ORIGINAL RESEARCH ARTICLE

The restorative effects of graded intensities of exercise training on the biochemical and nutritional status of obese induced male Wistar rats (*Rattus novegicus*)

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Abstract

The purpose of this study was to evaluate the specific effects of 12 weeks of intensive, graded intensities of exercise training on nutritional parameters and some plasma biochemical parameters in high-fat diet and sucrose obesity-induced (HFDS) Wistar rats. In evaluating the restorative effects of graded exercise training as an intervention in managing obesity, 25 male Wistar rats who were 7 weeks old were used. These 25 rats were broadly grouped into 5 rats as controls and 20 rats as experiments. In the first phase of the experiment, which involved the induction of obesity, the rats were fed on HFDS containing 35% total fat, 39% carbohydrates, 23% protein, 6% fiber per 100 grams, and 60% sucrose solution per 100 mL for 12 weeks, while the 5 rats in the control group were fed on a normal rodent diet containing 5% total fat, 39% carbohydrates, 23% protein, and 6% fiber per 100 grams. In the second phase, which was the interventional phase, the 20 HFDS-induced obese rats were randomly assigned into 4 groups: the control (sedentary group), low, medium, and high-intensity exercise training groups, and were subjected to various intensities of exercise training. All experimental groups received the HFDS throughout the exercise training period. After 84 days of exercise training intervention, the final body weight, plasma biochemical parameters including total cholesterol (TC), triglyceride (TG), and high-density lipoprotein cholesterol (HDLC) concentrations, and nutrition parameters including feed efficiency (FE), feed consumption (FC), and caloric intake (CI) were assessed. The study established that graded exercise training has a direct, inversely proportional reduction in TC, TG, and LDLC concentrations. In conclusion, medium- to high-intensity exercise training is associated with restoring the dyslipidemic profile in the serum of rats and also increased feed efficiency..

Keywords: High fat diet; Obesity; body mass index; lipid profile**.**

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1.0 Introduction

Obesity is a condition characterized by the excessive accumulation of visceral adipose tissue (Kim, 2016). It is defined by a body mass index (BMI) greater than or equal to 30 in adults and a weight-for-height greater than 3 standard deviations above the WHO child growth standard median in children (Apovian, 2016; Caterson & Gill, 2002). Obesity is a global public health concern due to the rising prevalence of 27.5% for adults and 47.1% for children (Apovian, 2016; Engin, 2017), which has been occasioned by an increase in the intake of energy-dense foods that are high in fat and sugar and an increase in physical inactivity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization (Njue et al., 2019; Shuster et al., 2012).

Further, the consumption of a high-fat diet combined with physical inactivity has resulted in an imbalance between the number of calories consumed and the number of calories expended. This imbalance has led to an increase in the accumulation of fat in the visceral adipose tissue (VAT), according to Ahmadi-Kani Golzar et al. (2019). Furthermore, VAT possesses unique biochemical characteristics that influence several normal and pathological processes in the human body (Tandon et al., 2018). In particular, excessive deposition of visceral adipose tissue raises the risk of serious medical conditions, including cardiovascular diseases, Alzheimer's disease, and type 2 diabetes (Liu et al., 2018). As such, visceral adiposity is a major contributor to the increased morbidity and mortality from chronic non-communicable diseases due to their associated complications (Chooi et al., 2019; Hruby & Hu, 2015). In addition, for every five-unit increase in BMI above 25 kg/m2, overall mortality increases by 29 percent, while vascular and diabetes mortality increase by 41 percent and 21 percent, respectively (Apovian, 2016).

The prevalence of obesity is increasing, with more than one billion people globally being obese, of which 650 million are adults, 340 million are adolescents, and 39 million are children. This high prevalence is associated with a global increase in high-fat diets, sucrose (HFDS) consumption, and physical inactivity (Engin, 2017).

