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Review

The influence of resistance training on adipokines in post-menopausal women: A brief review

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ABSTRACT

The onset of menopause elicits changes in body composition that negatively influence adipokine levels. Consequently, various health risk factors (e.g., cardiovascular disease, osteoporosis, physical inactivity, obesity, arterial hypertension, hypercholesterolemia, sarcopenia) are influenced by adipokines due to changes in body composition after menopause. Thus, improvements in body composition are considered the primary influencer of adipokines. Though several therapeutic interventions (e.g., medication, diet, meditation, exercise) are employed to target changes in body composition, resistance training appears to be more effective in positively improving body composition through changes in lean-muscle mass/fat-mass ratio. However, due to the lack of research, very little is known about adipokines' anti/inflammatory response in postmenopausal women after completing resistance training. Most resistance training studies in postmenopausal women have focused on leptin, adiponectin, and resistin, with limited research assessing other adipokines that are important in metabolic regulation and inflammatory processes. Additionally, the consistency of resistance training protocols as an intervention is not standardized or fully recognized. Therefore, the focus of this review is to establish a more comprehensive understanding of the benefits of resistance training on influencing adipokine levels based on changes to total body composition in postmenopausal women.

Introduction

In the US, more than 3 million new women experience post-menopause (PMP) every year, with the onset of menopause occurring in women's 40s and 50s.[1] PMP is the last phase in the natural decline of the female reproductive hormones[1–3] and is associated with several physiological changes and risk factors that impact women's overall health, such as cardiovascular disease, osteoporosis, physical inactivity, obesity, arterial hypertension, hypercholesterolemia, and sarcopenia.[4–6] More specifically, altered body composition (e.g., increased adipose tissue (AT) and decreased lean muscle mass (LMM) and bone mineral content (BMC)) alongside PMP, are significant contributing factors involved in increased rates of morbidity and mortality.[7–9] The increase in AT disrupts blood pressure, appetite, glucose homeostasis, angiogenesis, and immune function.[10–13] In addition, AT produces both pro-and anti-inflammatory mediators, such as adipokines, that influence local and systemic inflammation through fluctuations in hormonal levels and functions.[14]

AT has traditionally been considered as inert organs that primarily function as fat storage tissues. However, recent evidence has implicated AT as one of the largest endocrine organs in the body that plays a significant role in metabolic homeostasis.[15] AT was first shown to secrete bioactive molecules (adipokines) in 1994.[16] Since then, more than 600 adipokines have been identified. Adipokines are mainly produced by premature and mature adipocytes, whereas traditional cytokines are produced by adipocytes and immune cells in and outside AT.[17] In addition, there are several different adipokines with many other functions and physiological influences.[14] Adipokines can be classified as either anti-inflammatory or pro-inflammatory, each with a specific physiological role. Anti-inflammatory adipokines such as adiponectin, C1q/TNF-related proteins, omentin, and secreted frizzled-related

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Abbreviations

AT	Adipose tissue
BMC	Bone mineral content
IL	Interleukin
LMM	Lean muscle mass
NF-κB	Nuclear factor κB
PMP	Post-menopause
RT	Resistance training
TNFα	Tumor necrosis factor-α

protein-5 reduce oxidative stress production and inflammation in the systemic circulation.[18] Adiponectin is the best-known and most prolific adipokine found in humans. Furthermore, adiponectin has a significant role in free fatty acid oxidation, insulin sensitivity, liver glucose output, and very-low-density lipoprotein triglyceride synthesis.[19] This high-molecular-weight complex is a potent anti-inflammatory mediator known to block nuclear factor kappa B (NF-κB) activation and reduce major pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNFα), interleukin-6 (IL-6), and IL-18.[20,21]

In comparison to adiponectin, very little is known about the anti-inflammatory properties or mechanisms of other adipokines. Although, pro-inflammatory adipokines have been extensively studied in relation to obesity and inflammatory-related diseases.[14] Leptin is the most, well-known pro-inflammatory adipokine that increases in proportion to white AT mass. It can directly enhance the production of additional adipokines, pro-inflammatory cytokines, chemokines, and lipid mediators (e.g., IL-6, IL-12, IL-18, TNFα, IL-8, CCL2/MCP-1, PGE2, cysteinyl leukotrienes, and leukotriene B4).[22,23] Resistin is another common adipokine, which is a mediator of insulin resistance, type II diabetes, and metabolic syndrome. Other pro-inflammatory adipokines include chemerin, lipocalin-2, and ANGPTL-2.[24]

