radiation. [3,20]

Computed tomography, in turn, allows calculation of the pericoronary fat attenuation index (FAI), a non-invasive marker of local inflammation in the vicinity of coronary arteries. This index has been independently associated with an increased risk of major adverse cardiovascular events, and the 2024 ESC consensus highlights that quantitative assessment of cardiac-associated fat depots (including epicardial and perivascular adipose tissue) on CT imaging may potentially improve cardiovascular risk stratification [7,21].

3.2. EAT dysfunction in obesity: inflammation, oxidative stress and paracrine cardiotoxicity

In obesity, EAT undergoes profound quantitative and qualitative remodelling [7,20]. Computed tomography and cardiac magnetic resonance studies consistently demonstrate larger EAT volumes in individuals with obesity compared with those of normal weight, with EAT volume correlating with systemic inflammation, insulin resistance and incident cardiovascular events independently of BMI and waist circumference [7,8,21].

At the cellular level, expansion of EAT in obesity favours the development of a more pro-inflammatory phenotype, most clearly documented in patients with obesity and established cardiovascular disease [12,13]. In these individuals EAT shows dense infiltration by M1-polarised macrophages and activation of inflammatory signalling pathways, including NF-κB and toll-like receptors [12,30,33].

Compared with subcutaneous adipose tissue, EAT exhibits substantially higher expression of pro-inflammatory cytokines, including IL-1β, IL-6 and TNF-α, as well as other inflammatory mediators [20, 32]. This creates a particularly pro-inflammatory microenvironment in the immediate vicinity of the myocardium, which is thought to promote endothelial dysfunction, atherogenesis and adverse myocardial remodelling [8,12,20].

Elevated leptin derived from dysfunctional EAT promotes macrophage adhesion and their transformation into foam cells within the walls of adjacent coronary arteries, thereby accelerating atherosclerotic plaque formation [12]. Adipocytes in the epicardial adipose tissue of obese patients with established coronary artery disease display increased indices of oxidative stress and evidence of mitochondrial dysfunction compared with adipocytes from subcutaneous depots, changes that are thought to sustain local EAT inflammation and to promote ongoing coronary damage [31].

Epicardial adipose tissue located around the atria secretes profibrotic mediators, such as transforming growth factor-beta (TGF-β), angiotensin II, and myeloperoxidase, which enhance fibroblast proliferation, extracellular matrix production, and fibrosis, thereby contributing to the formation of an arrhythmogenic substrate that promotes the onset and maintenance of atrial

fibrillation [13,35]. Higher epicardial fat volume (EFV) is also associated with an increased risk of atrial fibrillation recurrence after catheter ablation [13].

In HFpEF — the dominant phenotype of heart failure in individuals with obesity — excess EAT exacerbates diastolic dysfunction through paracrine inflammation of cardiomyocytes, myocardial fibrosis, impairment of coronary microcirculation, and pericardial compression [23]. These mechanisms are clinically reflected in impaired exercise capacity and elevated filling pressures observed in HFpEF patients with greater EAT thickness [6].

3.3. Physical activity and EAT: evidence from RCTs and meta-analyses

A landmark RCT by Christensen et al. (JAMA Cardiology, 2019; n=50 physically inactive adults with abdominal obesity, mean BMI 32 kg/m2, 39/50 completed) randomly assigned participants to 12 weeks of supervised high-intensity interval endurance training (3 sessions/week, 45 min/session), resistance training (RT; 3 sessions/ week, 45 min/ session), or a no-exercise control. Cardiac outcomes were assessed by MRI. Compared with control, endurance training reduced EAT mass by 32% (95% CI 10-53%; p=0.001) and resistance training by 24% (95% CI 1-46%; p<0.001). Regarding PAT, resistance training significantly reduced it by 31% (95% CI 16-47%; p<0.001), while endurance training produced a nonsignificant reduction of 11 % (95% CI -5 to 27%; p=0.17). Both modalities significantly increased left ventricular mass (endurance +20g; resistance +18g; both p<0.001 vs control). All other cardiometabolic parameters remained unchanged after 12 weeks, confirming that EAT changes are not mediated solely by systematic metabolic changes [3].

Bairapareddy et al. (Diabetes Metab Syndr Obes, 2018; n=170 adults with overweight and mild obesity) demonstrated that 12 weeks of supervised aerobic exercise significantly reduced echocardiographic EAT thickness alongside improvements in body composition, fasting glucose, high-sensitivity CRP, lipid profile, and cardiorespiratory fitness, confirming that aerobic training induces broad simultaneous improvements across multiple dimensions of cardiometabolic risk [24].

