

Hypolipidemic and Antioxidant Effects of Methanolic *Cucumis sativus* Fruit Extract in Poloxamer 407-Induced Hyperlipidemic Rats

**Kenneth E. Omuekwu^{1*}, Akande Titilayo², Inalegwu Bawa³, Ogor A. Ogor⁴,
Pedro Akharenegebe⁵ & Abdullahi Iliyasu Hussaini¹**

¹Department of Biochemistry, Federal University of Lafia, Nasarawa State, Nigeria

²Department of Biochemistry, Federal University of Agriculture Makurdi, Benue State, Nigeria

³Department of Biochemistry, Federal University of Health Sciences, Otukpo, Benue State, Nigeria

⁴Department of Biochemistry, Benue State University, Makurdi, Nigeria

⁵Department of Science Laboratory Technology, Federal University of Lafia, Nasarawa State, Nigeria

Abstract

Cardiovascular disease is caused by several risk factors, including hyperlipidaemia and oxidative stress. Cardiovascular disease is a significant contributor to the morbidity and mortality rates across the world. No robust scientific evidence exists that proves the hypolipidemic efficacy of *Cucumis sativus* L. (cucumber), although it has been shown that it is capable of providing some antioxidant and pharmacological benefits. Hence, the researchers evaluated the hypolipidemic effect and antioxidant activity of methanolic cucumber fruit extract in Poloxamer 407 (P-407)-induced hyperlipidemic rats. A normal control, a hyperlipidemic control (P-407), two methanolic cucumber extracts at 250, 500 mg/kg, and simvastatin at 5 mg/kg were given to adult Wistar rats (180–220 g) (5 rats per treatment). Hyperlipidemia was induced in adult Wistar rats (hyperlipidemic control) by P-407, and the rats received their treatment samples orally for twenty-eight days. The lipid profile (Total cholesterol (TC), Triglycerides (TG), high-density lipid cholesterol (HDL-C), very low-density lipid cholesterol (VLDL-C) and low-density lipid cholesterol (LDL-C)) was evaluated along with superoxide dismutase (SOD), reduced glutathione (GSH) and catalase (CAT), using suitable biochemical methods. P-407 led to a remarkable hyperlipidemia and impaired antioxidant defense compared with normal Wistar rats. Hyperlipidemia and antioxidant depletion were dose-dependent controlled by cucumber extracts. Extracts at 500 mg/kg reduced the levels of TC by 33.37 %, TG by 28.37 % and LDL-C by 52.36 %, while an increase of 18.19 % in HDL-C was seen compared to the other hyperlipidemic models.

Keywords: *Cucumis sativus*, Hyperlipidemia, Oxidative stress, Poloxamer 407, Simvastatin

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Correspondences

K. E. Omuekwu ✉

omuekwuemeka@gmail.com

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Introduction

Hyperlipidemia represents increased circulating levels of total cholesterol (TC), triglycerides (TG) and low-density lipoprotein cholesterol (LDL-C), and lowered HDL-C. It constitutes a main modifiable risk factor for cardiovascular disease (CVD) [1]. Dyslipidemia plays key roles in atherogenesis, coronary artery disease, stroke and other cardiovascular pathologies [2]. Aside from lipid metabolism abnormalities, oxidative stress contributes not only to CVD initiation but also to progression, promoting lipid peroxidation, endothelial dysfunction, vascular inflammation and formation of foam cells [3, 4]. Thus, therapies combining lipid metabolism improvement with oxidative stress attenuation currently catch much attention to reduce cardiovascular risks. Statins, fibrates, cholesterol absorption inhibitors, bile acid sequestrants and PCSK9 inhibitors, which have emerged only in the last few years, comprise the main pharmacological treatments for clinical hyperlipidemia. While effective in lowering serum lipids and cardiovascular risk, long-term use of

these agents may entail adverse effects such as myopathy, hepatotoxicity, gastrointestinal disturbances and poor compliance [5].

