RESEARCH PAPER

Effect of Electrical Muscle Stimulation and Resistance Training on Body Mass Index and Regional Adiposity in Sedentary Type-II Diabetic Individuals: A Randomized Controlled Trial

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ABSTRACT

Background: Excess central adiposity and elevated body mass index (BMI) are key determinants of insulin resistance and cardiometabolic risk in individuals with type 2 diabetes mellitus (T2DM). Although progressive resistance training (RT) improves body composition, long-term adherence remains suboptimal in sedentary populations. Electrical muscle stimulation (EMS) represents a non-volitional alternative capable of eliciting contraction-mediated metabolic adaptations.

Objective: To compare the effects of EMS and supervised RT on BMI and regional adiposity in sedentary adults with T2DM.

Methods: In this randomized controlled trial, 66 sedentary individuals with T2DM were allocated to EMS (n=22), RT (n=22), or control (n=22) groups. EMS was delivered using Russian current (2500 Hz; 10 s ON/50 s OFF; 30 min/session; three sessions/week) for 12 weeks. The RT group performed supervised progressive resistance exercises three times weekly for 12 weeks. Primary outcome was BMI. Secondary outcomes included triceps, suprailiac, and abdominal skinfold thickness. Assessments were conducted at baseline, 12 weeks, and 3-month follow-up. Repeated-measures ANOVA evaluated time, group, and interaction effects.

Results: A significant group × time interaction was observed for BMI ($F(4,126) = 17.366$, $p<0.001$, $\eta^2=0.355$), indicating a large effect size. Both EMS and RT significantly reduced BMI compared with control ($p<0.01$), with no significant difference between intervention groups. Abdominal skinfold thickness demonstrated significant interaction effects ($F=9.554$, $p<0.001$, $\eta^2=0.233$), reflecting meaningful reductions in central adiposity in both intervention groups. Suprailiac skinfold thickness also showed significant interaction ($F=3.295$, $p=0.013$), while triceps skinfold exhibited a moderate between-group effect ($F=5.117$, $p=0.009$). No significant improvements were observed in the control group.

Conclusions: EMS and RT produced significant and comparable improvements in BMI and central adiposity in sedentary adults with T2DM. EMS may represent a clinically viable alternative for individuals unable to engage in conventional resistance training.

Keywords: Type 2 diabetes mellitus; Electrical muscle stimulation; Resistance training; Body mass index; Central adiposity; Randomized controlled trial.

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a progressive metabolic disorder marked by persistent hyperglycaemia arising from insulin resistance and relative insulin deficiency. Its global prevalence continues to increase substantially, largely driven by sedentary lifestyles, obesity, and population ageing [1]. Beyond disturbances in glycemic control, T2DM is closely associated with adverse alterations in body composition, particularly increased central adiposity and elevated Body Mass Index (BMI). These anthropometric changes are not merely secondary associations but actively contribute to metabolic dysregulation and heightened cardiovascular risk [2].

Central adiposity, especially visceral fat accumulation, plays a central role in the pathogenesis of insulin resistance. In individuals with T2DM, adipose tissue exhibits altered endocrine activity, characterized by increased secretion of pro-inflammatory adipokines such as tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), alongside reduced production of

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anti-inflammatory mediators including adiponectin [3]. This inflammatory environment disrupts insulin signalling pathways in skeletal muscle and hepatic tissues [4]. Additionally, enhanced lipolysis within visceral fat depots elevates circulating free fatty acid levels, which further impair glucose transport and promote ectopic lipid deposition in muscle and liver, thereby exacerbating insulin resistance [5]. Consequently, reduction of central adiposity is not solely an anthropometric objective but a primary therapeutic target in diabetes management aimed at improving insulin sensitivity and mitigating cardiometabolic complications [6].