According to Oppert et al. (2021), exercise training is a crucial component in the management of obesity. It is typically combined with other interventions such as caloric restriction, behavioral counseling, medication, and, in extreme cases, bariatric surgery. Together, these approaches aim to address the various factors that contribute to obesity and promote weight loss. Physical activity is defined in general terms as any bodily movement produced by skeletal muscles that results in energy expenditure (Shahidi et al., 2020), while exercise training is defined as a subdivision of physical activity that is scheduled, structured, and repetitive with a given purpose to maintain or promote physical fitness (Miko et al., 2020; Saris et al., 2003). Even though the value of physical activity and training exercise in promoting health and prevention of lifestyle diseases is well recognised in public health, its role in weight control in obese subjects remains debated both in the scientific and lay literature (Oppert et al., 2021). Further, how much exercise is needed to lose weight and what type of exercise training

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therapy should be performed to gain optimal benefits is a major question encountered by a clinician when patients ask for help. Although several researchers and organisations have speculated on the amount of physical exercise necessary to prevent weight gain and obesity, the American College of Sports Medicine (ACSM) position statement recommends 150–250 minutes per week of moderate to vigorous PA, with an energy equivalent of 1,200–2,000 kcals per week, while Saris et al. (2003) recommended that PA levels of 225–300 minutes per week were necessary to prevent the transition from normal weight to obesity.

2.0 Materials and method

2.1 The experimental rats and materials

In this study, a total of twenty-five male Wistar rats were obtained from the Lower Kabete Veterinary Animal House at the University of Nairobi. To ensure their welfare, the rats were housed in standard polycarbonate rodent cages and kept in a humid tropical environment under a 12-hour light-dark cycle, as described by Mwangi et al. (2023). The rats were provided with bedding in the form of shredded paper, which was changed every other day to maintain their living conditions. To ensure proper identification of the rats in the study, each cage was labeled with a card displaying essential information such as the experiment number, start date, exercise intensity, age, number of rats, species, and sex of the animal. This method was used to organize and differentiate the rats from one another and to ensure accurate data collection during the study. By using this labeling system, the researchers could easily track and analyze the data obtained from each specific rat, which was crucial for the success of the study.

The handling of the rats was conducted in compliance with the 8th edition of the Guide for the Care and Use of Laboratory Animals (Council, 2011). The study was approved by the University of Nairobi's Biosafety, Animal Use, and Ethics Committee under the reference number FVM BAUEC/2021/322 to ensure that ethical guidelines were followed.

2.2 Induction of obesity in the experimental group

In this study, we induced obesity in the experimental group by adopting a method that has been previously described by Mutiso et al. (2014) and Novelli et al. (2007). Specifically, the rats in the experimental group were fed a high-fat diet (HFD) comprising 30% fat and supplemented with a 60% sucrose solution. The rationale for choosing this diet was based on previous research that has shown it to be effective in promoting adipose tissue growth and weight gain in rats.

On the other hand, the control group was provided with a standard diet consisting of 5% fat, which has been shown to not induce significant metabolic changes or weight gain in rats. Throughout a 12-week period, we carefully monitored the rats' food intake and weight while they were kept on their respective diets.

To create the high-fat diet (HFD), we added 30 grams of fat (25 grams of vegetable oil and 5 grams of peanut oil) per 100 grams of commercially available standard rodent pellet food. The

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rodent pellets had a composition of 39% carbohydrates, 5% total fat, 23% protein, and 6% fiber per 100 grams. By adding the extra fat, the HFD provided a significantly higher fat content than the standard diet. The choice of vegetable oil was based on its high digestibility (99.7%), according to Council, 1995. The emphasis on dietary fat in this experiment meant that the essential minerals and vitamins required for rats were kept equal for both the HFD and normal diets, as described by Reeves et al. (1993). The metabolizable energy for this formulated diet was 12.56 kJ/g.

To prepare the 60% sucrose solution, 60 grams of cane sugar were dissolved in 100 ml of water. The standard rodent pellets' constituents were 5 grams of soybean oil per 100 grams. The successful induction of obesity in rats was determined using the BMI criterion, which was defined as having a value exceeding 0.68 g/cm2. This definition was established in a prior study by Novelli et al. (2007).

