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Original Article

Effects of upper-body resistance exercise training on serum nesfatin-1 level, insulin resistance, and body composition in obese paraplegic men

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ABSTRACT

Background: As a recently discovered adipokine, nesfatin-1 is conducive to insulin sensitivity, lipid profile, energy balance, and probably obesity.
Objective: The aim of the present study was to investigate the effect of upper-body resistance exercise training (RET) on nesfatin-1 levels, insulin resistance, lipid profile, and body composition in obese paraplegic men.
Methods: Twenty obese paraplegic men were randomly assigned into control and upper-body RET groups. Upper-body RET was performed for 8 weeks, 3 sessions per week at an intensity corresponding to 60–80% maximum amount of force that can be generated in one maximal contraction in 5 stations (bench press, seated rows, sitting lat pulldown, arm extension, and arm curls). Body fat percentage was determined according to 4-sites skinfold protocol of Durnin and Womersley and Siri equation. Obesity for spinal cord injury patients in the current study was set at BMI >22 kg/m^2. Data were statistically analyzed by paired and independent *t*-test ($P < 0.05$).
Results: We found significant improvements in serum levels of nesfatin-1 (21.13%), insulin sensitivity (8.95%), and high-density lipoprotein (10.87%). Other lipid profile markers, i.e. low-density lipoprotein (4.32%), cholesterol (8.20%), and triglyceride (15.10%) reduced significantly after upper-body RET. Moreover, upper-body RET led to a significant reduction in body mass index (2.36%), body fat percentage (2.79%), and waist-to-hip ratio (2.40%).
Conclusion: Upper-body RET improved insulin sensitivity, lipid profile, and body composition in paraplegic men. Serum nefastin-1 may be a potential marker of success in weight management in this population.

In a survey published by the World Health Organization, the average prevalence of disability was reported as 11.8% and 18% in higher and lower income countries, respectively.[1] In the United States, 22% of adults (or 53.3 million) are disabled.[2] These is a population consisting of people with limitations in physical mobility, and sensory and cognition functions. The largest subgroup of this population (13%) comprises individuals with physical disability.[3] People with disabilities are more likely to be inactive when compared to people without disabilities.[4] People with mobility impairments have a limited opportunity to cooperate in exercise training. Therefore, people with mobility disability might be more prone to obesity and obesity-related disease factors than non-disabled individuals.[5] In addition to energy storage, adipose tissue acts as an endocrine organ that secretes biologically active substances called adipocytokines.[6] Nesfatin-1 is one of these adipokines that is secreted from the adipose tissue.[7–10]

Nesfatin-1, a calcium and DNA binding peptide, originates from nucleobindin 2 (NUCB2) precursors.[7,8] In addition to being secreted

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from the central and peripheral nervous system, this peptide is expressed and released from endocrine cells in the pancreatic beta-cells, muscle and adipose tissue.[7] The circulation level of nesfatin-1 is regulated by hunger and satiety such that serum levels of this peptide increase after a meal and decrease during fasting.[8] It is considered as a new appetite-suppressant factor and modulator of energy balance that reduces body weight gain.[7,9] The recent literature highlights the anti-hyperglycemic effect of nesfatin-1.[8–12] In this context, it has been reported that intravenous injection of nesfatin-1 in a time-, dose-, and insulin-dependent manner significantly reduced blood glucose in hyperglycemic db/db mice.[9] In addition, increased expression of mRNA and protein contents of nesfatin-1 and NUCB2 have been shown in muscle and adipose tissues of patients with type 2 diabetes mellitus.[10]