Skeletal muscle represents the largest insulin-sensitive tissue in the human body and accounts for approximately 70–80% of postprandial glucose disposal. Muscle contraction stimulates glucose uptake through both insulin-dependent and contraction-mediated mechanisms involving AMP-activated protein kinase (AMPK), GLUT-4 translocation, and improved mitochondrial oxidative capacity [7]. For this reason, exercise-based interventions form a cornerstone of non-pharmacological management in T2DM. Among these strategies, Resistance Training (RT) has received considerable attention due to its capacity to increase lean muscle mass, enhance basal metabolic rate, and improve insulin sensitivity. Progressive resistance protocols have been shown to reduce visceral adiposity, improve body composition, and favourably modulate cardiometabolic markers [8].

Despite these established benefits, sustained adherence to structured resistance training remains challenging in sedentary diabetic populations [9]. Factors such as joint discomfort, obesity-related mobility restrictions, fatigue, limited time availability, and reduced exercise tolerance often hinder long-term participation. In older adults with T2DM or individuals with musculoskeletal comorbidities, traditional resistance-based exercise may be impractical or require extensive supervision [10]. These limitations have prompted interest in alternative or adjunctive modalities capable of eliciting comparable metabolic adaptations with reduced volitional effort.

Electrical Muscle Stimulation (EMS) has emerged as a promising non-volitional therapeutic approach in this context. EMS induces involuntary skeletal muscle contractions through externally applied electrical impulses delivered via surface electrodes. When administered at motor-level intensities, EMS activates large muscle groups, thereby increasing metabolic demand and reproducing certain aspects of voluntary muscle contraction [11]. Experimental evidence suggests that EMS can stimulate contraction-mediated glucose uptake, enhance lipid oxidation, and improve microcirculatory dynamics. Activation of AMPK signalling pathways and increased GLUT-4 translocation during electrically evoked contractions provide a mechanistic rationale for its metabolic effects [12]. Furthermore, EMS may augment regional blood flow and localized energy expenditure without imposing substantial mechanical stress on joints, making it particularly suitable for individuals with mobility limitations [13].

Most previous investigations examining EMS in diabetic populations have focused primarily on glycaemic indices or lipid profile modulation. While favourable effects on triglycerides and cholesterol fractions have been documented, relatively few studies have evaluated its impact on anthropometric outcomes such as BMI and regional adiposity distribution [14]. Given the pivotal role of abdominal fat in the development of insulin resistance and cardiovascular morbidity, assessing changes in central and peripheral skinfold thickness is of significant clinical relevance [15]. Moreover, direct comparisons between EMS and structured RT remain limited, particularly among sedentary adults with T2DM [16].

Determining whether EMS can induce body composition changes comparable to those achieved through resistance training has important translational implications [17]. If EMS demonstrates similar efficacy in reducing BMI and central adiposity, it may serve as a practical therapeutic alternative for individuals unable or unwilling to participate in conventional exercise programs [18]. Such an approach could broaden access to metabolic rehabilitation strategies and facilitate more individualized diabetes management.

Accordingly, the present study aimed to evaluate and compare the effects of Electrical Muscle Stimulation and Progressive Resistance Training on BMI and regional adiposity, specifically triceps, suprailiac, and abdominal skinfold thickness, in sedentary adults with T2DM. By incorporating a control group and longitudinal follow-up assessment, this investigation sought to determine both short-term and sustained effects of these interventions on anthropometric parameters. The null hypothesis (H_0) proposed that no significant differences would exist among EMS, RT, and control groups in altering BMI and regional adiposity. In contrast, the alternative hypothesis (H_1) posited that EMS and RT would significantly improve BMI and regional adiposity compared with control, with measurable reductions in central adiposity over time.

MATERIALS AND METHODS

Study Design

This investigation was conducted as a randomized, parallel-group, controlled experimental trial employing a repeated-measures design. The intervention period lasted 12 weeks, followed by a 3-month post-intervention follow-up assessment. The study comprised three arms: an Electrical Muscle Stimulation (EMS) group, a Progressive Resistance Training (RT) group, and a control group. Anthropometric outcomes were evaluated at three standardized time points: baseline (pre-intervention), immediately following completion of the 12-week intervention, and at 3-month follow-up to determine sustainability of effects.

