

## **PHYTOCHEMICAL INVESTIGATION AND ANTIDIABETIC ACTIVITY OF HERBAL FORMULATION OF *OUGEINIA OOJEINENSIS* PLANT EXTRACTS**

### **ABSTRACT**

The goal of this study was to assess the antidiabetic potential of produced herbal formulations of *Ougeinia oojeinensis* plant extracts. The results show that phospholipids complex-based drug delivery systems have a lot of promise for increasing the in-vivo antidiabetic activity of plant extracts. The phospholipids complex has a relationship with different physicochemical properties such as size and entrapment effectiveness. In compared to hydroalcoholic extract (200 mg/kg and 400 mg/kg), an improved phospholipids complex formulation can provide a substantial antidiabetic effect at a lower dose (100 mg/kg and 200 mg/kg). At a lower dosage than the hydroalcoholic extract, the extracts improved lipid profile and body weight. As a result, the dosage efficacy ratio was enhanced by using phospholipids complex formulation. The histological alterations of the pancreas and kidney in streptozotocin-nicotinamide caused diabetes were also prevented by an optimised phospholipids complex formulation of *Ougeinia oojeinensis* extract. As a result, the current investigation found that *Ougeinia oojeinensis* extract might be beneficial in the treatment of type 1 and type 2 diabetes with aberrant lipid profiles.

**Keywords:** *Ougeinia oojeinensis*, Antidiabetic, Herbal Formulation, Phospholipids complexes

### **INTRODUCTION**

Diabetes mellitus is a collection of disorders marked by elevated blood glucose levels caused by abnormalities in insulin synthesis, insulin action, or both, altered lipid, carbohydrate, and protein metabolism, and an increased risk of vascular disease complications<sup>1</sup>. It's the most prevalent endocrine condition, and it can lead to significant consequences and even death.

Diabetes is a disease that has been known for ages. It was mentioned as a sickness connected with the passing of a lot of urine in Eber's Egyptian Papyrus (1500 BC). Arataeus of Cappodica, a prominent Greek physician, invented the term diabetes (a syphon that defined it as the melting away of flesh and limbs into urine [1-4]).

During the 3rd and 6th centuries AD, scholars in China, Japan, and India described a polyurea-related ailment in which urine was "sweet and sticky." It wasn't until 1674 that Willis added the comment 'as it infused with honey and sugar', even though it had existed for decades. As a result, the word diabetes mellitus (mellitus-honey) was coined. Dobson, a century after Willis, proved

that the sweetness was attributable to sugar. From the period of the first known history of diabetes until the middle of the nineteenth century, progress in knowledge was gradual. A link was found between a disruption in the beta cells, which are concentrated as small tissue islets in the exocrine pancreas. During the nineteenth century, Brockman first recognised the occurrence of tiny islands in fish. Mering and Minkowski, two German biologists, discovered soon after that surgically removing the pancreas caused diabetes in dogs. In 1921, Fredrick Banting and Charles Best made a key advance in the development of insulin. Purified modified forms of insulin were accessible by 1952, and recombinant DNA insulin was available by 1982. As a result, diabetes, which had previously been associated with a deadly disease, has evolved into a more benign, manageable condition [5-8].



**Fig. 1: *Ougeinia oojeinensis***

The Fabaceae family includes *Ougeinia oojeinensis*. It is a herb that may be found all over the world, but especially in India's outer Himalayas and sub-Himalayan tracts from Jammu to Bhutan. Tinsa, Sandan, and Panjan are some of the common names for it. Anti-inflammatory and analgesic, hepatoprotective, antioxidant, anthelmintic, hypoglycemic, antidiabetic, and wound healing activities were found in the extract of the entire plant *O. oojeinensis*. Phytochemical studies on *O. oojeinensis* found lupeol, hydroxlupeol, betulin, and isoflavanones like dalbergioidin, homoferreirin, and ougenin, as well as lupeol, hydroxlupeol, betulin, and isoflavanones like dalbergioidin, homoferreirin, and ougenin. Jaundice, diarrhoea, dysentery, uorrhagia, diabetes, verminosis, leprosy, leucoderma, haemorrhages, fevers, ulcers, and other conditions are treated with the bark and leaves. The evaluation of literature on the *Ougeinia oojeinensis* (Roxb) Hochr has been done under many headings such as pharmacognostical review, phytochemical review, pharmacological review, and ethanomedical information for the same reasons. *Ougeinia oojeinensis* has the following botanical classification: Plant