Further, to ensure that only rats with induced obesity were included in the exercise training phase, those that did not meet the BMI criteria after twelve weeks of HFD induction were excluded. This approach allowed for precise control over the induction of obesity in the experimental group and ensured that the exercise training phase was conducted on a clearly defined group of rats. This methodology was intended to produce reliable and consistent results.

2.3 Exercise training protocol

The exercise training for swimming in this study was carried out according to the method described by Yang et al. (2016). Prior to the experimental exercise training, the rats first adapted to the water by swimming for 30 minutes, once per day, for five days. The water was heated and maintained at a temperature of 31.1°C, and the water tanks used had a height of 50 cm and a diameter of 30 cm. After the adaptation period, the experimental exercise training in rats was carried out by swimming for 60 minutes per day, five days a week, for eight weeks. A graded exercise protocol with incremental loads was used, and the exercise intensity was adjusted based on the percentage of the rats' body weight at the base of their tails. This was done to ensure that the exercise intensity was achieved in a shorter swim time.

During both the adaptation and experimental periods, both the control and experimental groups were maintained on a normal rodent diet containing 5% total fat, 39% carbohydrates, 23% protein, and 6% fiber per 100 grams. The intensity of swimming was determined based on the daily exercise time and workload. Low intensity was defined as 20 to 59 minutes per day with a 0% to 3% overload; moderate intensity was 60 to 89 minutes per day with a 0% to 5% overload; and high intensity was 90 minutes per day with more than 0% overload. The rats were removed from the water tank, and the loads were changed at intervals of approximately 15 to 18 seconds.

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During the exercise training, trained research personnel monitored the animals to ensure that they adhered to the swimming protocol. This was done to guarantee that the rats' swimming exercise training was consistent and comparable, allowing for an accurate evaluation of the effects of exercise on the rats' health. A rat was considered to have reached exhaustion when it displayed a loss of coordination in its movements and failed to return to the surface within 10 seconds for three consecutive times.

2.4 Nutrition determinations

The following nutritional parameters were calculated: In kilojoules per day, energy intake equals mean food consumption in grams multiplied by dietary metabolizable energy in kilojoules, whereas feed efficiency (FE) percentage equals mean body weight gain in grams multiplied by 100 divided by energy intake in kilojoules.

2.5 Total serum cholesterol, HDL, and triacylglycerol levels determination.

Total cholesterol (TC), triglyceride (TG), and high-density lipoprotein cholesterol (HDL-C) concentrations were measured using Pars Azmoun Company kits (Tehran, India). Further, the Friedwald equation was used to calculate very low-density lipoprotein (VLDL), low-density lipoprotein cholesterol (LDLC), and atherogenic indices as follows: VLDL = TG/5; LDLC = TC – HDLC – (TG/5).

2.6 Fasting blood glucose (FBS)

At the beginning and end of the study, rats fasted overnight, and a baseline blood draw from the lateral tail vein was collected for plasma fasting glucose. Glucose levels were measured with Precision OncoPlus test strips.

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3.0 Results

The results are expressed as mean values ± standard deviation, and the statistical significances were analysed using one-way ANOVA compared to the control, $P < 0.05$.

Table 1: Effect of Exercise Training on Nutritional parameters; Body Weights, Food intake, Feed Efficiency, and Energy Intake in HFDS induced Obese Wistar rats trained under low intensity exercise (LIE), Medium intensity exercise (MIE) and, High-intensity exercise (HIE)

	SED-HFDS n=5	HFDS-LIE n=5	HFDS-MIE n=5	HFDS-HIE n=5	F (3,16)	P-value
Initial Body weights (grams)	430.2±17.6	439.4±22.1	453.4±30.16	445.8±7.33	1.099	0.378
Terminal Body weights (grams)	471.2±20.91^{a}	396.2±38.38^{b}	375.2±18.^{bc}	347.8±19.88^{c}	21.264	<0.001
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	<0.001
Feed Efficiency (%)	9.57±3.3^{a}	-17.93±10.21^{b}	-17.54±7.38^{b}	-28.4±2.82^{b}	29.669	<0.001
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	<0.001

Key: g-grams, kJ- kilojoules.