Nesfatin-1 treatment (both in 1 μg/kg and 10 μg/kg doses) is reported to decrease blood glucose level and improve insulin sensitivity and lipid disorder by activating AMP-activated protein kinase in streptozotocin-induced type 2 diabetic mice.[8] Furthermore, it has been demonstrated that continuous peripheral infusion of nesfatin-1 increases insulin secretion both in vivo and in vitro in cultured MIN6 cells; it also up-regulates the phosphorylation of protein kinase B in pancreas and MIN6 islet cells.[11] At the cell surface, glucose transporter-4 (GLUT4) protein facilitates the diffusion of extracellular glucose into insulin-sensitive cells.[13] GLUT4 proteins are stored in vesicles inside the cells[13] and stimulate intracellular signaling cascades that result in the translocation of the GLUT4 storage compartments into the plasma membrane and GLUT4 mediated glucose uptake in the skeletal muscle and adipose tissue.[11] The notion of nesfatin-1improving insulin sensitivity is supported by the fact that continuous subcutaneous infusion of this peptide increased circulating insulin, reduced blood glucose and decreased circulating glucagon levels during an oral glucose tolerance test in male Fischer 344 rats.[12] Intravenously injected nesfatin-1 in freely fed leptin receptor-deficient db/db mice reduces blood glucose levels, indicating that nesfatin-1 improves insulin sensitivity.[9] Collectively, these findings suggest that nesfatin-1 improves insulin sensitivity and regulates intracellular glucose metabolism in the peripheral tissues.[8–12]

Exercise is an effective method for weight management and improvement of insulin resistance and obesity-induced risk factors in normal and obese individuals.[14–17] In this context, it has been reported that high-intensity interval training increases plasma nesfatin-1 levels significantly; however, fasting glucose, insulin and homeostasis model assessment-estimated insulin resistance (HOMA-IR) decreases significantly after this type of exercise training in overweight men.[18] Although high-fat diet results in reduced plasma levels of nesfatin-1, this decrease is controlled and suppressed by the running wheel in mice.[19] Additionally, moderate-intensity cycle ergometer raises plasma levels of nesfatin-1 in obese women.[20] Moreover, both short- and long-term aerobic exercise training was effective in reducing HOMA-IR and fat mass in the upper abdominal region of overweight/obese women.[21] A reduction in total cholesterol (TC), triglyceride (TG) and fat mass is also reported after both short-term aerobic and combined exercise trainings in overweight women.[22]

Furthermore, a meta-analysis reports that aerobic exercise training can significantly enhance the decrease of TG level in obese or overweight adults, but does not affect the TC, high density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and fasting blood-glucose.[23] Interestingly, an 8-week strength exercise training at an intensity corresponding to 50–80% of repetition maximum improves insulin sensitivity and HDL-C levels without changing body composition in overweight and obese individuals.[24] Finally, it has been recently reported that combined aerobic and resistance exercise using outdoor exercise machines improves fitness and HOMA-IR in elderly participants.[25]

Despite the well-known benefits of physical activity, people with impaired mobility are more likely to be inactive than populations without mobility limitations[4] and are prone to obesity.[5,26,27] Prevalence rates of obesity and extreme obesity in people with impaired mobility are 41.6% and 9.3%, respectively.[26,27] In addition, individuals with mobility limitations are at a relatively increased risk for total cholesterol, hypertension, cardiovascular disease, and diabetes compared to the healthy population.[28] Therefore, adults with mobility impairment are at an increased risk of premature death.[4] Given their limited ability to participate in aerobic exercise (such as jogging, walking and cycling), a sensible strategy would be to emphasize the upper body muscle strengthening.[28] Resistance exercise training improves insulin sensitivity in diabetic patients through increased GLUT4 translocation in the skeletal muscle.[15] In addition, resistance exercise training increases post-exercise oxygen consumption, energy expenditure during the recovery period[15] and causes a reduction in tumor necrosis factor alpha, interleukin-6, HOMA-IR, and glycosylated hemoglobin.[17] In individuals with increased insulin resistance, resistance exercises increase muscle mass and strength.[16] Moreover, a significant decline in fasting blood glucose, blood pressure, lipid profile, and subcutaneous adipose tissue compartments has been observed after resistance exercise regimen in patients with type 2 diabetes.[29,30] Therefore, resistance exercise training has been suggested for individuals with prediabetes and increased insulin resistance.[31] To the best of our knowledge, the effects of resistance exercise training on nesfatin-1, insulin resistance and lipid profile of obese paraplegic men have not yet been well examined. Thus, we have conducted the current study to investigate the changes of nesfatin-1, insulin resistance and lipid profile after resistance exercise training in obese paraplegic men with lower limb paralysis.

