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ROLE OF EXERCISE IN REGULATING SERUM VASPIN LEVELS IN LEAN AND OBESE ADULT ALBINO RATS

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ABSTRACT

Background: Exercise, which is the keystone of type 2 diabetes millets (T2DM) and obesity treatment, has revealed a great variance of effects on adipokines levels. Vaspin is a new adipocytokine with insulin-sensitizing effects, but its regulation in obesity and physical exercise are not clear yet.

Objective: Exploring the effect of continuous and intermittent exercise on circulating levels of vaspin and its relation to some metabolic changes and inflammatory markers in lean and obese rats.

Material and methods: A total number of 60 adult male local strain albino rats were used. Rats were divided into 2 main groups**:** group I" lean group, and group II "HFD group; Each group was further subdivided into 3 equal subgroups: group I A; control group with sedentary life, group I B; rats were exposed to continuous exercise, and group I C; rats were exposed to intermittent exercise. Serum vaspin, insulin, glucose, triglycerides, cholesterol, HDL, TNF and CRP levels were measured. In addition, BMI, HOMA-IR and atherogenic index were calculated in all groups.

Results: Serum vaspin levels were higher in obese (HFD) than in lean rats. Vaspin levels increased in continuous and intermittent exercised lean groups, and decreased in HFD induced obesity. Vaspin correlated negatively with inflammatory markers, HOMA-IR, TC TG and positively with HDL-C.

Conclusion: Serum vaspin levels were up regulated in HFD induced obesity. Intermittent exercise was more effective than continuous exercise in improving metabolic parameters and ameliorating inflammatory effect of obesity partially by modulating circulating vaspin levels in lean and obese rats.

Key words: vaspin, exercise, CRP, TNF.

INTRODUCTION

Over the last several decades, obesity has been reported as a public health problem due to its associated disorders, including insulin resistance, type II diabetes mellitus (T2DM), dyslipidemia, hypertension, and cardiovascular disease **(Deng and Scherer, 2010).** An undesirable metabolic consequence of obesity is because of buildup of adipose tissue in the abdominal cavity, rather than excess body fat (**Gui et al., 2004).**

Visceral adipose tissue in a rat model of abdominal obesity and T2DM secretes visceral adipose tissue-derived serpin (vaspin), which is an adipokine with insulin-sensitizing effects, and is a member of serine protease inhibitor family (**Hida et al., 2005).** Vaspin plays a role in the pathogenesis of metabolic syndrome (**Gulcelik et al., 2009).** Although, several studies demonstrated the significance of vaspin in insulin resistance and obesity in humans. The

correlation between serum vaspin levels and markers of insulin sensitivity, glucose metabolism, and obesity is still contro-versial **(Ko et al., 2013).** Non-pharmaceutical interventions (e.g. diet, exercise), which are the keystone of T2DM and obesity treatment, have shown a great variance of effects on adipokine levels (**Kadoglou et al., 2007). Youn et al. (2008)** declared that exercise was associated with elevated serum vaspin levels across normal glucose tolerant, impaired glucose-tolerant, and type 2 diabetic subjects**. Bashiri et al. (2014)** indicated that a sub-maximal aerobic workout does not result in significant changes in vaspin levels.

Therefore, this research was conducted to explore effect of diet- induced obesity, the continuous and intermittent exercises on serum vaspin levels and their relations to some metabolic changes and inflammatory markers.

MATERIAL AND METHODS

Animals: Sixty adult healthy male local strain albino rats (150-170 g), obtained from the animal house of Faculty of Veterinary Medicine- Zagazig University. Rats were kept in steel wire cages (40x 28x 18 cm 5/cage) under hygienic conditions. They had free access to water, and kept at comfortable temperature (20 to 24 ?C), and normal light/dark cycle (**Lesourd and Mazari, 1999).** All rats received care in accordance with the national health guidelines and the study protocol was approved by the Institutional Review Board and ethics committee of Faculty of Medicine-Zagazig University.