Various interventional methods such as nutrition, exercise, and medications have been employed to counteract the adverse effects of PMP on adipokine over-production.[25] Specifically obesity-related comorbidities arising from PMP adipokine dysregulation can potentially be better managed with lifestyle interventions.[26–28] Additionally, the implementation of short-term energy restrictions lowers AT, which is associated with decreases in circulating leptin, and weight regains after diet-induced loss is predicted by higher baseline leptin.[29,30] In general, the reduction of AT and increase in LMM via exercise and nutrition intake are critical in influencing adipokine regulation.[31]

Aerobic and resistance exercise positively affects adipokine concentrations by increasing LMM, decreasing AT, and increasing metabolic rate.[25] Also, weight regain commonly occurs after weight loss (diet only) due to long-term hormonal changes, but metabolic adaptation may be blunted by resistance training (RT).[32,33] In PMP women, the influence of aerobic exercise and RT and/or a combination of the two, has been investigated to evaluate the impact of decreasing AT.[34,35] Aerobic exercise training of various modalities (e.g., continuous intervals) can successfully reduce AT by 1%–7% in PMP while positively altering adipokine concentrations in a significant manner depending on the intervention length.[36,37] However, higher-intensity aerobic exercise produces greater oxidative stress in comparison to lower-intensity aerobic exercise, adding to the already elevated chronic inflammatory state of overweight and obese PMP women and potentially overwhelming cellular antioxidant capabilities.[38] In contrast, RT alters adipokines by increasing LMM and decreasing AT while stimulating lower oxidative stress production.[28]

Nevertheless, the effect of body composition on adipokines in PMP women has yet to be evaluated in the context of RT protocol length and intensity. These shortcomings indicate the need for a more detailed understanding of the topic and, hence, the current review's focus. This review aims to summarize the recent evidence for RT as an intervention to influence adipokine concentrations by decreasing AT and increasing LMM and BMC in PMP women.

Methods

A review of all published literature was conducted, regardless of the study design. The focus was to investigate the relationship between RT and adipokine concentrations isolated in any tissue in PMP women. While this review is not systematic, our search strategy was conducted in a structured manner that can be easily reproduced. PubMed, Medline, Sport Discus, and Google Scholar were searched from their inception through May 20th, 2022. Keywords and terms that pertained to RT and exercise in PMP women, such as "resistance training", "post-menopause", "older women", "female", "adipokine" and combined by Boolean operators. Additionally, the structured literature search was augmented by the consultation of article reference lists from retrieved papers. Unpublished theses or dissertations were not used in the literature search. The search produced a total of 479 citations based on our search terms. Upon intense scrutiny, six publications were accepted, meeting the full inclusion criteria (see Table 1). Only experimental studies were included, which consisted of PMP women receiving RT for varying lengths and intensities to elicit changes in adipokine concentrations.

Relationship between resistance training and adipokines

The primary role of RT is to promote increased muscular strength, endurance, balance, and power by conditioning and increasing LMM.[39,40] LMM consumes 13 kcal/kg of energy on a daily basis without the utilization of exercise.[41] Consequently, by increasing LMM, resting metabolic rate will assist in increasing the loss of AT.[41,42] Therefore, the basic recommendations for older women to participate in RT programs consist of two to three days a week of moderate-intensity, composed of 6–10 exercises, and 2 to 3 sets at 8–12 repetitions to promote healthy body composition.[43] However, the implementation of RT in PMP women as a therapeutic intervention to improve LMM and decrease AT to encourage changes in adipokines remains limited.[28,33] Currently, with only six studies focused on RT and PMP, there is a lack of specific adipokine focus. The antioxidant and inflammatory nature of adipokines in relation to body composition changes is difficult to ascertain. Within the current literature, the number of RT studies reporting on changes in adipokines is confined to adiponectin, adipsin, leptin, resistin, IL-4, IL-6, IL-15, TNF-α, osteonectin, and lipocalin-2 (See Table 2). Thus, the limited RT intervention protocols and outcomes, restrict comparisons of adipokine changes with RT in PMP women.