Methods

Participants

Twenty adult obese paraplegic men with chronic lower limb paralysis induced by spinal cord injuries were recruited from the Zahedan city, eastern Iran, to participate in this quasi-experimental study. The inclusion criteria were: 1) all lesions were complete and lesion levels were T9-T12; 2) all lesions were traumatic duo to physical trauma; 3) all subjects were physically inactive in training after occurrence of lower limb paralysis; 4) all participants were examined by a physician and received medical approval for participation in physical activities; 5) they were able to sit down while maintaining upper-body balance; and 6) all participants only used wheelchairs without any short leg braces + crutches, long leg braces + walker and crutches. They were free from pressure sores, bladder infections, and potentially damaging metabolic and cardiovascular limitations. The subjects were fully informed of the possible risks and benefits. Then, written voluntary consent form was signed by all subjects. In addition, it has been reported that the prevalence of adverse events during physical activity in patients with spinal cord injuries ranges from 0% to 4%.[32] Participants were screened for cardio-respiratory abnormalities using the Physical Activity Readiness Questionnaire (PAR-Q).[33] Participants were randomly allocated into equal groups of control (C) and resistance exercise training (RET). The demographic data of the subjects are shown in Table 1.

Table 1
The demographic data of the subjects.

Demographic variable	C (n = 10)	RET (n = 10)
Age (years)	25.50 ± 3.24	25.33 ± 3.02
Duration of paralysis (months)	117 ± 42	111 ± 51
Marital status		
Married	3 (30%)	2 (20%)
Single	7 (70%)	8 (80%)
Employment status		
Unemployed	9 (90%)	10 (10%)
Employed	1 (10%)	0 (0%)
Etiology		
Motor vehicle and car accident	9 (90%)	8 (80%)
Fall	1 (10%)	2 (20%)
Smoking		
Yes	3 (30%)	1 (10%)
No	7 (70%)	9 (90%)
Neurological status		
T9	2 (20%)	1 (10%)
T10	2 (20%)	2 (20%)
T11	0 (0%)	2 (20%)
T12	6 (60%)	5 (50%)
Height (cm)	174.50 ± 4.24	169.60 ± 1.95
Body weight (kg)	87 ± 4.66	84.95 ± 5.32

Values are means ± SD.
C: control, RET: resistance exercise training.

Anthropometric measurements

To determine anthropometric measurements, the participants were dressed in their usual indoor clothing without shoes. Height was segmentally measured from heel to knee, knee to hip, and hip to head using an elastic tape measure on the length board. In this condition, the participants' feet were placed in dorsal flexion and legs were straightened.[34] Body weight (nearest 0.1 kg) was measured using a digital scale (Sahand CO. Tabriz, Iran). Body Mass Index (BMI) was calculated for each patient by dividing the weight in kg by the height in m^2. Obesity in the current study was set at BMI >22 kg/m^2. The Waist-Hip Ratio (WHR) was calculated as the waist circumference in centimeters (at the smallest circumference of the waist, just above the belly button) divided by the hip circumference in centimeters (at the widest part of the buttocks). Body fat percentage was predicted from the skinfold measurement taken on the right side of the body using specialized calipers (Yagami model, Japan). A 4-sites skinfold (triceps, biciptal, subscapular and suprailiac) protocol of Durnin and Womersley was used to estimate body density.[36] Finally, body fat percentage was calculated using the Siri equation.[36] All anthropometric measurements were measured before and after the upper-body RET protocol.

Upper-body RET protocol

Initially, the subjects were familiarized with the correct lifting techniques. Then, one-repetition maximum (1-RM) of all the movement was determined according to Brzycki Formula.[37] 1-RM is the maximum amount of weight that a person can possibly lift in one repetition.[37] The subjects exercised on five resistance exercise machines in the following order on the basis of overload principle: bench press, seated rows, sitting lat pulldown, arm extension, and arm curls (8 weeks, three times a week, 3 sets/exercise, 12 repetitions/set). Upper-body RET started at an intensity of 60% 1-RM and gradually increased to 80% 1-RM in the final sessions. At the beginning and end of upper-body RET, warm-up and cool-down were performed for 10 min. Participants in the RET group were not allowed to perform any additional exercises during the training period. In addition, participants in the C group were asked to maintain their usual lifestyle pattern and to refrain from any new exercise programs during the study. Attendance of the participants in the current protocol was recorded by a physical education instructor. Adherence to 24 sessions of upper-body RET was 100% for all the participants in the RET group.