Methods: Rats were accommodated to laboratory conditions for three weeks before starting the experimental regimen (**Gui et al., 2004).** They were divided into 2 main equal groups as follows **: Group I" lean group"** had free access to standard chow of commercial rat standard chow [25.8 % protein, 62.8 % carbohydrate and 11.4 % fat]**). Group II "HFD** had free access to HFD (protein 20%, carbohydrates 35% and fat 45%, mainly in form of lard and soy bean) for 9 weeks **(Cha et al., 2000)**. Each group was further subdivided into 3 equal subgroups: group I A; control group with sedentary life, group I B; rats exposed to continuous exercise, and group I C; rats exposed to intermittent exercise**.**

Experimental protocol: After 21 days of acclimatization, exercise programs consisted of continuous and intermittent swimming performed in a plastic container that was 100 cm high, filled to 60 cm with water, and maintained at a temperature of 28–32 °C for four weeks **(Perse et al., 2009)**. Continuous training protocol consisted of swimming, one hour/day, five days/week for four weeks **(Gobatto et al., 2001 and Crespilho et al., 2010).** The animals of the continuous group swam for 15, 30, and 45 min on the first, second, and third days to adapt. The swimming period then increased to 60 min/day **(Sene-Fiorese et al., 2008).** The animals of the intermittent group swam three sessions for 10, 20, and 30 min on the first, second, and third days to adapt. The swimming period was 60 min/day, divided into three daily sessions (3 × 20 min/ day) with 4 h of controlled intervals between the exercise periods throughout the experimental period. The rats were kept in their home cages all the time between the exercise periods, performed at 7 am, 11:30 am, and 3:30 pm. The

exercise protocols were performed for 5 days/week for four weeks (**Voltarelli et al., 2002)**. At the end of the study, all rats were weighed and BMI were calculated according to the equation: body weight (g)/ length^2 (cm^2) -(nose to anus length (**Novelli et al., 2007**).

Blood collection: At the end of the experiment, after overnight fasting, rats were anesthetized using ether (ADWIC Laboratory Chemicals, Egypt). Blood was collected from orbital sinus. Samples were allowed to clot at room temperature, then centrifuged for 15 minutes at approximately 3000 rpm.

Biochemical assay:

- Serum Vaspin levels: was measured by enzyme Amplified Sensitivity Immunoassay using (WKEA MED SUPPLIES CORP, 450 11th Ave, New York, NY 10123, USA) Kits (**Tietz, 1995).**
- Serum TNF-α was measured by enzyme linked immunosorbent assay kit (Abcam, Catalog No. ab100785, USA) (**Hosseini-Tabatabaei et al., 2009).** - Serum C reactive protein (CRP) level was measured by Monobind Inc Lake Forest, Ca 92630, USA **(Ridker, 2001).**
- Serum glucose was measured by using glucose enzymatic **(**GOD-PAP)-liquizyme rat Kits (Biotechnology, Egypt) (**Tietz, 1995).**
- Serum Insulin was measured by KAP1251-INS-EASIA (Enzyme Amplified Sensitivity Immunoassay) rat Kits (BioSource Europe S.A., Belgium) **(Temple et al., 1992).**
- Serum total cholesterol (TC**)** was measured by enzymatic colorimetric method using (BioSource Europe S.A.-Rue de l'Industrie, 8-B- 1400 Nivelles-Belgium) kits (**Tietz, 1995).**
- Serum Triglycerides (TG) were measured by using triglycerides ESPAS SL kits (Elttech S.A., Sees, France) **(Fossati, 1982).**
- Serum high density lipoprotein cholesterol (HDL-C) was measured by enzymatic colorimetric method using (BioSource Europe S.A.-Rue de l'Industrie, 8-A-1340 Nivelles-Belgium) kits (**Nauck et al., 1997).**
- Calculation of athrogenic index (AI) from the formula: (AI) = [Log (triglycerides / HDL- cholesterol)] (**Karthik and Ravikuma, 2011).**
- Calculation of the homeostasis model of assessment of insulin resistance (HOMA-IR) = [insulin (μIU/ ml) x glucose (mmol/l) / 22.5] or [HOMA-IR = insulin (μIU /ml) x glucose (mg/dl) /405] (**Matthews et al., 1985).**

Statistical analysis: Data were presented as mean ± S.D Statistical significance was determined by one way analysis of variance (ANOVA) followed by LSD test. P values less than 0.05 were considered to be significant. The correlations between parameters were analyzed using Pearson's correlation.

In statistical analysis, SPSS version 19 program for Windows (SPSS Inc. Chicago, IL, USA) was used.

