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Homeostatic Effect of *Phoenix Dactylifera* on Alloxan-Induced Diabetes and Aluminium-Induced Neurotoxicity in Male Wistar Rats

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ABSTRACT

Introduction: This study assessed homeostatic effect of *Phoenix dactylifera* on alloxan-induced diabetes and aluminium-induced neurotoxicity in male wistar rats divided into 5 groups of 5 rats each.
Methods: Group A; control, Group B; diabetic group, Group C; $AlCl_3$, Group D Alloxan+ *P. dactylifera*, Group E; $AlCl_3$ + *P. dactylifera* (0.5ml each). Diabetes was experimentally induced in groups B-D. The animals were left to acclimatize for 2 weeks and treatments were dissolved in distilled water and given orally for 2 weeks (4weeks). Blood sample was collected via ocular puncture for analysis of fasting blood glucose level. At the end of 4 weeks, the animals were sacrificed and blood samples taken for estimation of oxidative stress markers. Blood and brain samples were collected for neuromuscular function markers.
Results: The results were statistically significant at 95% confidence interval. The results obtained when compared to control from showed group D and E exhibited decrease levels of lipid peroxidation (MDA), increased levels of antioxidant enzymes (SOD, CAT, GPx and GSH). There was also decrease BGL2, indicating positive glycemic control. When compared to the diabetic group, the treatment group showed a significant reduction in MDA levels and significant increase in the levels of GSH, CAT, SOD, and GPX. It also showed significant decrease in CKMM levels (less muscle activity and damage), and increase in AChE indicating nerve activity and stress was observed in control group too. Significant reduction in FBGL2 were shown. There was no significant difference in FBG1 levels.
Conclusion: *P. dactylifera* aqueous extract exhibits potential antidiabetic and antioxidant effect in Alloxan-induced diabetic and aluminium-induced toxicity in male wistar rats.

Key words: *Phoenix dactylifera*; Neurologic; Diabetes; Phytochemical; Therapy

INTRODUCTION

Homeostasis, a concept first introduced by Walter Cannon in the 1930s, refers to the

ability of living organisms to maintain internal stability despite external changes [1]. It is a fundamental principle that governs the physiological processes necessary for life. *Phoenix dactylifera* has been cultivated for thousands of years and have played a significant role in human nutrition and traditional medicine across various cultures [2]. Diabetes mellitus is a metabolic disorder characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. It is a metabolic disorder affecting millions of people worldwide. The *P. dactylifera* palm is often referred to as the "*Tree of life*" due to its ability to thrive in arid climates and provide sustenance and medicinal benefits to communities in these regions. Dates are rich in essential nutrients, including carbohydrates, vitamins (such as vitamin A, vitamin B6, and vitamin K), minerals (such as potassium, magnesium, and copper), and dietary fibres [3]. Additionally, *P. dactylifera* contain various bioactive compounds, including polyphenols, flavonoids, carotenoids, and triterpenes, which contribute to their antioxidant, anti-inflammatory, and potential therapeutic properties [1][2]. Alloxan, a compound derived from uric acid, is commonly used in experimental models to induce diabetes due to its selective cytotoxicity to pancreatic beta cells [1][4]. Alloxan-induced diabetes in rodents closely resembles certain aspects of human diabetes, making it a valuable tool for studying the pathophysiology of the disease and evaluating potential therapeutic interventions [4]. Understanding the mechanisms underlying alloxan-induced diabetes and exploring natural remedies, such as dates, for managing this condition is of significant scientific interest and clinical relevance. Aluminium is a ubiquitous metal found in various environmental sources, including food, water, and consumer products. Despite its abundance, aluminium exposure has raised concerns due to its potential neurotoxic effects. Accumulating evidence suggests a possible link between aluminium exposure and the development of neurodegenerative diseases, particularly Alzheimer's disease [5][6]. The mechanisms underlying aluminium neurotoxicity are complex and multifaceted, involving oxidative stress, inflammation, and disruptions in neuronal function [7]. Given the increasing prevalence of neurodegenerative diseases and the potential role of environmental factors, such as aluminium exposure, understanding the mechanisms of aluminium neurotoxicity and exploring interventions to mitigate its effects is a critical area of research. This study evaluates the potential homeostatic effects of *P. dactylifera* on alloxan-induced diabetes in male Wistar rats and aluminium-induced neurotoxicity, shedding light on its therapeutic utility in these conditions.