Diet

A week before the protocol initiated, a 24-h diet recall interview was conducted for three consecutive days to analyze participants' diet. During the interview, the subjects were asked to recall all the drinks and food they had consumed in the past 24 h.[38] The results of the nutritional analysis demonstrated that the calorie intakes from carbohydrate, protein and fat were 57%, 15%, and 28%, respectively. Participants were asked to maintain their normal eating habits and avoid any antioxidants and nutrition supplements for stimulating lipolysis during the training weeks. In addition, they were instructed not to consume meals with a high fat content.[39] Moreover, none of the subjects had a history of using lipid-lowering drugs.

Biochemical assays

Fasting blood samples were taken from the antecubital vein before and 48 h after the last exercise training session. Blood samples were then centrifuged (Eppendorf Centrifuge, Mini Spin R, Germany) at 3000 rpm for 15 min at 4 °C. Serum was isolated and stored at - 80 °C until analyses were performed. The serum nesfatin-1 (Phoenix Pharmaceutical, Inc., California, USA) and insulin (Demeditec Diagnostics GmbH., Lise-Meitner-Straße, Germany) levels were measured using the ELISA kits according to the company manual. The glucose and lipid profile levels were measured by enzymatic methods (Parsazmoon Co., Karaj, Iran). Finally, insulin resistance was calculated using HOMA-IR.[40]

Statistical analysis

Statistical analyses were performed with Statistical Package for the Social Sciences (SPSS) version 16. Normality of data was confirmed by Kolmogorov–Smirnov test. Paired *t*-test and independent *t*-test were used to examine the intra- and inter-group differences, respectively. All data are expressed as means ± standard deviation. P values < 0.05 were considered statistically significant.

Results

In the context of anthropometric measurements, the results indicated no significant differences between the baseline values of BMI (P = 0.828), WHR (P = 0.802), and body fat (%) (P = 0.741) in C and RET groups. Upper-body RET resulted in significant reduction in BMI (P = 0.010), WHR (P = 0.015) and body fat (%) (P = 0.019). However, our results revealed only significant differences in WHR between C and RET groups after upper-body RET (P = 0.027). The changes in anthropometric measurements are shown in Table 2.

The differences in lipid profile measurements are shown in Table 3. Baseline levels of TG (P = 0.667), TC (P = 0.083), LDL (P = 0.875), and HDL (P = 0.376) did not differ between C and RET groups. Upper-body RET significantly reduced TG (P = 0.001), TC (P = 0.001), and LDL (P = 0.001) levels. In turn, serum HDL levels (P = 0.001) increased significantly following upper-body RET. In addition, our results revealed significant differences in TG (P = 0.001), TC (P = 0.001), and LDL (P = 0.001) between C and RET groups after upper-body RET.

Table 2
Anthropometric measurements of the subjects.

Variables	Group	Pre-test	Post-test
BMI (kg/m^2)	C	24.91 ± 0.98	25.14 ± 1.01
	RET	25.33 ± 1.37	24.73 ± 1.24 [a]
WHR (cm)	C	0.83 ± 0.14	0.84 ± 0.02
	RET	0.83 ± 0.02	0.81 ± 0.01 [b,a]
Body fat (%)	C	32.60 ± 2.17	32.90 ± 2.23
	RET	32.20 ± 2.08	31.30 ± 2.21 [a]

Values are means ± SD.
C: control, RET: resistance exercise training.
[a] : significantly different within group by the paired *t*-test,[#]: P < 0.05.
[b] Significantly different by the independent *t*-test,[*]: P < 0.05.

Table 4
Insulin resistance measurements of the subjects.

