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ASSESSMENT THE EFFECT OF VASPIN AND ADIPONECTIN ON OBESE DM PATIENTS AND OBESE NON DM WOMEN

Rayahin T. Yousef[1]*, Hanaa S. Kadhum[2] and Mahdi M. Thuwaini[1]

[1]College of Health and Medical Technology, Southern Technical University, Basra, Iraq.
[2]Department of Pathological Analysis, College of Science, Basra University, Iraq.
*e-mail : Mahdi.Murshd@stu.edu.iq

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ABSTRACT : This cross-sectional, descriptive study was conducted at Al-Fayha Teaching Hospital, Basra. The goal of this study was to estimate vaspin in addition to adiponectin levels in obese women with or without T2DM. 58 obese diabetic patients having DM were included in the study. During a period beginning from November 2020 to February 2021. The study included 35 healthy obese and non-diabetic women as a control group. Blood was drawn from each person and the serum was then isolated and placed (the blood) at a temperature of -20°C until the analysis was performed to assess the level of blood sugar, HbA1c, Lipid profile Addition of serum vaspin and adiponectin by ELISA Reader. In 93 subject's serum vaspin and adiponectin levels were measured with or without T2DM. Then the analysis was performed between groups with or without diabetic patients. We found that the serum adiponectin level was significantly higher in obese patients with diabetes compared with non-obese and non-diabetic patients (P = 0.0001). The serum vaspin levels showed weakness raised significant variation in obese patients with diabetes than in non-obese subjects without diabetes (P=0.05). The serum T.G levels showed no statistical differences in obese patients with diabetes than in non-obese subjects without diabetes (P=0.231). There was a statistically significant positive correlation between adiponectin and vaspin in both obese diabetic patients (P=0.027) and obese non-diabetic control (P=0.000). It also found a positive statistically significant relationship between T.G, BMI, and HDL in obese diabetic patients and non-obese non-diabetic. This pattern mimic between LDL and HDL in obese non-diabetic control (P=0.011) and also between RBS and HBAIC in obese diabetic patients (P=0.001). In conclusion, direct proportional correlation between cholesterol and LDL (P=0.0001) and T.G (P=0.023) in obese women with diabetic patients, while in obese nondiabetic control same correlation but with BMI (P=0.003) and LDL (P=0.000). In conclusion; this study refers that decreased serum concentration of adipocytokines (vaspin, adiponectin) is a risk factor or predictable indicator for the diagnosis and advancement of T2DM.

Key words : Typ2DM, Vaspin, adiponectin, obese, lipid profile, adipose tissue, fat cells, blood vessels, adiponectin and obesity.

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INTRODUCTION

Diabetes mellitus is a group of metabolic disorders of hyperglycemia resulting from insulin secretion, insulin malfunction, or both. Chronic hyperglycemia caused by diabetes is associated with a long-term injury to the eyes, liver, nerves, heart and blood vessels, inflammation and multiple organ dysfunction (Diabetes, 2007). There are many causal disease processes involved in the development of diabetes. The extent of the autoimmune destruction of beta cells in the pancreas and the consequent lack of insulin to abnormalities that may lead to resistance to the action of insulin. The basis of those abnormalities in carbohydrate, lipid and protein metabolism in diabetic patients is the lack of insulin activity on target tissues, where insufficient insulin action results from insufficient insulin secretion and/or decreases tissue response to insulin at one or more points in a complex. The pathways of action of these hormones (Diabetes mellitus, 2007). Generally, the American Diabetes Association lists five categories within the group of disorders that represent diabetes syndrome (Cudario, 2010). It included: diabetes mellitus of both types I and type I. However Diabetes is associated with contributing clinical conditions, diseases, medications and/or chemicals. - Pregnancy diabetes. Diabetes-related malnutrition (Cudario, 2010). Obesity is defined as a mass of chronic excess fat. It is important to emphasize that obesity is a risk factor for the emergence of many metabolic disorders because of the chronic imbalances between both the