Furthermore, high costs limit access to certain lipid-lowering drugs, especially in low- to middle-income countries. This drives the search for medicinal plants as promising alternatives, offering affordability, availability, and diverse bioactive constituents [6, 7]. *Cucumis sativus* L. (cucumber), a Cucurbitaceae member, enjoys widespread cultivation and consumption as both a vegetable and a functional food [8]. Its fruit is rich in phytochemicals such as flavonoids, phenols, phytosterols, tannins, saponins and cucurbitacins, correlating with antioxidant, anti-inflammatory, hepatoprotective and cardioprotective activities [8, 9]. Despite evidence of *C. sativus*' antioxidant and general pharmacological properties, few studies have comprehensively assessed its hypolipidemic potential in experimentally validated models of non-dietary hyperlipidemia. Moreover, dose-dependent impacts of methanolic whole-fruit extracts



on serum lipid profile and antioxidant defense systems remain insufficiently explored. Among hyperlipidemia models, Poloxamer 407 (P-407)-induced hyperlipidemia is a dependable choice. This non-ionic surfactant causes severe hyperlipidemia. It inhibits lipoprotein lipase activity, hindering triglyceride-rich lipoprotein clearance, while boosting hepatic cholesterol biosynthesis by increasing HMG-CoA reductase activity [10, 11].

Unlike diet-induced models, which demand long feeding periods and are susceptible to dietary variation, the P-407 model ensures rapid and predictable lipid profile changes. This renders it suitable for testing hypolipidemic candidates and associated antioxidant effects [10, 11]. Though evidence supports *C. sativus*' pharmacology, data on its efficacy against severe P-407-induced hyperlipidemia and oxidative stress are scarce. Its therapeutic effect relative to statins like simvastatin has seen limited comparison. Addressing these gaps is crucial for establishing the scientific basis for the potential therapeutic role of *C. sativus* in dyslipidemia. In contrast to other studies, this one examined the dose-dependent hypolipidemic and antioxidant effects of methanolic *Cucumis sativus* fruit extract in P-407-induced hyperlipidemic rats. It further gauged efficacy against simvastatin, through changes in serum lipid profile parameters and antioxidant biomarkers, aiming to provide further proof. These findings will underpin future mechanism-based elucidation, phytochemical characterization and clinical investigations, while providing substantial therapeutic backing for medicinal plant research.

Materials and Methods

Plant material collection, authentication and extraction

Fresh mature cucumber fruits were collected from a local farmers' market in Lafia, Nasarawa State. They were then subjected to botanical identification and authentication at the Federal University of Lafia, Nigeria, where a voucher specimen (Voucher No: FUL/PSB/H. Lab/0092) was deposited into the departmental herbarium for future reference [12]. Afterwards, the cucumber fruits were washed thoroughly with distilled water to remove extraneous substances, dirt and dust. The fruits were then cut into slices and air-dried in the shade at room temperature ($25 \pm 2^\circ\text{C}$) for approximately 14 days, or until a constant weight was attained. At the end of the drying period, they were chopped using a grinder to a coarse powder and then ground further in a laboratory blender. The resulting powder was subjected to extraction using a 10:1 ratio of 99.8 % methanol as the solvent and allowed to macerate for 72 h with intermittent shaking [13]. The extract was filtered using Whatman No. 1 filter paper, and the filtrate was concentrated under reduced pressure at 40°C using a rotary evaporator. The resulting crude methanolic extract yielded 9 % w/w and was stored in an airtight amber glass container at 4°C until required for experimental use.

Experimental animals and ethical approval

Healthy adult male Wistar rats aged 8-10 weeks and weighing 180-220 g were obtained from the Animal House Facility and housed in clean polypropylene cages with sterile wood-shaving bedding in well-ventilated animal rooms under standard conditions of a 12 h light/dark cycle, a temperature of $25 \pm 2^\circ\text{C}$ and a relative humidity of 50-60 %. Standard laboratory rodent pellet food and fresh drinking water were supplied ad libitum [14]. All animals were allowed a conditioning period of 7 days in the laboratory to adapt to the laboratory conditions prior to the onset of the experiment. Treatment and handling of the animals were conducted in accordance with the Animal Research: Reporting of In Vivo Experiments (ARRIVE) guidelines [15] and internationally recognized principles for the care and use of laboratory animals, with ethical approval number FUL/DIRECT/ECC/2022/LS: 0008.

Experimental design

The acclimatized rats were then assigned to five groups of five animals each ($n=5$) [16]. For successful induction of hyperlipidemia, a single intraperitoneal injection of 500 mg/mL Poloxamer 407 dissolved in 0.9 % normal saline was administered to the rats [17-19]. Twenty-four hours after induction, oral treatment with the extract began and continued daily for 28 consecutive days [20].

The groups were treated as follows:

Group I (Normal Control): Treated orally with 2 mL/kg body weight of distilled water daily.

Group II (Hyperlipidemic Control): This group received an intraperitoneal injection of 500 mg/kg Poloxamer 407. No other treatment was provided [21].

Group III (Low Dose Test Extract): Rats in this group were administered 500 mg/kg Poloxamer 407 intraperitoneally, followed by oral administration of 250 mg/kg body weight of methanolic extract of *Cucumis sativus* fruit.

