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The Ameliorative Effect of Liraglutide (Saxenda® and Victoza®) Treatment on Overweight and Induced Diabetes Mellitus Type 2 in Male Rats

Article Info.

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Abstract

Recent researches are increasingly focused on incretin-based therapies, which is use the body's natural "incretin effect" to manage diabetes and overweight. The study's goal is to utilize the incretin-mimicking medications to leverage their efficiency. 42 male rats were allocated into 7 groups (6 rats per group); G1 (Control) received an IP injection of NS for 4 weeks. G 2 (HFD group) is categorized into 3 subgroups. The positive control group received daily injections of normal saline for 4 weeks. Victoza and Saxenda-treated groups were administered a graduated Sc dose from 0.6 to 1.2 mg/kg daily for 4 weeks. G3 Induced diabetes mellitus type 2 (IDM) is categorized into three categories: positive control: received daily injections of NS (1 ml/kg) for 4 weeks. Victoza and Saxenda-treated groups received Sc injections of graduated doses from 0.6 to 1.2 mg/kg daily for 4 weeks. The results showed a significant increase in incretin hormone in the HFD and IDM Victoza groups compared to the HFD and IDM Saxenda groups; while the insulin hormone showed a significant decrease in HFD and IDM Victoza groups compared to the same groups treated with Saxenda. HDL showed a significant elevation in groups IDM and HFD treated with Victoza, more than those treated with Saxenda. LDL levels appeared significantly decreased in the IDM and HFD Victoza groups compared to the same groups treated with Saxenda. The study shows that these two medications work by acting as agonists at the GLP-1 receptor, which in turn increases “insulin secretion and improves incretin signaling”

Keywords:Glucagon-Like-Polypeptide-1 (GLP-1), Glucose inhibitory peptide (GIP), Liraglutide, Saxenda®, and Victoza®.

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Introduction

Modulating insulin secretion in response to fluctuations in glucose levels, incretin is a peptide hormone secreted postprandially and generated in the gut. Glycemic regulation has been greatly improved by incretin-based therapy, which was initially developed for the management of Diabetes mellitus type 2. In the fight against overweight, it shows great promise. There has been recent evidence that these medications significantly improve cardiovascular and kidney health (1)**.** The insulin-stimulating effects of two extensively researched incretins, glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), are caused by separate G-protein-coupled receptors that are abundantly expressed on islet β cells. Many cell types outside of islet cells express the GLP-1 and GIP receptors (2).

Specialized enteroendocrine cells, called L cells, are mostly located in the colon and distal intestine, and they secrete glucagon-like peptide-1 (GLP-1). During fasting, GLP-1 secretion is low and continuous at a basal level; after eating, it increases to a bolus release, around two to three times its baseline level. It improves glucose regulation by increasing the generation of insulin that is dependent on glucose and decreasing the release of glucagon from pancreatic α-cells (3)**.** Several bodily systems and organs include the GLP-1 receptor, including the gastrointestinal tract, brain, pancreas, and fat (4). In response to food intake, K cells in the large intestine secrete glucose-dependent insulinotropic polypeptide, also known as GIP, which plays an essential role in maintaining glucose balance by enhancing the generation of insulin from pancreatic β-cells that is dependent on glucose. This is achieved by attaching to the GIP receptor and activating the cAMP signaling pathway (5)**.** Restoring GIP deficiency using a GIP receptor agonist would be an intriguing area for future studies, as GIP's insulinotropic effects are reduced in people with Diabetes mellitus Type 2 (6)**,** One important aspect of GIP's regulatory effect on glucose homeostasis is that, unlike GLP-1, which inhibits glucagon synthesis, it stimulates α-cells to enhance glucagon release (7)**.**

Liraglutide is a glucagon-like peptide-1 receptor antagonist initially authorized for the treatment of Diabetes Mellitus type 2. Research assessing liraglutide for overweight management employed a dosage exceeding that sanctioned for diabetic treatment. The advised maintenance dosage of liraglutide for diabetes is 1.2 mg or 1.8 mg, delivered subcutaneously once daily, contingent upon glycemic response (8)**.** The Saxenda®, a glucagon-like peptide 1 (GLP-1) agonist, is injected subcutaneously at a high dosage of 3.0 mg daily. It received FDA approval in 2014 and EMA authorization in 2015 for the long-term management of weight. Victoza, a glucagon-like peptide 1 (GLP-1) agonist approved in 2010 for the treatment of type 2 diabetes mellitus, is administered at a dosage of 1.8 mg subcutaneously (SC) once daily.