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RESULTS

Serum vaspin levels were significantly higher in obese group (IIA) than lean group (IA) (P< 0.001). These high levels were accompanied by significant high levels of TNF-α, C reactive protein (CRP), glucose, insulin, HOMA-IR, total cholesterol (TC), triglycerides (TG), atherogenic index (AI) and calculated body mass index (BMI), while high density lipoprotein (HDL) levels were significantly lower. Continuous and intermittent exercised lean subgroups (IB and IC) showed significant higher serum vaspin levels than control group (IA). On the other hand, continuous and intermittent exercised obese subgroups (IIB and IIC) revealed significant lower serum vaspin levels than control group (IA). In addition to the significant changes in serum vaspin levels in both continuous and intermittent exercised, there were significant improves in both glucose and lipid profiles with decrease in pro-inflammatory markers (significant decrease in glucose, insulin, HOMA, TC, TG, AI , BMI, TNF- α and CRP with significant increase in HDL). Furthermore, in group IA; vaspin levels significantly positive correlated with BMI, CRP, and insulin. In group IB, while vaspin levels significantly positive correlated with BMI, insulin and HDL, it correlated negatively with CRP, glucose, HOMA, TC and TG. In group IC while vaspin levels significantly positive correlated with HDL, it correlated negatively with BMI, CRP, glucose, insulin, HOMA, TC, TG, and AI. In group IIA vaspin levels significantly positive correlated with BMI, TNF- α. CRP, glucose, insulin, HOMA, TC, HDL and AI. In group IIB while vaspin levels significantly positive correlated with BMI, and HDL, it correlated negatively with TNF- α, CRP, insulin, HOMA, TC and AI. In group IIC, while vaspin levels significantly positive correlated only with HDL it correlated negatively with BMI, TNF- α, CRP, glucose, insulin, HOMA, TC, and AI (Tables 1, 2 and 3).

Table (1): **Circulating vaspin, TNF-α ,CRP and calculated BMI in the all studied groups (Mean ±SD).**

Groups / Parameters	Group I (lean)			Group II (obese)		
parameter	**Group IA**	**Group IB**	**Group IC**	**Group IIA**	**Group IIB**	**Group IIC**
Vaspin (ng\ml)	0.40±0.03	0.45 ±0.039[a]	0.507 ±.079[a,b]	0.732±.0.06[a]	0.517±0.065[c]	0.455±0.03[c,d]
BMI (gm/cm^2)	0.52 ±0.05	0.477± 0.02[a]	0.49±0.026[a,b]	1.01±0.09[a]	0.86±0.13 [c]	0.72±0.07 [c,d]
	r= 0.994	**r**=+0.939[*]	**r**= -0.985[*]	**r**=0.910[*]	**r**[=] 0.947[*]	**r**[=] -0.986[*]
TNF-α (pg/ml)	13.31±1.49	11.66±1.126[a]	11.42±0.927[a]	55.30±5.056 [a]	31.70±5.25 [c]	23.80±3.96 [c,d]
	r= -0.021	**r**= -0.406	**r**= -.572	**r**= 0.920[*]	**r**= -0.988[*]	**r**= -0.971[*]
CRP (mg/dl)	0.227± 0.02	0.169± 0.01[a]	0.13± 0.03[a,b]	5.54 ±0.69 [a]	3.16 ±0.70 [c]	2.53±0.46 [c,d]
	r=0 .996[*]	**r**= -0.849[*]	**r**= -0.941[*]	**r**= 0.928[*]	**r**= -0.931[*]	**r**= -0.959[*]

([a]) = significant when compared with group IA. ([b])= significant when compared with group IIA, ([c]) = significant when compared with group IB. ([d]) = significant when compared with group IIB r = correlation with vaspin levels

Table (2): Parameters related to glucose metabolism in the all studied groups (Mean ±SD).