Group IV (High Dose Test Extract): Rats were made hyperlipidemic by intraperitoneal injection of 500 mg/kg Poloxamer 407 (ice-cold), followed by oral administration of 500 mg/kg body weight of methanolic fruit extract of *Cucumis sativus*.

Group V (Positive Control): The animals were also made hyperlipidemic by intraperitoneal injection of 500 mg/kg Poloxamer 407, and after successful induction, they were treated orally with 5 mg/kg body weight of simvastatin.

The sample size used in this experiment is similar to the sample size in other previously conducted studies of hyperlipidemia models [22]. Hence, it is optimal for detecting significant differences between the hyperlipidemic condition and the treated animals.

Blood collection and biochemical analysis

After completion of treatment, the animals were fasted overnight (12 h) and then anaesthetized with a light anaesthetic prior to sacrifice. Blood was collected by cardiac puncture into plain tubes, allowed to clot for 30

min, and then centrifuged at $1500 \times g$ for 10 min at 4°C . Serum was separated and stored at -20°C until biochemical analysis [13]. Serum levels of TC, TG, and HDL-C were determined using diagnostic kits purchased from Randox (Randox Laboratories Ltd., Crumlin, United Kingdom) according to the manufacturer's instructions. VLDL-C (very low-density lipoprotein cholesterol) levels were calculated using the equation: $\text{VLDL-C} = \text{TG}/5$ [18]. LDL-C (low-density lipoprotein cholesterol) levels were measured using the following Friedewald's equation: $\text{LDL-C} = \text{TC} - (\text{HDL-C} + \text{VLDL-C})$.

Serum oxidative stress marker measurement (SOD, CAT, and GSH)

The activities of superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) in serum were measured spectrophotometrically according to previously described methods [23-24].

Statistical analysis

The data were analyzed using a one-way analysis of variance (ANOVA) with SPSS software. Homoscedasticity of the data was assessed using Levene's test for homogeneity of variance. Normality was checked using the Shapiro-Wilk test. Values are

expressed as mean $\pm$ standard deviation (SD). Differences between groups were considered statistically significant at $P < 0.05$. Tukey's post hoc test (two-sided P-value) was used where appropriate for comparing means.

Results and Discussion

Effect of *Cucumis sativus* extract on oxidative stress biomarkers

Administration of P-407 to albino Wistar rats impaired antioxidant enzyme activity (Table 1). SOD, GSH and CAT levels were decreased by 50.48, 38.10 and 27.28 %, respectively, in the hyperlipidemic group relative to the control group ($P < 0.05$). *C. sativus* extract improved these levels to an appreciable extent at the low dose (250 mg/kg), and restored them most effectively at the higher dose (500 mg/kg). The SOD level increased from 34.37 ± 2.87 to 62.70 ± 5.45 $\mu\text{mol/min}$ with administration of 500 mg/kg extract, representing an increase of 82.43 % ($P < 0.05$). Likewise, GSH was elevated by 32.09 % and CAT by 19.59 %. The antioxidant levels in rats treated with *C. sativus* extract (500 mg/kg) were statistically similar to those in simvastatin-treated animals ($P > 0.05$).

Table 1: Effect of methanolic *Cucumis sativus* extract on serum antioxidant biomarkers in Poloxamer 407-induced hyperlipidemic rats

Parameter	Group I	Group II	Group III	Group IV	Group V
SOD ($\mu\text{mol/min}$)	69.41 ± 1.91^b	34.37 ± 2.87^a	55.57 ± 2.40^{ab}	62.70 ± 5.45^{ab}	69.67 ± 1.50^b
GSH ($\mu\text{g/mL}$)	24.67 ± 0.55^b	15.27 ± 0.47^a	18.40 ± 0.56^{ab}	20.17 ± 0.13^{ab}	23.47 ± 0.55^{ab}
CAT (Catalase activity)	22.18 ± 1.81^b	16.13 ± 0.73^a	17.56 ± 0.40^a	19.29 ± 0.31^{ab}	21.67 ± 1.26^b

Data are presented as mean $\pm$ SD ($n = 5$); ^a $P < 0.05$ vs normal control group; ^b $P < 0.05$ vs hyperlipidemic control group;

Group I: Normal control; Group II: Hyperlipidemic control (Poloxamer 407, 500 mg/kg); Group III: Poloxamer 407 + methanolic *Cucumis sativus* extract (250 mg/kg); Group IV: Poloxamer 407 + methanolic *Cucumis sativus* extract (500 mg/kg); Group V: Poloxamer 407 + simvastatin (5 mg/kg)