energy intake and energy expenditure, for example, but not limited to type 2 diabetes. In addition, it has been well proven by many researchers that the risk of developing several types of malignant tumors increases in obese individuals (Tumminia *et al*, 2019). Therefore, obesity has been identified as a major public health challenge and has also been considered the reason behind preventable mortality and morbidity (Di Angelantonio *et al*, 2016). Obesity is often associated with metabolic defects that may favor not only the initiation of cancer but its development as well. These abnormalities include low-grade adipose tissue inflammation, which involves the production of specific inflammatory adipocytes, oxidative stress, and peripheral insulin resistance with hyperinsulinemia and dyslipidemia (Vigneri *et al*, 2016). It has been scientifically established that most patients with DM2 are obese, and the obesity epidemic among the world's population has largely explained the dramatic increase in the incidence and prevalence of type 2 diabetes over the past twenty years (Centers for Disease Prevention and Control, 2011). Anyhow, the accurate mechanisms associate that the two conditions stay indistinct. Various factors could explain why there are more restrictions on movement in obese and/or diabetic people: increased physical inactivity, increased incidence of arthritis and comorbidities associated with T2D and obesity, and a change in body composition with loss of muscle mass and muscle quality (Duclos, 2016). Researchers have observed that obesity is closely related to a wide range of pathophysiological consequences including insulin resistance (IR), type 2 diabetes (T2D), atherosclerosis, hypertension, and hyperlipidemia. Note that this association of obesity with T2D has been recognized for decades, and most importantly is the ability of obesity to mainly generate insulin resistance (Kahn and Flier, 2000). As lipids are stored in adipocytes in the form of TAGs, adipocytes become overwhelmed when plasma FFA levels are too high. Therefore, fats accumulate in the cells of other organs in the form of lipid droplets and this can lead to lipotoxicity under certain conditions. Moreover, free fatty acids are directly and indirectly related to the production of inflammatory molecules and the regulation of inflammation, a disease known in T2DM. Dyslipidemia may also affect cellular membranes, thereby impairing cellular functions. All these aspects were considered as cofactors in FFA-induced insulin resistance, considering also the differential effects of different types of lean fatty acids. As against, some of the complications associated with T2DM are consequences of dysregulated FFA metabolism (Sobczak *et al*, 2019). Adipose tissue (AT): includes white adipose tissue and brown adipose tissue, which are two separate sections. Adipose tissue is not only a reservoir of energy but also an immune organ. When talking about obesity, it is now recognized that the development of insulin resistance is initiated by inflammation of the adipose tissue. However, the initial events that lead to this inflammation remain unclear, as a combination of endocrine and immune factors regulate this adipose tissue microenvironment (Stolarczyk, 2017). More than 50 adipokines have been identified; they have diverse roles, but there is compelling evidence for the effects of leptin, adiponectin, vaspin, and apelin on adipocyte metabolism and diabetes mellitus, and thus on their role in obesity and associated metabolic disorders. Adiponectin has the main physiological function which can increase insulin sensitivity as well as decrease plasma concentrations in relation to insulin resistance. The anti-inflammatory ability of adiponectin results in the protection of blood vessels, heart, lung, and colon. In addition to its anti-inflammatory properties, adiponectin directly defends the heart, blood vessels, kidneys and colon during periods of stress and injury. Adiponectin also possesses profound anti-inflammatory properties, preventing fibrosis in both the liver and the skin during injury and disease (Fang and Judd, 2011). Vaspin (visceral adipose tissue-derived serpin, SerpinA12) is a member of the serine protease inhibitor family of serpins, which has recently been proposed as a well-reliable biomarker, along with other pro-inflammatory adipokines, in evaluating disease-exposure associations (Eichelmann *et al*, 2007). 2017). regarding this issue, while several studies have shown an association between angiogenesis and obesity (Zhou *et al*, 2010; Dai *et al*, 2016; Tan *et al*, 2008; Yoon *et al*, 2008). Furthermore, Yun *et al* (2008) Analysis of the effect of body mass index and body fat on vascular production in normal glucose-tolerant Caucasian subjects. The serum vessel concentration was lower in subjects who showed a normal weight than in subjects who demonstrated overweight. This value, in turn, was lower than that of obese subjects. This association has also been found in people with metabolic syndrome (Dai *et al*, 2016). This study aims to investigate the serum levels of vaspin and adiponectin and their relationship to the glycemic and lipid profile characteristics of obese women with diabetes.