Groups / Parameters	Group I (lean)			Group II (obese)		
parameter	**Group IA**	**Group IB**	**Group IC**	**Group IIA**	**Group IIB**	**Group IIC**
Glucose (mg/ dl)	81.80±12.90	74.90±9.69[a]	70.90±8.44[a]	224.0±27.59 [a]	192.60±20.17 [c]	168.00±15.73 [c,d]
	r=0.976[**]	**r**= -0.985[**]	**r**= -0.930[**]	**r**= -0.711[*]	**r**= -0.455	**r**= -0.737[*]
Insulin (µIU/ml)	14.89± 2.45	13.44±2.21	12.20±1.54	39.70±4.69 [a]	28.86±2.68 [c]	25.96±2.09 [c]
	r= 0.977[*]	**r**= 0.981[*]	**r**= -0.895 [*]	**r**= 0.664[*]	**r**= -0.667[*]	**r**= -0.986[*]
HOMA-IR	2.77± 0.18	2.56±0.18[a]	2.17±0.52[a,b]	22.14±2.78 [a]	13.79±0.196 [c]	10.71±0.60 [c,d]
	r= 0.528	**r**=-0.688[*]	**r**= -0.911 [**]	**r**= 0.674[*]	**r**=- 0.856[*]	**r**= -0.785[*]

([a]) = significant when compared with group IA. ([b])= significant when compared with group IIA, ([c]) = significant when compared with group IB. ([d]) = significant when compared with group IIB r = correlation with vaspin levels .

Table (3): Parameters related to lipid metabolism in the all studied groups (Mean ± SD).

Groups / Parameters	Group I (lean)			Group II (obese)		
parameter	**Group IA**	**Group IB**	**Group IC**	**Group IIA**	**Group IIB**	**Group IIC**
TC (mg/dl)	100.30±5.23	90.50±5.08[a]	89.10±3.45[a]	208.5±11.09 [a]	191.2±11.46 [c]	175.80±8.25 [c,d]
	r= 0.304	**r**= -0.972 [**]	**r**= -0.668 [*]	**r**= 0.659[*]	**r**= -0.929[**]	**r**= -0.961[**]
HDL (mg/dl)	50.50±2.07	59.20±4.10[a]	63.40±3.69[a,b]	38.80±3.99 [a]	50.0±3.97 [c]	55.50±4.60 [c,d]
	r= 0.538	**r**= 0.673[*]	**r**= 0.873[*]	**r**=0.910[*]	**r**= 0.784[*]	**r**= 0.991[*]
TG (mg/dl)	55.0 ±8.18	48.20±6.45 [a]	44.3±3.47[a]	120.1±11.84 [a]	111.60±3.68 [c]	102.10±3.17 [c,d]
	r= -0.066	**r**= -0.651[*]	**r**= 0.978[**]	**r**= 0.519	r= -0.167	r= -0.171
AI	0.98 ±0.12	0.53±0.11[a]	0.41±0.11[a,b]	4.44±0.79 [a]	2.86±0.50 [c]	2.197±0.41 [c,d]
	r=0.609	**r**= -0.214	**r**= -0.659 [*]	**r**= 0.908[*]	**r**= -0.884[*]	**r**= -0.984[*]

([a]) = significant when compared with group IA. ([b])= significant when compared with group IIA, ([c]) = significant when compared with group IB. ([d]) = significant when compared with group IIB r = correlation with vaspin levels.

DISCUSSION

Obesity, a rising health concern, is linked with insulin resistance and characterized by low-grade inflammation **(Sigal et al., 2006)**. Exercise training is recommended to improve long term health metabolic outcomes. One of the physiological changes responsible for health and cardio- metabolic benefit of exercise could be mediated by altering the secretion of adipokines which is suggested to be concerned with inflammation. As regard in HFD induced obesity (group

IIA) in the present study, a significant high circulating vaspin levels were detected compared with the lean group (IA) accompanied by positive correlation with BMI.