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 $P < 0.05$

Table 1 shows the results of a one-way between-subjects ANOVA that was conducted to compare the effects of exercise training on obesity in the following nutritional parameters: initial and terminal body weight, food intake, feed efficiency, and energy intake at low, medium, and high intensities of exercise training.

There was a significant effect of terminal weight on exercise training at the $p < 0.05$ level for the four groups [$F (3, 16) = 21.26$, $p < 0.001$]. The 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 different when compared to the HFDS-LIE (M = 375.2, SD = 18.13), HFDS-MIE (M = 396.2, SD = 38.38), and HFDS-HIE (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 feed consumption from exercise training at the $p < 0.05$ level for the four groups [$F (3, 16) = 37.884$, $p < 0.001$]. The post hoc comparisons using the Tukey HSD test indicated that the mean feed consumption for the SED HFDS control group (M = 34.32, SD = 1.51) was significantly different compared to the HFDS-LIE (M = 28.66, SD =1.35), HFDS-MIE (M = 27.4, SD = 1.15), and HFDS-HIE (M = 26.18, SD = 1.22). Also, statistical significance was

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noted between HFDS-LIE (M = 28.66, SD = 1.35), HFDS-MIE (M = 27.4, SD = 1.15), and HFDS-HIE (M = 26.18, SD = 1.22). Therefore, these results mean that exercise training decreases food consumption, and as you increase the intensity of exercise, the food consumption decreases. Further, there was a significant effect on feed efficiency from exercise training at the $p < 0.05$ level for the four groups [$F (3, 16) = 29.669$, $p < 0.001$]. The 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 different compared to 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).

Furthermore, there was a significant effect on energy intake from exercise training at the p.05 level for the four groups [$F (3, 16) = 37.884$, $p < 0.001$]. The post hoc comparisons using the Tukey HSD test indicated that the mean energy intake for the SED HFDS control group (M = 431.06, SD = 18.91) was significantly different compared to the HFDS-LIE (M = 359.97, SD = 16.91), HFDS-MIE (M = 344.14, SD = 14.36), and HFDS-HIE (M = 328.82, SD = 15.11). Also, there was a statistical difference between the HFDS-LIE (M = 359.97, SD = 16.91) and the HFDS-HIE (M = 328.82, SD = 15.11). Therefore, this study's findings show that energy intake decreases as you increase the intensity of exercise training.

Table 2. Effects of graded intensities of Exercise Training on lipid profile and Fasting Blood Sugars in HFDS Induced Obese Wistar rats

	SED-HFDS n=5	HFDS-LIE n=5	HFDS-MIE n=5	HFDS-HIE n=5	F (DF 3,16)	P-Value
TG (mg/dl)	46.94±2.04^{a}	42.54±3 ^{a}	42.1±2.86 ^{ab}	31.56±3.11^{b}	5.549	0.008
TC (mg/dl)	129.39±1.87^{a}	119.33±5.78^{ab}	116.34±3.67^{b}	71.93±1.07^{c}	50.468	0.001
HDLC(mg/dl)	23.36±0.63 ^{a}	25.52±2.46 ^{ab}	31.79±1.31^{bc}	34.57±2.25 ^{c}	8.347	0.001
LDLC	83.14±0.82 ^{a}	82.37±1.15 ^{a}	80.28±3.43 ^{a}	46.404±6.42^{b}	.346	<0.001
Initial FBS	4.84±.18	4.7±.34	4.92±.26	4.58±.27	1.580	0.233
Terminal FBS	5±.26 ^{a}	3.4±.19 ^{a}	3.88±.22 ^{b}	3.88±.18 ^{b}	51.486	<.001

Key: TG-Total Triglyceride, TC-Total Cholesterol, HDLC-High Density Lipoproteins Cholesterol, LDLC – low-density lipoproteins, FBS - Fasting Blood Sugar. 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

Table 2 shows the results of a one-way between-subjects ANOVA that was conducted to compare the effects of exercise training on lipid profiles and fasting blood sugars at low, medium, and high intensities of exercise training in HFDS-induced obese Wistar rats.