The primary function is to regulate blood glucose by suppressing glucagon secretion and promoting insulin synthesis from pancreatic β-cells in a glucose-dependent fashion (9). The present study intends to evaluate the efficacy of the recently introduced treatments Saxenda® and

Victoza®, which replicate the composition and effects of incretin, and to investigate the impact of these treatments to determine their relative efficiency.

Material and Methods

Experimental animals

The present study was carried out at the laboratory animal house in the College of Veterinary Medicine, University of Basrah. 42 male rats weighing (80±25 gm) and aged (3 months) were used for the present study. The animals were acclimated for two weeks before the start of the experiments. All the experimental animals were maintained under optimum conditions (24±2°C), and a 12/12-hours light/dark cycle throughout the study. The food and drinking water were administered *ad libitum* throughout the experimental period. The Ethical Committee of the College of Veterinary Medicine, University of Basrah, accepted the study protocol, which was carried out between December/2024 and July/2025.

High-fat diet (HFD) and Diabetes Mellitus Type 2 (DM type 2) Induction and protocol of the study

A total of 42 adult male rats; Three groups of rats were randomly assigned; Group 1, serving as the negative control, was administered a low-fat diet. Group 2 was administered a high-fat diet for 12 weeks and was subdivided into three subgroups: positive control, Victoza-treated, and Saxenda-treated groups. Group 3 was administered a high-fat diet (HFD) for four weeks and induced type 2 diabetes mellitus via a single intraperitoneal injection of streptozotocin (STZ) at a dosage of 35 mg/kg, dissolved in a freshly produced sodium citrate buffer (0.1M, pH 4.5)(10). Both STZ and citrate buffer solutions were maintained at low temperatures using ice bags in heat-insulated cases, as STZ is sensitive to heat and light and unstable even under acidic conditions. Following STZ injection, a 10% sucrose solution was administered as a substitute for drinking water for 48 hours to prevent potentially lethal hypoglycemia resulting from extensive β-cell necrosis and the abrupt release of a substantial amount of insulin(11). The blood glucose meter (CONTOUR®PLUS Self-Monitoring Blood Glucose) was employed to assess blood glucose levels seven days post-STZ injection. Animals exhibiting glucose levels over 250 mg/dl were incorporated into the study and subsequently categorized into three subgroups: positive control, Victoza-treated, and Saxenda-treated groups.

Blood sample preparation and biochemical assays

Following the administration of 1.9% breathed diethyl ether for anesthesia. Cardiac puncture was performed to collect 5 ml blood samples from each rat using a disposable 10 ml syringe (12). The samples were placed in gel tubes and centrifuged at 3000 rpm for 15 minutes to achieve serum

separation into a clear, non-hemolyzed supernatant, which was then divided into two portions and stored in polyethylene Eppendorf tubes at –20 °C for subsequent hormonal and biochemical analysis. The serum samples were analyzed with an automated Mindray analyzer for hormonal and biochemical parameters, encompassing Incretin, Insulin, Leptin hormones, and lipid profile components, including Total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL).

Statistical analysis

The current studies' data had been analyzed in the computerized SPSS (Statistical Packages for the Social Sciences) V.13, programmed using univariate analysis of variance (ANOVA). The threshold of significance was set at $P<0.05$. The data were presented in the form of mean ± standard deviation. The least significant difference (LSD) test was used to compare groups (13).

Results

Effect of Liraglutide Treatment on Digestive Hormones in Overweight and Induced Diabetes Mellitus Type 2 Male Rats

The data of incretin hormone in table (1) showed a significant ($P\leq0.05$) elevation in HFD Victoza and IDM Victoza treated groups than the positive control and the negative control groups. In contrast, the HFD, Saxenda, and IDM Saxenda-treated groups showed statistically significant ($P \leq 0.05$) higher levels of this hormone compared to the positive control DM and negative control groups. Insulin hormone data reported a significant ($P \leq 0.05$) decrease in the IDM Victoza and HFD Victoza treated groups compared to the positive control, the negative control, and all treated groups. In contrast, the IDM Saxenda and HFD Saxenda groups still had higher insulin hormones than all groups treated with Victoza, but lower than the HFD positive control. The leptin hormone appeared to decrease significantly ($P\leq0.05$) in IDM Victoza as compared to the positive control group and HFD Saxenda-treated groups more than all other groups, except the IDM positive control group. Still, levels of this hormone showed no reported differences between all other groups.