MATERIALS AND METHODS

Ethical consideration

This study was approved by the Research Ethics Committee of Madonna University Nigeria, Ref: MAU/DRC/HD/E/PHY/2024/ 002.

Animal collection

Twenty-five (25) male wistar rats (*Rattus norvegicus*) weighing 160g to 180g and aged 2 weeks were purchased and housed in the research animal facility, Department of Human Physiology, Madonna University, Elele, Rivers State, Nigeria. The animals were allowed to acclimatize for a period of 2 weeks and exposed to 12/12 hours' light/dark cycle. The animals had access to feed and water *ad libitum.*

Induction and Confirmation of Diabetes Mellitus

The induction of diabetes using alloxan is a widely used method in research to create experimental models of diabetes mellitus in animals, particularly rodents. Alloxan is a cytotoxic agent that selectively destroys

insulin-producing β-cells in the pancreas, leading to hyperglycaemia. The materials used include, Alloxan monohydrate, Syringes and needles, Glucometer and test strips, Rodents [8], Cages and standard laboratory animal care equipment. Typical dose for inducing diabetes in rodents ranges from 120 to 200 mg/kg of body weight. For example, a common dose is 150 mg/kg [8][9]. Alloxan was dissolved in 120ml of H2O to prepare a working solution. The rats were fasted overnight, The Rodents should be fasted for 12-16 hours before alloxan administration to ensure uniform absorption and effectiveness [10]. The alloxan administration was via intraperitoneal injection. Using a syringe, the calculated dose of alloxan solution was injected into the peritoneal cavity of each animal. Alloxan can also be administered via intravenous (IV) or subcutaneous (SC) routes, depending on the experimental requirements and animal model used. After injection, the animals were monitored closely for signs of distress or adverse reactions. food and water were provided ad libitum. Blood glucose levels was measured 48-72 hours post-injection using a glucometer. Animals with blood glucose levels exceeding 250 mg/dL (13.9 mmol/L) are considered diabetic [10][11]. Regular monitoring of blood glucose levels was done to ensure the maintenance of the diabetic state. Adjustments in diet and care such as provision of clean water was done to manage hyperglycemia and associated complications. To confirm the successful induction of diabetes, blood glucose levels were compared with established diagnostic criteria for diabetes in the specific species or rat strain

Procurement of *phoenix dactylifera*

The *Phoenix dactylifera* used for this study were obtained from Ogoja in Cross River state, Nigeria.

Concentration and Dosage of treatments

Concentration of *P. dactylifera* administered

40000 mg of *P. dactylifera* was dissolved in 250ml of water

16.00mg/ml of *P. dactylifera* was administered to the rats

0.5ml were administered to the rats.

Concentration of Alloxan administered

LD_{50} 0f alloxan is 150mm/kg

120g of alloxan is dissolved in 120ml of water

0.5 ml was administered to the rats.

Concentration of Aluminium chloride ($AlCl_3$)

1000 mg of aluminium was dissolved in 250ml of water, the concentration is 2mg/ml

0.5ml was administered to the rats.

Table 1: Study design

Groups	Treatment
A	Feed + Water
B	Alloxan 0.5ml
C	Aluminium 0.5ml
D	Alloxan + *P. dactylifera* 0.5ml
E	Aluminium + *P. dactylifera 0*.5ml

***N*=5**

Study duration

After 2 weeks of acclimatization, this study lasted for 4 weeks (28 days).

Administration of Extract

Phoenix dactylifera powder was dissolved in water to create the same composition of mixture, and then was administered to the rats orally using an orogastric cannula. This method of delivery ensured direct and accurate delivery of the *P. dactylifera* powder mixture to the gastrointestinal tract of the rats, allowing for optimal absorption and potential evaluation of its effects. By using the orogastric cannula, accurate control over the administration process was achieved, reducing any potential variations and ensuring consistency in the dosage delivered to each rat.