MATERIALS AND METHODS

This study was carried out among women attending Al Fayhaa Center for Endocrinology, Diabetes and Metabolism, Basra, Iraq. Study criteria included women with a BMI-obesity range (30.02 - 54.82 kg/m^2) aged 45 years and over. On the contrary, pregnant women, impaired liver and kidneys, hypertension, gastrointestinal,

cardiovascular, thyroid, autoimmune diseases, and women with infectious diseases were excluded. For the period from November 2020 to February 2021. The study included 58 obese patients with diabetes. In addition, the study included 35 obese and non-diabetic women as a control group. The questionnaire included writing down demographic information [age, body mass index (BMI), educational level, marital status, and occupation]. Blood samples were taken from all the women. Then separated by centrifugation at 3000 rpm for 15 minutes and then serum was isolated and placed at -200°C, until analyzed for determination of blood sugar, glycated hemoglobin, lipids, and complex Blood angiogenesis and adiponectin by Elisa Rieder (Human, Germany). Data are reported as means ± standard deviation (SD). Differences between group means were tested in a chi-square test. The correlations between the variables were also determined. All statistical analyzes were performed using SPSS for Windows 20 (version 23, USA). The non-parametric Kruskal-Wallis test and the Mann Whitney test were performed. Normal distribution, use one-way ANOVA. A value of $P<0.05$ was considered statistically significant and $P>0.05$ was considered not statistically significant.

RESULTS

As shown in Table 1 about the basic characteristics of the study population, a total of 93 women participated in this investigation, the obese diabetic patients comprised the majority of cases (62.36%, n=58) than that of the non-obese diabetic patients (37.63%, n=35).

Table 1 : The Frequencies and percentage of the study population.

	Frequency	Percent%
Obese Diabetic Patients	58	62.36
Obese Non Diabetic Control	35	37.63
Total	93	100.0

As illustrated in Table 2, no significant difference was recorded in the Age when comparing between obese diabetic Patients group and Control Obese non-diabetic group (P=0.830), On the other hand, the data indicated that BMI was significantly higher (P = 0.0001) than the control groups.

As shown in the same table, it appeared that significant variations were recorded in the levels of HbA1C and FBS at (P=0.0001), between the obese Diabetic Patients group and Control Obese non-diabetic group.

Furthermore, the data of this study show that results were revealed statistically a significant decrease of Vaspin and Adiponectin in the Obese Diabetic Patients group (P=0.0001) in comparison with the Obese Non-Diabetic Control group.

Regarding the lipid profile, the result appeared that LDL and Cholesterol were highly significant increase in the Obese Diabetic Patients group (P=0.0001) in comparison with Obese Non-Diabetic Control. Whereas no significant between obese diabetic patients group and obese non-diabetic control in level of TG despite there is an increase in its level of obese diabetic patients group in comparison with Obese Non-Diabetic Control (p=0.231). Nevertheless, the results also appeared that HDL level was a highly significant decrease in Obese Diabetic Patients group in comparison with Obese Non-Diabetic Control (P=0.0001). As shown in Table 3, there was a statistically significant direct proportional correlation between TG and BMI within the obese diabetic patients and obese non-diabetic control group (P=0.036, P=0.020) Straight. In addition, the direct proportional correlation was noticed in obese non-diabetic patients between TG and HDL (0.002), in the obese diabetic patients, that correlation was changed to the inverse correlation between TG and HDL (P=0.007). Though, there was a

Table 2 : Shows the comparison of all parameters between the obese diabetic patient's group and obese non-diabetic control. Values were expressed mean ±SD.

Groups / Parameters	Obese non-diabetic control(n=35)		Obese diabetic patients(n=58)		P-value
	Mean ±SD	Median (min-max)	Mean ±SD	Median (min-max)	
Age (year)	56.63±9.268	56.00(45-73)	55.98±7.642	53.50(45-70)	0.830
BMI (kg/m²)	25.1966±2.17247	25.0900(22.12-29.17)	35.9260±5.22689	34.6900(30.02-53.57)	0.0001
FBS(mg/dl)	90.261±1.773	92.00(67-120)	224.47±86.512	220.00(64-530)	0.0001
HBA1C%	5.0229±0.60880	4.9000(4-46)	9.6017±1.89315	9.6500(6.20-15.20)	0.0001
HDL(mg/dl)	44.98±10.789	44.50(21-82)	32.11±12.290	29.00(16-66)	0.0001
LDL(mg/dl)	121.29± 46.37	113.50(44-233)	158.03±42.84	151.00(94-223)	0.0001
T.G (mg/dl)	154.91±84.046	137.00(59-341)	174.22±87.167	150.50(64-511)	0.231
Chol.(mg/dl	217.60±38.251	163.00(108-280)	274.34±44.913	209.00(149-285)	0.0001
Adipo.(pg/ml)	.4477±0.12204	.4230(.26-.79	.3104±0.08315	.3040(.10-.56)	0.0001
Vaspin(pg/ml)	.25069±0.100189	.23750(.022-.755)	.21697±0.043917	.21000(.131-.290)	0.055

Table 3 : Spearman's Nonparametric Correlations among vaspin, adiponectin, and anthropometric and biochemical indices.