classification is the division of plants into categories and subgroups. Binomial nomenclature generally indicates in naming a plant in terms of its genus name, and species names. The foundation of taxonomical is principally put forth by International code of botanical nomenclature. A significant number of plant families have common properties that allow researchers to study crude drugs from all of them at the same time. It is scientific way of naming, describing and arranging the plants in an orderly manner [8-13].

## **Material & Method**

### **Collection and authentication of Plant material**

The leaves of the plant *Ougeinia oojeinensis* were gathered and verified. The plants were dried in the shade, pulverised, and stored in an airtight container.

### **Screening for phytochemicals**

#### **Screening for Qualitative Phytochemicals**

The extracts of *Ougeinia oojeinensis* were subjected to preliminary phytochemical screening. Standard techniques were used to screen the crude extracts for the presence or absence of secondary metabolites such as alkaloids, steroidal chemicals, phenolic compounds, flavonoids, saponins, and tannins [3-4].

#### **Alkaloid testing [3]**

The chemical tests were conducted using a neutral or slightly acidic drug solution; alkaloids provide the following types of chemical tests:

##### **Dragendorff's test:**

The plant extract was treated with Dragendorff's reagent (potassium bismuth iodide). The presence of alkaloids was established by the appearance of an orange red coloured precipitate.

##### **Mayer's test:**

Adequate amount of plant extract was taken and few drops of Mayer's reagent (Potassium mercuric iodide) were added. Appearance of creamy-white coloured precipitate, confirmed the presence of alkaloids.

##### **Hager's test:**

Adequate amount of plant extract was taken and few drops of Hager's reagent (Saturated aq. solution of picric acid) were added. Appearance of crystalline yellow coloured precipitate, confirmed the presence of alkaloids.

**Wagner's test:**

Few drops of Wagner's reagent (Dilute iodine solution) were added to the plant extract. Appearance of reddish-brown coloured precipitate, confirmed the presence of alkaloids.

**Tannic Acid test:**

Few drops of tannic acid solution were added to the plant extract. Appearance of buff coloured precipitate, proved the presence of alkaloids.

**Test for carbohydrates [4]****Charring test:**

The plant extract was placed in a test tube and heated in the presence of concentrated  $H_2SO_4$  to induce charring and a burning sugar-like odour, indicating the presence of carbohydrate.

**Molish test:**

A few drops of Molish reagent (-naphthol) and conc.  $H_2SO_4$  were introduced from the sidewall of the test tube to the plant extract in a test tube. The presence of carbs was established by the appearance of a purple ring at the junction.

**Iodine test:**

A few drops of iodine solution were applied to the plant extract. The presence of starch was shown by the appearance of a blue colour that vanished on heating and returned on cooling. Polysaccharides are the focus of this test.

**Barfoed test:**

2 mL of Barfoed reagent (Cupric acetate, acetic acid, and water) was added to the plant extract and heated. The presence of monosaccharide was disclosed by the appearance of brick red precipitate in 5 minutes, whereas the presence of disaccharide was revealed in 7 minutes. It's a tool for distinguishing between monosaccharides and disaccharides.

**Test for a Fehling solution:**

The test consists of two solutions that are combined in situ and is mostly used for reducing sugars. Fehling solution A contains 0.5 percent copper sulphate while Fehling solution B contains sodium potassium tartarate. Equal amounts of Fehling A and Fehling B solutions (1 ml each) were combined, and 2 ml of plant extract was added before boiling on a water bath for 5-10 minutes. Appearance of reddish brown coloured precipitate due to formation of cuprous oxide, confirmed the presence of reducing sugar.