Vaspin, as an adipocytokine; is mainly expressed in visceral adipose tissue. So, the elevated circulating vaspin levels in HFD fed rats can be explained by diet induced obesity. Insulin resistance and its metabolic effects are a second possibility to explain the rise in circulating vaspin level in HFD induced obesity. Accordingly, the results showed significant high levels of glucose, insulin and calculated HOMA-IR in HFD as compared with the lean control group. Moreover, circulating vaspin levels were significantly and positively correlated with circulating glucose levels, circulating insulin levels and calculated HOMA-IR in HFD group. This finding could be explained by vaspin being insulin sensitizing, is increased as a compensatory mechanism to impaired glucose tolerance (**Hida et al., 2005 and Zhang et al., 2011),** This was supported by other investigators who reported that serum vaspin levels were found to be significantly high in T2DM and correlated positively with markers of insulin resistance and glycemic control such as fasting glucose, HOMA-IR, insulin, and HbA1c % (**Zhan et al., 2011; Hida et al. 2012 and Alghannam et al., 2013).** Moreover, vaspin messenger RNA expression was found to be absent in lean normal glucose tolerant individuals and was more frequently detected in T2DM patients **(Kloting et al., 2007).** Glucose was found to induce a significant dose-dependent increase in vaspin net protein production and secretion from human omental adipose tissue explants **(Tan et al., 2008).** In addition, patients on peripheral glucose uptake enhancer had less serum vaspin levels than patients who were not taking it (**Gulcelik et al., 2009**).

The present results showed also significant high levels of plasma total cholesterol (TC), and triglycerides (TG) along with significant low levels of HDL-C inducing high athergenic index (AI) in the HFD control group when compared with that of lean control group. Moreover, circulating vaspin levels were found to be significantly and positively correlated with TG, TC, and AI. These results were in line with the reported positive correlation between vaspin levels and triglycerides levels in control subjects. However, diabetic patients had no correlation between vaspin and lipid profile (**Seeger et al., 2008** and **Atya et al., 2013).** Moreover, **El-Mesallamy et al**. (**2011)** reported that vaspin may play a role in lipid metabolism or might be induced by diabetic dyslipidemia as a compensatory mechanism. Vaspin levels in T2DM significantly correlated with some markers of lipid metabolism such as TG, TC. However, **Giomisi et al. (2011)** showed a negative association between vaspin levels and lipid profile in pregnant women.

Regarding role of exercise in the present study, the significant improvement of metabolic parameters with reduction in BMI, TNF-alpha, and CRP detected in continuous and intermittent exercise in lean and HFD rats making exercise an effective strategy to reverse almost all atherosclerotic risk factors linked to HFD obesity and was proved in multiple other studies **(Dekker et al., 2010 and Abd El-Kader et al., 2014& 2015).** Majority of

studies have reported significant decrease in insulin resistance index after both acute and chronic endurance exercise (**Magkos et al., 2008 and Kim et al., 2011)**. However, **Jamurtas et al. (2006)** reported a positive correlation between insulin and insulin resistance index in overweight males after acute exercise**.** Previous studies indicated that the mechanism, by which insulin resistance decreases, is independent from insulin concentration. Exercise could increase the expression of intracellular insulin signaling pathway components, in particular of glucose transporters (GLUT4) in skeletal muscle (**Lehnen et al., 2012**). **Luqut et al. (2005)** reported that physical exercise increases the number of capillaries and oxidative fibers in muscle, enhancing lipolysis, which allows free flow of fatty acid to the tissue, reducing its concentration in plasma, which is an indicator of its uptake and oxidation by tissues**.** The exercise activates an alternative pathway, ie. the mitogen-activated protein kinases (AMPK) in the liver, muscle and adipocytes by increasing fatty acid oxidation, decreasing cholesterol synthesis, lipogenesis , and modulating insulin secretion on pancreatic islets (**Viollet et al., 2009) .**

Statistically significant decrease in AI in continuous and intermittent exercised lean and obese groups was detected in the present study when compared with lean sedentary group and obese sedentary group respectively. These results were in line with **Stranska et al. (2011)** who reported that aerobic exercise has beneficial impact on AI in sedentary overweight and obese subjects.

Moreover, intermittent exercise was more efficient than continuous exercise in reducing the adverse effects of high-fat diet and sedentarism because intermittent exercise induces significant more improvement in lipid profile and glucose homeostasis, decrease AI and proinflamatory markers (CRP and TNF-alpha) with reduction in BMI when compared with that induced by continuous exercise**.** This was in line with **Sene-Fiorese et al. (2008)** who stated that intermittent exercise has more efficient health beneficial effects than continuous exercise. The potential mechanisms for the anti-inflammatory effect of exercise include reduced percentage of body fat and macrophage accumulation in adipose tissue, muscle-released interleukin-6 inhibition of TNF-alpha, and the cholinergic anti-inflammatory pathway (**Woods et al., 2006 and Abd El-Kader et al. 2013&2014)**.

