ORIGINAL RESEARCH ARTICLE

The ameliorative effects of graded intensities of exercise training on anthropometrical parameters on high fat diet and sucrose-induced obesity in Wistar rats

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ABSTRACT

Obesity is a condition characterised by the expansion of visceral adipose tissue (VAT) depots, leading to its abnormal function, which is associated with the development of insulin insensitivity, cardiovascular disorders, and cancer. This study aims to evaluate the effects of graded intensities of exercise training on visceral adipose tissue weight in the various depots and anthropometrical parameters in Wistar rats with a high-fat diet and sucrose (HFDS)-induced obesity. The study had two phases: the induction of obesity and the intervention phase. In the induction phase, 25 male Wistar rats, 7 weeks old, were randomly grouped into the control group (c) of five rats and the experimental group of 20 rats. The control group was given free access to a normal rodent diet containing 5% fat. The experimental group received a high-fat diet containing 30% fat and drank 60% sucrose ad libitum (HFDS) for 12 weeks. In the second phase, the HFDS-induced rats were randomly grouped into four groups, as follows (n = 5 per group): sedentary, low, medium, and high-intensity exercise training groups. The exercise training was done by swimming in modified pools of 50 cm in height and 30 cm in diameter as follows: low intensity 20 to 59 min/day with a 0% to 3% overload, moderate intensity 60 to 89 min/day with a 0% to 5% overload, and high intensity 90 min/day with more than 10% overload for five days/week for eight weeks. The terminal body weight, body mass gain, and body mass index (BMI) were lower in all training and exercise group rats than in the sedentary group. In addition, the training groups significantly decreased the VAT weights in the perigonadol, mesenteric, and retroperitoneal depots. Exercise training at medium-to-high intensities ameliorates the weight of visceral tissue and attenuates anthropometrical parameters.

Keywords: High fat diet; obesity; body mass index; visceral adipose tissue, exercise training

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1.0 Introduction

Excessive deposition of visceral adipose tissue (VAT), also known as visceral obesity, is a global public health concern that is on the increase (Shuster et al., 2012; West-Eberhard, 2019). Recently, VAT, mainly a white adipose tissue depot, has been considered the single most important factor associated with a systemic low-grade chronic pro-inflammatory metabolic state (Revelo et al., 2014; Verboven et al., 2018). For some metabolic signals, specialised cells, including adipocytes, T-cells, and macrophages, are responsible for the activation of systemic chronic inflammatory responses through the production of various adipokines and cytokines (Itoh et al., 2011). Furthermore, there is evidence that the amount of VAT in various depots may be an essential determinant of inflammatory cytokine production, including interleukin-1β, tumour necrosis factor-alpha (TNF-alpha), and interleukin-6 (Verboven et al., 2018).

Additionally, various lifestyle diseases have been associated with chronic systemic low-grade inflammation, such as insulin resistance, dyslipidemia, type II diabetes, and cardiovascular disease (Itoh et al., 2011). As such, visceral adiposity is a key contributor to the increased morbidity and mortality from chronic non-communicable diseases due to their associated complications like increasing insulin resistance in type II diabetes, cardiovascular disease complications leading to sudden heart attacks, as well as triggering the onset of various forms of cancers ((Chooi et al., 2019; Hruby & Hu, 2015).

Further, most of the studies on obesity are geared toward the preventive or alternative effects of VAT accumulation, because the latter has been associated with a chronic, low-grade inflammatory process. Exercise training, one of the non-pharmacological management strategies for visceral obesity, has been shown to reduce the chronic systemic low-grade inflammatory process in both experimental animals and humans (Gonzalo-Encabo et al., 2021; Park et al., 2014). Therefore, increasing physical activity has emerged as a crucial aspect of the non-pharmacological management of visceral obesity.

Though various methods of controlling the deposition of lipids in visceral adipose tissue have been developed, adopting the most effective intensity of training exercise is always a challenge due to the paucity of data on the effectiveness of the various treatment methods. As such, the contextual framework of this study is to evaluate the restorative effects of exercise training on the anthropometric parameters and visceral adipose tissue depot weights in HFDS obesity-induced Wistar rats. Further, we sought to establish the relationships between anthropometric measurements and visceral adiposity.