The study protocol received approval from the Institutional Ethical Committee (WWET/2023/IEC-AP/03) and was prospectively registered with the Clinical Trials Registry of India (CTRI/2023/09/057825). All procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment. The trial incorporated standardized intervention protocols, blinded outcome assessment, allocation concealment, and predefined statistical procedures to ensure methodological rigor, internal validity, and reproducibility.

Participants

A total of 66 sedentary adults diagnosed with Type 2 Diabetes Mellitus (T2DM) were recruited and equally allocated into three groups (n = 22 per group). Participants were recruited through outpatient diabetic clinics and community referrals. Prior to enrolment, all individuals underwent comprehensive medical screening conducted by an internal medicine specialist to confirm eligibility and ensure safety for participation.

Inclusion Criteria

Participants were included if they were aged between 45 and 65 years, had a confirmed diagnosis of T2DM managed with stable oral hypoglycaemic medication, demonstrated a sedentary lifestyle as classified by the International Physical Activity Questionnaire (IPAQ) low-activity category, and were medically stable without acute diabetic complications.

Exclusion Criteria

Individuals were excluded if they were receiving insulin therapy; had unstable cardiovascular conditions such as uncontrolled hypertension, arrhythmias, or recent myocardial infarction; presented with neuromuscular disorders contraindicating electrical stimulation; had implanted electronic devices (e.g., pacemakers) or metallic implants incompatible with EMS; exhibited dermatological conditions preventing safe electrode placement; or were engaged in regular structured exercise programs.

Following baseline assessment, participants were randomized using a computer-generated allocation sequence. Allocation concealment was achieved using sealed opaque envelopes prepared by an independent investigator not involved in outcome assessment or intervention delivery, thereby minimizing selection bias.

Sample size estimation was performed a priori using G*Power software (version 3.1), based on repeated-measures ANOVA (within–between interaction). Assuming a large effect size ($f = 0.74$), $\alpha = 0.05$, power ($1-\beta$) = 0.80, and three groups with three measurement points, the required sample size was calculated as 60. To account for potential attrition, 66 participants (22 per group) were enrolled [19].

Intervention Protocols

All interventions were administered over a 12-week period, with supervised sessions conducted three times weekly on non-consecutive days.

Electrical Muscle Stimulation (EMS) Group

Participants assigned to the EMS group received neuromuscular electrical stimulation using an 8-channel stimulator (Johri Digital TR841). Russian current with a carrier frequency of 2500 Hz was applied in accordance with established neuromuscular stimulation protocols. Stimulation parameters were standardized as follows: a duty cycle of 10 seconds contraction (ON) followed by 50 seconds rest (OFF), a session duration of 30 minutes, and a frequency of three sessions per week over 12 weeks. Surface electrodes were positioned bilaterally over the quadriceps, hamstrings, and gluteus maximus muscles according to standardized motor point placement guidelines. Two muscle groups were stimulated simultaneously in 10-minute intervals per pair to ensure uniform exposure across targeted regions [20].

Stimulation intensity was progressively adjusted to achieve visible, tolerable tetanic contractions at motor-level intensity without inducing discomfort. Intensity progression was individualized based on participant tolerance while maintaining consistent contraction quality. All EMS sessions were supervised by a licensed physiotherapist to ensure safety, adherence, and protocol fidelity.

Resistance Training (RT) Group

Participants in the RT group underwent supervised progressive resistance training targeting major bilateral muscle groups, including hip extensors and flexors, knee extensors and flexors, shoulder flexors, elbow flexors, and plantar flexors. Each session began with a standardized 10-minute warm-up consisting of low-intensity dynamic stretching and mobility exercises. Participants performed two sets of 10 repetitions per targeted muscle group at an intensity equivalent to three-repetition maximum (3RM).

One-repetition maximum (1RM) was estimated using the Brzycki formula:

1RM = weight lifted / (1.0278 − (0.0278 × repetitions)) [21]

Load progression was implemented when participants were able to comfortably complete 20 repetitions, at which point resistance was increased incrementally by approximately 0.5 kg. Training sessions were conducted three times per week for 12 weeks under direct physiotherapy supervision to ensure correct technique, progressive overload, and participant safety [22].