The present study proved that continuous and intermittent exercises significantly increase serum vaspin levels in lean (IB and IC groups), and significantly decreased serum vaspin levels in HFD (IIB and IIC) groups. Moreover, intermittent exercise was significantly more effective than continuous exercise in regulating serum vaspin levels.

Modulation of serum vaspin levels can be explained by the changes in metabolic parameters and vice versa, as serum vaspin levels correlated negatively with the reduced calculated HOMA- IR, TC and TG levels in continuous and intermittent exercises groups of both lean and HFD fed groups. There were positive correlations between serum vaspin levels

and both elevated HDL-cholesterol levels and reduced BMI in continuous and intermittent exercises of HFD groups. This was in line with **Lee et al. (2010)** who reported that serum vaspin is correlated negatively with HOMA-IR in diabetic patients and obese children.

Vaspin treatment was found to be associated with sustained glucose-lowering effects in leptin receptor - deficient mice (**Alghannam et al., 2013 and Andrade-Oliveira et al., 2015)**. Furthermore, **Heiker et al. (2013)** provided an evidence that the serpin function of vaspin is essential for its physiologic effects and demonstrated that the anti-diabetic vaspin effects in vivo was based on an insulin-stabilizing effect most likely by inhibiting human kallikrein 7-mediated insulin degradation. Moreover, **Auguet et al. (2011)** found that serum vaspin levels were inversely correlated with levels of lipocalin-2 (LCN2) and interleukin-6 (IL-6). It has been reported that LCN2 is an adipokine that seems to be an independent risk factor for hyperglycemia and insulin resistance in humans. **Al-Azzam et al. (2013)** proved that statins therapy (antihyperlipidemic agents) increases plasma vaspin levels in addition to have a lipid-lowering effect. This could be a mechanism underlying the pleiotropic effects seen with statins, including their cardioprotective and antiatherosclerotic effects. In the same context, **Hida et al. (2005)** reported that administration of recombinant human vaspin improved insulin sensitivity, glucose tolerance and suppressed the expression of the genes related to insulin resistance such as resistin and TNF-α, in diet-induced obese mice, whereas it increased that of the glucose transporter-4 and adiponectin in WAT. **Shaker and Sadik (2013)** reported an up-regulation of visceral vaspin expression in diet induced obesity s associated with insulin resistance and rise in serum leptin level.

Moreover, **Youn et al. (2008)** reported that circulating vaspin was significantly correlated with leptin serum concentrations, and **Shangani et al. (2013)** proved that incremental resistance training reduced body fat percent and serum leptin levels in sedentary overweight females. In addition, **Gonz?lez et al. (2009)** found that leptin administration was able to partially reverse the lower WAT vaspin levels after fasting. **Nia et al. (2009)** proved that exercise training inducing reduction in leptin levels have been attributed to alterations in energy balance, and improvements in insulin sensitivity alterations in lipid metabolism. Another indirect regulatory effect of exercise on circulating vaspin levels may be through increasing PGC-1α in exercise which up regulates the expression and secretion of vaspin in adipocytes **(Kajimura et al., 2008)** as exercise training increase PGC-1α mRNA expression in rat white adipose tissue **(Nia et al., 2009) .** the expression and secretion of interleukin 6 (IL-6) from skeletal muscle has been shown to increase dramatically during exercise (**Pedersen and Febbrio, 2008).** Interestingly, IL-6 has been shown to activate 5'AMP activated protein kinase **,** a reputed mediator of PGC-1α mRNA expression (**Jager et al., 2007).** In addition, decrease TNF-α in exercise which proved in our study and was reported by **Zehsaz et al. (2014)** who stated that after exercise training, both insulin sensitive index and serum TNF-α concentration were decreased significan-

tly. **Yano et al. (2010)** proved that both the TNF-α and interferon -α concentrations in the plasma of exercised mice were significantly lower than those in the plasma of non exercised via toll-like affecting receptor (TLR) 7. Moreover, long-term exercise carried out concurrently with consumption of a HFD could alleviate HFD-induced non alcoholic steatohepatitis. These may be partly attributable to the attenuation of hepatic TNF-α over expression (**Federico et al., 2014).** In the present study, the modulated vaspin levels in exercised groups were correlated negatively with the significantly reduced CRP levels in continuous and intermittent exercised groups of lean obese rats when compared with lean sedentary group and obese sedentary group respectively. This was in agreement with **Kadoglou et al. (2013)** who reported that in obese subjects with Type2 diabetes, all exercise training modalities decrease high-sensitivity CRP. In addition, **Abd El-Kader (2011)** reported that aerobic exercise intervention, but not resistance exercise, reduces serum inflammatory cytokines including IL-18, CRP and IL-6. Exercise has the potential to lower the inflammatory status by the reduction of high-sensitivity CRP and TNF-α (**Zhou et al., 2015).** Taken together, these findings might suggest that vaspin has an anti-inflammatory effect.