Table 2: Effect of methanolic *Cucumis sativus* extract on serum lipid profile in Poloxamer 407-induced hyperlipidemic rats

Parameter	Group I	Group II	Group III	Group IV	Group V
TG (mg/dL)	22.56 ± 0.78^b	38.52 ± 1.62^a	26.11 ± 0.94^{ab}	27.59 ± 0.62^{ab}	30.15 ± 0.94^{ab}
HDL-C (mg/dL)	85.53 ± 1.78^b	53.49 ± 2.32^a	61.65 ± 0.59^{ab}	63.22 ± 1.79^{ab}	71.92 ± 1.43^{ab}
TC (mg/dL)	149.33 ± 2.71^b	208.43 ± 14.14^a	140.14 ± 1.87^b	138.87 ± 2.00^b	143.15 ± 2.62^b
VLDL-C (mg/dL)	4.51 ± 0.72^b	7.70 ± 1.60^a	5.22 ± 0.92^b	5.52 ± 0.60^b	6.03 ± 0.93^{ab}
LDL-C (mg/dL)	59.30 ± 0.99^b	147.23 ± 1.22^a	73.25 ± 1.89^{ab}	70.14 ± 0.19^{ab}	65.20 ± 1.87^{ab}

Data are presented as mean $\pm$ SD ($n = 5$); ^a $P < 0.05$ compared with the normal control group; ^b $P < 0.05$ compared with the hyperlipidemic control group; Group I: Normal control; Group II: Hyperlipidemic control (Poloxamer 407, 500 mg/kg); Group III: Poloxamer 407 + methanolic *Cucumis sativus* extract (250 mg/kg); Group IV: Poloxamer 407 + methanolic *Cucumis sativus* extract (500 mg/kg); Group V: Poloxamer 407 + simvastatin (5 mg/kg)

Effect of *Cucumis sativus* extract on serum lipid profile

On treatment of Poloxamer 407, significant changes were seen in serum lipid levels as compared to that of the normal control (Table 2). A significant increase ($P < 0.05$) was observed in TC, TG, VLDL-C, and LDL-C, along with a notable reduction in HDL-C. Compared

to the normal control, increases were observed in the levels of TC, TG, VLDL-C, and LDL-C by 39.58, 70.74, 70.73, and 148.28 %, respectively, while a decrease in the level of HDL-C (27.46 %) was observed. On treatment with the methanolic extract of *Cucumis sativus*, these levels decreased dose-dependently. At dose of 500 mg/kg, decrement was



observed in the serum level of TC- 208.43 ± 14.14 to 138.87 ± 2.00 (33.37 %), LDL-C -147.23 ± 1.22 to 70.14 ± 0.19 (52.36 %) and also decrement in the serum level of TG- 38.52 ± 1.62 to 27.59 ± 0.62 (28.37 %) and increment in HDL-C in greater extent than untreated hyperlipidemia group ($P < 0.05$). Lipid-lowering activity of the methanolic extract of *Cucumis sativus* was comparable to that of simvastatin.

The work aimed to investigate the hypolipidemic and antioxidant effects derived from methanolic *Cucumis sativus* fruit extract in Poloxamer-407 (P-407)-induced hyperlipidemic rats. It was found that the P-407-treated group increased the concentrations of serum total cholesterol (TC), triacylglycerol (TG), low-density lipoprotein-cholesterol (LDL-C), and very low-density lipoprotein-cholesterol (VLDL-C), whilst simultaneously reducing high-density lipoprotein-cholesterol (HDL-C). This is in agreement with previous studies, which report that P-407 promotes dyslipidemia by inhibiting lipoprotein lipase activity and stimulating hepatic cholesterol biosynthesis by increasing the rate of HMG-CoA reductase. P-407 causes hyperlipidemia through the inhibition of lipoprotein lipase activity and also an increase in hepatic cholesterol biosynthesis with heightened HMG-CoA reductase activity. The P-407-treated group was also discovered to have suppressed activities of antioxidant enzymes, namely superoxide dismutase (SOD), catalase (CAT) and reduced glutathione (GSH), thereby indicating the presence of oxidative stress in these animals. The lipid-lowering actions could also be attributed to the bioactive phytochemicals found in previous research to be present at high concentrations in *Cucumis sativus*, including phytosterols, flavonoids, phenolic compounds, tannins, saponins and cucurbitacins [8], which could influence lipid metabolism through the inhibition of cholesterol uptake within the intestine, the enhancement of cholesterol clearance and antioxidant activity. While these compounds were neither identified nor quantified in this work, future work will address both identification and quantification. Although these compounds were neither identified nor measured in the present study, they could influence cholesterol metabolism, inhibit intestinal cholesterol absorption, enhance cholesterol clearance and provide antioxidant activity.