2.0 Materials and method

2.1 The experimental rats and materials

In this study, a total of 25 male Wistar rats from the Lower Kabete Veterinary Animal House at the University of Nairobi were employed. These rats were housed in typical polycarbonate rodent cages and kept in humid tropical environments with 12-hour light-dark cycles. Paper shavings were utilised as bedding, which was changed every other day. Each cage was identified with a card that included the experiment number, start date, caloric inhibition level,

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exercise intensity, age, species, and sex of each animal. The experimental group was fed a high-fat diet (HFD) and a 60% sucrose solution ad libitum. The control group was put on regular rat pellets from Unga Limited and water as needed. The rats were handled according to the guidelines for the care and use of laboratory rats (Council, 2011).

2.2 Induction of obesity in the experimental group

Induction of obesity in Wistar rats was carried out as per the procedure outlined by Mutiso et al., 2014, and Novelli et al., 2007. The rats in the experimental group were fed on an HFD containing 30% fat, supplemented with a 60% sucrose solution, while the control group was fed on a normal diet consisting of 5% fat for 12 weeks.

The HFD was prepared daily in the morning and was composed of 30 grams of fat per 100 grams of rodent pellets (25 grams of vegetable oil and 5 grams of peanut oil). To formulate the HFD feeds, vegetable oil was thoroughly mixed with the commercially standard rodent pellet food obtained from Unga Feeds Kenya Limited, Nairobi, Kenya. The vegetable oil was used due to its palatability and high digestibility of 99.7%. The 60% sucrose solution was comprised of 60 grams of cane sugar dissolved in 100 ml of water. The standard rodent pellets contain 5 grams of soybean oil per 100 grams. All the essential minerals and vitamins required for rats were kept equal for both the HFD and normal diet feeds. Successful obesity induction in the rats was determined by a BMI of greater than 0.68 g/cm^2 as previously described by Novelli et al., 2007. After twelve weeks of HFD, rats that did not achieve the BMI requirements were removed from the experimental group.

2.3 Exercise training protocol

2.3.1 Procedure for anthropometric measurements

The anthropometric measurements were done using the procedure described by Novelli et al., 2007. The following anthropometric measurements were made: the abdominal circumference (AC), which was taken immediately anterior to the forefoot; the thoracic circumference, which was measured immediately behind the foreleg; and body length, which was measured from nose-to-anus or nose–anus length. These anthropometric measurements were determined in all rats on days 0, 84, and 140. The measurements were made in anesthetized rats (0.1 mL intraperitoneal of 1% sodium barbiturate). Throughout the investigation, the body weights were measured every day using an electronic weighing device with excellent precision. These body weights and body lengths were used to determine the following anthropometrical parameters: BMI = body weight (g) / length (cm^2); Lee index = cube root of body weight (g) / nose-to-anus length (cm) (Bernardis, 1970). The specific rate of body mass gain was calculated using Bernardis' (1970) formula as follows: g/kg = dM/Mdt, where dM represents the gain of body weight during dt = t2−t1, and M is the rat's body weight at t1 (initial weight), while t2 was the terminal body weight.

The exercise training for swimming was adopted and implemented as per Emami et al.'s (2016) protocol. Before starting the experimental exercise training, the rats were first trained in the water. This adaptation involved swimming in water that was heated and kept at 31.1 C for 30

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minutes, once a day for five days, in water tanks that were 50 cm in height and 30 cm in diameter. Following adaptation, the rats underwent experimental exercise training by swimming for 60 minutes each day, five days a week, for eight weeks. A graded exercise protocol was used, with incremental loads (percentages of the rats' body weight) adjusted at the base of the rats' tails to guarantee the exercise intensity was attained in a shorter duration. During the experimental period, the rats were fed a normal rodent diet containing 5% fat.

The following levels of daily exercise time and workload were used to determine the intensity of swimming:

Table 1: Showing Intensity, Duration, and Weight Overload in Percentage During Swimming Exercise.