**Benedict's test:**

1 ml Benedict's solution (copper sulphate and sodium hydroxide) was added to the plant extract and heated almost to boiling, resulting in the appearance of green, yellow, orange, red, or brown colour in the test solution as the concentration of simple sugar in the solution increased, indicating the presence of reducing sugars due to the formation of cuprous oxide.

### **Protein and amino acid testing [3]:**

#### **Test for biuret:**

The plant extract was treated with a few drops of Biuret reagent (potassium hydroxide, copper sulphate, and sodium potassium tartrate). The presence of proteins was shown by the appearance of violet or pink colour.

#### **Millons test:**

Millons reagent (mercuric and mercurous ions in nitric and nitrous acids) was added to the plant extract in a few drops. The presence of proteins was demonstrated by a white precipitate that became crimson following heating over a hot water bath.

#### **Ninhydrin test:**

The plant extract was mixed with a few drops of a 5% Ninhydrin solution and cooked in a boiling water bath for 10 minutes. The presence of proteins was indicated by the presence of a purple or bluish colour.

#### **Lead sulphide test:**

To the plant extract, 2 mL of 10% NaOH solution was added, followed by a few drops of lead acetate solution. The solution was shaken before being heated for a few minutes in a water bath. The existence of proteins was confirmed by the appearance of black precipitate in the presence of sulfur-containing amino acids.

#### **Xanthoproteic test:**

2 ml dil. HNO<sub>3</sub> was added to a sufficient volume of plant extract. The presence of aromatic ring containing amino acids such as tyrosine, tryptophan, and others was shown by the appearance of yellow precipitate.

#### **Test for steroid use [3]:**

Plant extract was first extracted with chloroform, then a few drops of acetic anhydride were added, followed by conc. H<sub>2</sub>SO<sub>4</sub> from the test tube's side wall. At the intersection of two liquids, a violet to blue ring emerged, indicating the existence of a steroid entity.

#### **Salkovaski test:**

Plant extract was extracted first using chloroform, then conc.  $H_2SO_4$  was added from the test tube's sidewall. The presence of steroid was established by the appearance of a yellow ring at the intersection of two liquids, which became red after 2 minutes.

**Trichloro acetic acid test:**

Adequate amount of plant extract was taken and saturated solution of trichloro acetic acid was added. Colored precipitate appeared that proved the presence of steroid moiety.

**Test for glycosides [3]:**

**Borntrager's test:**

Dil. HCl was added to the appropriate amount of plant extract, heated, and filtered. To the filtrate, an equal volume of chloroform was added and thoroughly mixed. Ammonia was added after the chloroform layer was separated. The presence of anthraquinone was shown by the appearance of crimson in the ammonical layer.

**Modified test devised by Borntrager is as follows:**

A suitable amount of plant extract was obtained and diluted, then 5 ml  $FeCl_3$  was added. The filtrate was extracted with  $CCl_4$  or benzene and an equivalent amount of ammonia solution was added after boiling for 10 minutes on a water bath. The presence of anthraquinone moiety was indicated by the appearance of pink to red colour.

**Keller Killiani test:**

An equal volume of water and 0.5 ml of strong lead acetate solution were added to the appropriate amount of plant extract, shaken, and filtered. An equivalent volume of chloroform was used to extract the filtrate. The chloroform extract was evaporated to dryness, and the residue was dissolved in 3 mL glacial acetic acid, then a few drops of  $FeCl_3$  solution were added. The resulting solution was poured into a test tube with 2 mL of concentrated  $H_2SO_4$ . The existence of digitoxose was established by the appearance of a reddish brown coloured layer that became blue green after standing.

**Legal test:**

A suitable amount of plant extract was extracted, along with an equal proportion of water and a lead acetate solution, which was shaken and filtered. Filtrate was extracted with an equivalent amount of chloroform, which was then evaporated to dryness. To make alkaline, the residue was dissolved in 2 ml pyridine and 2 ml sodium nitropruside was added, followed by the addition of NaOH solution. The presence of glycosides or aglycone moiety was indicated by the pink colour.