Sample collection

The animals were anaesthetized with chloroform before an incision was made in the cranial region, the skull was opened and the brain was carefully removed and Placed on Ice.

Laboratory Analysis

Brain Tissue Homogenization

All animals were anaesthetized with chloroform (0.5 ml I.p.) and perfused with normal Saline (0.9% NaCl) followed by 4% Paraformaldehyde (PFA) in phosphate buffer (PB; 0.1 M pH 7.4). The brains were removed from the skull and placed in normal saline for biochemical analysis.

Biochemical analysis

Oxidative stress markers assayed f. Oxidative stress markers used include Glutathione Peroxidase (GPx), Superoxide dismutase (SOD), Catalase (CAT), Glutathione Reductase (GR) and Acetylcholinesterase

Assay for Glutathione Peroxidase (GPx)

Using Rotruck, et al., method, the assay measures the amount of glutathione that is left over after measuring the actin of glutathione peroxidase at 412 nm. U/g is the unit of measurement. The assay mixture, which has a total volume of 2.0 ml, includes 0.5 ml of sodium phosphate buffer, 0.1 ml of sodium aside, 0.2 ml of reduced glutathione, 0.1 ml of H2O2, 0.5 ml of sample dilution at a ratio of 1:10, and 0.6 ml of d/w. After three minutes of incubation at 37°C, 0.5 milliliter of 10% TCA was added to stop the reaction. One milliliter of the supernatant was combined with 4.0 milliliters of disodium hydrogen phosphate and one milliliter of DTNB to ascertain the amount of remaining glutathione. At 412 nm, the color developed was measured.

Assay for Superoxide dismutase (SOD)

Using Mista and Fridouich technique, this reaction serves as the foundation for a straightforward assay for this dismutase because of SOD's ability to prevent the auto-oxidation of adrenaline at PH 10.2. U/g is the unit of measurement. 200ul of the diluted sample was added to 2.5ml of 0.05m carbonate buffer (pH10.2) after 0.2ml of the sample was diluted in d/w to create a 1:10 dilution. After that, 0.3 ml of freshly made 0.3 mm epinephrine was added to the mixture, and it rapidly mixed by inversion. For a duration of 30 to 2.5 minutes, measure and document the rise in absorbance at 480 nm.

Assay for Catalase (CAT)

Using Clairborne method, the sample's catalase broke down the hydrogen peroxide, which was detected at 240 nm. The quantity of protein required to convert 1µmol H_2O_2/min is equal to one unit of catalase activity. U/g is the unit of measurement.0.2ml of sample was added to phosphate buffer containing 100mm of H_2O_2, in a total of1ml. Incubated for 2 mins at 37'c. It was then Read and record change in absorbance at 240nm.

Assay for Reduced Glutathione (GSH)

Using Sedlak & Lindsay method, the majority of cellular non-protein sulfhydryl groups were, in most cases, composed of the reduced form of glutathione. Thus, the foundation of this procedure is the formation of a comparatively stable yellow color upon the addition of Ellman's reagent, 5,5 dithiobis-2-nitrobenzoic acid, to sulfhydryl compounds. At 412 nm, the chromorphoric product of Ellman's reagent reacting with the reduced GSH is detected. 1.8 ml of d/w, 3 ml of the precipitating solution, and 0.2 ml of the sample were combined. After five minutes of standing, centrifuge at 4000 rpm for ten minutes. 4 ml of 0.1 m phosphate buffer were mixed with 1 ml of the filtrate. Finally, 0.5ml of DTNB was added. To zero the spectrophotometer, read and record the absorbance at 412 nm using a prepared blank.

Assay for Acetylcholinesterase (AChE)

Using Ellman et al method, it observes the formation of the yellow ion of 5-thio-2-nitrobenzoic acid (TNB) and is based on the reaction between thiols and chromogenic 5,5'-dithiobis-2-nitrobenzoic acid (DTNB). (U/L) is the unit of measurement. The reaction mixture (3 ml) contains 50 mM phosphate buffer (pH 7.4), 0.5 mM DTNB, and 0.50 mM ATI in a quartz cuvette with a 1 cm path length. At intervals of 30 seconds, the optical density change was monitored for three minutes at 412 nm. Extinction coefficient $13.6x10^3M$ ʉ¹ cm ʉ´ was used to calculate the AChE activity.

