

## Effects of Fasting on Glucagon-like peptide-1 hormone (GLP-1), and Lipid Profile Indices in Obese and Thin Women

Shahrbanoo Haghighi<sup>1</sup>, Seyed Reza Attarzade Hosseini<sup>2</sup>, Masoud Saleh Moghaddam<sup>1</sup>, Majid Rajabian<sup>1</sup>, Mohammad Ali Kiani<sup>3</sup>, \*Habibolah Taghizade Moghaddam<sup>4</sup>, Seyed Majid Sezavar Kamali<sup>5</sup>

<sup>1</sup>Department of Biochemistry, Payame Noor University, Mashhad, Iran. <sup>2</sup>Department of Sport Physiology, Faculty of Physical Education and Sports Sciences, Ferdowsi University of Mashhad, Mashhad, Iran.

<sup>3</sup>Department of Pediatrics, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran.

<sup>4</sup>Department of Biochemistry, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran.

<sup>5</sup>PhD in Laboratory Science, Mashhad, Iran.

### Abstract

#### Background

Glucagon-like peptide-1 hormone (GLP-1) contributes to the regulation of insulin and glucose concentration. However, the effects of fasting on GLP-1 response in different people has not been determined yet. The aim of the present research was to investigate the effect of fasting on GLP-1 and the lipid profile of obese and thin women.

**Materials and Methods:** In this research, 25 obese and thin women whose age ranged from 35 to 45 years were selected through a convenient sampling method and were divided into two groups of obese (n=12, body mass index >30 kg/m<sup>2</sup>) and thin (n=13, body mass index=18-20 kg/m<sup>2</sup>). GLP-1 in both groups was measured in four phases: 3 days before the beginning of Ramadan, 14 days after the beginning of Ramadan, 28 days after the beginning of Ramadan and 2 weeks after the end of Ramadan. Repeated –measure ANOVA was used to statistically analyse the data.

#### Results

GLP-1 was reduced from phase 1 to 3 of the research. However, it was increased after Ramadan. In the thin group, GLP-1 was increased in 14 days of fasting, but did not show any change at the end of Ramadan, and also two weeks after this month. However, none of these changes were statistically significant. The two groups did not diverge from each other significantly in any of the phases.

#### Conclusion

The present findings showed that fasting has no significant effect on the GLP-1 and lipid profile indices of the obese and thin women.

**Key Words:** Fasting, Glucagon-like peptide-1 hormone, Obese, Women.

\*Please cite this article as: Haghighi Sh, Attarzade Hosseini SR, Saleh Moghaddam M, Rajabian M, Kiani MA, Taghizade Moghaddam H, et al. Effects of Fasting on Glucagon-like peptide-1 hormone (GLP-1), and Lipid Profile Indices in Obese and Thin Women. *Int J Pediatr* 2019; 7(3): 9095-9102. DOI: **10.22038/ijp.2018.36085.3147**

#### \*Corresponding Author:

Habibolah Taghizade Moghaddam, Department of Biochemistry, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran.

Email: Taghizade moghaddamh1@mums.ac.ir

Received date: Feb.14, 2018; Accepted date: Oct. 22, 2018

## 1- INTRODUCTION

In recent years, the hormones involved in adjusting energy in intestines have been discovered one of which is the Glucagon-like peptide-1 hormone (GLP-1). It has shown to inhibit food absorption and weight gain (1). This peptide is known as a type of incretin. Incretins are a group of metabolic hormones secreted in response to glucose consumption by intestinal cells and play a key role in glucose homeostasis (2). Moreover, these hormones account for the secretion of 50-70% of the insulin secreted upon consuming oral glucose (3).

GLP-1, on the one hand, directly increases the gene expression of insulin and its synthesis through the existing receptors on pancreas cells (4), and on the other hand, directly affects its own receptors and pancreas beta cells indirectly through the vagus nerve and hepatic portal vein and increases insulin secretion and decreases blood glucose (5). Upon entering the portal blood stream, GLP-1 activates a glucose sensor in the portal vein which sends signals to the central nervous system and the brain through vagus nerves and then again through vagus nerves sends signals to the pancreas which increases the secretion of insulin in pancreas. It also directly affects pancreas alpha cells and reduces the secretion and release of glucagon (5). Moreover, GLP-1 reduces the speed of stomach evacuation and causes a slow absorption and prevents the sudden increase of blood glucose. It creates a feeling of satiation and thus cuts down on food consumption (6).