**Baljeet test:**

The plant extract was treated with a sodium picrate solution. The presence of aglycones or glycosides is evidenced by a yellow to orange colour.

**Saponins can be detected using the following test:****Haemolysis test:**

A drop of blood was combined with a few drops of plant extract on a slide. The existence of ruptured RBCs was shown by their appearance.

**Foam test:**

10-20 ml of water was added to the plant extract and shaking was done for few minutes, Appearance of froth which persisted for 60-120 s, confirmed the presence of saponins.

**Test for flavonoids:****Ammonia test:**

After dipping a piece of filter paper in the plant extract, it was exposed to ammonia vapour. The presence of flavonoids was shown by the appearance of a yellow spot on filter paper.

Magnesium turning and dil. HCl were added to the plant extract in the Shinoda test (a). The presence of flavonoids was shown by the appearance of a red colour.

(b) Plant extract was treated with Zn turning and diluted HCl. The presence of dihydro flavonoids was detected by the deep red to magenta colour.

**Test using vanillin and hydrochloric acid:**

Hydrochloric acid of vanillin was added to the plant extract. The presence of flavonoids was shown by the pink colour.

**Check for phenolic compounds [4]:**

5 percent ferric chloride solution: 5 percent  $\text{FeCl}_3$  was added to a suitable amount of plant extract. The presence of deep blue-black colour was proven by its appearance.

**Lead acetate solution:** Lead acetate solution was added to plant extract and appearance of white colored ppt., confirmed the presence of phenolic compounds.

**Gelatin solution:** Gelatin solution was added to the plant extract. Appearance of white ppt., revealed the presence of phenolic compounds.

**Potassium dichromate:** Appearance of red ppt., revealed the presence of phenolic compounds.

**Dilute iodine solution:** Appearance of transient red colour, revealed the presence of phenolic compounds.

**Dilute nitric acid:** Appearance of reddish to yellow colour, revealed the presence of phenolic compounds.

**Acetic acid solution:** Appearance of red colour solution, revealed the presence of phenolic compounds.

**Dil. potassium permanganate solution:** Appearance of de coloration, revealed the presence of phenolic compounds.

**Test for fixed oils [5]:**

**Spot test:**

Small quantity of plant extract was taken and pressed between two filter papers. Oil stains on the paper, showed the presence of fixed oil.

**Saponification test:**

Few drops of 0.5 N alc. KOH and phenolphthalein were added to the plant extract. Mixture was heated on a water bath for 1-2 h. Soap formation or partial neutralisation takes place that confirmed the presence of fixed oils.

**Preparation of Phospholipids complex**

The antisolvent precipitation technique, rotary evaporation technique, and solvent evaporation technique were used to produce phospholipid complexes of plant extract in 1:1 molar ratio trial batches [14-15].

**Technique of antisolvent precipitation**

Methanol was used to dissolve the specific quantities of plant extract and soya lecithin, whereas DCM was used to dissolve the cholesterol. To achieve a concentrate, the mixture was refluxed for 2 hours at a temperature of not more than 60°C. The precipitate was filtered, collected, and kept in vacuum desiccators overnight after an anti-solvent, Hexane, was applied purposely with stirring. The powdered compound was stored in an amber glass bottle in the refrigerator.

***In-vivo* anti diabetic activity**

The anti-diabetic activity in vivo was assessed using a technique previously described in the literature [13]. The animals were kept in conventional polypropylene cages at room temperature of 30 2°C and 60–65 percent relative humidity and fed a regular food and water ad libitum under usual laboratory conditions.

**Oral glucose tolerance test (OGTT)**

Glucose tolerance is a test that determines how effectively our body's cells are prepared to

take glucose, which is our major source of energy. The oral glucose resilience test (OGTT) is used to diagnose metabolic disorders, track the progression of prediabetes to Type 2 diabetes, and assess the metabolic effects of cardiovascular and metabolic medications.