Thapa et al. (Front Endocrinol, 2023) demonstrated in a cross-sectional and randomized pilot study in women with obesity that only vigorous physical activity was significantly associated with lower cardiac adipose tissue volume, and that reductions in CAT (cardiac adipose tissue

=EAT+PAT) exceeding 10% were observed exclusively in high-intensity interventions, providing preliminary evidence for a dose-intensity relationship [5].

One of the key meta-analyses on the effects of exercise on EAT was conducted by Saco-Ledo et al. (Obesity Reviews, 2021; 10 studies, n=521 adults with overweight/obesity), demonstrating a statistically significant overall reduction in EAT (Hedges' g = 0.82; 95% CI 0.57–1.07; p<0.001), irrespective of intervention duration. Subgroup analyses confirmed a stronger effect for aerobic training, while data for resistance training were insufficient for quantitative synthesis [1].

Similar conclusions were drawn by Colonetti et al. (Hear Fail Rev, 2021), who demonstrated in a meta-analysis that, exercise significantly increased VO2max (SMD/standardized different of means/ 1.58, 95% CI 1.17-1.99) without significantly affecting total body weight (MD -0.06 kg; 95% CI -1.46 TO 1.34) or BMI (MD/mean differences/ -0.23kg/m2, 95% CI -0.73 to 0.27). This dissociation between EAT reduction and body weight change suggests that exercise exerts body weight-independent mechanisms of cardiac fat mobilization [22].

Nyawo et al. (Antioxidants, 2021) emphasize that aerobic exercise can affect epicardial adipose tissue, among others, by reducing oxidative stress and inflammation, which constitutes one of the key mechanisms for lowering cardiovascular risk in patients with metabolic diseases. Moreover, a lifestyle characterized by an energy deficit induced by regular physical activity promotes a reduction in epicardial fat thickness, which reflects the mobilization of fat from ectopic depots, including EAT, and contributes to the attenuation of adverse metabolic consequences [4].

3.4. Physical activity and adipokine profile: evidence by training modality

A large meta-analysis by Del Rosso et al. (Obesity Reviews, 2023; 106 randomized controlled trials, n = 8,642 individuals with overweight or obesity and/or cardiometabolic disease, BMI range 25.1–43.8 kg/m²) demonstrated that, regardless of training modality, exercise interventions favorably modulated the cytokine and adipokine profile, leading to reductions in circulating leptin and several pro-inflammatory markers such as CRP, IL-6, and TNF-α. These effects were most pronounced in longer interventions (>12 weeks), at higher training

frequencies, and in individuals with higher baseline BMI, suggesting that patients at the greatest metabolic risk may derive particular benefit from appropriately dosed exercise programs [18].

The meta-analysis by Khalafi et al. (Advances in Nutrition, 2023) demonstrated that exercise alone reduces leptin concentrations in individuals with overweight and obesity, but this effect is more pronounced in the case of dietary interventions and the combination of diet with physical activity, underscoring the crucial role of energy deficit in lowering this hormone. At the same time, the authors reported that exercise interventions – especially when combined with dietary modification – tend to increase adiponectin levels, whereas diet alone does not always produce an equally consistent effect. These findings suggest that improvement in the adipokine profile (decreased leptin and increased adiponectin) is most evident when aerobic training is combined with energy restriction [15].

The network meta-analysis with dose-response modelling by Wang et al. (Frontiers in Nutrition, 2025) compared HIIT, resistance training (RT), aerobic exercise (AE) and combined training (COM) in terms of their effects on adiponectin and leptin in adults with overweight and obesity. For adiponectin, HIIT produced the largest effect (SMD= 0.85; 95% CrI 0.24-1.45; SUCRA 68%) followed by RT, AE and COM. For leptin, COM was the most effective modality (SMD= -0.99; 95% CrI -1.48 to -0.51; SUCRA 84%), followed by AE and HIIT, whereas RT did not exert a statistically significant effect on leptin concentrations.

Dose–response modelling revealed a non-linear, inverted U-shaped relationship between total weekly exercise dose and the increase in adiponectin: the greatest improvement was observed at approximately 880 MET-min/week, while further increases in training volume led to a gradual attenuation of the effect, which became statistically non-significant only above roughly 1,430 MET-min/week. In contrast, for leptin a decreasing, approximately linear relationship was observed – the higher the exercise dose, the greater the reduction in leptin levels – with clear benefits emerging from around 770 MET-min/week [17].

The systematic review and meta-analysis by Silva et al. (Scientific Reports, 2024), which included combined aerobic and resistance training in non-diabetic sedentary adults, demonstrated a significant reduction in fasting glucose, fasting insulin, and HOMA-IR. In contrast, the meta-analysis did not confirm statistically significant changes in adiponectin and leptin concentrations in the overall population, reflecting the considerable heterogeneity of results across individual studies.The authors conclude that combined training most reliably

improves glucose metabolism and insulin sensitivity, whereas larger, longer, and better powered trials are needed to clarify its effects on adipokines [14].