Table 1
Inclusion and exclusion criteria for study eligibility.

Parameter	Inclusion	Exclusion
Population	Post-menopausal women	Physical disabilities
Intervention	Post-menopausal women receiving RT for varying lengths and intensities to elicit changes in adipokine concentrations	
Outcome	Plasma or serum adipokine concentrations (e.g., adiponectin, leptin, resistin, PAI-1, LP-2, osteonectin, MCP-1, TNF-a, IL-4, IL-6, IL-15)	
Study Design	Experimental studies (e.g., RCT's, Crossover) published in English	Abstracts, Conference proceedings, Unpublished Theses and Dissertations

CVD = cardiovascular disease, CKD = chronic kidney disease, RT = resistance training, PAI-1 = plasminogen activator inhibitor-1, LP-2 = lipocalin-2, MCP-1 = monocyte chemoattractant protein-1, TNF-a = tumor necrosis factor-alpha, IL = interleukin, RCT = randomized controlled trial.

Table 2
Resistance training protocols and adipokine outcomes.

References	Participants	Training Status	Intervention	Exercise Protocol	Biomarker Type (Sample Extraction)	Adipokines Analyzed	Outcomes
Prestes (2018)	26 elderly POM women, 62.6 ± 6.7 yr And BMI = 28.1 ± 4.8 kg/m^2.	Sedentary	Four-month longitudinal RT program.	RT consisted of 2 whole-body sessions per week with increasing intensity and lower volume by using 6–14 repetitions max. Body weight exercises were always performed until exhaustion in 2 sets. Gradually increased loads throughout trial based on a weekly assessment.	Plasma; 3 ml taken from AV, centrifuged at 800 g for 10 min, Sampled before, immediately after, 24 h, and 48 h after first RT session, then at same time points post-intervention	IL-4, leptin, & resistin	Post-intervention leptin levels were sig. ↓ in LR. Both groups displayed a ↓ in resistin values after RT, but only a simple main effect was observed for LR. There was no time effect and no sig. Interaction between the responsiveness and time on IL-4 levels.
Park (2019)	35 PRM 47.5 ± 6.1 yr & POM 57.8 ± 5.7 yr. BMI 26.8 ± 4.6 kg/m^2 (PRM) & 24.7 ± 2.5 (POM).	–	RT for 12 weeks. The goal of the RT was to burn 230–260 kcal. They were divided into PRM (n = 15) and POM (n = 20) groups.	Moderate-intensity RT (55%–65% of 1-RM) was performed for 60 min per day, 3 days per week for 12 weeks. RT consisted of circuit training, 12 reps, & 3 sets.	Serum; OF (12 h) with blood sample taken from MCV at 07:00, centrifuged at 3 000 g for 10 min, sampled pre- and post-intervention, post-intervention data was sampled 48–72 h after last RT session	Adiponectin, leptin, IL-6, & IL-15	There was a sig. Diff. In IL-6 and leptin levels between the two groups after training. IL-6, IL-15, and adiponectin levels were sig. ↑ in both groups post-training vs. pre-training, although leptin levels were sig. ↓post-exercise in the PRM group.
Caneiro (2021)	LIRT (n = 20), average age 61 yr, BMI 26.6 kg/m^2. HIRT (n = 20) average age 61.9 yr, BMI 26.9 kg/m^2.	Physically active	15-weeks of RT, 3x a week.	3 sets of 8 dynamic exercises. LIRT (high reps 30–35) or HIRT (low reps 8–12). Load was increased to 2%–5% upper body and 5%–10% for lower body: if the upper limits of the established repetition zone was reached in the first set of the exercise.	Plasma; OF (8–10 h) with 12 ml taken between 08:00–09:30, centrifuged at 3 500 g for 10 min, post-intervention samples were collected 48–72 h after the last RT session	Leptin & adiponectin	LIRT (L did not sig. Change, while A sig. ↓) and HIRT (L did not sig. Change, while A sig. ↓)
Ward (2020)	55 POM women, age 55.5 ± 5.0 yr, BMI 26.7 ± 3.6 kg/m^2 (control) and 28.1 ± 3.8 kg/m^2(RT).	Sedentary	Women were randomized to a 15-week RT program (n = 26) or remain sedentary as control (n = 29).	RT was performed 3x week. RT consisted of 6 seated exercises performed 15–20 repetitions in 2 sets in week 1–3, and 8–12 reps in 2 sets week 4–15. Body-weight exercises were performed until exhaustion in 2 sets for the entire study.	Plasma; Fasted blood sample in EDTA vacutainer, centrifuged at 1 500 g, sampled pre- and post-intervention	Adiponectin, leptin, PAI-1, LP-2, osteonectin, resistin, & MCP-1	After 15 weeks, sig. lower plasma levels of adiponectin ($p < 0.001$), LP-2 ($p < 0.01$) and resistin ($p = 0.04$) were found.
Prestes (2009)	35 POM women, 63.18 ± 4.8 yr; BMI 57.84 ± 7.70 kg/m^2.	Sedentary	16 weeks of periodized RT.	RT consisted of two weekly sessions of three sets of 6–14 repetition maximum. Intensity was done according to this rep x set scheme: (3 sets x 12 to 14 reps = 60 s of rest between sets, 3 × 10 to 12 = 80 s, 3 x 8 to 10 = 100 s, 3 x 6 to 8 = 120 s).	Plasma; 3 ml taken from AV, centrifuged at 2 500 rpms for 20 min, sampled before, immediately after, 24 h, and 48 h after first RT session, then at same time points post-intervention	TNF-α, IL-6, IL-15, leptin, & resistin	There were no changes in IL-15. Leptin, resistin, and IL-6 was sig. ↓ when compared to baseline after 16 weeks.
Botero (2013)	23 POM women, age 63.0 ± 4.4 yr, BMI 28.0 ± 1.0 kg/m^2	Sedentary	12 months of periodized RT.	RT consisted of 2 x week and 3 sets of 6–14 reps. Loads were monitored in each session and were adjusted to maintain the number of max reps. Number of reps were reduced as intensity increased. In all weeks, maximal reps to concentric failure were performed.	Plasma; 3 ml taken from AV, centrifuged at 2 500 rpms for 10 min, samples taken pre- and post-intervention	Resistin & leptin	The was a sig. ↓ in leptin & resistin levels after the 12 months of RT ($p < 0.05$).