60 minutes before the oral glucose load (2.0 g/kg), different dosages of plant extracts will be given. The animals were divided into six groups of six animals each at random.

**Group I:** Control received Glucose (2g/kg)

**Group II:** received Glucose + Hydroalcoholic extract (200mg/kg)

**Group III:** received Glucose + Hydroalcoholic extract (400mg/kg)

**Group IV:** received Glucose + Phospholipids complex of Hydroalcoholic extract (100mg/kg)

**Group V:** received Glucose + Phospholipids complex of Hydroalcoholic extract (200mg/kg)

**Group VI:** received Glucose + Glibenclamide (10 mg/kg)

Blood samples will be taken from each group right before glucose injection (0 minutes), as well as 30, 60, and 120 minutes afterwards. Glucometer will be used to estimate blood glucose levels.

#### **Diabetic model produced by streptozotocin and nicotinamide**

2-Deoxy-2-[[[(methylnitrosoamino) - carbonyl] amino] streptozotocin is a streptozotocin with the chemical name 2-Deoxy-2-[[[(methylnitrosoamino) - carbonyl] amino] streptozotocin. -D-glucopyranose is a naturally occurring molecule that is highly harmful to the pancreas' insulin-producing cells [12]. It was first discovered as an antibiotic in the late 1950s [14].

#### **Experimental Protocol**

Diabetic animals were randomly assigned into following groups of six animals each.

**Group I:** Diabetic control received vehicle (Normal saline)

**Group II:** Diabetic animals received Hydroalcoholic extract (200mg/kg)

**Group III:** Diabetic animals received Hydroalcoholic extract (400mg/kg)

**Group IV:** Diabetic animals received Phospholipids complex of Hydroalcoholic extract (100mg/kg)

**Group V:** Diabetic animals received Phospholipids complex of Hydroalcoholic extract (200mg/kg)

**Group VI:** Diabetic animals received Glibenclamide (10 mg/kg)

#### **Treatment**

Experiments were carried out on rats that had been starved for the previous 24 hours (deprived of food for at least 12 h but allowed free access to water). For 21 days, the medication solution and vehicle were given orally once a day. In fasting animals, the impact of vehicle, extract, and standard medication on blood glucose and body weight was measured at 0, 7, 14, and 21 days following oral treatment. At the 21<sup>st</sup> day, blood samples were taken from all of the animals through retro-orbital plexus and heart puncture under moderate anaesthesia, and biochemical parameters were calculated using a diagnostic kit (ERBA Diagnostic Mannheim, Germany) in an auto-analyzer.

## RESULT & DISCUSSION

### Qualitative phytochemical screening

Table 1, shows the results of qualitative phytochemical screening.

**Table 1: Results of qualitative phytochemical screening of *Ougeinia oojeinensis***

S.NO.	Class of compound		HECE
1.	Carbohydrates	Reducing sugars	+ve
		Monosaccharides	+ve
		Pentose sugar	+ve
		Hexose sugar	+ve
		Molish test	+ve
2.	Glycosides	Keller killiani	-ve
		Legal test	-ve
3.	Proteins	Biuret test	+ve
		Million's test	+ve
4.	Steroids	Salkowski test	-ve
		Liebermann-Burchard test	-ve
5.	Tannin	Extract + Iodine solution	+ve
		Extract + Acetic acid	
		Extract + 5% ferric chloride	
		Lead acetate test	+ve

			+ve +ve
6.	Flavonoids	Extract + Lead acetate Shinoda test Extract + NaOH	+ve +ve +ve
7.	Amino acids	Ninhydrin test	+ve
8.	Alkaloids	Dragendorff 's test Mayer 's test Hager 's test Wagner 's test	+ve +ve +ve +ve
9.	Saponins	Foam test	+ve

### ***In vivo* anti diabetic activity**

#### **Oral glucose tolerance test (OGTT)**

##### **OGTT of optimized formulation of *Ougeinia oojeinensis***

Blood glucose levels nearly doubled from its initial level of control within 30 minutes of commencing the glucose tolerance test. This hyperglycemia was sustained for 60 minutes, after which it began to decline (Table 2). At dosages of 200 and 400 mg/kg, *Ougeinia oojeinensis* considerably reduced the rise in blood glucose levels after 60 minutes of glucose delivery. The phospholipid compound of *Ougeinia oojeinensis* reduced blood glucose levels from rising after 60 minutes. Glibenclamide also prevented blood glucose levels from rising after 30 minutes.