There was a significant effect of TC on exercise training at the $p < 0.05$ level for the four groups [$F (3, 16) = 5.549$, $p = 0.008$]. The post hoc comparisons using the Tukey HSD test indicated that the mean weight for the SED HFDS control group (M = 46.94, SD = 2.04) was significantly different compared to the HFDS-HIE (M = 31.56, SD = 3.11). However, the SED HFDS did not

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significantly differ from the HFDS-LIE and HFDS-MIE. "Taken together, these results suggest that high-intensity exercise training does affect total cholesterol. Specifically, these results suggest that when you perform high-intensity exercise training, you lose more total cholesterol. However, it should be noted that to have optimal total cholesterol reduction, the exercise intensity must be high. Low and medium intensity levels do appear to decrease total cholesterol, though not optimally.

4.0 Discussion.

In the current study, the BMI for Wistar rats during the induction phase ranged between 0.55 and 0.69 g/cm^2 after 12 weeks of feeding with HFDS. This agrees with a study by De Moura e Dias et al. (2021), whose findings showed that 2 weeks of HFDS is adequate for the induction of visceral obesity in Wistar rats.

The Wistar rats in the HFDS control group (sedentary) had significantly higher body weights and food intake than those in the training exercise, which is consistent with the findings of Dupas et al. (2018). This significant lower body weight in exercise-trained rats could be attributed to the reduction of the amount of adipose tissue, resulting in decreased generation of sex hormones, glucose, and leptin, increased levels of appetite-suppressing neuropeptide hormones such as nesfatin-1 and peptide YY, and negative energy and fat balance linked with increased energy expenditure and fat oxidation during exercise (Speretta et al., 2012). However, Mohammadi et al. (2006) showed that the increased body weight of rabbits after 8 weeks of high-fat diet feeding was not altered by chronic exercise.

Obesity and hyperlipidemia are the major risk factors for cardiovascular diseases. Further, excess LDLC promotes the formation of atherosclerotic plaque. In the present study, the plasma levels of TC, TG, VLDL, and LDLC in the HFDS group were increased due to an increase in both de novo synthesis and intestinal absorption of cholesterol (Nicolantonio, James J. 2018). HFDS causes the synthesis of reactive oxygen species (ROS), which cross-react with lipoproteins to produce oxidative states, thus decreasing the cell's uptake of lipids from the blood. These results are similar to those of other studies. However, these parameters decreased in the exercise training groups when compared with the HFDS group. The decrease in lipids may be caused by the inhibition of impaired lipid digestion and absorption, improvements in glucose and lipid metabolism, enhancement of insulin sensitivity, increased antioxidant defense, and down-regulation of lipogenic enzymes. Several studies have demonstrated that exercise decreases blood lipid and lipoprotein levels in humans and rats (Wang & Xu, 2017).

In the present study, hypertriglyceridemia was present in the sedentary control group and low-intensity group, which is one of the criteria for the diagnosis of metabolic syndrome. This hypertriglyceridemia could be attributed to the high free fatty acid influx to the liver due to the expansion of visceral adipose tissue mass. This expansion can cause excess production of triglyceride-rich very low-density lipoproteins (VLDL), which results, in turn, in high circulating

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levels of triglycerides (Gordon et al., 2014; Jastrzebski et al., 2021). Further, hypertriglyceridemia is also a reflection of the insulin-resistant condition, which was demonstrated in current studies where the control group (HSFD) had significantly higher blood sugar as compared to the exercise training group (medium and high-intensity group) (Table 2).
Furthermore, throughout a 12-week period of consuming a high-fat diet, the Wistar rats displayed signs of insulin resistance, which was characterized by the inability of insulin to facilitate glucose uptake and metabolism in insulin-sensitive tissues. On the other hand, in the exercise group rats, FBS had normalized at the end of the study, as was the case for hypertriglyceridemia, as reported by previous studies using the same HFD (Badalzadeh et al., 2014; Lira et al., 2012; Nounou et al., 2012).