Effect of Liraglutide Treatments on Lipid Profile in Overweight and Induced Diabetes Mellitus Type 2 Male Rats

In table (2) effect of liraglutide treatments on lipid profile in Overweight and IDM type 2 male rats is shown, TC levels increased significantly ($P\leq0.05$) in group IDM that treated with Saxenda than group treated with Victoza and negative control and positive control IDM, but when compared with HFD group that same treated showed decrement in HFD treated with Saxenda than HFD

treated with Victoza, but these groups showed a significant decrease as compared with the positive control groups.

The results of TG level in groups of IDM showed a significant (P≤0.05) lowering in the group treated with Victoza and Saxenda than in the positive control IDM group, but still more than in the negative control group. When comparing these groups with groups of HFD treatments, there was a significant (P≤0.05) decrement compared to the HFD Victoza group, but more than in the HFD Saxenda and positive control HFD groups. While in groups of HFD, TG levels appeared statistically (P≤0.05) lower in the group treated with Saxenda than in the group treated with Victoza.

Table (1): Effect of Liraglutide Treatment on Digestive Hormones in Overweight and Induced Diabetes Mellitus Type 2 Male Rats (Mean ±SD).

Parameters	Groups	Mean	Std. Deviation
Incretin (pg/mL)	-Control	46.33 c	22.54
	+ Control IDM	27.91 d	10.48
	IDM Victoza	110.53 a	15.64
	IDM Saxenda	59.98 b	6.79
	+ Control HFD	49.28 c	3.76
	HFD Victoza	107.34 a	18.46
	HFD Saxenda	72.01 b	14.66
Insulin (ng/mL)	-Control	1.32a	0.17
	+ Control IDM	1.90 a	0.40
	IDM Victoza	0.48 b c	0.02
	IDM Saxenda	1.15 a	0.13
	+ Control HFD	0.92b	0.12
	HFD Victoza	0.11c	0.03
	HFD Saxenda	0.93 a	1.35
Leptin (ng/mL)	-Control	0.42b	0.04
	+ Control IDM	0.50 a	0.011
	IDM Victoza	0.40 b	0.008
	IDM Saxenda	0.41b	0.008
	+ Control HFD	0.35b	0.03
	HFD Victoza	0.45 a	0.018
	HFD Saxenda	0.49a	0.02

a, b, c……sign to significant differences between groups within (P≤0.05).

The levels of HDL affected by Liraglutide treatments recorded in table (2), which showed significantly (P≤0.05) elevation in groups IDM and HFD treated with Victoza more than to that treated with Saxenda, and with group negative control and positive control IDM and HFD, but when comparing these groups with those groups in HFD group treatments appeared significantly (P≤0.05) lower HDL levels.

The LDL appeared significantly (P≤0.05) decrement in groups IDM and HFD groups treated with Victoza as compared to other groups of IDM and negative control groups and lesser than to those groups of HFD treated with same treatments, but in group of HDF groups appeared significantly (P≤0.05) decrease in group treated with Victoza than to that treated with the Saxenda and positive control HFD group, but higher to that levels in the negative control group.

The VLDL levels were lowered in groups of IDM treated with Victoza and Saxenda to similar levels, but lower than those in the positive control IDM and higher than in the negative control, and the data appeared to be no significant differences between these groups and the group HFD Saxenda treatment, but lower than that level in the group HFD treated with Victoza, but in IDM-treated groups, the VLDL level significantly (P≤0.05) lowers in the group treated with Saxenda than in the group treated with Victoza and also compared to the positive control HFD group, but higher than in the negative control group.