Control Group

Participants allocated to the control group received standardized educational counselling regarding diabetes management, including dietary recommendations, lifestyle modification strategies, and glycaemic control guidance. They were encouraged to engage in light-to-moderate physical activity such as walking and routine household activities but did not receive supervised exercise intervention during the study period. No modifications were made to prescribed pharmacological regimens in any group throughout the intervention phase to prevent confounding effects.

Outcome Measures

All primary and secondary outcomes were assessed at baseline, immediately post-intervention (12 weeks), and at 3-month follow-up by a blinded assessor to minimize measurement bias.

Primary Outcome

Body Mass Index (BMI): BMI was calculated as body weight in kilograms divided by height in meters squared (kg/m^2). Body weight was measured using a calibrated digital weighing scale with participants barefoot and wearing light clothing. Height was recorded using a stadiometer to the nearest 0.1 cm.

Secondary Outcomes

Regional Adiposity Assessment: Skinfold thickness was measured using a calibrated Harpenden skinfold calliper at standardized anatomical sites: triceps (posterior midline of the upper arm), suprailiac (immediately superior to the iliac crest along the mid-axillary line), and abdominal (approximately 2 cm lateral to the umbilicus). Measurements were recorded in millimetres, and the mean of three consecutive readings was used for statistical analysis to enhance reliability. All measurements were performed by the same trained assessor to reduce inter-observer variability.

Statistical Analysis

Data were analysed using the Statistical Package for the Social Sciences (SPSS), version 25.0 (IBM Corp., USA). Normality of distribution was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation. Repeated-measures Analysis of Variance (ANOVA) was employed to examine within-group (time effect), between-group (group effect), and interaction (group × time effect) differences across the three assessment points. Mauchly's test of sphericity was used to assess homogeneity of variance assumptions. Where sphericity was violated, Greenhouse–Geisser corrections were applied. Bonferroni-adjusted post-hoc comparisons were conducted to determine specific pairwise differences. Effect sizes were reported as Partial Eta Squared (η^2), interpreted as small (0.01), medium (0.06), and large (≥ 0.14). Statistical significance was established at $p < 0.05$.

RESULTS

Participant Characteristics

Sixty-six participants were randomized equally into three groups (EMS, RT, and Control; n = 22 each), and all completed the 12-week intervention and 3-month follow-up assessments. There were no dropouts, protocol deviations, or reported adverse events. Baseline values were comparable between the EMS and RT groups across anthropometric variables, whereas the control group demonstrated relatively higher baseline adiposity indices. All outcome variables satisfied normality assumptions (Shapiro–Wilk $p > 0.05$).

Body Mass Index

Mean BMI values over time are presented in Table 1 and illustrated in Figure 1. At baseline, BMI was 25.84 ± 3.07 kg/m^2 in the EMS group, 26.14 ± 3.84 kg/m^2 in the RT group, and 29.00 ± 3.42 kg/m^2 in the control group. After 12 weeks of intervention, BMI decreased to 24.91 ± 3.10 kg/m^2 in the EMS group and to 25.37 ± 3.86 kg/m^2 in the RT group, while the control group showed a slight increase to 29.20 ± 3.49 kg/m^2. At 3-month follow-up, further reductions were observed in the EMS (24.79 ± 3.13 kg/m^2) and RT groups (24.98 ± 3.97 kg/m^2), whereas the control group demonstrated a continued increase to 29.36 ± 3.55 kg/m^2.

The EMS and RT groups achieved sustained BMI reductions of −4.06% and −4.44%, respectively, over the study period, whereas the control group exhibited a net increase of +1.24%. Repeated-measures ANOVA demonstrated a significant main effect of time ($F(2,126) = 30.375$, $p < 0.001$, $\eta^2 = 0.325$), a significant group × time interaction ($F(4,126) = 17.366$, $p < 0.001$, $\eta^2 = 0.355$), and a significant between-group effect ($F(2,63) = 8.865$, $p < 0.001$, $\eta^2 = 0.220$). Bonferroni-adjusted post hoc comparisons confirmed that both EMS and RT groups differed significantly from the control group ($p < 0.01$), while no statistically significant difference was detected between EMS and RT ($p > 0.05$).