Variables	Group	Pre-test	Post-test
Glucose (mg/dl)	C	98.10 ± 25.54	97.90 ± 24.19
	RET	98.30 ± 11.43	91.50 ± 7.70
Insulin (uμ/ml)	C	16.80 ± 3.59	16.30 ± 3.27
	RET	15.93 ± 2.81	15.58 ± 2.81
HOMA-IR	C	7.27 ± 2.09	7.02 ± 1.88
	RET	6.92 ± 1.27	6.30 ±1.01[a]

Values are means ± SD.
C: control, RET: resistance exercise training.
[a] : significantly different within group by the paired *t*-test,[#]: P < 0.05.

The results of the changes in insulin resistance markers, both within and between the groups, are shown in Table 4. Our results showed no significant differences in baseline levels of glucose (P = 0.982), insulin (P = 0.555), and HOMA-IR (P = 0.666) between C and RET groups. However, within-group comparisons showed that upper-body RET only resulted in a significant reduction of HOMA-IR (P = 0.029). Upper-body RET resulted in non-significant reduction in glucose (P = 0.092) and insulin levels (P = 0.198). In contrast, no significant differences were observed in glucose (P = 0.436), insulin (P = 0.608), and HOMA-IR (P = 0.298) between the two groups after intervention.

In the context of nesfatin-1, the exercise group showed a significant improvement (p = 0.001) from 11.26 ± 2.52 to 13.64 ± 2.02 ng/dl, after 8 weeks. However, the control group did not show a significant difference in nesfatin-1 levels between pre- (11.63 ± 2.23 ng/dl) and post-intervention (10.71 ± 1.75 ng/dl) (P = 0.076). Statistically significant differences were found in nesfatin-1 levels between the two groups after intervention (P = 0.003). Nesfatin-1 change is depicted in Fig. 1.

Discussion

Sedentary lifestyle contributes to the epidemic of obesity and obesity-associated diseases. However, exercise is a practical intervention in improving insulin sensitivity and managing body composition in obese population with inactive lifestyle.[21] Here, an experimental model RET was shown for the first time to result in significant increases in nesfatin-1 concentration and insulin sensitivity in obese paraplegic men with lower limb paralysis. In addition, we observed an improvement in lipid profile and body composition after upper-body RET.

There is now a substantial body of evidence suggesting that exercise training improves insulin sensitivity through increasing bioactive factors released from adipose tissue in healthy individuals.[14,18,21] Nesfatin-1 is synthesized in the central and peripheral nervous system as well as in the adipose tissue and is subsequently released into circulation.[7] Nesfatin-1 exerts multiple actions at the level of nervous and digestive systems, and plays a role in stress response and thermogenesis. Nesfatin-1 not only suppresses food intake and appetite but also affects energy expenditure and glucose homeostasis.[41] Li and colleague[11] reported that nesfatin-1 alters glucose homeostasis by mechanisms which increase glucose uptake and insulin sensitivity. In this regard, our findings showed that serum nesfatin-1 and insulin sensitivity increased after upper-body resistance exercise training in obese paraplegic men. Results of the present study are consistent with other studies that showed concurrent increase in nesfatin-1 and insulin sensitivity following high-intensity interval training[18] and moderate-intensity cycle ergometer[20] in overweight/obese women and men without mobility limitations.[18,20] Moreover, Chaolu et al.[19] observed that the reduction in nesfatin-1 induced by high-fat diet is suppressed by the running wheel in mice. Intriguingly, Le et al.[21] reported that both short- and long-term aerobic exercise training protocols improve HOMA-IR and fat mass in the upper abdominal region of overweight/obese women. Nesfatin-1 reduces blood glucose by activating AMP-activated protein kinase, up-regulating the phosphorylation of protein kinase B in pancreas, and increasing glucose transporter-4 membrane translocation in skeletal muscle and adipose tissue which in turn improves insulin sensitivity.[8,21] In addition, a significant positive correlation has been reported between plasma nesfatin-1 and insulin sensitivity in response to exercise in overweight men[14] and polycystic ovary syndrome patients.[42] Hence, these pieces of evidence suggest that changes in insulin sensitivity may be associated with increase in nesfatin-1 levels after upper-body resistance exercise trainings in obese paraplegic men.