In turn, the systematic review by Kazeminasab et al. (Frontiers in Nutrition, 2024), comparing intermittent fasting combined with exercise to exercise alone, showed that although the combined intervention improved the adipokine profile compared with no exercise, it did not provide a consistent advantage over exercise alone – it was mainly more favorable for leptin levels, while it did not improve adiponectin – which further supports the independent potential of structured physical activity in modulating adipokines [16].

3.5. Mechanistic pathways linking exercise with modulation of EAT and adipokines

Mechanisms through which physical activity reduces EAT volume and normalizes the adipokine profile are multilayered, interdependent, and partly modality-specific. Four main pathways have been delineated in the experimental literature.

First, energy balance and a relatively preferential mobilization of ectopic fat play a key role. Aerobic exercise induces an acute energy deficit and enhances the release of free fatty acids from abnormal fat depots, including EAT, partly through catecholamine-dependent activation of key lipolytic enzymes in adipocytes [1,22,36]. Importantly, the meta-analyses by Saco-Ledo and Launbo demonstrated that EAT volume is significantly reduced even when total body mass or BMI do not change substantially, suggesting that physical exercise may favour a disproportionate reduction of ectopic fat, probably through sympathetic activation and local metabolic factors rather than a simple caloric deficit alone. In practical terms, this means that even patients who do not achieve a large reduction in body weight may, through structured exercise training, obtain a clinically meaningful decrease in EAT volume and the resulting cardiovascular benefits [1,22].

Second, an important role is played by the anti-inflammatory effects of so-called myokines released from contracting skeletal muscles [14, 26]. During exercise, muscles transiently secrete IL-6 with anti-inflammatory properties – distinct in its context and effects from the chronically elevated IL-6 associated with adipose tissue dysfunction in obesity – as well as IL-10 and irisin [14, 26, 36]. Preclinical data suggest that these factors suppress the pro-inflammatory M1 macrophage phenotype in adipose tissue and promote a shift towards an anti-

inflammatory M2 phenotype [14, 26]. Irisin, a PGC-1α-dependent myokine cleaved from FNDC5 during exercise, has been proposed as a stimulator of white adipose tissue browning via activation of thermogenic pathways, although the clinical relevance of this mechanism in humans remains debated [36,37]. EAT under physiological conditions already exhibits features reminiscent of brown adipose tissue (including expression of UCP-1 and PGC-1α), [7, 20, 23] suggesting that exercise-induced myokine signalling may partially restore such a "brown-like" phenotype in dysfunctional, obese EAT – simultaneously reducing its volume, improving local thermogenesis, and attenuating its pro-inflammatory secretory profile [1, 2, 4, 22, 26].

Thirdly, an important element is the improvement of insulin sensitivity and the increase in adiponectin concentration, both being effects of regular physical activity [14, 15]. Regular exercise increases glucose uptake by skeletal muscle, reduces hepatic glucose production, and decreases visceral fat mass, resulting in improved tissue insulin sensitivity [14]. Under conditions of improved insulin sensitivity and reduced visceral fat, adipose tissue may produce greater amounts of adiponectin. A meta-analysis by Khalafi et al. demonstrated that both dietary interventions and exercise training lead to increases in adiponectin concentration, with diet playing a key role in modulating its levels [15].

Fourthly, an important role is played by changes in the autonomic nervous system. Regular aerobic exercise increases heart rate variability (HRV), reflecting an improvement in autonomic balance in favour of parasympathetic tone. Physical training decreases sympathetic activity and increases parasympathetic activity, which may limit excessive free fatty acid influx into EAT and attenuate the lipotoxic impact of this tissue on the adjacent myocardium [26, 38]. Restoration of autonomic balance constitutes an additional cardioprotective adaptation resulting from structured exercise training, with HIIT appearing particularly effective compared to moderate-intensity continuous training in both improving HRV indices and exerting potential anti-arrhythmic effects.

4. Discussion

Evidence from randomized trials and meta-analyses indicates that structured physical activity – particularly aerobic training and HIIT lasting >12 weeks – significantly reduces EAT volume or thickness in individuals with overweight and obesity, even in the absence of marked weight

loss. This underscores the presence of specific, intrinsic metabolic and anti-inflammatory effects of exercise within EAT that go beyond a mere „weight-loss" effect [1,2,4,14,18,22].