Triceps Skinfold Thickness

Triceps skinfold thickness values are summarized in Table 2. Baseline measurements were 14.14 ± 4.01 mm (EMS), 15.05 ± 3.20 mm (RT), and 16.59 ± 2.08 mm (Control). At 3-month follow-up, values were 13.82 ±

4.06 mm in the EMS group, 14.91 ± 3.28 mm in the RT group, and 17.00 ± 3.21 mm in the control group. Between-group analysis revealed a statistically significant difference (F(2,63) = 5.117, p = 0.009, η² = 0.140). Post hoc testing demonstrated significantly higher triceps skinfold thickness in the control group compared with EMS (p = 0.007), while differences between EMS and RT were not statistically significant.

Suprailiac Skinfold Thickness
Baseline suprailiac thickness was 15.73 ± 4.62 mm in the EMS group, 16.59 ± 4.29 mm in the RT group, and 19.55 ± 4.17 mm in the control group. At 3 months, values were 15.14 ± 4.62 mm (EMS), 16.68 ± 4.53 mm (RT), and 20.18 ± 4.34 mm (Control). A significant group × time interaction was observed (F(4,126) = 3.295, p = 0.013, η² = 0.095), indicating differential changes over time among the groups, with reductions primarily observed in the intervention groups and progressive increase in the control group.

Abdominal Skinfold Thickness
Abdominal skinfold thickness values are presented in Table 3 and depicted in Figure 2. At baseline, mean values were 29.68 ± 5.27 mm (EMS), 29.09 ± 6.08 mm (RT), and 34.41 ± 6.39 mm (Control). After 12 weeks, reductions were observed in the EMS (27.64 ± 5.05 mm) and RT groups (27.68 ± 5.88 mm), whereas the control group remained largely unchanged (34.32 ± 6.52 mm). At 3-month follow-up, abdominal thickness was 27.77 ± 5.10 mm in the EMS group and 27.73 ± 5.92 mm in the RT group, while the control group increased to 34.77 ± 6.64 mm.

Net reductions at follow-up were −6.43% in the EMS group and −4.67% in the RT group, compared with a +1.05% increase in the control group. Repeated-measures ANOVA demonstrated a significant main effect of time (F(2,126) = 27.892, p < 0.001, η² = 0.307) and a significant group × time interaction (F(4,126) = 9.554, p < 0.001, η² = 0.233). Both EMS and RT differed significantly from control (p < 0.01), with no statistically significant difference between the two intervention groups.

Overall, both EMS and RT produced significant and sustained reductions in BMI and central adiposity, with effect sizes indicating moderate-to-large intervention effects, whereas the control group demonstrated stable or progressive increases across anthropometric measures.

Table 1: Mean BMI values over time

Group	**Baseline**	**12 Weeks**	**3 Months**
EMS	25.84 ± 3.07	24.91 ± 3.10	24.79 ± 3.13
RT	26.14 ± 3.84	25.37 ± 3.86	24.98 ± 3.97
Control	29.00 ± 3.42	29.20 ± 3.49	29.36 ± 3.55

Table 2. Triceps and Suprailiac Skinfold Thickness (mm)

Group	Triceps Baseline	Triceps 12 weeks	Triceps 3 Months	Suprailiac Baseline	Suprailiac 12 weeks	Suprailiac 3 Months
EMS	14.14 ± 4.01	13.77 ± 4.05	13.82 ± 4.06	15.73 ± 4.62	15.05 ± 4.44	15.14 ± 4.62
RT	15.05 ± 3.20	15.23 ± 3.15	14.91 ± 3.28	16.59 ± 4.29	16.36 ± 4.40	16.68 ± 4.53
Control	16.59 ± 2.08	16.45 ± 3.96	17.00 ± 3.21	19.55 ± 4.17	20.00 ± 4.29	20.18 ± 4.34

Table 3. Abdominal Skinfold Thickness (mm)