**Table 2: Oral glucose tolerance test of optimized formulation of *Ougeinia oojeinensis***

Groups/ Treatment	Time (min)				
	0	30	60	120	180
(I) Diabetic control (Glucose)	70.25 ± 4.43	168.51 ± 5.43	182.35 ± 2.86*	179.32 ± 4.85*	168.41 ± 1.56*
(II) Glucose + HEOO 200mg/kg	76.32 ± 4.47	188.47 ± 4.52	192.32 ± 2.74*	152.32 ± 4.72*	132.33 ± 4.34*
(III) Glucose+HEOO 400mg/kg	98.47 ± 3.45	192.43 ± 4.21	198.5 ± 3.54*	141.44 ± 5.85*	119.53 ± 5.23*
(IV) Glucose+Phospholipids of HEOO 100mg/kg	72.5 ± 3.21	171.54 ± 4.72	180.50 ± 3.71*	143.5 ± 2.52*	122.25 ± 3.83*
(V) Glucose+Phospholipids of HEOO 200mg/kg	90.75 ± 3.57	158.54 ± 4.74	160.7 ± 4.32*	132.5 ± 4.72*	113 ± 2.81*
(VI) Glucose+ Glibenclamide	78.45 ± 4.37	125.54 ± 2.85	118.72 ± 3.56*	97.35 ± 2.52*	89.35 ± 2.85*

All values represent means ± S.D of the mean (n=6) , \*p<0.05 vs diabetic control group

UNDER PEE

## **Streptozotocin-Nicotinamide Induced Diabetic Model**

### **STZ model for *Ougeinia oojeinensis***

#### **Effect of *Ougeinia oojeinensis*, phospholipids complex of *Ougeinia oojeinensis* and glibenclamide on serum glucose level in Streptozocin-nicotinamide induced diabetic rats**

The effect of 21 days treatment of HEOO (200mg/kg & 400mg/kg), phospholipid complex of HEOO (100mg/kg & 200mg/kg) and glibenclamide (10 mg/kg) on serum glucose level in streptozotocin- nicotinamide (STZ+NIC) induced diabetic rats is depicted in Table 3

The dose was introduced to the animals once a daily for 21 days. After 21 days blood samples were collected by retro-orbital plexus under mild anesthesia. Diabetic control group with no drug treatment showed no significant difference in the fasting serum glucose level after 21 days treatment as compared to the initial day treatment. However treated diabetic groups showed gradual and consistent fall in serum glucose level.

UNDER PEER REVIEW

**Table 3: The effect of 21 days treatment of HEOO (200mg/kg & 400mg/kg), phospholipids of HEOO (100mg/kg & 200mg/kg) and glibenclamide (10 mg/kg) on serum glucose level in streptozotocin-nicotinamide (STZ+NIC) induced diabetic rats**