Table 3
Lipid profile measurements of the subjects.

Variables	Group	Pre-test	Post-test
TG (mg/dl)	C	159.80 ± 9.70	161.20 ± 9.78
	RET	158.20 ± 6.28	134.30 ± 7.58 [b,a]
TC (mg/dl)	C	185 ± 4	186.50 ± 4.24
	RET	180.30 ± 7.02	165.50 ± 5.89 [b,a]
LDL(mg/dl)	C	109 ± 4.59	110.80 ± 3.76
	RET	108.70 ± 3.74	104 ± 1.94 [b,a]
HDL(mg/dl)	C	44.60 ± 4.32	45.10 ± 4.45
	RET	43.20 ± 2.25	47.90 ± 3.63 [a]

Values are means ± SD.
C: control, RET: resistance exercise training.
[a] : significantly different within group by the paired *t*-test,[#]: P < 0.05.
[b] : significantly different by the independent *t*-test,[*]: P < 0.05.

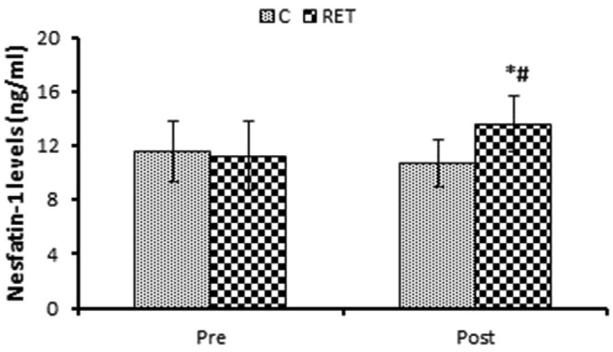

Fig. 1. Effect of upper-body RET on nesfatin-1 level in obese paraplegic men. The asterisk (*) indicates a significant difference from the baseline value in the same group. The hash sign (#) indicates a significant difference between groups.

Gupta et al.[35] considered spinal cord injury patients with BMI of 30 kg/m^2 or above as obese. However, it has been reported that the mean BMI in patients with spinal cord injury ranges from 23.1 to 25.7 kg/m^2 whereas body fat percentage in these patients ranges from 27.5 to 36.3%.[34] This finding suggests that BMI underestimates obesity. In addition, Laughton et al.[34] reported that people with chronic spinal cord injury and BMI values > 22 kg/m^2 should be considered as being at high risk for obesity and obesity-related chronic diseases. Therefore, obesity for spinal cord injury patients in the current study was set at BMI >22 kg/m^2. Individuals with mobility disability present higher BMI and body fat over time than non-disabled individuals.[5] However, our findings showed that upper-body resistance exercise training resulted in a significant reduction in BMI, WHR, and body fat percentage in obese paraplegia men. These findings are consistent with studies that demonstrate significant reduction in weight and BMI in overweight or obese individuals with permanent impaired mobility after long-term lifestyle intervention (i.e., reduced calorie and fat intake and increased exercise).[26] Nesfatin-1 as a recently discovered hormone acts upon energy balance and probably obesity.[42] Nesfatin-1 reduces food intake by exerting an impact on hypothalamic nuclei involved in the regulation of food intake, i.e., supraoptic nucleus, lateral hypothalamic area, arcuate nucleus, and paraventricular nucleus.[43] In this context, it has been reported that dose-dependent injection of nesfatin-1 into the third brain ventricle reduces the dark phase food intake in freely fed rats.[44] Moreover, nesfatin-1's food intake inhibitory effect is mediated by increasing vagal afferent activity, which in turn inhibits gastric emptying and reduces gastroduodenal motility.[41,43] In addition to its role in food intake regulation, nesfatin-1 participates in the regulating energy expenditure.[41] In this regard, it has been demonstrated that nesfatin-1 results in an increase in core body temperature by enhancing the expression of uncoupling protein 1 in the brown adipose tissue.[45] According to a literature review, nesfatin-1 is likely to respond significantly to long-term exercise training and a significant loss of weight and fat percentage.[46] On the other hand, increased levels of this adipocytokine induced by exercise can act as stimuli to reduction of visceral and subcutaneous fat.[47] As such, a negative correlation has been reported between nesfatin-1 and variables such as BMI and body fat percentage.[48] Interestingly, it has been demonstrated that intravenous injection of nesfatin-1 increases the expression of enzymes involved in fatty acid oxidation, i.e., phosphorylated AMP-activated protein kinase and phosphorylated acetyl CoA carboxylase in the skeletal muscle of type 2 diabetic mice.[8] In reality, these findings point out to overall beneficial aspects of nesfatin-1 on lipid metabolism through promoting peripheral lipid utilization.[49] Taken together, a reduced level of BMI, WHR, and body fat percentage induced by the present upper-body resistance exercise training may be due to modification in nesfatin-1.