Abbreviations: Adiponectin (A), Adipsin (AD), Body Fat (BF), Body Mass Index (BMI), Grams (g), High-intensity resistance training (HIRT), High Responders (HR), Leptin (L), Lipocalin-2 (LP-2), Low-intensity resistance training (LIRET), Low Responders (LR), Osteonectin (O), Pre-menopause (PRM), Post-menopause (POM), resistance training (RT), Resistin (R), Total adiponectin (TA), High molecular weight adiponectin (HMW-A), Antecubital vein (AV), Median cubital vein (MCV), Overnight fast (OF), Rotations per minute (rpms), Hour (h), Milliliters (ml), Seconds (s) and information not reported (–), No significant changes (*), significantly increased $p < 0.05$ (↑), and significantly decreased $p < 0.05$ (↓).

Resistance training intervention length and protocol

The identified research studies utilized either short-term (< 6 months) or long-term (> 6 months) RT interventions in PMP women. Interventions consisting of < 6 months of RT resulted in conflicting adipokine outcomes. Studies lasting < 12 weeks observed an increase in IL-6 and IL-15, while a decrease or no change was seen in leptin and resistin levels. No difference was demonstrated in IL-4, and the changes in adiponectin were equivocal. Prestes et al.[44] reported on both low vs. high-responders of muscular strength who participated in a 12-week RT intervention. RT in low-responders led to significantly lower leptin and resistin concentrations, and no change was observed in IL-4 concentrations compared to baseline. In contrast, high-responders had no changes in leptin, resistin, and IL-4 concentrations compared to baseline. However, short-term studies lasting 15 to 16 weeks resulted in a decrease in IL-6 and lipocalin-2 and no change in TNF-α, adipsin, and osteonectin. Also, there was a reduction in adiponectin and resistin after the RT intervention in both studies.[35,45]