In conclusion: Serum vaspin levels are up regulated in HFD induced obesity. Intermittent exercise is more effective than continuous exercise in improving metabolic parameters and ameliorating inflammatory effect of obesity partially by modulating circulating vaspin levels in lean and obese rats.

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خلفية البحث: تعد التمارين الرياضية حجر الزاوية في علاج الداء السكري من النوع الثاني T2DM، وعلاج السمنة، وقد كشفت الأبحاث عن وجود تباين كبير في تأثيرها على مستويات الاديبوكاينزز. "فاسبين" الذي يزيد الحساسية لهرمون الإنسولين، ولكن تنظيم مستوياته في مصل الدم في حالة السمنة وحاله ممارسة الرياضة البدنية ليست واضحة بعد.

هدف البحث: دراسة تأثير ممارسة التمارين الرياضية المستمرة والمتقطعة على تنظيم مستويات الفاسبين في مصل الدم وعلاقاتها و بعض التغيرات الأيضية ودلائل الإلتهابات في الجرذان البيضاء البالغة النحيلة والسمينة.

مواد وطرق البحث: تم استخدام ستون من ذكور الجرذان البيضاء البالغه ،وقد قسمت الجرذان إلى مجموعتين رئيسيتين: المجموعة الأولى: المجموعة النحيلة والمجموعة الثانية: مجموعة: خضعت لنظام غذائي عالي الدهون .وقد تم تقسيم كل مجموعة الى 3 مجموعات فرعية متساوية: مجموعة IA; المجموعة الضابطة، ومجموعة IB; تعرضت الفئران فيها إلى ممارسة بروتوكول التمارين الرياضية المستمرة (السباحة) ساعة واحدة/ يوميا، خمسة أيام في الأسبوع لمدة أربعة أسابيع، والمجموعة Ic ; تعرضت الفئران فيها لممارسة بروتوكول السباحة المتقطعة خمسة أيام في الأسبوع لمدة أربعة أسابيع . وقد تم قياس مستوي الفاسبين، والإنسولين والجلوكوز، والدهون الثلاثية ، والكوليسترول، ومستويات دهون عالية الكثافة ، وعامل نخر الورم TNF ، و CRP في مصل الدم. علاوه علي قياس مؤشر كتلة الجسم، و دليل مقاومة الإنسولين، ومؤشر تصلب الشرايين في جميع المجموعات.

النتائج: وجد إرتفاعا ذا دلالة احصائية فى مستويات الفاسبين في المصل مقارنة بالمجموعة الضابطة، وكانت أعلى في المجموعة الثانية التي تعاني السمنه والتي خضعت لنظام غذائي عالي الدهون مما كان عليه في المجموعة الأولى النحيلة. وبعد اتمام بروتوكول الرياضة المستمرة والمتقطعة وجدت زيادة في مستويات القاسبين في المجموعة الاولي النحيلة ، بينما وجد إنخفاضا ذا دلالة إحصائية في المجموعة الثانية التي تعاني السمنة التي خضعت لنظام غذائي عالي الدهون. كما وجد إرتباطا سلبيا بين مستويات الفاسبين مع دلائل الإلتهابات، ودليل مقاومه الإنسولين، والدهون الثلاثية، وإيجابية مع مستويات دهون عالية الكثافة .

الإستنتاج: ممارسة التمارين الرياضية المتقطعة هي أكثر فاعلية من الممارسة المستمرة في تحسين معايير الأيض وتخفيف تأثير الإلتهابات التي تسببها السمنة جزئيا وذلك عن طريق تنظيم مستويات الفاسبين في الفئران النحيلة و التي تعانى من السمنة.