Exercise intensity	Duration (minutes)	Overload (%)
Low intensity	20-59	0-3
Moderate	60-89	0-5
High	90	>10

The intervals needed to remove the rats from the water tank and change the loads often lasted between 15 and 18 seconds. During the test, trained research personnel familiar with the performance protocol observed the animals performing the exercise. When an animal showed a loss of coordination and failed to emerge within 10 seconds three times in a row, it was deemed to have attained fatigue; hence, it was removed from the swimming tank, dried, and returned to its cage.

3.0 Results

Table 2 shows the results of a one-way between-subjects ANOVA that was conducted to compare the effect of exercise training on obesity in the following anthropometric measurements: terminal body weights, body weight gain or loss, specific rate of body mass gain or loss (g/kg), NAL, AC, TC, Lee index, and BMI in low, medium, and high intensities of exercise training.

There was a significant effect on the terminal weight following exercise training for the four groups [F (3, 16) = 21.26, p <.001]. Post hoc comparisons using the Tukey HSD test indicated that the mean weight for the SED HFDS control group (M = 471.2, SD = 20.911) was significantly (p =.001) different from that of the HFDS-LIE group (M = 375.2, SD = 18.13), the HFDS-MIE group (M = 396.2, SD = 38.38), and the HFDS-HIE group (M = 347.8, SD = 19.88). However, the LIE (M = 375.2, SD = 18.13) did not significantly differ from the HFDS-MIE and HFDS-HIE. Further, there was a significant effect on body weight gain or loss in g/day on exercise training at the p<.05 level for the four groups [F (3, 16) = 32.558, p <.001].

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Post hoc comparisons using the Tukey HSD test indicated that the mean feed efficiency for the SED HFDS control group (M = 9.57, SD = 3.3) was significantly (p=0.001) different than the HFDS-LIE (M = -17.93, SD = 10.21), HFDS-MIE (M = -17.54, SD = 7.38), and HFDS-HIE (M = -28.4, SD = 2.82).

Table 2: Effect of graded intensities exercise training on Anthropometric parameters in HFDS obesity-induced Wistar rats

	SED-HFDS	**HFDS-LIE**	**HFDS-MIE**	**HFDS-HIE**	**F (3,16)**	**P-value**
Initial Body weights (g)	430.2±17.6	439.4±22.1	453.4±30.16	445.8±7.33	1.099	0.378
Terminal Body weights (g)	471.2±20.91^{a}	375.2±18.13^{b}	396.2±38.38^{c}	347.8±19.88^{c}	21.264	0.000
Feed Consumption (g/day)	34.32±1.51^{a}	28.66±1.35^{b}	27.4±1.15^{bc}	26.18±1.2^{c}	37.884	.000
Energy intake (kJ/day)	431.06±18.91^{a}	359.97±16.9^{b}	344.14±14.36^{bc}	328.82±15.11^{c}	37.884	.000
The specific rate of body mass gain/loss (g/kg)	0.1±0.03^{a}	-0.126±0.06^{b}	-0.14±0.07$^{b c}$	-0.22±0.04^{c}	35.332	0.000
NAL (cm	23.86± 0.89	24.38± 0.91	24.26±0.68	24.24± 0.6	.415	0.745
AC (cm)	17.06±0.46^{a}	15.32±0.62^{b}	14.48±0.64^{bc}	13.96±0.21^{c}	13.47	0.000
TC (cm)	13.98± 0.37^{a}	13.22± 0.24^{b}	13.16±0.51^{b}	13.18±0.26^{b}	5.565	0.006
Lee index	0.326± .01^{a}	0.296± 0.02^{b}	0.303±0.01^{b}	0.29±0.01^{b}	16.339	0.001
Initial BMI (g/cm^2)	0.4460±0.04	0.4427±0.01	0.4241±.06	0.4430±.04	0.297	0.827
Terminal BMI (g/cm^2)	0.828±0.03^{a}	0.673± 0.06^{b}	0.635± 0.07^{b}	0.594±0.06^{b}	9.655	0.000
AC/TC	1.22±0.02^{a}	1.1±0.04$^{a b}$	1.16±0.01$^{b c}$	1.06±0.07^{c}	10.865	0.000