Group	**Baseline**	**12 Weeks**	**3 Months**
EMS	29.68 ± 5.27	27.64 ± 5.05	27.77 ± 5.10
RT	29.09 ± 6.08	27.68 ± 5.88	27.73 ± 5.92
Control	34.41 ± 6.39	34.32 ± 6.52	34.77 ± 6.64

Table 4: A summary of the repeated-measures ANOVA findings

Outcome	Effect	F-Value	p-value	Partial η²
BMI	Time	30.375	<0.001	0.325
BMI	Group × Time	17.366	<0.001	0.355
BMI	Between Groups	8.865	<0.001	0.220
Triceps SFM	Between Groups	5.117	0.009	0.140
Suprailiac SFM	Group × Time	3.295	0.013	0.095
Abdominal SFM	Time	27.892	<0.001	0.307
Abdominal SFM	Group × Time	9.554	<0.001	0.233

Figure 1. Mean body mass index (BMI) across baseline, 12 weeks, and 3-month follow-up. Significant group × time interaction (F(4,126) = 17.366, $p < 0.001$).

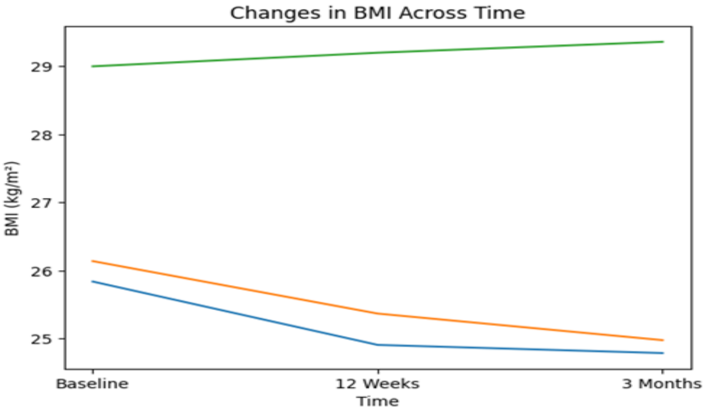
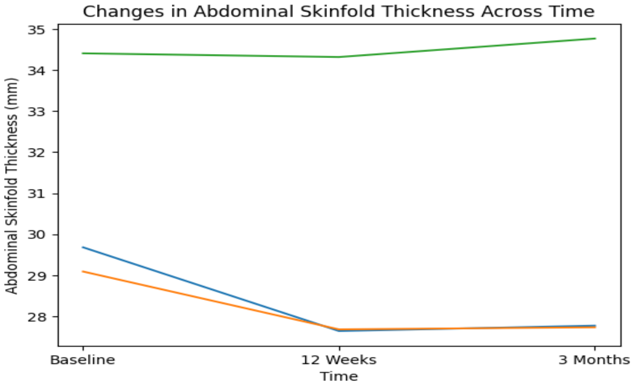


Figure 2. Changes in abdominal skinfold thickness across time points demonstrating significant intervention effects (F(4,126) = 9.554, $p < 0.001$).



DISCUSSION

The present randomized controlled trial demonstrates that both Electrical Muscle Stimulation (EMS) and Progressive Resistance Training (RT) elicit significant reductions in Body Mass Index (BMI) and regional adiposity in sedentary individuals with Type 2 Diabetes Mellitus (T2DM). The magnitude of the group × time interaction for BMI ($\eta^2 = 0.355$) reflects a large effect size, indicating that the observed changes were not only statistically significant but also clinically meaningful. Both active interventions were superior to standard care, while no significant difference was detected between EMS and RT, suggesting that electrically evoked muscle contractions may induce metabolic adaptations comparable to those achieved through conventional resistance exercise.

Interpretation of BMI Reduction

BMI reduction serves as a global anthropometric marker of improved metabolic status. The significant time and interaction effects indicate that the reductions were intervention-driven rather than attributable to spontaneous fluctuation or regression to the mean [23]. In individuals with T2DM, even modest reductions in BMI are associated with enhanced insulin sensitivity, improved glycaemic control, and decreased cardiovascular risk. Adiposity reduction contributes to lower hepatic glucose output, improved skeletal muscle glucose uptake, and attenuation of chronic low-grade inflammation [9].