In this study, the significant increase in glucose in HFDS-fed animals can be explained as being due to insulin production defects, decreased insulin sensitivity, insulin resistance, decreased adiponectin hormone level, and defective Na+-K+-ATPase activity (Oppert et al., 2021). It indicated that HFDS leads to insulin resistance through oxidative stress. Exercise training significantly reduced the increased blood glucose levels due to the increased adiponectin levels and insulin sensitivity.

5.0 Conclusion

The study reveals that feeding Wistar rats a high-fat diet (HFDS) induces obesity, oral glucose intolerance, and hyperlipidemia. Nevertheless, moderate-to-high intensity exercise training can alleviate these negative effects by suppressing obesity and improving lipid profiles. These findings underscore the potential benefits of exercise in preventing obesity-related tissue damage and promoting overall health.

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6.2 Declaration of interest

None

6.3 Conflict of interest

The authors declare no conflict of interest.

7.0 References

Ahmadi-Kani Golzar, F., Fathi, R., & Mahjoub, S. (2019). High-fat diet leads to adiposity and adipose tissue inflammation: the effect of whey protein supplementation and aerobic exercise training. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition et Metabolisme*, *44*(3), 255–262. https://doi.org/10.1139/apnm-2018-0307

Apovian, C. M. (2016). Obesity: definition, comorbidities, causes, and burden. The

URL: https://ojs.jkuat.ac.ke/index.php/JAGST
ISSN 1561-7645 (online)
doi: 10.4314/jagst.v22i3.4

American Journal of Managed Care, 22(7 Suppl), s176-85. https://pubmed.ncbi.nlm.nih.gov/27356115/

Badalzadeh, R., Shaghaghi, M., Mohammadi, M., Dehghan, G., & Mohammadi, Z. (2014). The effect of cinnamon extract and long-term aerobic training on heart function, biochemical alterations and lipid profile following exhaustive exercise in male rats. *Advanced Pharmaceutical Bulletin*, *4*(Suppl 2), 515–520. https://doi.org/10.5681/apb.2014.076

Caterson, I. D., & Gill, T. P. (2002). Obesity: epidemiology and possible prevention. Best Practice & Research. Clinical Endocrinology & Metabolism, 16(4), 595–610. https://doi.org/10.1053/beem.2002.0228

Chooi, Y. C., Ding, C., & Magkos, F. (2019). The epidemiology of obesity. Metabolism: Clinical and Experimental, 92, 6–10. https://doi.org/10.1016/j.metabol.2018.09.005

Council, N. R. (1995). Nutrient Requirements of Laboratory Animals,: Fourth Revised Edition, 1995. The National Academies Press. https://doi.org/10.17226/4758

Council, N. R. (2011). Guide for the Care and Use of Laboratory Animals: Eighth Edition. The National Academies Press. https://doi.org/10.17226/12910

de Moura e Dias, M., dos Reis, S. A., da Conceição, L. L., Sediyama, C. M. N. de O., Pereira, S. S., de Oliveira, L. L., Gouveia Peluzio, M. do C., Martinez, J. A., & Milagro, F. I. (2021). Diet-induced obesity in animal models: points to consider and influence on metabolic markers. Diabetology & Metabolic Syndrome, 13(1), 32. https://doi.org/10.1186/s13098-021-00647-2

Dupas, J., Feray, A., Guernec, A., Pengam, M., Inizan, M., Guerrero, F., Mansourati, J., & Goanvec, C. (2018). Effect of personalized moderate exercise training on Wistar rats fed with a fructose enriched water. Nutrition & Metabolism, 15(1), 69. https://doi.org/10.1186/s12986-018-0307-6

Engin, A. (2017). The Definition and Prevalence of Obesity and Metabolic Syndrome. Advances in Experimental Medicine and Biology, 960, 1–17. https://doi.org/10.1007/978-3-319-48382-5_1