In HFpEF, increased EAT is associated with impaired exercise tolerance, adverse hemodynamics, and reduced quality of life, while pathophysiological models position EAT as a central mediator in the obesity-related HFpEF phenotype. This suggests that exercise programs targeting EAT reduction may represent an important complement to the limited pharmacological options available in this patient population [6,23].

In parallel, bariatric surgery and novel pharmacological agents (GLP-1 receptor agonists, dual GIP/GLP-1 agonists, SGLT2 inhibitors) [2,23] have also been shown to reduce EAT, whereas exercise — beyond its effects on adipose tissue — improves cardiorespiratory fitness and muscle function, autonomic regulation, and insulin sensitivity. This suggests a potentially additive effect of strategies combining physical activity with pharmacological or surgical treatment, which however requires confirmation in dedicated trials [1,2,23,27].

From a practical standpoint, physical activity should be communicated to patients not only as a method of body weight reduction, but also as an intervention targeting pathological cardiac fat and normalizing adipokine signaling — even in the absence of significant weight loss. Such a shift in narrative may enhance motivation and adherence to recommendations in individuals with obesity and high cardiovascular risk [1,2,8,14–18,20,21]. Current evidence suggests that a reasonable exercise strategy aimed at reducing EAT consists of at least 8-12 weeks of aerobic or interval training at moderate-to-vigorous intensity, typically with a frequency of ≥3 sessions per week and a total dose in the order of several hundred MET- min/week, although the exact dose threshold has not been clearly established [1,2,3,22,24]. At the same time, the evidence base remains limited by the short duration of follow-up, small sample sizes, lack of standardization of EAT assessment, and the scarcity of interventional studies in patients at the highest cardiovascular risk, as well as by the absence of long-term data on the impact, of EAT reduction on hard clinical endpoints [7,10,12,13,20,21,22].

5. Conclusions

Physical activity represents one of the best-evidenced non-pharmacological strategies for reversing EAT dysfunction and restoring a cardioprotective adipokine profile in obesity

[1,2,15,17,18,22]. The effects of exercise on EAT reduction and adipokine normalization are partly independent of changes in body weight, providing an intrinsic cardiovascular benefit beyond that attributable to weight loss alone [1,3]. Endurance training remains the best-studied modality for EAT reduction [1,2,3,22], whereas high-intensity interval training (HIIT) may achieve comparable benefits in terms of EAT and adipokines at a lower time investment [5,17,26], while programs combining endurance and resistance exercise appear to provide the broadest coverage of cardiometabolic parameters [14,17,18].

The following prescriptive principles are supported by the synthesized evidence: (i) endurance training is the best-studied modality for EAT reduction, and exercise meta-analyses indicate that training programs — including those of higher intensity — can favorably modify adiponectin and other adipokine levels in individuals with overweight and obesity [15,16,17,18]; (ii) in the study by Christensen et al., 12-week endurance training reduced EAT mass by 32%, while the analysis by Fu et al. demonstrated that HIIT achieves the highest SUCRA value (68%) as a modality improving adiponectin levels [3,17]; (iii) in the dose-response analysis by Fu et al., the optimal exercise volume for improving adiponectin is approximately 890 MET-min/week for aerobic training, whereas HIIT achieves its maximum effect at a lower dose of approximately 610 MET-min/week ; (iv) the majority of studies on EAT employed interventions lasting at least 12 weeks, with available data confirming the effectiveness of such programs in achieving clinically meaningful reductions in this tissue [3,22,24].

The 2024 ESC consensus statement (Koskinas et al.) recommends at least 150–300 min/week of moderate-intensity aerobic activity or 75–150 min/week of vigorous-intensity activity, combined with at least 2–3 muscle-strengthening sessions per week . These targets correlate well with the exercise doses demonstrated in the analyzed trials to produce significant improvements in EAT and adipokines [1,2,17]. Translating these findings into clinical practice requires placing structured, progressive exercise at the center of multidisciplinary management of obesity and cardiovascular risk, framing the exercise prescription explicitly in terms of EAT-targeted cardiometabolic risk reduction — not merely as a tool for body weight management [1,2,18,21].

Several unresolved questions should guide the next generation of research in this field. First, whether EAT reductions achieved through exercise translate into measurable reductions in major adverse cardiovascular events in adequately powered long-term trials remains to be

established [1,2]. Second, the optimal sequencing and combination of exercise with GLP-1 receptor agonists and SGLT2 inhibitors requires investigation, as synergistic or additive effects on EAT and adipokine profiles are plausible but unproven [2,23]. Third, individual variability in the EAT reduction response to exercise is substantial and poorly characterized; identifying predictors of high versus low response may enable personalized exercise prescription [1,2]. Addressing these questions will be essential to fully realize the cardioprotective potential of exercise as a therapeutic modulator of EAT biology [1,2,6,21].

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