Key: *Values are expressed as mean ± standard deviation*
The comparisons for one-way between-subject across the rows and the values with a different alphabet superscript indicate a statistical difference at P<0.05
SED- No Exercise Sedentary, LIE-Low-Intensity Exercise, MIE- Medium Intensity Exercise, HIE-High-Intensity Exercise, NAL-Nasal Anal Length, AC-Abdominal Circumference, TC- Thoracic Circumference, BMI- Body Mass Index;

Table 3 shows the results of a one-way between-subjects ANOVA that was conducted to compare the effect of exercise training on visceral adipose tissue in the retroperitoneal, perigonadol, and mesenteric depots of obese rats. There was a significant effect of

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retroperitoneal weight depot on exercise training at the p < .05 level for the four groups [F (3, 16) = 12.285, p < .001]. Post hoc comparisons using the Tukey HSD test indicated that the mean retroperitoneal fat weight for the SED HFDS control group (M = 7.413, SD = 1.01) was significantly different from the HFDS-LIE (M = 6.751, SD = 0.64), HFDS - MIE (M = 4.971, SD = 1), and HFDS - HIE (M = 4.275, SD = 0.47). However, the LIE (M = 6.751, SD = 0.64) did not significantly differ from the HFDS-MIE and HFDS-HIE. Further, there was a significant effect of exercise training on perigonadol fat depot at the p<.05 level for the four groups [F (3, 16) = 52.831, p < .001].

Post hoc comparisons using the Tukey HSD test revealed that the SED HFDS control group's mean feed efficiency (M = 8.171, SD =.39) was significantly (p =.001) different from the HFDS-LIE (M = 7.459, SD = 0.7), HFDS - MIE (M = 3.161, SD = 0.69), and HFDS - HIE (M = 2.381, SD = 0.39).

Furthermore, there was a significant effect of exercise training on the mesenteric fat depot at the p<.05 level for the four groups [F (3, 16) = 12.722, p < .001]. Post hoc comparisons using the Tukey HSD test indicated that the mean feed efficiency for the SED HFDS control group (M = 4.97, SD =.19) was significantly (p=.001) different than the HFDS - LIE (M = 3.67, SD = 0.48), HFDS - MIE (M = 3.64, SD = 0.5), and HFDS - HIE (M = 3.317, SD = 0.3).

Table 3 Effects of graded intensities Exercise Training on the weight of the various Visceral Adipose Tissue depots

	SED-HFDS	HFDS-LIE	HFDS-MIE	HFDS-HIE	F (3,16)	P-value
RPF Wt	7.413±1.01 [a]	6.751±0.64 [b]	4.971±1 [b]	4.275±0.47 [b]	12.285	.000
PGF Wt	8.171±.39 [a]	7.459±0.7 [b]	3.161± 0.69 [b]	2.381±0.39 [b]	52.831	.000
MF Wt	4.97±0.19 [a]	3.67±0.48 [b]	3.64±0.5 [b]	3.317±0.3 [c]	12.722	.000

Key: *Values are expressed as mean± standard deviation of the mean*
The comparisons for one-way between-subjects ANOVA were conducted across the rows and the values with a different alphabet superscript indicate a statistical difference at P < 0.05
Retroperitoneal (RPF), Perigonadol (PGF), and Mesenteric (MF) Depot.

4.0 Discussion

In this study, induction of obesity was successful using HFDS feeding for 12 weeks in Wistar rats; this is in agreement with previous studies that have established that 12 weeks of HFDS are adequate for the induction of visceral obesity, body weight, and an altered lipid profile in rats (Marques et al., 2016; Rasool et al., 2018). In addition, the HFDS diets have also been shown to impair glucose metabolism, stimulate abnormal gluconeogenesis, and promote both insulin resistance and a systemic chronic low-grade pro-inflammatory metabolic state (Rasool et al., 2018; Yang et al., 2012). Further, the body weight and food intake of Wistar rats fed the

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HFDS were significantly increased compared to controls on a normal diet (Malafaia et al., 2013; Yang et al., 2012).