Discussion

The data showed a significant increase in incretin in the IDM and HFD Victoza-treated groups compared to all treated and positive control groups. In the first stages of Diabetes Mellitus Type 2, the production of GLP-1 and GIP may rise as a compensatory response to counteract insulin resistance. Chronic elevation of glucose levels can lead to incretin resistance and subsequent reduction due to diminished GLP-1 secretion. Liraglutide, a GLP-1 receptor agonist, has extended activity compared to natural GLP-1 and elicits incretin effects, including enhanced insulin secretion, reduced glucagon levels, and delayed stomach emptying (14). The result agrees with (15), which showed in diabetic animals indicates that liraglutide mitigates diabetes progression, enhances insulin production, and improves β-cell mass/function, aligning with an elevated incretin response in treated, induced-diabetes subjects. Liraglutide (Victoza) is a GLP-1 receptor agonist that simulates endogenous GLP-1, potentially maintaining or augmenting incretin signaling despite metabolic impairment The HFD alone may hinder incretin secretion; however, the addition of liraglutide could reinstate GLP-1 activity. HFD promotes the proliferation of endocrine cells in the duodenum, leading to the differentiation into K cells and excessive secretion of GIP. The gradual increase in GIP levels may signify the trigger for insulin hypersecretion. Moreover, Victoza impedes nutritional transit and gastrointestinal sensitivity, hence augmenting natural incretin release, which improves endogenous secretion. This result agrees with (16), which showed that exogenous injection of GLP-1 has been observed to reduce stomach emptying, intestinal transit speed, and pancreatic exocrine secretion, thereby augmenting endogenous GLP-1 secretion and increasing incretin levels.

Table (2): Effect of Liraglutide Treatments on Lipid Profile in Overweight and Induced Diabetes Mellitus Type 2 Male Rats (Mean ±SD).

Parameters	Groups	Mean	Std. Deviation
TC (mg/dl)	-control	30.55 d	2.11
	+ control IDM	162.93 a	6.08
	IDM Victoza	50.64 c	2.14
	IDM Saxenda	79.57 b	3.17
	+ control HFD	160.32a	5.12
	HFD Victoza	75.54 b	3.59
	HFD Saxenda	71.16 b	3.13
TG (mg/dl)	-control	29.43 c	5.98
	+ control IDM	176.91 a	11.06
	IDM Victoza	64.59 b	12.14
	IDM Saxenda	64.00 b	15.98
	+ control HFD	160.96a	5.99
	HFD Victoza	78.04 b	4.92
	HFD Saxenda	57.61 b	3.27
HDL (mg/dl)	-control	26.53c	1.18
	+ control DM	19.41 c	5.90
	IDM Victoza	41.43 a	5.30
	IDM Saxenda	37.93 ab	6.92
	+ control HFD	11.24c	2.79
	HFD Victoza	41.31 a	7.81
	HFD Saxenda	34.53b	5.16
LDL (mg/dl)	-control	19.87c	1.50
	+ control DM	98.91a	5.15
	IDM Victoza	17.36 c	4.45
	IDM Saxenda	28.84 b c	3.74
	+ control HFD	93.97a	6.73
	HFD Victoza	37.29 b	7.89
	HFD Saxenda	41.73b	1.13
VLDL (mg/dl)	-control	7.32c	1.76
	+control DM	35.08 a	5.28
	IDM Victoza	12.92 b	2.42
	IDM Saxenda	12.80 b	3.19
	+ control HFD	32.21a	5.05
	HFD Victoza	14.22 b	2.55
	HFD Saxenda	11.61 b	1.51

a, b, c……sign to significant differences between groups within (P≤0.05)

The IDM and HFD treated with Saxenda exhibit reduced incretin levels relative to the positive control and the corresponding groups treated with Victoza, as exogenous GLP-1 receptor agonists like liraglutide can downregulate the synthesis of endogenous proglucagon-derived peptides (PGDPs), including endogenous GLP-1. This impact seems to manifest independently of weight loss and presumably indicates direct negative feedback on the cells (e.g., L-cells) responsible for producing these incretin hormones (17).

These results agree with (18), which showed that Liraglutide significantly downregulates Proglucagon-Derived Peptides (PGDPs), and normalizing their levels may yield further metabolic, weight reduction, and glycemic management advantages in the future.

Our findings demonstrated a significant decrease in insulin levels in both the IDM and HFD Victoza-treated groups as compared to the control and other treated groups. Victoza reduces insulin levels, facilitates normal blood sugar regulation, and preserves β-cells from fatigue through enhanced insulin sensitivity. Furthermore, weight reduction achieved via Victoza diminishes insulin resistance over time (19). The results agree with (20), which found that administering Liraglutide to obese or overweight individuals with type 2 diabetes resulted in anticipated weight loss in the majority of participants, a subsequent reduction in body mass index (BMI) and fat mass percentage, and an enhancement in glucose homeostasis, surpassing the effects of caloric restriction.