Category			BMI	HDL	LDL	TG	CHOL	HbA1C	FBS	VASSP	ADIPO
Obese diabetic patients	Age(year)	Sig.	.853	.068	.734	.209	.778	.283	.128	.385	.788
	BMI(kg/m^2	Sig.		.758	.346	.036	.317	.928	.929	.270	.440
	HDL	Sig.			.429	.007	.112	.322	.118	.273	.201
	LDL	Sig.				.079	.000	.935	.884	.130	.091
	TG	Sig.					.023	.868	.724	.639	.260
	Chol.	Sig.						.922	.603	.203	.227
	HBA1C	Sig.							.001	.485	.973
	FBS	Sig.								.459	.127
	VASPIN	Sig.									.027
Obese Non Diabetic Control	Age(year)	Sig.	.964	.762	.610	.626	.624	.102	.620	.614	.576
	BMI	Sig.		.239	.243	.020	.003	.617	.783	.925	.766
	HDL	Sig.			.011	.002	.762	.563	.489	.180	.335
	LDL	Sig.				.197	.000	.923	.997	.556	.517
	TG	Sig.					.113	.310	.467	.401	.238
	Chol	Sig.						.418	.538	.141	.147
	HBAIC	Sig.							.141	.676	.531
	FBS	Sig.								.333	.670
	VASPIN	Sig.									.000

statistically significant inverse correlation between LDL and HDL of obese non-diabetic (P-0.011). However, the same table reveals in obese diabetic patients there is a direct proportional correlation between cholesterol and LDL and TG (P=0.000, P=0.023) respectively and a high direct proportional correlation between cholesterol and BMI (P=0.003), while the study appeared highly direct portion correlation between cholesterol and LDL (P=0.000) within obese non-diabetic control. Also, the same table shows there is a strong direct proportional correlation between RBS and HBA (P=0.001). Ultimately, the data of this study show a direct proportional correlation between adiponectin and vaspin (P=0.000).

DISCUSSION

This study showed that different lipid concentrations depend on the presence or absence of obesity in patients newly diagnosed with T2DM. Therefore, it is assumed that the levels of fat cells in the blood serum may indicate an established pathophysiological problem that varies among diabetic patients. In order to give an informed understanding of the role of these cells in the relationship between obesity and diabetes, the present study was carried out in order to investigate the levels of adipose cells in newly diagnosed T2DM patients, as this study showed that these patients have different degrees of obesity. The results of this study also showed the appearance of low serum concentrations of adiponectin and vaspin in obese and diabetic patients. Moreover, a confounding adipocytokine profile appeared in obese T2DM patients, and this gives us an indication that obesity and T2DM are interrelated, and on this basis, adipocytokine concentrations are regulated, which in turn could be a promising method for treating patients with T2DM (Wei *et al*, 2020).

However, in the current study, the mean age for obese diabetic patients was 55.98±7.642 years and obese non-diabetic control was 56.63±9.268 years with no significant difference. Hence, the two groups were age-matched**.** According to the WHO, middle-aged and older adults are still at the highest risk for progression T2DM. According to IDF (2009), adults aged 45–64 were the most diagnosed age group for diabetes. In our study, regardless of the glycemic status, but the serum adiponectin levels are lower in obese diabetic patients compared to obese non-diabetics obese and the difference is the lower statistical significance (P= 0.0001). Where, a study by Saltevo *et al* (2009) indicated that the women who have adiponectin concentrations are decreased relatively more than the men across individuals with prediabetes and T2DM. In the present study, similarly, serum vaspin levels in obese non-diabetic women were significantly increased (P=0.055) in comparison to obese diabetic women, this our results are consistent with other study gave the same results (Teresa *et al*, 2011).

Conversely, other workers confirm different results regarding Vaspin, who found a higher statistically significant concentration in obese diabetic patients (Mohammed *et al*, 2020).