Assay for Malondialdehyde (MDA)

Ohkawa & Ohishi method unit umol/ml, MDA, which is created when fatty acid membranes peroxide, reacts with 2-thiobabituric acid, a chromogenic reagent, in an acidic environment to generate a pink complex that can be detected at 532 nm. 0.4 ml of supernatant was separated from 1.6 ml of Tris-Kcl buffer, and 0.5 ml of 30% TCA was added. Subsequently, 0.5 milliliter of 0.75 percent TBA was introduced and cooked for one hour. This was centrifuged at 4000 rpm after being chilled in ice. After collecting the clear supernatant, the absorbance was measured at 532 nm, with d/w serving as the blank. • A 10 µL sample was added to different wells. After adding 100 µL of the reconstituted reagent, the plate was tapped to combine it. After that, it was incubated at either room temperature of 37 °C. Within 20 minutes, the glutathione present in the Substrate Solution completely activated CK. Two readings of OD340nm were taken at 20 and 40 minutes.

Assay for Protein Carbonyl (PC)

Using Levine method, 2,4 dinitrophenylhydrazine (DNPH) and protein carbonyls react to form a Schiff base, which in turn yields the equivalent Hydrazone, which is spectrophotometrically measurable. mmol/g is the unit of measurement. 200µl of the sample was put into a 2 ml plastic tube. 800µl of DNPH was added to the sample test tube. The tube was then vortexed once every 15 minutes while it was incubated in the dark at room temperature. One millilitre of 20% TCA was added to the test tubes and vortexed. After being incubated for five minutes on ice, the tube was centrifuged at 10,000 x g for ten minutes at 4°C in a microcentrifuge. The supernatant was discarded, and the pellet was suspended in a 1:1 Ethyl acetate is ethanol. blend. Using a spatula to manually suspend the pellets, they were extensively vortexed. After the final wash, the protein pellets were suspended in 500 µl of guanidine hydrochloride by vortexing. The tube was centrifuged again at 10,000 x g for 10 mins at 4 °C in a microcentrifuge to remove any remaining debris. 220 µl of the supernatant was transferred to two wells of the 96-well plate. The absorbance was measured at a wavelength of 360–385 nm using a plate reader.

Blood Function Markers

Assay for fasting blood glucose (FBG)

Assaying fasting blood glucose levels involves measuring the concentration of glucose in a blood sample taken after a period of fasting. Here is a general principle and procedure for assaying fasting blood glucose according to ADA, 2021. Fasting blood glucose measurement typically employs enzymatic methods based on the reaction of glucose with glucose oxidase or glucose dehydrogenase. These enzymes catalyze the oxidation of glucose, producing hydrogen peroxide (H_2O_2). The generated H2O2 is then quantified using a colorimetric or electrochemical detection system, which allows for the determination of glucose concentration. Patient was fasted for a specified period, avoiding food and drink except water. venous blood sample was collected using sterile needle. Sample was allowed to clot, then separated into plasma by centrifugation. Reagents were prepared as

instructed, including enzyme reagents, substrate solutions, and glucose standards. A standard curve was created using known glucose concentrations. patient's sample, glucose standards, and control samples were added to reaction cuvettes, enzymatic reagent were added and incubated. Absorbance was measured using electrochemical analyzer. A standard curve was used to convert measured signal into glucose concentrations. Fasting blood glucose concentration was reported in milligrams per deciliter (mg/dL) or millimoles per liter (mmol/L).

Assay for glycated haemoglobin (HbA1c)

The assay for glycated hemoglobin, also known as HbA1c, is an important diagnostic test for monitoring long-term blood glucose control in individuals with diabetes. Here is a general principle and procedure for assaying fasting blood glucose according to IFCC, 2012. The principle of the assay is based on the fact that glucose molecules can non-enzymatically bind to haemoglobin in the red blood cells. The binding occurs on the N-terminal valine of the beta chain of haemoglobin, forming a stable adduct known as HbA1c. The level of HbA1c in the blood is directly proportional to the average blood glucose concentration over the lifespan of the red blood cells. Venous blood sample was collected in an EDTA anticoagulant tube to prevent clotting. The blood sample was haemolysed to release haemoglobin by adding a suitable lysing agent, then centrifuged to separate cellular debris. HbA1c fraction was separated from other haemoglobin forms using techniques like HPLC, ion-exchange chromatography, or capillary electrophoresis. HbA1c was detected and quantified using immunoassays with specific antibodies. Results was expressed as a percentage of glycated haemoglobin to total haemoglobin.