The results of the current study showed a significant decrease in leptin level for the IDM treated with Victoza and an increase in HFD treated with Saxenda compared to the positive control groups. Liraglutide (Victoza) enhances β-cell functionality, leading to a slight restoration of insulin secretion. Insulin is a crucial regulator of leptin production; even minor elevations in insulin can provoke leptin release from adipocytes (21). The results agree with (22), who reported that indicators of peripheral adiposity, including insulin and leptin, are essential as they convey the body's energy status to the brain and facilitate their metabolic effects through signal transduction in hypothalamic areas that govern food intake, energy expenditure, and glucose metabolism. Changes in insulin and leptin signaling may lead to diabetes and obesity. The primary signaling interaction between insulin and leptin is crucial for sustaining adequate energy homeostasis.

The results of the present study demonstrate that the level of TC and TG in the IDM Victoza is lower than IDM Saxenda and the positive control because the Victoza group is experiencing a more moderate weight loss. Their weight reduction is adequate to enhance cholesterol levels; however, not so quick as to induce a substantial increase in lipid mobilization. Consequently, their overall cholesterol levels are reduced. The Saxenda-treated group, on a trajectory towards improved long-term metabolic health, is in a heightened catabolic state at the time of measurement. The temporary rise in lipids from liberated free fatty acids counteracts the fundamental enhancement, leading to an elevated TC measurement relative to the Victoza group (23). While the TC and TG in the treated group with Victoza showed higher levels than the treated groups with Saxenda due to Liraglutide reducing postprandial triglycerides by inhibiting chylomicron (apoB48) production and enhancing its clearance, kinetic studies indicate an approximately 60% reduction in apoB48 synthesis and a decrease in the triglyceride area under the curve after meals (24). The results agree with (25), who demonstrated that GLP-1 has a significant influence on blood glucose regulation, and its impact on lipid metabolism is more efficient.

The results of the current study showed a significant increase in HDL levels in IDM treated with Victoza compared to IDM treated with Saxenda and the positive control, Victoza's mechanism in diabetes diminishes hyperglycemia and resistance to insulin, resulting in reduced triglyceride synthesis and enhanced clearance. As triglyceride levels decrease, the biochemical suppression of HDL is alleviated, resulting in a substantial increase in HDL levels from a markedly low baseline. Saxenda, while advantageous, prioritizes calorie limitation, appetite control, and weight reduction. In IDM animals, this intensified catabolic condition may diminish hepatic HDL synthesis, notwithstanding the reduction in TG and LDL levels (26). The results agree with (27) GLP-1 enhances glucose-stimulated insulin levels without affecting glucose tolerance, systemic insulin sensitivity, or circulating cytokine levels. GLP-1R agonists suppress stomach and small intestine motility. GLP-1 decreases postprandial chylomicron production and reduces circulating triglyceride levels.

The results of the current study showed a significant decrease in LDL and VLDL levels in the IDM and HFD-treated groups with Victoza compared to the IDM and HFD groups treated with Saxenda and the positive control. Saxenda induces a significant caloric deficit and weight reduction, while adipose tissue degrades triglycerides, liberating a considerable quantity of free fatty acids (FFAs) into the circulatory system. The liver absorbs these FFAs, and it encapsulates the abrupt surge of lipids into VLDL for exportation, which is converted into IDL and subsequently into LDL. Consequently, an elevation in VLDL production frequently results in a corresponding rise in LDL-C levels, particularly in the short to medium term(28).The results agree with (29); who showed that in the liver, fatty acids originate from the absorption of plasma-free fatty acids released by adipose tissue from triglycerides that have undergone lysosomal lipolysis following the uptake of triglyceride-rich remnant lipoproteins by the liver, and from de novo lipogenesis of fatty acids derived from acetyl-CoA. The use of triglycerides includes the oxidation of fatty acids, primarily inside mitochondria, and the production of VLDL. When synthesis exceeds consumption, lipid droplets form from the cytosolic aspect of the endoplasmic reticulum (ER) membrane and accumulate in the cytoplasm. Enhancing the liver's capacity to release VLDL might mitigate hepatic lipid buildup.