Moreover, GLP-1 can independently improve the accumulation of hepatic glycogen, increase glucose absorption and decrease the concentration of triglycerides (7). A particular nutritional condition facing all Muslims worldwide on a yearly scale is fasting in Ramadan. The procedure involves refraining from eating, drinking and smoking from the outset of sunrise up until sunset. They are allowed to consume

foods and drinks in the time between sunset and the next sunrise. In other words, the regular meal pattern would change to two meals a day in Ramadan (8), which can contribute to the diet plan of inactive obese individuals and improve their lipid profile. How fasting affects blood biochemical indices is not determined yet and is under investigation. Fasting in Ramadan is a perfect opportunity for correcting one's diet and can affect fasting blood sugar (FBS), insulin concentration, insulin resistance, total cholesterol (TC), triglyceride (TG), Low-density lipoprotein (LDL-C), High-density lipoprotein (HDL-C), and proportion of total cholesterol to high-density lipoprotein (TC/HDL-C), and proportion of low-density lipoprotein to high-density lipoprotein (LDL-C/HDL-C). There have been contradictory research findings on how fasting affects blood fats in healthy individuals with no extra weight (8-11). The role GLP-1 plays in obesity pathogenesis is not clarified yet.

In obesity, GLP-1 level and insulin level have been suggested to correlate negatively. That is to say that resistance to insulin may cause a weak GLP-1 response and lead to the gain of more weight (12). The reaction of GLP-1 to one meal after a long-term loss of weight and after fasting is not known yet. Only a few investigations looked into the acute effect of the weight lost in response to a diet on the basic and fasting levels of GLP-1.

Verdict et al. (2001) indicated the poor response of fasting GLP-1 in obese subjects which was significantly increased after a six-month weight loss through a diet. There seems to be a mediating factor involved between GLP-1 response in thin and obese people and this mediating factor is GLP-1 response to weight loss (12). A body of research reported a lower plasma level of fasting GLP-1 and lower GLP-1 response to food consumption in obese individuals (13-15). Some other research reported an increased GLP-1 response to

consuming food stuff in obese human and rats (16). As for the loss of weight, other investigations that involved a weight loss intervention reported the reduction (17-19), and no change (20) of GLP-1 level after the weight loss through a diet. Yet, the effect of fasting on GLP-1, and lipid profile values in thin and obese people is not determined and requires further investigation. Thus, the present research aimed to investigate the effect of fasting on the GLP-1 and lipid profile of thin and obese women.

## 2- MATERIALS AND METHODS

The research methodology followed in this study was semi-experimental with repeated measures and two case groups. The sample was comprised of women from Mashhad city, Iran. Among those who met the inclusion criteria, 25 women whose age ranged between 21 and 51 years, and formerly filled out the written consent form were selected through convenient sampling method. They were divided into two groups of obese (n=12, body mass index [BMI] >30 kg/m<sup>2</sup>), and thin (n=13, BMI=18-20 kg/m<sup>2</sup>). These subjects were diagnosed as healthy according to a demographic information questionnaire, and their medical record.

They had no experience of smoking, cardio-vascular, renal, respiratory, hepatic and metabolic diseases. The study was conducted in Ramadan (2017), and it lasted for seven weeks (one week before until two weeks after the end of Ramadan). It is noteworthy that due to the long-term fasting, voluntary participation in the study, considering the ethical issues and continuous attention to people's health, one subject was excluded from the thin, and one from the obese group as they did not meet the research conditions (travel, absence for more than three sessions of exercise and omission of the Sahar meal) during the research procedures. Their previous information was omitted too.