Electrolyte Assay

To measure calcium, one uses a potentiometer. This technique measures the potential difference that forms between an ion selective electrode's inner and outer phase. A substance that selectively permits the passage of calcium ions makes up the electrode. By comparing the potential to that of the reference electrode, the potential is determined. The voltage difference between the two electrodes is due to the concentration of ionized calcium in the sample, as the reference electrode maintains a constant potential (Ilochi & Chuemere, 2021). mg/dl is the unit of measurement. Using a Centronics GmbH kit, the concentrations of sodium ions (Na^+) and potassium (K^+) in serum were determined using the turbidimetric determination method developed by Hillmann et al. [11] and Tietz [12]. For Na^+, the unit is mmol/L, and for K^+, it is meq/L.

STATISTICAL ANALYSIS

The data were presented as mean ± SEM, with statistical significance defined as $P<0.05$. With the use of IBM®SPSS Version 21.0, the data from this study were analyzed using Post Hoc analysis and One-Way analysis of variance (ANOVA). Using techniques from Chuemere et al. [13], the percentage change (%c) was also displayed in conventional charts and tables. Chuemere, et al. (2018) employed a suitable technique to compute the percentage change (%c) by utilizing the formula V_2-V_1/V_1 X 100 [13][14].

RESULTS

Table 2: GC-FID Phytochemical analysis of *P. dactylifera*

Component	Concentration	Unit
Caprylic	2.3013	ug/ml
Flavanol	12.1201*	Ppm
Pelargolic	0.7420	ug/ml
Undecyclic	1.1070	ug/ml
Anthraquinone	4.3102	ug/ml

Naringin	7.1437	ug/ml
Terpenoids	6.0212	ug/ml
Anthocyanin	12.7115*	ug/ml
Naringenin	1.1242	ug/ml
Resveratol	3.022	ug/ml
Sapogenin	2.0320	ug/ml
Phenol	15.6310*	Ppm
Lunamarin	8.1401	ug/ml
Flavanones	27.2422*	Ppm
Steroids	5.6077	Ppm
Kaempferol	4.0112	ug/ml
Flavone	11.2200	Ppm
Lectin	1.0320	ug/ml
Lunamarine	2.7130	ug/ml
Oxalate`	7.1217	ug/ml
Catechin	6.4311	ug/ml

Flavanones

This phytochemical has a concentration of 27.2422, The 15-carbon skeleton of flavanones is made up of two heterocyclic rings and two phenyl rings. Eriocitrin, naringin, and hesperidin are important flavanones. The most common sources of these chemicals are citrus fruits and their juices, as well as some herbs. Due to their strong antioxidant qualities, flavanones aid in the body's defense against free radicals. Unstable chemicals called free radicals can harm cells, resulting in aging and illnesses including cancer and heart disease. Flavanones' antioxidant properties aid in lowering oxidative stress and providing protection from these harmful consequences. Additionally, these substances have anti-inflammatory properties that help lessen inflammation throughout the body. Regular consumption of citrus flavanones has been shown to considerably lower the risk of cardiovascular disorders, according to a study published in the "American Journal of Clinical Nutrition" [6][7]. A research article that appeared in "Cancer Letters" revealed that flavanones such as naringin and hesperidin may prevent some cancer cell types from growing [10]. Additionally, flavanones support stronger immune system performance, better skin health, and better metabolic health. In nutritional research, their function in enhancing general health and wellbeing is generally acknowledged.