One 12-week study showed an increase in IL-15,[28] while a 15-week study showed no change in IL-15.[45] Likewise, Ward et al.[35] observed no change in leptin after 15 weeks of RT, Prestes et al.[45] observed a significant decrease in leptin after 16 weeks of RT (see Table 2). In addition, short-term studies lasting 15 to 16 weeks that utilized RT three vs. two times per week observed variations in the outcomes of the same adipokines (leptin and TNF-α) assessed. For the 15-week study, leptin and TNF-α were not altered, whereas the 16-week study demonstrated a decrease in leptin and TNF-α.[35,44,46] Furthermore, the 15-week research study implemented progressively higher volume, more repetitions, and bodyweight exercises, whereas the 16-week study utilized more repetitions to failure and moderate volume. When comparing all short-term RT studies, the studies that implemented RT twice per week with moderate-intensity and higher repetitions to failure observed similar adipokine changes. Comparably, studies that implemented RT three days per week with higher volume, more repetitions, and bodyweight exercises observed comparable outcomes regardless of short-term intervention length. Therefore, the difference in adipokines was due more to the number of training days per week rather than the extra week of intervention, which may be largely due to the increased amount of total volume.

Botero et al.[33] implemented the longest RT intervention in PMP women to determine changes in adipokines. The RT intervention lasted for 12 months, which was 8 to 9-month longer than the short-term studies (3 to 4 months). Leptin and resistin concentrations decreased from pre-to post-training, indicating that PMP women responded similarly to the short-term RT interventions as the long-term intervention. However, there appeared to be similar discrepancies in adipokine outcomes when comparing the long-term study to the short-term RT studies. For example, resistin decreased with 12 months of RT but increased, did not change, or decreased with 12 to 16-week RT studies. In addition, leptin was not significantly altered in the 12 to 15-week studies but significantly decreased in the 12-month study. Also, previously mentioned 12- and 16-week studies that implemented RT twice a week with moderate-intensity and higher repetitions to failure demonstrated the same outcomes as the 12-month study that implemented a similar RT protocol.[33,44,45] Therefore, based on the high variance in RT protocols and the resulting adipokine outcomes, it is difficult to make a clear consensus on the effectiveness of RT influence on altering adipokine levels in PMP women.

Adipokine outcomes related to changes in muscular strength

There appears to be a relationship between RT-associated improvements in muscular strength and the expression of adipokines; although, the response may not be linear. Muscular strength was assessed in five of the six reviewed studies. On average, half of the studies with short-term (12–16 weeks) RT protocols demonstrated a 16% lower gain in muscular strength when compared to long-term protocols. Prestes et al.[44,45] performed two separate studies implementing similar RT protocols lasting 12 and 16 weeks in PMP women. The 12-week protocol increased skeletal muscle strength by 42% in high-responders vs. 23% in low-responders. The 16-week protocol resulted in similar increases in muscle strength with an average increase of 20% in total muscle strength and an increase of 31% in lower-body muscular strength. Both short-term studies implemented similar weekly protocols (2 weekly sessions, 3 sets, and 6–14 repetitions) compared to the other two short-term RT protocols (3 weekly sessions, 3 sets, and 8–12 repetitions). Unsurprisingly, increases in muscle strength were substantially greater following long-term RT when compared to the ST studies. On average, Botero et al.[33] reported an increase in muscular strength by 31% in the bench press, 100.9% in the leg press, and 26.4% in the biceps curl. The long-term RT protocol consisted of two sessions per week with 3 sets and 6–14 repetitions, similar to the protocols implemented by Prestes et al.[44,45] in the short-term studies.

Interestingly, Prestes et al.[23] reported a novel finding of the responsiveness in IL-4 and resistin, which is believed to be related to changes in muscular strength. The increase in relative muscle strength was positively correlated with an increase in IL-4 and a decrease in resistin 48-h after an acute bout of RT. These results support the potential for acute changes in adipose biomarkers with RT. However, there is no consensus on the influence of RT on improving muscular strength and altering adipokine levels in PMP women due to the large variability in adipokine outcomes.