So, the idea associated with obesity and diabetic Mellitus can assist in explaining the indecision of disease in patients with T2DM and the prospect of facilitating the personalized treatment. Notably, concentrations of Serum adiponectin have been shown to be directly proportional correlated with Vaspin in obese with diabetic patients (Qadir *et al*, 2015). This is consistent with the results of previous studies (Stêpieñ *et al*, 2013; Neuparth *et al*, 2013; Darabi *et al*, 2015). The present study illustrated that a lower level of adiponectin was occur in obese with diabetic patients than in those who had normal BMI and non-diabetic. Furthermore, noted that the diversity that will happen between those groups was constant after variation for BMI, which refers that there is a relevance between both adiponectin and BMI status in obese women patients with diabetes. Adiponectin is believed to have anti-diabetic and anti-inflammatory effects. Therefore, it is acceptable to assume that patients with T2DM are obese than patients with T2DM who have a normal BMI. However, obesity was described as repeated comorbidity in women patients with T2DM and it has been evaluated that a minimum of 90% of these patients is obese or overweight (Qadir *et al*, 2015). In addition to, the abundant complications and comorbidities (e.g., cardiovascular illness and chronic kidney diseases) due to the huge increase in women patients with diabetic mellitus type2 who suffering from concomitant obesity. (Stêpieñ *et al*, 2013; Neuparth *et al*, 2013; Darabi *et al*, 2015). However, a recent study of newly identified diabetic cases in Japan showed that serum adiponectin levels are inversely associated with the incidence of diabetes (Yamamoto *et al*, 2014).

Our findings propose that in obese diabetic patients, adipocytokine concentrations both of adiponectin and Vasspin vary between patients with non-obese) and those who are obese. Patients with T2DM who were obese exhibited a profile of disordered adipocytokine in the form of significantly decreased vaspin concentration and decreased level of adiponectin, in comparison to the patients with T2DM who had a normal BMI (non-obese diabetic). After time studies are needed to determine the causal relationships implicated and to determine whether a treatment that organizes adipocyte levels can help personalized accession to diabetes administration. It is noteworthy, scientists are looking forward to several adipocytokines, especially adiponectin, as a future treatment for diabetes.

As shown in Table 3, there was a statistically significant direct proportional control (p=0.008), but not in obese DM. Conversely, a directly proportional correlation between cholesterol with LDL in obese D. and obese non-D. (p=0.0001), TG in obese diabetic patients (p=0.023), while inverse significant correlation between cholesterol, BMI and HDL (p=0.003, p= 0.0001) respectively in women obese without diabetic control. Direct proportional association between obesity and Age in women obese without diabetic control group only (p=0.008). Also, data are found there is an inverse significant correlation between LDL with both BMI and HDL (p=0.003, p=0.0001) in the order in obese non-diabetic control otherwise obese diabetic patients. Furthermore, inverse significant correlation in obese diabetic patients between TG and HDL (p=0.007) in addition to obese non-diabetic control with age (p=0.049) and positive with BMI (P=0.000l) in the same group. Direct proportional significant correlation between FBS and HBA1C (p=0.00l) in obese diabetic otherwise in obese without diabetic Control. However, in obese women diabetic patients and obese without diabetic control, there is a direct significant correlation between Adiponectin with Vasspin, cholesterol, and LDL in obese with diabetic and obese without diabetic (p=0.027, p=0.001, p=0.002) respectively, only between Adiponectin and HDL appear inverse significant correlation in obese non-diabetic control only (p=0.047). On the other hand, there is an inverse correlation between Apelin and Vasspin (p=0.005) in obese diabetic patients, while direct proportional with Vasspin (p=0.0001) in obese non-diabetic. However, the same table was revealed that there is a direct proportional significant correlation between Leptin with Vasspin and Apelin (p=0.033, p=0.004) in the order in the obese non-diabetic control group.

Conflict of interest : None

Source of findings : self-findings.

Ethical clearance : This research was carried out with the patient's verbal and analytical approval before the sample was taken.

REFERENCES

Cho Jin-Kyung, Tae-Kyung Han and Hyun-Sik Kang (2010) Combined effects of Body Mass Index and cardio/Respiratory fitness on serum vaspin concentrations in Korean Young Men. *Europ. J. Appl. Physiol.* **108**(2), 347–353.

Codairo (2010) Type 2 Diabetes, Pre-Diabetes and the metabolic syndrome. *Springer Science & Business Media.*

Dai Rongfeng, Zhenhua Dong, Yu Qian and Yan Han (2016) Obese type 2 diabetes mellitus patients have higher serum Vaspin concentrations. *J. Diab.* **8**(3), 445–447.

Darabi H, Raeisi A and Kalantarhormozi M R (2015) Adiponectin as

a protective factor against the progression toward type 2 diabetes mellitus in postmenopausal women. *Medicine* **94**, e1347.