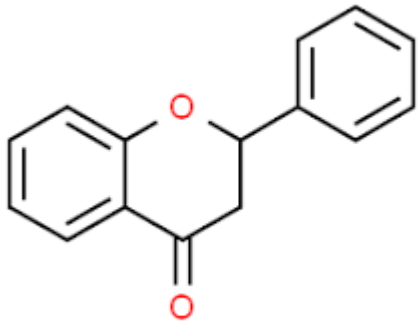

Fig. 1: The structure of flavonone

Source: http://www.chemspider.com/Chemical-Structure.9833.html

Phenol

This phytochemical has a concentration of 15.6310* Phenol, also known as carbolic acid, is an aromatic organic compound with the molecular formula [7][8]. It consists of a phenyl group bonded to a hydroxyl group (OH). Phenol is an important industrial commodity as well as a key starting material in the synthesis of many compounds. Phenol is a white crystalline solid that is volatile. It has a distinct, sweet, tar-like odor and is slightly soluble in water, forming a weakly acidic solution. The hydroxyl group attached to the

benzene ring makes phenol more acidic than alcohols but less acidic than carboxylic acids. The acidity of phenol is due to the resonance stabilization of the phenoxide ion formed after losing a proton [16]. Phenol is toxic and can cause severe burns upon contact with the skin. It is also harmful if inhaled, ingested, or absorbed through the skin. Chronic exposure to phenol can lead to systemic effects and damage to the liver and kidneys. Due to its toxicity, handling phenol requires appropriate safety measures, including the use of personal protective equipment (PPE) and proper ventilation [14].

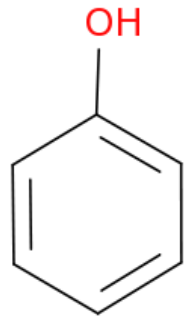

Fig. 2: The structure of phenol
Source: https://www.molinstincts.com/formula/phenol-cfml-CT1001494742.html

Anthocyanins

This phytochemical has a concentration of 12.7115*, Anthocyanins are glycosides composed of an anthocyanidin aglycone and one or more sugar moieties attached via glycosidic bonds. The aglycone part consists of a flavonoid structure with conjugated double bonds and hydroxyl groups, making it highly reactive and responsible for its color. The sugar moieties attached can vary, affecting the stability and color of the anthocyanin molecule [16][17]. Anthocyanins are found in many parts of plants, including fruits (e.g., berries, grapes, cherries), vegetables (e.g., red cabbage, eggplant), leaves (e.g., red autumn leaves), and flowers (e.g., roses, pansies). Their presence often serves as a visual signal to attract pollinators or seed dispersers, and they provide protection against environmental stressors such as UV radiation [16][17][18]. Studies suggest that regular consumption of anthocyanin-rich foods may contribute to cardiovascular health, improve cognitive function, and reduce the risk of chronic diseases such as cancer and diabetes [17][18].

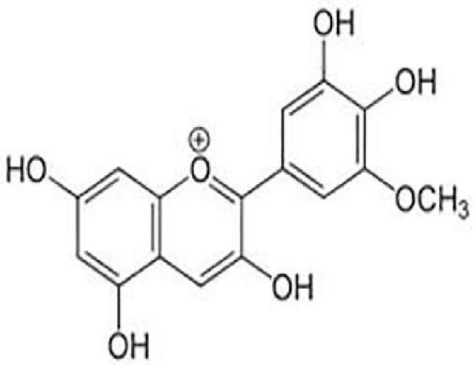

Fig. 3: The structure of Anthocyanin
Source: Molecular structure of anthocyanin. | Download Scientific Diagram (researchgate.net)

Flavanols

This phytochemical has a concentration of 12.1201, Flavanols are a class of flavonoids, which are polyphenolic compounds widely distributed in the plant kingdom. They are recognized for their significant health benefits and are found in a variety of foods, including tea, grapes, apples, and cocoa. The most well-known flavanols include catechins and epicatechins, which are often found in foods and beverages like green tea and chocolate [19]. The presence of flavanols contributes to the bitterness and astringency of these foods, and they play a role in the plant's defence mechanisms against pathogens and UV radiation [20]. Flavanols have been shown to improve endothelial function, reduce blood pressure, and enhance blood flow. Studies indicate that regular consumption of flavanol-rich foods can reduce the risk of cardiovascular diseases [21] [22]. As powerful antioxidants, flavanols help neutralize free radicals and reduce oxidative stress. This activity is beneficial in preventing chronic diseases and aging-related [23] [24]. Emerging evidence suggests that flavanols may improve cognitive function and protect against neurodegenerative diseases. They are thought to enhance brain perfusion and promote neurogenesis [25].