<b>Groups</b>	<b>0 day</b>	<b>7 day</b>	<b>14 day</b>	<b>21 day</b>
<b>(I) Diabetic control (Glucose)</b>	276.20 ± 5.80	274.40 ± 7.30	274.40 ± 8.75	275.00 ± 11.49
<b>(II) Glucose + HEOO 200mg/kg</b>	258.20 ± 6.62 <sup>*</sup>	247.60 ± 8.18 <sup>*</sup>	229.00 ± 7.84 <sup>*</sup>	207.40 ± 2.97 <sup>*</sup>
<b>(III) Glucose + HEOO 400mg/kg</b>	240.40 ± 4.23 <sup>**</sup>	225.08 ± 2.36 <sup>**</sup>	201.08 ± 1.95 <sup>**</sup>	199.04 ± 1.32 <sup>**</sup>
<b>(IV) Glucose + Phospholipid of HEOO 100mg/kg</b>	260.35 ± 3.42 <sup>*</sup>	221.96 ± 4.32 <sup>*</sup>	199.06 ± 2.52 <sup>*</sup>	182.56 ± 3.46 <sup>*</sup>
<b>(V) Glucose + Phospholipid of HEOO 200mg/kg</b>	246.87 ± 2.13 <sup>*</sup>	218.57 ± 3.45 <sup>*</sup>	189.34 ± 5.21 <sup>*</sup>	162.86 ± 3.40 <sup>*</sup>
<b>(VI) Glucose + Glibenclamide</b>	214.40 ± 1.82 <sup>*</sup>	197.06 ± 0.60 <sup>*</sup>	178.35 ± 2.95 <sup>*</sup>	151.70 ± 4.30 <sup>*</sup>

All values represent means ± S.D of the mean (n=6) , <sup>\*</sup> p<0.05; <sup>\*\*</sup> p <0.01vs diabetic control group

### **Effect of HEOO, phospholipids of HEOO and glibenclamide on body weight in streptozotocin-nicotinamide induced diabetic rats**

In Streptozotocin nicotinamide induced diabetic rats, the effects of HEOO, phospholipids complex of HEOO, and glibenclamide on body weight are shown in Table 3.

When comparing the initial day therapy to the 21-day treatment, the diabetic control group with no medication treatment exhibited a significant change in body weight. The loss of body weight in untreated diabetic rats is related to fat catabolism and protein wastage, as well as a reduction in glucose availability for energy usage. The diabetic rat, on the other hand, showed a substantial ( $p < 0.05$ ) fall in body weight. The diabetic rat treated with glibenclamide (10 mg/kg) gained weight gradually and consistently.

### **Effect of HEOO, phospholipid complex of HEOO and glibenclamide on serum cholesterol, triglycerides, HDL-C, VLDL-C and LDL-C level in streptozotocin-nicotinamide induced diabetic rats**

At doses of 100 mg/kg and 200 mg/kg, treated diabetic rats demonstrated substantial reductions in serum cholesterol, triglycerides, VLDL-C, LDL-C, and rise in HDL-C levels after 21 days of therapy, as shown in Table 4. In comparison to the control group, the conventional medication glibenclamide (10mg/kg) demonstrated a decrease in serum cholesterol, triglycerides, VLDL-C, LDL-C, and an increase in HDL-C level.

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**Table 4: Effect of HEOO, phospholipid of HEOO and glibenclamide on serum cholesterol, triglycerides, HDL-C, VLDL-C and LDL-C level in streptozotocin- nicotinamide induced diabetic rats**

<b>Groups</b>	<b>Total cholesterol (mg/dl)</b>	<b>Triglycerides (mg/dl)</b>	<b>HDL-C (mg/dl)</b>	<b>LDL-C (mg/dl)</b>	<b>VLDL-C (mg/dl)</b>
<b>(I) Diabetic control (Glucose)</b>	257 ± 1.2	298 ± 2.43	42.1 ± 0.27	156.2 ± 1.12	58.7 ± 1.16
<b>(II) Glucose + HEOO 200mg/kg</b>	225.3 ± 1.41*	244 ± 1.25*	45 ± 0.13*	130.4 ± 0.52*	52 ± 2.17*
<b>(III) Glucose + HEOO 400mg/kg</b>	214 ± 1.06*	231 ± 1.76*	46 ± 1.82*	115.2 ± 0.35*	50.2 ± 1.64*
<b>(IV) Glucose + Phospholipid of HEOO 100mg/kg</b>	207 ± 2.3*	211 ± 2.84*	46.2 ± 1.04*	108.8 ± 0.26*	47.1 ± 1.32*
<b>(V) Glucose + Phospholipid of HEOO 200mg/kg</b>	191 ± 2.49*	202 ± 3.16*	47.5 ± 1.74*	102.3 ± 0.21*	44.6 ± 1.85*
<b>(VI) Glucose + Glibenclamide</b>	189 ± 1.53*	194 ± 0.31*	48 ± 2.52*	101.2 ± 0.18*	43.8 ± 1.39*