Therefore, the thin group ended up with 12 and the obese group with 11 subjects examined in four phases (3 days before Ramadan, 14 days after the beginning of Ramadan, the end of Ramadan and 14 days after Ramadan). The anthropometrics used in this research included: standing height (Seca stadiometer made in Germany, precision of 5 mm), weight (Seca scale, precision of 100 gr), body composition (impedance bioelectrical scale, Inbody 720, made in South Korea).

All these measurements were done in the morning time. The subjects were formerly asked to avoid intensive physical activities two days before the test. The measurement was to be done when the subjects' bladder, bowels and stomach were emptied. All the tests were given from 1:00 p.m. to 2:30 p.m. Moreover, in the present research, 7ml blood samples were taken from median cubital vein from 4:00 to 6:00 p.m. after at least 12 hours of fasting in Dr. Sezavar's medical diagnostic lab.

The serum of these samples was immediately centrifuged. Then an automatic analyzer as well as Pars-Azmoon kits were used to measure HDL, TG, TC, and LDL. GLP-1 was measured by GDV-ELISA Reader. Once the data were collected, they entered SPSS software version 24.0 for the required statistical analysis. The data were appropriately labeled and analyzed through descriptive and inferential statistics.

The former included mean scores, distribution indices and plots. The latter involved exploratory Shapiro-Wilk and Levene tests which examined the normality of data and homogeneity of group variances, respectively. Repeated-measure ANOVA was used to compare within-group and inter-group mean scores. To make statistical decisions, the significance level was set at  $p < 0.05$ .

### 3- RESULTS

The mean and standard deviation of obese subjects' age was  $44.5 \pm 4.9$  years, while that of the thin group was  $30.84 \pm 8.99$  years. According to **Table.1**, within-group variation in the means cores of weight, BMI and body fat were not statistically significant in either group ( $p > 0.05$ ). Only the between-group proportion of waist circumference to hip circumference and the percentage of fat were significantly different ( $p < 0.05$ ).

The results presented in **Table.2** indicate significant within-group changes in total cholesterol, low-density lipoprotein and high-density lipoprotein in the obese group and only high-density lipoprotein in the thin group ( $p < 0.05$ ). Within-group variation in the mean scores of GLP-1 and triglyceride was not statistically significant in either group. Similarly, the within-group variation in total cholesterol and low-density lipoprotein was not statistically significant in the thin group ( $p > 0.05$ ).

**Table-1:** Within- and between-group anthropometric differences in the thin (n=12), and obese (n=11) groups

Anthropometric measure	Group	Phases				Within-group variation		Between-group variation	
		3 days before Ramadan	2 <sup>nd</sup> week of Ramadan	4 <sup>th</sup> week of Ramadan	2 weeks after Ramadan	F	P-value	F	P-value
Body weight (kg)	Obese	82.88±8.6	82.13±8.62	82.23±9.03	82.03±9.34	1.42	.253	94.85	.001
	Thin	53.53±5.91	53.16±5.72	53.50±5.63	53.59±5.83	1.99	.133		
BMI (kg/m <sup>2</sup> )	Obese	33.62±3.43	33.36±3.53	33.39±3.62	33.31±3.75	.96	.423	154.92	.001
	Thin	20.31±1.32	20.17±1.28	20.30±1.30	20.30±1.33	1.44	.245		
Waist /hip circumference (cm)	Obese	1.01±.06	1. ±.08	1.0±.07	1.0±.07	1.61	.205	68.33	.001
	Thin	.81±.03	.80±.03	.81±.03	.82±.03	5.95	.002		
Body fat (%)	Obese	45.43±5.27	44.89±5.68	45.05±5.04	44.90±5.01	.91	.445	48.05	.001
	Thin	30.40±5.61	30.51±5.51	30.47±5.49	30.29±5.41	.05	.983		

BMI: Body mass index.