Gordon, B., Chen, S., & Durstine, J. L. (2014). The effects of exercise training on the traditional lipid profile and beyond. Current Sports Medicine Reports, 13(4), 253–259. https://doi.org/10.1249/JSR.0000000000000073

Hruby, A., & Hu, F. B. (2015). The Epidemiology of Obesity: A Big Picture. PharmacoEconomics, 33(7), 673–689. https://doi.org/10.1007/s40273-014-0243-x

Jastrzebski, D., Toczylowska, B., Zieminska, E., Zebrowska, A., Kostorz-Nosal, S., Swietochowska, E., Di Giulio, C., & Ziora, D. (2021). The effects of exercise training on lipid profile in patients with sarcoidosis. Scientific Reports, 11(1), 5551. https://doi.org/10.1038/s41598-021-84815-4

Kim, S. (2016). The Definition of Obesity. Korean Journal of Family Medicine, 37, 309. https://doi.org/10.4082/kjfm.2016.37.6.309

Lira, F. S., Carnevali, L. C., Zanchi, N. E., Santos, R. V. T., Lavoie, J. M., & Seelaender, M. (2012). Exercise Intensity Modulation of Hepatic Lipid Metabolism. Journal of Nutrition and Metabolism, 2012, 809576. https://doi.org/10.1155/2012/809576

URL: https://ojs.jkuat.ac.ke/index.php/JAGST
ISSN 1561-7645 (online)
doi: 10.4314/jagst.v22i3.4

Liu, L., Feng, J., Zhang, G., Yuan, X., Li, F., Yang, T., Hao, S., Huang, D., Hsue, C., & Lou, Q. (2018). Visceral adipose tissue is more strongly associated with insulin resistance than subcutaneous adipose tissue in Chinese subjects with pre-diabetes. Current Medical Research and Opinion, 34(1), 123–129. https://doi.org/10.1080/03007995.2017.1364226

Mwangi, A., Kweri, J. K., Kamau, C. K. ., Kanyoni, J. M., Kigundu, A. M., Mwangi, E., & Marera, D. (2023). The pregnancy outcomes of female albino rats (Rattus Norvegicus) exposed prenatally to varied doses of lamotrigine . Journal of Agriculture, Science and Technology, 22(2), 22–33. https://doi.org/10.4314/jagst.v22i2.3

Miko, H.-C., Zillmann, N., Ring-Dimitriou, S., Dorner, T. E., Titze, S., & Bauer, R. (2020). [Effects of Physical Activity on Health]. Gesundheitswesen (Bundesverband der Arzte des Offentlichen Gesundheitsdienstes (Germany)), 82(S 03), S184–S195. https://doi.org/10.1055/a-1217-0549

Mohammadi, M., AliPpour, M. R., & Vatankhah, A. M. (2006). Effect oF High Choresterol Diet and Parallel Chronic Exercise on Erythrocyte Primary Antioxidant Enzymes and Plasma Total Antioxidant Capacity in Dutch Rabbit. International Journal of Endocrinology and Metabolism, 4(1), 30–40.

Mutiso, S. K., Rono, D. K., & Bukachi, F. (2014). Relationship between anthropometric measures and early electrocardiographic changes in obese rats. BMC Research Notes, 7, 931. https://doi.org/10.1186/1756-0500-7-931

Nicolantonio, James J, J. H. (2018). Effects of dietary fats on blood lipids: a review of direct comparison trials. Open Heart, 5(2). https://doi.org/10.1136/openhrt-2018-000871

Njue, R. M., Mburu, C., & Muchiri, F. (2019). Factors Influencing Sedentary Lifestyle Prevalence Among Workers In Selected Institutes in Kenya Agricultural Livestock and Research Organisation. Journal of Agriculture, Science and Technology, 19(1),84–99.