All values represent means ± S.D of the mean (n=6) , \*p<0.05 vs diabetic control group

## **CONCLUSION**

The current study found that *Ougeinia oojeinensis* extract may be beneficial in the treatment of type 1 and type 2 diabetes with aberrant lipid profiles. These plants' anti-diabetic properties might be attributable to bioactive triterpenoids, steroids, and flavonoids. In a nutshell, phospholipids complex-based formulations might be a helpful technique for improving therapeutic effectiveness, dose reduction, and dosing regimen modification. To make a claim for their antidiabetic effectiveness, more research has to be done in human volunteers.

## **Ethical Approval**

The experimental procedure was authorised by the institutional Animal Ethical Committee, and the study was carried out and completed with formal approval from the in-house ethical committee.

## **COMPETING INTERESTS DISCLAIMER:**

**Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.**

## **References**

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1. Devi VK, Jain N, Valli KS. Importance of novel drug delivery systems in herbal medicines. *Pharmacogn Rev* 2010;4(7):27-31.
2. Ansari SH, Islam F, Sameem M. Influence of nanotechnology on herbal drugs: A review. *J Adv Pharm Technol Res* 2012;3(3):142-6.
3. Shaikh MS, Derle ND, Bhamber R. Permeability enhancement techniques for poorly permeable drugs: A review. *J Appl Pharm Sci* 2012;02(06):34-9.
4. Kesarwani K, Gupta R, Mukerjee A. Bioavailability enhancers of herbal origin: An overview. *Asian Pac J Trop Biomed* 2013;3(4):253-66.

5. Chaturvedi M, Kumar M, Sinhal A, Saifi A. Recent development in novel drug delivery systems of herbal drugs. *Int J Green Pharm* 2011;5(2):87-94.
6. Jain S, Jain V, Mahajan SC. Lipid based vesicular drug delivery systems. *Adv Pharm* 2014;2014:1-14.
7. Kidd PM. Bioavailability and activity of phytosome complexes from botanical polyphenols: the silymarin, curcumin, green tea, and grape seed extracts. *Altern Med Rev* 2009;14(3):226-46.
8. Amin T, Bhat SV. A review on phytosome technology as a novel approach to improve the bioavailability of nutraceuticals. *Int J Adv Res Technol* 2012;1(3):1-5.
9. Semalty A, Semalty M, Rawat BS, Singh D, Rawat MSM. Pharmacosomes: the lipid-based novel drug delivery system. *Expert Opin Drug Deliv* 2009;6(6):599-612.
10. Fresta M, Cilurzo F, Cosco D, Paolino D. Innovative Drug Delivery Systems for the Administration of Natural Compounds. *Curr Bioact Compd* 2007;3(4):262-77.
11. Bombardelli E, Cristoni A, Morazzoni P. Phytosomes in functional cosmetics. *Fitoterapia* 1994;65(5):387-401.
12. Sarika D, Khar RK, Chakraborty GS, Saurabh M. Phytosomes: A Brief overview. *J Pharm Res* 2016;15(2):56-62.
13. Afanaseva YG, Fakhretdinova ER, Spirikhin LV, Nasibullin RS. Mechanism of interaction of certain flavonoids with phosphatidylcholine of cellular membranes. *Pharm Chem J* 2007;41(7):354-6.
14. Van Meer G, de Kroon AI. Lipid map of the mammalian cell. *J Cell Sci* 2011;124(Pt 1):5-8.
15. Bruce A, Alexander J, Julian L, Martin R, Keith R, Peter W. Molecular Biology of the Cell. 4th ed. New York; Garland Sciences: 2002.