Bonferroni post-hoc test results showed statistically significant within-group variation (in the thin group) in the mean scores of waist /hip circumference between the 2<sup>nd</sup> phase (2<sup>nd</sup> week of Ramadan) and the fourth phase (2 weeks after Ramadan), high density lipoprotein between the 2<sup>nd</sup> phase (2<sup>nd</sup> week of Ramadan) and the third phase (4<sup>th</sup> week of Ramadan), and also between the second phase (2<sup>nd</sup> week of Ramadan), and the fourth phase (2 weeks after Ramadan). In the obese group, within group variances revealed significant changes in the mean scores of high-density

lipoprotein between the first phase (3 days before Ramadan) and the second phase (2<sup>nd</sup> week of Ramadan), in the low-density lipoprotein between the first phase (3 days before Ramadan) and the fourth phase (2 weeks after Ramadan), and in the total cholesterol between the second phase (2<sup>nd</sup> week of Ramadan), and the fourth phase (2 weeks after Ramadan). According to the results presented in **Table.2**, variation in within-group means cores was not statistically significant in any of the target variables in either group ( $p > 0.05$ ).

**Table-2:** Within- and between-group biochemical differences in the thin (n=12), and obese (n=11) groups

Biochemical measure	Group	Phases				Within-group variation		Between-group variation	
		3 days before Ramadan	2 <sup>nd</sup> week of Ramadan	4 <sup>th</sup> week of Ramadan	2 weeks after Ramadan	F	P-value	F	P-value
GLP-1	Obese	185.06±185.85	179.46±189.35	166.66±109.03	185.08±183.60	.96	.43	.97	.331
	Thin	278.07±252.73	310.85±250.22	295.74±277.79	291.20±268.16	.56	.63		
TG (mg/dl)	Obese	126.22±41.09	127.81±26.70	125. ±43.85	122.27±38.18	.07	.975	1.27	.272
	Thin	113.23±40.48	107.07±38.52	105.46±35.67	115.76±42.99	.76	.524		
TC (mg/dl)	Obese	207.41±32.87	216.75±28.45	201.72±35.19	178.09±20.36	5.41	.005	2.76	.111
	Thin	178.46±37.31	184.07±36.14	173.61±32.33	170.69±31.78	1.64	.195		
LDL (mg/dl)	Obese	121.68±26.24	113.36±19.97	105.27±25.32	101.81±24.02	4.87	.007	3.18	.088
	Thin	98.53±26.93	94.69±24.31	92.46±23.09	93. ±23.09	.72	.545		
HDL (mg/dl)	Obese	51.12±6.85	61.66±9.56	52.90±8.97	53.41±7.92	6.10	.002	.42	.520
	Thin	51.34±8.84	58.07±6.73	50.38±10.03	50.84±7.34	4.74	.007		

GLP-1: Glucagon-like peptide-1; TG: triglyceride; TC: total cholesterol; LDL: low density lipoprotein; HDL: high density lipoprotein.

#### 4- DISCUSSION

The present research aimed to investigate the effect of fasting on the GLP-1 and lipid profile of thin and obese women. The results showed that in neither group (thin or obese), fasting had any significant effect on GLP-1. In some other research, it was found that the thin and obese groups diverged significantly in terms of GLP-1 catabolism. This difference was associated with the activity of IV dipeptide peptidase (15). However, in the present research, the thin and obese groups did not diverge from each other in terms of GLP-1 changes. Quite many investigations (21-27) reported an increase in incretin after the stomach bypass surgery. It is not yet determined whether calorie deficiency or rapid weight loss are involved in the change of incretin level since the weight loss induced by a diet is correlated with high GLP-1 level (even to a limited

degree) (12). GLP-1 has been observed to be increased significantly in response to oral glucose one month after the bypass surgery (28). GLP-1 level tends to be reduced after a nutritional diet though not to a significant degree. This finding is in contrast with what Verdich et al. found in terms of the increased GLP-1 level (even to some degree) during one meal after loss of weight among men (12). The time spent after the surgery which has differed from one research to the other can be a key variable as a body of research has shown that increased GLP-1 is temporary and does not continue in 6-12 months of surgery (29). The effect of fasting in Ramadan was observed in the present research more on the lipid indices of obese subjects than the thin. This difference can be probably explained by higher basic levels of TG, TC, and LDL of the obese group than the thin. With this respect,

some other research with 96 subjects afflicted with hyperlipidemia who received consultation on correcting their lifestyle reported that the effect size of controlling diet on TC and LDL levels is correlated with their basic levels (30). Therefore, the lower basic level in the thin group might have limited the effect size of fasting on lipid indices in this group. How fasting in Ramadan affects blood biochemical indices is not determined yet. Overall, fasting in Ramadan is a good opportunity for correcting one's nutritional diet and can affect LDL, TG, TC and HDL. Moreover, there have been contradictory findings concerning the effect of fasting on the lipids of healthy people.