The comparable magnitude of BMI reduction observed in EMS and RT groups suggests that sufficient skeletal muscle activation—irrespective of whether contraction is volitional or electrically induced—may be the principal determinant of metabolic improvement [24]. This finding supports the concept that contraction-mediated metabolic signalling, rather than voluntary effort per se, underlies adiposity reduction in sedentary diabetic populations [25].

Central Adiposity and Regional Fat Distribution

Among regional adiposity measures, abdominal skinfold thickness demonstrated the most pronounced response, with a large time effect ($\eta^2 = 0.307$) and a moderate-to-large interaction effect ($\eta^2 = 0.233$). Central adiposity is particularly relevant in T2DM due to its close association with visceral fat accumulation and cardiometabolic risk. Abdominal adipose depots exhibit high lipolytic activity, increased free fatty acid release, and elevated secretion of pro-inflammatory adipokines, all of which contribute to insulin resistance and endothelial dysfunction [26]. The observed reduction in abdominal skinfold thickness suggests improved regulation of central fat stores. Although skinfold measurements do not directly quantify visceral adipose tissue, reductions at the abdominal site are strongly correlated with improvements in truncal adiposity and metabolic risk indices [27]. Suprailiac and triceps measurements demonstrated smaller yet statistically significant changes, indicating that both central and peripheral fat depots were influenced by the interventions. The more substantial response in abdominal adiposity may be attributable to activation of large proximal muscle groups (quadriceps, hamstrings, gluteus maximus), which were targeted in both EMS and RT protocols. Recruitment of these high-mass muscle groups increases systemic energy expenditure and may preferentially influence truncal fat redistribution [28].

Resistance training promotes metabolic improvement through several well-established physiological mechanisms. Skeletal muscle hypertrophy increases lean body mass, thereby elevating basal metabolic rate and total glucose disposal capacity [29]. Given that skeletal muscle accounts for approximately 70–80% of insulin-mediated glucose uptake, hypertrophy directly enhances glycaemic regulation. RT also increases mitochondrial density and oxidative enzyme activity, facilitating fatty acid oxidation and reducing ectopic lipid accumulation [30]. Additionally, repeated voluntary contractions stimulate GLUT-4 translocation and improve insulin receptor signalling efficiency, collectively contributing to reduced adiposity and improved metabolic homeostasis [31].

EMS induces involuntary skeletal muscle contractions via motor-level electrical impulses. The comparable efficacy observed in this trial suggests that electrically evoked contractions activate similar metabolic pathways to voluntary exercise [11]. EMS stimulates contraction-mediated glucose uptake through activation of AMP-activated protein kinase (AMPK), promoting GLUT-4 translocation independent of insulin signalling. Neuromuscular stimulation also enhances regional blood flow, thereby improving nutrient delivery and lipid mobilization [12]. Repeated tetanic contractions elevate local metabolic demand and energy expenditure, contributing to adipose tissue reduction [32].

Unlike RT, EMS does not require sustained volitional effort, which may explain its feasibility in sedentary or mobility-limited individuals [33]. The absence of statistically significant differences between EMS and RT implies that adequate motor-unit recruitment and contraction intensity may be sufficient to initiate metabolic adaptations necessary for adiposity reduction [34].

Comparison with Existing Literature

Prior investigations examining EMS in diabetic populations have primarily focused on glycaemic indices and lipid profiles, with reported improvements in triglycerides and glucose tolerance [35]. Fewer studies have directly compared EMS with structured RT for anthropometric outcomes. Resistance training has consistently demonstrated reductions in visceral fat and improvements in body composition among individuals with T2DM [36]. The present findings align with this established evidence and extend it by demonstrating that EMS may achieve comparable reductions in BMI and regional adiposity [37].

These results support emerging evidence that contraction intensity and muscle mass recruitment, rather than voluntary exertion alone, are central determinants of metabolic adaptation. Consequently, EMS represents a viable adjunctive or alternative modality in metabolic rehabilitation for individuals unable to participate in traditional exercise programs.