Consequently, these previously reported mechanisms may partly explain the improvements in serum lipid parameters observed following extract administration. The antioxidant activity observed may reflect the presence of phenolic and flavonoid compounds previously reported in *C. sativus*. Although these constituents were not characterized in the present study, earlier reports suggest that they possess free-radical scavenging and antioxidant properties that may have contributed to the restoration of endogenous antioxidant enzymes. The dose-dependent improvements observed throughout this study further support the biological activity of the extract. Rats receiving 500 mg/kg methanolic *C. sativus* extract consistently demonstrated greater improvements in both lipid profile and

antioxidant biomarkers than those treated with 250 mg/kg, suggesting that the therapeutic response increases with dose within the range investigated. Although the higher extract dose produced effects approaching those observed in the simvastatin-treated group, the present findings should not be interpreted as evidence of pharmacological equivalence. Rather, they suggest that the extract possesses considerable lipid-lowering and antioxidant potential worthy of further investigation. Unlike simvastatin, which primarily acts through competitive inhibition of HMG-CoA reductase, *C. sativus* likely acts through multiple complementary mechanisms involving modulation of cholesterol absorption, enhancement of cholesterol clearance, antioxidant activity, and possibly anti-inflammatory effects [22, 23]. Such multi-target actions are characteristic of many medicinal plants and may provide therapeutic advantages in cases such as complex metabolic disorders.

The findings of this study contribute further experimental evidence to support the use of *C. sativus* as a powerful natural therapeutic agent in the management of hyperlipidemia and oxidative stress. By simultaneously improving lipid metabolism and restoring antioxidant defense, the active extract of *C. sativus* may also reduce the pathological processes associated with dyslipidemia and cardiovascular disease. Further research, however, needs to be conducted on the isolation and characterization of the potentially responsible chemicals, as well as the elucidation of specific anti-hyperlipidemic and antioxidant mechanisms, before clinical testing can begin to establish long-term safety and efficacy.

Study limitations

The data obtained from this research are fascinating and certainly suggest strong anti-hyperlipidemic and antioxidant activities of methanolic *C. sativus* fruit extract; however, it must be acknowledged that limitations remain. Given the small sample size and the 28-day treatment duration, these findings cannot necessarily be generalized to a larger population, but they provide a solid foundation for doing so. Regarding histopathology, no tests could be performed on heart and muscle cells to assess the extent to which the extract affected them. Several biomarkers were not investigated; the extent of hepatic function was also not examined, as ALT and AST were left out completely. Furthermore, no studies were conducted on the extract's actions within lipid metabolism or on inflammation, including LDL receptors, TNF- α , or IL-6. This experiment also did not attempt to identify the active compounds through techniques such as liquid chromatography-mass spectrometry. Further experimentation is needed to fully uncover the effects of *C. sativus*.

Conclusion

Methanolic fruit extract of *Cucumis sativus* has shown significant hypolipidemic and antioxidant activities in hyperlipidemic rats. It exerted a positive modulatory

role against hyperlipidemia induced by Poloxamer 407 and improved serum lipid levels. Its treatment decreased serum total cholesterol, triglycerides (TG), low-density lipoprotein-cholesterol (LDL-C), and very low-density lipoprotein-cholesterol (VLDL-C), while serum levels of high-density lipoprotein cholesterol (HDL-C) increased. The antioxidant activity of the plant was demonstrated by increased activity levels of some endogenous enzymes, namely catalase (CAT), superoxide dismutase (SOD), and reduced glutathione (GSH). The 500 mg/kg dose showed greater effects, and the results were closer to those achieved with simvastatin. The above findings suggest that phytochemical components present in the fruit extract of *Cucumis sativus* possess therapeutic potential that can be utilized to combat hyperlipidemia, and it may serve as an alternative therapy in treating patients with the disease. However, more studies are required to investigate the role that those different molecules/chemicals present in the extract may play in therapeutic activity and the underlying molecular mechanisms. Further investigations into their mechanisms of action are required alongside preclinical studies evaluating their long-term safety. Once this stage is completed, clinical trials in patients could be conducted to determine the optimal dosages and other clinical outcomes.

Conflict of interest: The authors declare that there is no conflict of interest in this study.

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