TC is among the key biochemical indices and includes a certain category of lipids which, if exceeds a natural range, is accompanied by the risk of atherosclerosis. Therefore, examining its variation profile during fasting in Ramadan is of a great significance. According to a body of existing research, Haghdoost and Poorranjbar (2009), and Yarahmadi et al. (2003) reported an increase in TC during Ramadan (10, 31). On the contrary, Boobes et al. (2009), and Mansi (2007) reported the reduction of TC (9, 32). Lipoproteins rich in cholesterol such as LDL may be increased during Ramadan. However, several researchers proved to the contrary (33-35). Recent findings attested to the lacking variation in HDL concentration during Ramadan. Some others, yet found a significant increase in HDL concentration (33-36).

Changes in blood lipid level can be correlated with the amount of food consumed among those who eat excessively during the day (37). Lower energy consumption in Ramadan is correlated with lower TC and plasma LDL that act as cardiovascular factors (38, 39). Nevertheless, contradictory findings can be explained by the fact that Ramadan follows a lunar calendar rather than a solar

calendar cycle. Therefore, the duration of fasting limited to the hours of day differ across countries and varies across years. It depends on whether the fasting is done during hot and long summer days or in short and cold winter days. Moreover, ethnic/national differences as well as socioeconomic differences can be involved that affect nutritional diets. These factors can tremendously affect the measured variables in different studies conducted in previous years.

#### **4-1. Limitations of the study**

One limitation of the present study is the absence of a control group as it was impossible to find healthy subjects not fasting in Ramadan. Another limitation was that the subjects' nutritional diet was not controlled and they could eat freely at night. Moreover, the target research population was 25 subjects which made it rather insufficient for the required statistical analyses.

#### **5- CONCLUSION**

According to the results, variation (GLP-1, and lipid profile) in within-group means cores was not statistically significant in any of the target variables in obese and thin groups. The present findings showed that fasting has no significant effect on the GLP-1 and lipid profile indices of the obese and thin women.

#### **6- CONFLICT OF INTEREST: None.**

#### **7- REFERENCES**

1. Barrera JG, Sandoval DA, D'alessio DA, Seeley RJ. GLP-1 and energy balance: an integrated model of short-term and long-term control. *Nature Reviews Endocrinology*. 2011;7(9):507.
2. Creutzfeldt W. The incretin concept today. *Diabetologia*. 1979;16(2):75-85.
3. Brown J, Dryburgh J, Ross S, Dupre J, editors. Identification and actions of gastric inhibitory polypeptide. *Proceedings of the*