Adipokine outcomes related to changes in body composition

Body composition improvement from RT is known to influence adipokines in various populations positively.[3] However, when evaluating the six present studies, over half did not report specific changes in total body composition other than weight change after participants completed the designated RT protocol (Table 3). Two studies reported changes in LMM, three reported changes in AT, and five reported total weight changes. No studies reported BMC values in relation to adipokine levels.

Alterations in LMM were reported in both short-term (12 weeks) and long-term (12 months) studies. The long-term research study resulted in a 1.6% increase in LMM, whereas the short-term study demonstrated a 1% increase. Neither study demonstrated clinical significance due to the 1.5% standard deviation of the DEXA. However, these outcomes are interesting due to the substantial changes in adipokines post-RT, potentially indicating an existing correlation between increased skeletal muscle force production and adipokine mechanisms.

Reductions in AT were more substantial than improvements in LMM. Two 12-week studies reported a decrease in AT ranging from 0.5% to 4.9%, while the 12-month study reported a decrease of 2.64% in AT.[28,33,46] It is important to note that the study with the smallest change in AT contained PMP women with the lowest baseline percent AT (22.2%–23.8%). The study with the greatest AT loss had a larger baseline AT percentage (35.3%)—indicating that PMP women with higher amounts of AT may lose more AT when compared to those with lower amounts of AT at baseline. In addition, Park et al.[28] observed a linear

Table 3
Body composition and muscular strength outcomes in relation to adipokines.

Citation	Study groups	Intervention	Baseline BMI (kg/m^2)	Body weight (kg)	Weight Change (%)	% BF	AT Change (%)	LMM Change (%)	Muscular Strength Changes (%)	Adipokine Changes (sig.)
Prestes (2018)	RT ($n = 26$)	12 Weeks	HR (28.6 ± 5.1) LR (27.6 ± 4.7)	HR (66.6 ± 12.1) LR (64.8 ± 8.6)	HR (- 1.1) LR (+2.1)	HR (38.4 ± 7.3) LR (39.1 ± 7.3)	–	–	HR (+42.2) LR (+23.0)	HR (IL-4 *, L *, R *) LR (IL-4 *, L ↓, R ↓)
Park (2019)	RT for both PRM ($n = 15$) POM ($n = 20$)	12 Weeks	PRM (26.8 ± 4.6) POM (24.7 ± 2.5)	PRM (66.7 ± 12.9) POM (60.6 ± 6.6)	PRM (- 3.2) POM (- 2.0)	PRM (73.3 ± 6.1) POM (35.3 ± 5.3)	PRM (- 7.9) POM (- 4.9)	PRM (+3.2) POM (+1.0)	PRM (+9.0) POM (+8.4)	PRM (IL-6 ↑, IL-15 ↑, L ↓, A ↑) POM (IL-6 ↑, IL-15 ↑, L *, A ↑)
Caneiro (2021)	LIRT ($n = 20$) HIRT ($n = 20$)	15 Weeks	LIRT (26.6) HIRT (26.9)	LIRT (65.5) HIRT (66.0)	–	LIRT (22.2) HIRT (23.8)	LIRT (- 0.5) HIRT (- 0.6)	–	LIRT (+19.1) HIRT (+22.1)	LIRT (L *, A ↓) HIRT (L *, A↓)
Ward (2020)	Sedentary Control ($n = 29$). RT ($n = 26$)	15 Weeks	Control (26.7 ± 3.6) RT (28.1 + 3.8)	Control (72.3 ± 11.5) RT (76.5 ± 11.5)	Control (−) RT (- 1.0)	–	–	–	–	Control (A *, AD *, LP-2 *, O *, L *, R *, TNF-α *) RT (A ↓, AD *, LP-2 ↓, O *, L *, R ↓, TNF-α *)
Prestes (2009)	Periodized RT ($n = 35$)	16 Weeks	RT (77.6 ± 4.5)	64.8 ± 8.6	1.39	–	–	–	Bench Press + 20.5 Leg Press + 30.8 Arm Curl + 9.5	IL-6 ↓, IL-15 *, TNF- α ↓, L ↓, R ↓
Botero (2013)	Periodized RT ($n = 23$).	12 Months	RT (28.0 ± 1.0)	67.56 ± 2.26	−1.5	37.8 ± 1.3	−2.64	1.6	Bench Press + 30.8 Leg Press + 100.9 Arm Curl + 26.4	L ↓ and R ↓