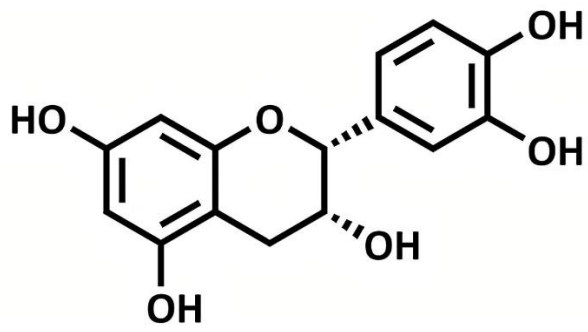

Figure 4: Structure of flavanol
Source: [About Cocoa Flavanols | Cocoa Flavanol Science]

4.1.2 Total Antioxidant Capacity of *Phoenix dactylifera*

Table 3: Total Antioxidant Capacity of *P. dactylifera* extract

Conc. of P. *dactylifera* extract	Absorbance
20mg/ml	0.270
40mg/ml	0.320
60mg/ml	0.386
80mg/ml	0.426
100mg/ml	0.581

Table 4: Total antioxidant capacity of standard

Conc. of ascorbic acid	Absorbance
20mg/ml	0.301
40mg/ml	0.373

At 20mg/ml and 40mg/ml the absorbance of *P. dactylifera* was 0.270 and 0.320 and that of Ascorbic Acid (A well-known antioxidant which was used as a standard), 0.301 and 0.373 respectively. Consequently, an increased concentration of *P. dactylifera* (100mg/ml) showed an absorbance of 0.581 indicating a positive correlation in the absorbance of *P. dactylifera* and Ascorbic acid. This indicates that *P. dactylifera* has a potential antioxidant activity to ascorbic acid.

Table 5: Mineral analysis of *P. dactylifera*

Mineral	Composition (Mg/kg)
Na	103.40
Ca	217.20
K	41.80
Mn	2.31
Mg	4.22
Cu	0.02
Zn	Nil

Mineral analysis of *P. dactylifera* shows that there are high levels of Na and Ca present in the extract

Calcium (Ca)

The growth and upkeep of healthy bones and teeth depend on the mineral calcium. Moreover, it is essential for blood coagulation, neuron transmission, muscle contraction, and cell signalling. Blood pressure and hormone secretion are two physiological processes that calcium is involved in controlling. The mineral

content of Pheonix dactylifera is measured in order to evaluate its contribution to the mineral makeup of date. Date calcium content can change based on a number of variables, including the kind of soil, the climate, and farming methods.

Sodium (Na)

Sodium is an essential mineral and electrolyte crucial for various physiological functions in the human body. It is primarily obtained from table salt (sodium chloride) and is naturally present in many foods. Na regulates water movement in and out of cells, maintaining blood volume and pressure, It also aids in generating and transmitting electrical impulses for communication between the brain and body. Na works with potassium to ensure proper muscle contraction and relaxation, and also helps maintain the body's acid-base balance. Date sodium content can change based on a number of variables, including the kind of soil, the climate, and farming methods.

4.1.4 UV-VIS (Ultraviolet visible spectrophotometry) Analysis

Table 6: UV-VIS wavelengths and absorbance of *P. dactylifera*

Wavelength (nm)	*P. dactylifera*
200	0.184
220	0.42
260	0.465
290	0.37
310	0.305
330	0.359
380	0.4
400	0.421
420	0.439
450	0.5
470	0.966
500	1.24
550	1.02
580	0.2
600	1.11
620	0.44
700	0.223
750	0.111
800	0.057
850	0.032
870	0.022
900	0.014