- 1974 Laurentian Hormone Conference; 1975: Elsevier.
4. Inagaki N, Seino Y, Takeda J, Yano H, Yamada Y, Bell GI, et al. Gastric inhibitory polypeptide: structure and chromosomal localization of the human gene. *Molecular endocrinology*. 1989;3(6):1014-21.
  5. Kreymann B, Ghatei M, Williams G, Bloom S. Glucagon-like peptide-1 7-36: a physiological incretin in man. *The Lancet*. 1987;330(8571):1300-4.
  6. Gutzwiller J, Göke B, Drewe J, Hildebrand P, Ketterer S, Handschin D, et al. Glucagon-like peptide-1: a potent regulator of food intake in humans. *Gut*. 1999;44(1):81-6.
  7. Tahrani AA, Bailey CJ, Del Prato S, Barnett AH. Management of type 2 diabetes: new and future developments in treatment. *The Lancet*. 2011;378(9786):182-97.
  8. Al Hourani HM, Atoum MF, Akel S, Hijjawi N, Awawdeh S. Effects of Ramadan fasting on some haematological and biochemical parameters. *Jordan J Biol Sci*. 2009;2(3):103-8.
  9. Boobes Y, Bernieh B, Al Hakim MR. Fasting Ramadan in kidney transplant patients is safe. *Saudi Journal of Kidney Diseases and Transplantation*. 2009;20(2):198.
  10. Haghdooost A, Poorranjbar M. The interaction between physical activity and fasting on the serum lipid profile during Ramadan. *Singapore Med J*. 2009;50(9):897-901.
  11. Hallak MH, Nomani MZA. Body weight loss and changes in blood lipid levels in normal men on hypocaloric diets during Ramadan fasting. *The American journal of clinical nutrition*. 1988;48(5):1197-210.
  12. Verdich C, Toubro S, Buemann B, Madsen JL, Holst JJ, Astrup A. The role of postprandial releases of insulin and incretin hormones in meal-induced satiety—effect of obesity and weight reduction. *International journal of obesity*. 2001;25(8):1206.
  13. Ranganath L, Beety J, Morgan L, Wright J, Howland R, Marks V. Attenuated GLP-1 secretion in obesity: cause or consequence? *Gut*. 1996;38(6):916-9.
  14. Tomasik P, Sztefko K, Malek A. GLP-1 as a satiety factor in children with eating disorders. *Hormone and Metabolic Research*. 2002;34(02):77-80.
  15. Lugari R, Dei Cas A, Ugolotti D, Barilli A, Camellini C, Ganzerla G, et al. Glucagon-like peptide 1 (GLP-1) secretion and plasma dipeptidyl peptidase IV (DPP-IV) activity in morbidly obese patients undergoing biliopancreatic diversion. *Hormone and metabolic research*. 2004;36(02):111-5.
  16. Fukase N, Igarashi M, Takahashi H, Manaka H, Yamatani K, Daimon M, et al. Hypersecretion of truncated glucagon-like peptide-1 and gastric inhibitory polypeptide in obese patients. *Diabetic medicine*. 1993;10(1):44-9.
  17. Adam TC, Lejeune MP, Westerterp-Plantenga MS. Nutrient-stimulated glucagon-like peptide 1 release after body-weight loss and weight maintenance in human subjects. *British journal of nutrition*. 2006;95(1):160-7.
  18. De Luis D, Sagrado MG, Conde R, Aller R, Izaola O. Decreased basal levels of glucagon-like peptide-1 after weight loss in obese subjects. *Annals of Nutrition and Metabolism*. 2007;51(2):134-8.
  19. Sumithran P, Prendergast LA, Delbridge E, Purcell K, Shulkes A, Kriketos A, et al. Long-term persistence of hormonal adaptations to weight loss. *New England Journal of Medicine*. 2011;365(17):1597-604.
  20. Svendsen PF, Jensen FK, Holst JJ, Haugaard SB, Nilas L, Madsbad S. The effect of a very low calorie diet on insulin sensitivity, beta cell function, insulin clearance, incretin hormone secretion, androgen levels and body composition in obese young women. *Scandinavian journal of clinical and laboratory investigation*. 2012;72(5):410-9.
  21. Lauritsen K, Christensen K, Stokholm K. Gastric inhibitory polypeptide (GIP) release and incretin effect after oral glucose in obesity and after jejunoileal bypass. *Scandinavian journal of gastroenterology*. 1980;15(4):489-95.
  22. Clements RH, Gonzalez QH, Long CI, Wittert G, Laws HL. Hormonal Changes After Roux-en Y Gastric Bypass For Morbid Obesity and the Control of Type-II Diabetes

Mellitus/DISCUSSION. The American surgeon. 2004;70(1):1.

23. Morínigo R, Moizé V, Musri M, Lacy AM, Navarro S, Marín JLs, et al. Glucagon-like peptide-1, peptide YY, hunger, and satiety after gastric bypass surgery in morbidly obese subjects. The Journal of Clinical Endocrinology and Metabolism. 2006;91(5):1735-40.