Nounou, H. A., Deif, M. M., & Shalaby, M. A. (2012). Effect of flaxseed supplementation and exercise training on lipid profile, oxidative stress and inflammation in rats with myocardial ischemia. Lipids in Health and Disease, 11, 129. https://doi.org/10.1186/1476-511X-11-129

Novelli, E. L. B., Diniz, Y. S., Galhardi, C. M., Ebaid, G. M. X., Rodrigues, H. G., Mani, F., Fernandes, A. A. H., Cicogna, A. C., & Novelli Filho, J. L. V. B. (2007). Anthropometrical parameters and markers of obesity in rats. Laboratory Animals, 41(1), 111–119. https://doi.org/10.1258/002367707779399518

Oppert, J.-M., Bellicha, A., van Baak, M. A., Battista, F., Beaulieu, K., Blundell, J. E., Carraça, E. V, Encantado, J., Ermolao, A., Pramono, A., Farpour-Lambert, N., Woodward, E., Dicker, D., & Busetto, L. (2021). Exercise training in the management of overweight and obesity in adults: Synthesis of the evidence and recommendations from the European Association for the Study of Obesity Physical Activity Working Group. Obesity Reviews : An Official Journal of the International Association for the Study of Obesity, 22 Suppl 4(Suppl 4), e13273. https://doi.org/10.1111/obr.13273

Reeves, P. G., Nielsen, F. H., & Fahey George C., J. (1993). AIN-93 Purified Diets for

URL: https://ojs.jkuat.ac.ke/index.php/JAGST
ISSN 1561-7645 (online)
doi: 10.4314/jagst.v22i3.4

Laboratory Rodents: Final Report of the American Institute of Nutrition Ad Hoc Writing Committee on the Reformulation of the AIN-76A Rodent Diet. The Journal of Nutrition, 123(11), 1939–1951. https://doi.org/10.1093/jn/123.11.1939

Saris, W. H. M., Blair, S. N., van Baak, M. A., Eaton, S. B., Davies, P. S. W., Di Pietro, L., Fogelholm, M., Rissanen, A., Schoeller, D., Swinburn, B., Tremblay, A., Westerterp, K. R., & Wyatt, H. (2003). How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obesity Reviews : An Official Journal of the International Association for the Study of Obesity, 4(2), 101–114. https://doi.org/10.1046/j.1467-789x.2003.00101.x

Shahidi, S. H., Stewart Williams, J., & Hassani, F. (2020). Physical activity during COVID-19 quarantine. Acta Paediatrica (Oslo, Norway : 1992), 109(10), 2147–2148. https://doi.org/10.1111/apa.15420

Shuster, A., Patlas, M., Pinthus, J. H., & Mourtzakis, M. (2012). The clinical importance of visceral adiposity: a critical review of methods for visceral adipose tissue analysis. The British Journal of Radiology, 85(1009), 1–10. https://doi.org/10.1259/bjr/38447238

Speretta, G. F. F., Rosante, M. C., Duarte, F. O., Leite, R. D., Lino, A. D. de S., Andre, R. A., Silvestre, J. G. de O., Araujo, H. S. S. de, & Duarte, A. C. G. de O. (2012). The effects of exercise modalities on adiposity in obese rats. Clinics (Sao Paulo, Brazil), 67(12), 1469–1477. https://doi.org/10.6061/clinics/2012(12)19

Tandon, P., Wafer, R., & Minchin, J. E. N. (2018). Adipose morphology and metabolic disease. The Journal ofExperimental Biology, 221(Pt Suppl 1). https://doi.org/10.1242/jeb.164970

Wang, Y., & Xu, D. (2017). Effects of aerobic exercise on lipids and lipoproteins. Lipids in Health and Disease, 16(1), 132. https://doi.org/10.1186/s12944-017-0515-5

Yang, X.-Q., Yuan, H., Li, J., Fan, J.-J., Jia, S.-H., Kou, X.-J., & Chen, N. (2016). Swimming intervention mitigates HFD-induced obesity of rats through PGC-1α-irisin pathway. European Review for Medical and Pharmacological Sciences, 20(10), 2123–2130.

URL: https://ojs.jkuat.ac.ke/index.php/JAGST
ISSN 1561-7645 (online)
doi: 10.4314/jagst.v22i3.4