Conclusion

According to the research, these two medications work by acting as agonists at the GLP-1 receptor, which in turn improves incretin signaling, increases insulin production, decreases glucagon, and slows down stomach emptying. Victoza® may be better suited for diabetic patients looking for better glycemic control and lipid profile due to its chemical formulations (including different preservatives: m-cresol in Victoza vs. benzoic acid in Saxenda). At the same time, Saxenda® may be better for overweight patients managing their weight loss while carefully monitoring lipid profile parameters during rapid weight loss.

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Conflict of interest: There is no need to declare a conflict of interest.

Ethical Clearance

This work is approved by The Research Ethical Committee

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التأثير المحسن لعلاج ليراجلوتيد (ساكسيندا® وفيكتوزا®) على زيادة الوزن والسُكَّري المُحْدَث من النوع الثاني في ذكور الجرذان.

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الخلاصة

تُركز الأبحاث الحديثة بشكل متزايد على العلاجات القائمة على هرمونات الإنكرتين، والتي تستخدم "تأثير الإنكرتين" الطبيعي في الجسم لإدارة مرض السكري والوزن الزائد من خلال تعزيز إفراز الأنسولين بعد الوجبات. تهدف الدراسة إلى استخدام الأدوية المحاكية للإنكرتين حديثة التطور (Saxenda® وVictoza®) للاستفادة من كفاءتها. تم توزيع اثنين وأربعين من ذكور الجرذان البالغة عشوائياً إلى 7 مجموعات (6 جرذان في كل مجموعة): المجموعة الأولى (السيطرة) تلقت حقناً داخل غشاء البطن بمحلول ملحي طبيعي لمدة 4 أسابيع. المجموعة الثانية (مجموعة النظام الغذائي عالي الدهون) تم تقسيمها إلى 3 مجموعات فرعية. المجموعة االسيطرة الموجبة تلقت حقناً يومياً بمحلول ملحي طبيعي لمدة 4 أسابيع. بينما تلقت مجموعات العلاج بـ Victoza وSaxenda جرعات تحت الجلد متدرجة من 0.6 إلى 1.2 مجم/كجم يومياً لمدة 4 أسابيع. المجموعة الثالثة (مجموعة السكري المُستحث نمط 2) تم تقسيمها إلى ثلاث فئات: السيطرة الموجبة: تلقت حقناً يومياً بمحلول ملحي طبيعي (1 مل/كجم) لمدة 4 أسابيع. بينما تلقت مجموعات العلاج بـ Victoza وSaxenda حقناً تحت الجلد بجرعات متدرجة من 0.6 إلى 1.2 مجم/كجم يومياً لمدة 4 أسابيع. أظهرت النتائج زيادة معنوية في هرمون الإنكرتين في مجموعات النظام الغذائي عالي الدهون والسكري المُستحث المعالجة بـ Victoza مقارنة بمجموعات النظام الغذائي عالي الدهون والسكري المُستحث المعالجة بـ Saxenda؛ بينما أظهر هرمون الأنسولين انخفاضاً معنوياً في مجموعات النظام الغذائي عالي الدهون والسكري المُستحث المعالجة بـ Victoza مقارنة بنفس المجموعات المعالجة بـ Saxenda. كما أظهرت مستويات البروتين الدهني عالي الكثافة (HDL) ارتفاعاً معنوياً في مجموعات السكري المُستحث والنظام الغذائي عالي الدهون المعالجة بـ Victoza، أكثر من تلك المعالجة بـ Saxenda. بينما ظهر البروتين الدهني منخفض الكثافة (LDL) منخفضاً بشكل معنوي في مجموعات السكري المُستحث والنظام الغذائي عالي الدهون المعالجة بـ Victoza مقارنة بنفس المجموعات المعالجة بـ Saxenda. الملخص تظهر الدراسة أن هذين الدوائين يعملان من خلال العمل كمواد ناهضة لمستقبلات GLP-1، مما يزيد بدوره من إفراز الأنسولين، ويقلل من الجلوكاجون، ويحسن إشارات الإنكرتين.

الكلمات المفتاحية: البيتيد الشبيه بالجلوكاجون-1, البيتيد الإنسولينى المنشط المعتمد على الجلوكوز,ليراجلوتيد ,ساكسندا®,فيكتوزا®