Di Angelantonio Emanuele *et al* (2016) Body-Mass Index and All-Cause Mortality: Individual-Participant-Data Meta-Analysis of 239 prospective studies in four continents. *The Lancet* **388**(10046), 776–86.

Diabetes Description O F (2007) Diagnosis and classification of Diabetes mellitus. *Diabetes Care* **30**(Suppl. 1), S42–S47.

Eichelmann F *et al* (2017) Novel adipokines: Methodological utility in human obesity research. *Int. J. Obes.* **41**(6), 976–81.

Fang Han and Robert L Judd (2018) Adiponectin regulation and function. *Comprehensive Physiology* **8**(3), 1031–63.

International Diabetes Federation (IDF) (2009) *Diabetes Atlas.* **4**th ed. Brussesls: International Diabetes Federation; 2009.

Kahn Barbara B and Jeffrey S Flier (2000) Obesity and insulin resistance. *J. Clin. Invest.* **106**(4), 473–481.

Mohammed Ismael Dawood, Kalaf Abdullah Sayer and Mohanned H Alwan (2020) Association between serum vaspin levels and diabetes in obese T2DM patients. *Annals Trop. Med. Publ. Hlth* **23**(7), 839-843. http://doi.org/10.36295/ASRO.2020.2374

Neuparth M J, Proença J B and Santos-Silva A (2013) Adipokines, oxidized low-density lipoprotein, and C-reactive protein levels in lean, overweight and obese Portuguese patients with type 2 diabetes. *ISRN Obes.* **2013**, 142097.

Qadir M I and Ahmed Z (2017) lep expression and its role in obesity and type-2 diabetes. *Crit. Rev. Eukaryot. Gene Expr.* **27**, 47–51.

Saltevo J, Kautiainen H and Vanhala M (2009) Gender differences in adiponectin and low-grade inflammation among individuals with normal glucose tolerance, prediabetes and type 2 diabetes. *Gend. Med.* **6**, 463–470.

Sobczak Amélie I S, Claudia A Blindauer and Alan J Stewart (2019) Changes in Plasma free fatty acids associated with type2 diabetes. *Nutrients* **11**(9), 1–42.

Stêpieñ M, Stêpieñ A and Wlaze³ R N (2013) Obesity indices and adipokines in non-diabetic obese patients with early stages of chronic kidney disease. *Med. Sci. Monit.* **19**, 1063–1072.

Stolarczyk Emilie (2017) Adipose tissue inflammation in obesity: A metabolic or immune response? *Curr. Opinion Pharmacol.* **37**, 35–40. http://dx.doi.org/10.1016/j.coph.2017.08.006.

Tan Bee K *et al* (2008) Metformin decreases the Adipokine Vaspin in overweight women with Polycystic Ovary Syndrome concomitant with improvement in insulin sensitivity and a decrease in insulin resistance. *Diabetes* **57**(6), 1501–1507.

Teresa Auguet, Yunuen Quintero, David Riesco, Beatriz Morancho, Ximena Terra, Anna Crescenti, Montserrat Broch, Carmen Aguilar, Montserrat Olona, José Antonio Porras, Mercè Hernandez, Fátima Sabench, Daniel del Castillo and Cristóbal Richart (2011) New adipokines vaspin and omentin. Circulating levels and gene expression in adipose tissue from morbidly obese women. *BMC Med. Gen.* **12**, 60.

Tumminia Andrea *et al* (2019) Adipose tissue, obesity and adiponectin: Role in endocrine cancer risk. *Int. J. Mol. Sci.* **20**(12).

Wei Liu M D, Xianghai Zhou, MD Yufeng Li, MD, Simin Zhang, MD Xiaoling Cai, MD, Rui Zhang, MD, Siqian Gong, MD, Xueyao Han MD and Linong Ji MD (2020) Serum leptin, resistin, and adiponectin levels in obese and non-obese patients with newly diagnosed type 2 diabetes mellitus. *Medicine* **99**(6), e19052.

Yamamoto S, Matsushita Y, Nakagawa T, Hayashi T, Noda M and Mizoue T (2014) Circulating adiponectin levels and risk of type 2 diabetes in the Japanese. *Nutr. Diabetes* **4**, e130.

Youn Byung-Soo *et al* (2008) Serum Vaspin concentrations in human obesity and type 2 diabetes. *Diabetes* **57**(2): 372–377.