Clinical Implications

The translational relevance of these findings is substantial. EMS may be particularly beneficial for elderly individuals with T2DM who frequently present with sarcopenia, reduced exercise tolerance, balance impairment, and mobility limitations that restrict participation in conventional resistance training. By eliciting muscle contraction without substantial mechanical loading, EMS offers a practical strategy to stimulate metabolic activity in this vulnerable population [38].

Obese individuals with joint pain or degenerative musculoskeletal conditions may also benefit, as EMS minimizes orthopaedic strain while activating large muscle groups [39]. Furthermore, EMS may improve engagement in individuals with low adherence to structured exercise programs due to motivational or logistical barriers [40]. As a non-volitional modality, EMS can serve as an introductory intervention, facilitating gradual transition to voluntary resistance or aerobic training [41]. Importantly, EMS should not be viewed as a replacement for progressive resistance training but rather as a complementary or alternative strategy when conventional exercise is contraindicated or poorly tolerated [42]. The ability to achieve comparable reductions in BMI and central adiposity broadens therapeutic accessibility and supports individualized metabolic rehabilitation in T2DM [43].

Despite methodological rigor, several limitations warrant consideration. Regional adiposity was assessed using skinfold thickness measurements rather than advanced imaging modalities such as Dual-Energy X-ray Absorptiometry (DEXA) or Magnetic Resonance Imaging (MRI). Although validated and clinically practical, skinfold measurements provide indirect estimates of fat distribution and cannot precisely quantify visceral adipose tissue.

The 3-month follow-up period limits conclusions regarding long-term sustainability of adiposity reduction. Chronic metabolic adaptations require extended monitoring to evaluate durability. Biochemical markers of metabolic regulation, including insulin resistance indices (e.g., HOMA-IR), inflammatory cytokines, and adipokines, were not assessed, limiting mechanistic confirmation of metabolic improvements. Additionally, although standardized education was provided, strict dietary monitoring was not implemented, and nutritional variability may have influenced anthropometric outcomes. Finally, the study population comprised sedentary middle-aged adults with T2DM, which may limit generalisability to younger individuals, highly active populations, or those with advanced diabetic complications.

Future research should incorporate advanced body composition assessment techniques such as DEXA or MRI to precisely quantify changes in visceral adiposity and fat redistribution. Extended follow-up durations (≥12 months) are necessary to evaluate long-term sustainability of intervention effects. Inclusion of metabolic biomarkers—such as HOMA-IR, inflammatory cytokines, and adipokines—would strengthen mechanistic interpretation of physiological adaptations.

Combination protocols integrating EMS with aerobic training should be explored to assess potential synergistic effects on metabolic outcomes. Dose–response studies are needed to determine optimal stimulation parameters, including frequency, intensity, and session duration. Furthermore, evaluating the safety and efficacy of EMS in individuals with advanced diabetic complications, including neuropathy and cardiovascular comorbidities, will help define its role within comprehensive metabolic rehabilitation frameworks.

CONCLUSION

This randomized controlled trial demonstrates that both Electrical Muscle Stimulation (EMS) and Progressive Resistance Training (RT) produce significant reductions in Body Mass Index and regional adiposity in sedentary individuals with Type 2 Diabetes Mellitus. The magnitude of change, particularly in central adiposity, indicates clinically meaningful metabolic improvement. Notably, EMS achieved outcomes comparable to structured resistance training, suggesting that electrically induced muscle contraction is sufficient to activate metabolic pathways associated with adipose reduction. These findings support EMS as a viable non-volitional therapeutic alternative for individuals with limited exercise tolerance, musculoskeletal constraints, or low adherence to conventional exercise programs. Incorporation of EMS into physiotherapy-based diabetes management strategies may expand access to effective, non-pharmacological interventions aimed at improving body composition and reducing cardiometabolic risk. Further long-term studies incorporating advanced body composition imaging and metabolic biomarkers are warranted to confirm durability of effects and elucidate underlying mechanisms.

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