Abbreviations: Adiponectin (A), Adipsin (AD), High-intensity resistance training (HIRT), High Responders (HR), Leptin (L), Lipocalin-2 (LP-2), Low-intensity resistance training (LIRET), Low Responders (LR), Osteonectin (O), Pre-menopause (PRM), Post-menopause (POM), resistance training (RT), Resistin (R), Total adiponectin (TA), High molecular weight adiponectin (HMW-A), and information not reported (−), No significant changes (*), significantly increased $p < 0.05$ (↑), and significantly decreased $p < 0.05$ (↓).

decrease in leptin and adiponectin concentrations when comparing the loss of AT post-RT, which is considered a normal trend with leptin, but not adiponectin. It is known that serum adiponectin concentrations are decreased by abdominal obesity (visceral AT) while leptin concentrations are greatly associated with subcutaneous AT levels.[28] Visceral and subcutaneous AT were not assessed in any of the reviewed studies, which limited the effectiveness of determining RT influence on adipokines in PMP women.

A decrease in total body weight was observed in four of the five studies reporting total body weight change. Prestes et al.[44] observed an increase in total body weight in the low-responders group and a decrease in the high-responders group. Changes in LMM and AT were not reported, therefore decreases in adipokines that were reported cannot be correlated to specific changes in total body composition. A 23% increase in muscular strength was observed in the low-responders group, indicating a potential increase in LMM. The absence of sufficient data presents limitations to understanding adipokine outcomes and body composition changes in PMP women after participating in an RT protocol.

Conclusion

The systemic and local effects of RT on adipokine concentrations are a developing area of study. Due to obstacles in stratifying manipulating factors that influence adipokines, there is still no consensus on the involvement of RT in altering adipokine levels. Prior studies demonstrate equivocal conclusions regarding adipokine levels in PMP women who participate in interventional RT therapy. Collectively, adipokine concentrations have a varied response when comparing pre-and post-RT levels in PMP women. Half of the studies assessed reported only adiponectin, leptin, and resistin, leaving the majority of adipokines unassessed in PMP women. Furthermore, body composition outcomes relating to AT and LMM were not reported in most of the studies. Therefore, significant gaps remain in our understanding of the influence of RT on body composition and adipokine levels in PMP women. Future research is needed to fill in the highlighted gaps regarding RT protocol development and the inclusion of more adipokine biomarkers for a more comprehensive assessment. Forthcoming studies should assess changes in body composition (AT, LMM, and BMC) to strengthen the collective knowledge on the effectiveness of RT in altering adipokines in PMP women.

Submission statement

All authors have read and agreed to the manuscript content. This manuscript has not been published and is not under consideration for publication elsewhere.

Authors' contributions

All authors contributed to the writing and editing of this manuscript. Conceptualization - J.S.F., L.F., A.G., and P.K.Methodology – T.CL., J.S.F., L.F., and P.K.Software - J.S.F. and T.CL.Validation - J.S.F., J.L.H., P.K., K.A.R. and A.I.Formal analysis - J.S.F., D.W., T.CL.Investigation - J.S.F., J.L.H., P.K., K.A.R., L.F., A.G., and A.I.Resources - J.S.F., A.G., P.K., and L.F.Data curation - J.S.F., and T.C.LWriting - original draft preparation - J.S.F., T.CL., D.W., E.F., A.I., and J.L.H.Writing—review and editing - J.S.F., J.L.H., P.K., K.A.R., L.F., A.G., T.CL., D.W., E.F., and A.I.Supervision - J.S.F., J.L.H., P.K., K.A.R., L.F., A.G. and A.I.Project administration - J.S.F., T.CL., and J.L.H.

Conflict of interest

The authors have no conflict of interest to report.

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