The current study showed that the body weights of all HFDS groups, both sedentary and exercise-trained, significantly increased until the end of the experiment compared with the body weights of the normal diet groups. However, the trained groups had a lower body weight than the sedentary groups. These results are similar to the study by Speretta et al. (2012). Therefore, changes in diet are more related to body weight changes than exercise training. Food consumption was low in the HFDS group; this was associated with an increased caloric content in feed. These findings suggest that rats are sensitive to both energy and the HFDS contents of the diet with regards to controlling their food intake by satiety. Moreover, the study finding that the swimming training groups had a lower food consumption and calorie intake compared with the sedentary group agrees with previous studies by Dupas et al. (2018) that showed similar results. Further, Bobbo et al.'s (2021) findings showed that exercise training raises the expression of interleukin-6 (IL-6) and interleukin-10 (IL-10) in the hypothalamus, which in turn raises leptin and insulin sensitivity through the decrease of IkappaB kinase (IKKb) and restricting endoplasmic reticulum (ER) stress, leading to a reduction in food intake. Therefore, the high energy expenditure in exercise training and the lower caloric intake could be attributed to the lower body weight gain found in the exercise-trained HFDS groups.

In the present study, the HFDS diet also promoted higher relative weights of all adipose tissues and a high adipose weight of the perigonaldol, retroperitoneal, and mesenteric adipose tissues. Other studies have demonstrated similar findings (Boa et al., 2017; Speretta et al., 2012). In the HFDS exercise-trained groups, the relative weight of the perigonadol adipose tissue was lower compared with that of the sedentary group. Furthermore, high- to moderate-intensity swimming reduced visceral adipose weight. Our findings are inconsistent with other studies that did not observe a significant decrease in visceral tissue relative to weight in HFDS-induced obesity in exercise-trained rats (Riahi & Riyahi, 2016; Rocha et al., 2016; Vissers et al., 2013). This discrepancy can be attributed to the load that was used in the swimming exercise in the current study (6%–10%) of the rat's body weight. As previously described by Gobatto et al. (2001), these weights on the base of the tail of the rats transitioned the exercise training intensity between aerobic and anaerobic metabolism in rats, promoting a significant rise in catecholamine levels; VAT is highly sensitive to catecholamine because they pose a high number of alpha and beta-adrenergic receptors (Gobatto et al., 2001; Voltarelli et al., 2002):In view of these facts, we highly speculate that the load used by the obese rats in the low-intensity group during swimming was not enough to promote a significant increase in catecholamine levels, resulting in a smaller mobilisation of visceral adipose tissue during the swimming exercise, and that the load did not correspond to the maximum steady-state lactate level, which acts as the gold standard for the identification of the anaerobic threshold in obese rats and is 6% of the rats' body weights.

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Furthermore, the decrease in body weight in high- and medium-intensity exercised trained rats could be attributed to a reduction in the amount of visceral adipose tissue depots, which results in a decrease in the production of sex hormones, leptin-12, and glucose (Giolo De Carvalho & Sparks, 2019). As well as an increase in appetite-suppressing neuropeptide hormone levels, such as nesfatin-1 and peptide YY levels, as well as a negative energy and fat balance associated with increased energy expenditure and fat oxidation during exercise training (Emami et al., 2016; Giolo De Carvalho & Sparks, 2019).

5.0 Conclusion

In conclusion, the present study has demonstrated that both medium and high-intensity exercise training positively attenuated anthropometric parameters, body weight, and adipose weight in obese rats. These findings support that both medium and high exercise intensity seem to be effective in controlling the effects of the consumption of a high-fat diet. However, the influence of each modality on the analysed parameters seems to be different. Therefore, we can suggest that swimming exercise at a high to moderate intensity could offer a great choice for the prevention and/or treatment of obesity. Furthermore, even though exercise has been shown to alter various mechanisms of obesity, many questions remain unanswered. Therefore, more studies are required in the field of histostereology to evaluate the exercise-induced changes associated with obesity.

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None

6.2 Declaration of Interest

None

6.3 Conflict of Interest

None.

7.0 Reference

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