24. Green BD, Flatt PR. Incretin hormone mimetics and analogues in diabetes therapeutics. Best Practice and Research Clinical Endocrinology and Metabolism. 2007;21(4):497-516.

25. Morínigo R, Lacy AM, Casamitjana R, Delgado S, Gomis R, Vidal J. GLP-1 and changes in glucose tolerance following gastric bypass surgery in morbidly obese subjects. Obesity surgery. 2006;16(12):1594-601.

26. Valverde I, Puente J, Martín-Duce A, Molina L, Lozano O, Sancho V, et al. Changes in glucagon-like peptide-1 (GLP-1) secretion after biliopancreatic diversion or vertical banded gastroplasty in obese subjects. Obesity surgery. 2005;15(3):387-97.

27. Korner J, Bessler M, Inabnet W, Taveras C, Holst JJ. Exaggerated glucagon-like peptide-1 and blunted glucose-dependent insulinotropic peptide secretion are associated with Roux-en-Y gastric bypass but not adjustable gastric banding. Surgery for Obesity and Related Diseases. 2007;3(6):597-601.

28. Laferrère B, Heshka S, Wang K, Khan Y, McGinty J, Teixeira J, et al. Incretin levels and effect are markedly enhanced 1 month after Roux-en-Y gastric bypass surgery in obese patients with type 2 diabetes. Diabetes care. 2007;30(7):1709-16.

29. Laferrère B, Tran H, Egger J, Teixeira J, McGinty J, Yap K, et al. The increase in GLP-1 levels and incretin effect after Roux-en-Y gastric bypass surgery (RYGBP) persists up to 1 year in patients with type 2 diabetes mellitus (T2DM). Obesity. 2007;15:7.

30. Henkin Y, Shai I. Dietary treatment of hypercholesterolemia: can we predict long-term success? Journal of the American College of Nutrition. 2003;22(6):555-61.

31. Yarahmadi S, Larijani B, Bastanagh M, Pajouhi M, Baradar RJ, Zahedi F, et al. Metabolic and clinical effects of Ramadan fasting in patients with type II diabetes. Journal of the College of Physicians and Surgeons--Pakistan: JCPSP. 2003;13(6):329-32.

32. Mansi KMS. Study the effects of Ramadan fasting on the serum glucose and lipid profile among healthy Jordanian students. Am J Appl Sci. 2007;4(8):565-9.

33. Maislos M, Abou-Rabiah Y, Zuili I, Iordash S, Shany S. Gorging and plasma HDL-cholesterol—the Ramadan model. European journal of clinical nutrition. 1998;52(2):127.

34. Adlouni A, Ghalim N, Benslimane A, Lecerf JM, Saïle R. Fasting during Ramadan induces a marked increase in high-density lipoprotein cholesterol and decrease in low-density lipoprotein cholesterol. Annals of nutrition and metabolism. 1997;41(4):242-9.

35. Maislos M, Khamaysi N, Assali A, Abou-Rabiah Y, Zvili I, Shany S. Marked increase in plasma high-density-lipoprotein cholesterol after prolonged fasting during Ramadan. The American journal of clinical nutrition. 1993;57(5):640-2.

36. Uysal AR, Erdogan MF, Sahin G, Kamel N, Erdogan G. Clinical and metabolic effects of fasting in 41 type 2 diabetic patients during Ramadan. Diabetes care. 1998;21(11):2033.

37. Kassab S, Abdul-Ghaffar T, Nagalla D, Sachdeva U, Nayar U. Interactions between leptin, neuropeptide-Y and insulin with chronic diurnal fasting during Ramadan. Annals of Saudi medicine. 2004;24:345-9.

38. Khoshdel A, Kheiri S, Nasiri J, Saedi E, Mobasheri M. The effect of Ramadan fasting on lipid profile in pregnant women. Journal of Fasting and Health. 2015;3(2):81-5.

39. Pirsaeheb S, Pasdar Y, Navabi SJ, Rezaei M, Darbandi M, Niazi P. Fasting consequences during Ramadan on lipid profile and dietary patterns. Asia Oceania J Nucl Med Biol. 2013;1(2):6-12.