In the context of lipid profile, upper-body RET regimen dramatically increased HDL levels in obese paraplegic men, while it significantly reduced LDL, cholesterol, and triglyceride levels. Our findings are consistent with other studies that showed a significant reduction in cholesterol, TG and LDL after moderate and high-intensity resistance training intensity,[50] jogging,[51] and short-term combined exercise training[22] in overweight/obese adults. Moreover, results of the present study are consistent with Jiménez and colleagues' findings[24] that report a significant increase in HDL-C levels without changing body composition among overweight/obese individuals who had no mobility disorders. However, Akkurt et al.[52] reported that short-term aerobic arm ergometer exercise (3 days/week; 1.5 h/week, at an intensity corresponding to 50–70% maximal oxygen uptake) had no significant effect on metabolic syndrome parameters (TG, TC, HDL, LDL and fasting blood sugar) in patients with spinal cord injury. Mann et al. in a review, concluded that increased volume of resistance training (in terms of increased number of sets and repetitions) has a greater effect on the lipid profile than increased intensity of resistance exercise training.[53] Moreover, resistance exercise training at an intensity corresponding to 50–85% 1-RM had a greater influence on lipid profile than resistance exercise training at the intensity over 85% 1-RM.[53] These findings confirm the efficacy of our upper-body resistance exercise training on lipid profile of obese paraplegic men due to the suitable volume and intensity applied in the current study. It is hypothesized that resistance exercise training reduces plasma lipid levels by enhancing the ability of skeletal muscles to utilize lipids[54] and increasing lipoprotein lipase activity.[55] Moreover, in a recently published study, the chronic infusion of nesfatin-1 in mice fed with normal or high fat diet reduced plasma levels of triglyceride and cholesterol as well as the diameter of lipid droplets.[56] Thus, one of the mechanisms involved in the observed reduction of lipid profiles in the current study may be due to the lipid metabolism effects of nesfatin-1.

One of the limitations of this study was the lack of a controlled diet. The participants were asked to maintain their normal eating habits, caloric intake and nutrient type; however, there is no documentation that these patients kept the same dietary habits. Amount of consumed calories and type of nutrition can affect the circulating nesfatin-1 levels, lipid profile, and body composition[57] and nesfatin-1 influences food intake.[43] Future research should include a detailed record of food intake throughout the study.

Conclusion

The present study emphasized the fact that more attention should be paid to exercise training in people with mobility impairments. In fact, the novel aspect of the current study is that upper-body RET improves insulin sensitivity, lipid profile, and body composition in obese paraplegic men. In addition, the results of the current study highlight the beneficiary effects of upper-body RET on serum nefastin-1 as a potential marker of weight management success. Therefore, upper-body resistance exercise training at moderate intensity can be used as a preventive and therapeutic approach to improve body composition and weight management and reduce obesity-related risk factors in obese people with lower limb paralysis.

Author contributions statement

All authors conceived the study and its design and coordination. MM, HT, SAP-B and AT were involved in the data collection, data analysis, and drafting of the manuscript. Finally, all authors read and approved the final version of the manuscript, and agreed with the order of presentation of the authors.

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Conflicts of interest

There are no conflicts of interest.

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