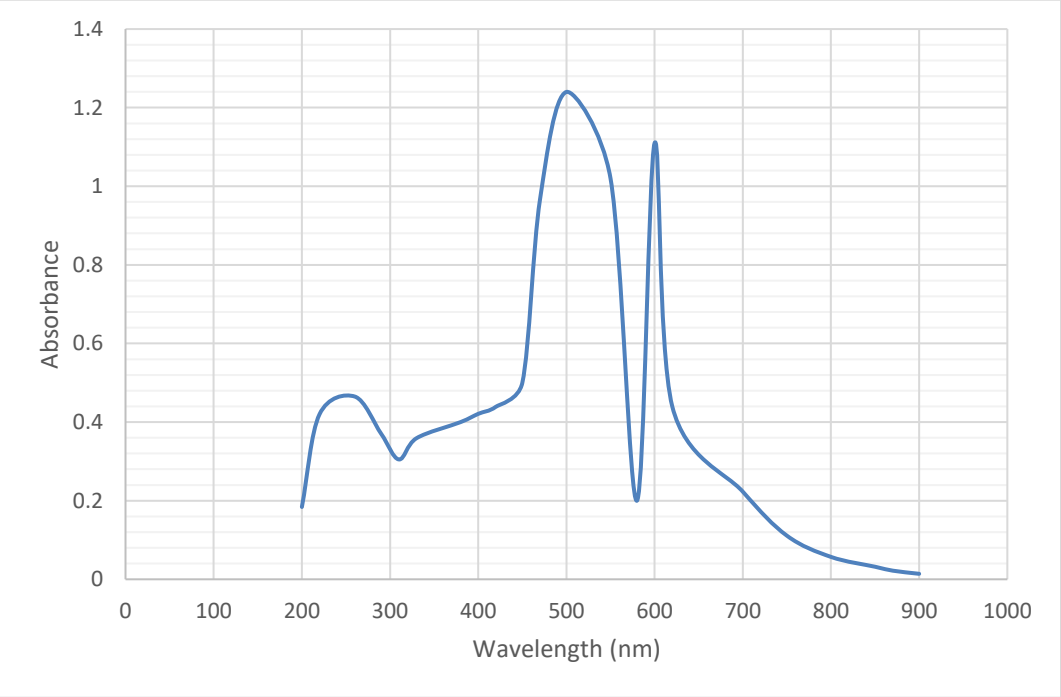

FIG: 5: The UV-Vis spectra analysis of *P. dactylifera*

The UV-Vis spectra analysis of the date samples showed distinct absorbance peaks at wavelengths of 220 nm, 260 nm, 470 nm, 500 nm, and 600 nm, with corresponding absorbance values of 0.42, 0.465, 0.966, 1.24, and 1.11, respectively. Absorbance in the UV region, particularly around 220 nm and 260 nm, is often indicative of the presence of phenolic compounds and flavonoids. Phenolic compounds generally exhibit strong absorbance in the UV region due to their aromatic ring structures. For instance, gallic acid, a common phenolic acid, shows maximum absorbance near 220 nm, while catechins and other flavonoids absorb strongly around 260 nm [26]. The peak observed at 470 nm could be attributed to carotenoids. Carotenoids, including beta-carotene, lutein, and zeaxanthin, typically absorb light in the range of 400-500 nm, with a prominent peak around 470 nm [27]. The absorbance at 500 nm is also within the range typical for some carotenoids and anthocyanins. Anthocyanins are flavonoid pigments that exhibit absorbance maxima between 475 and 550 nm, depending on their structure and pH. The peak at 500 nm could suggest the presence of these pigments, contributing to the red and orange coloration of the date samples [28][29]. The absorbance peak at 600 nm is characteristic of some anthocyanins, especially in more basic or neutral pH environments. These compounds can shift their absorbance to longer wavelengths under different conditions, indicating their presence in the sample [30]. The observed peaks and their corresponding absorbance values suggest that the date samples contain a variety of phenolic compounds, flavonoids, carotenoids, and anthocyanins. High peaks at these wavelengths indicate a significant concentration of these compounds. The strong absorbance at 470 nm and 500 nm particularly points to a high content of carotenoids and anthocyanins, which are known for their antioxidant properties. The presence of these compounds is consistent with previous research on the phenolic and flavonoid content of dates [31].

4.2 Results of biochemical Analysis

Table 7: Brain stress markers; SOD, CAT, GPx and GSH, levels in the cerebral cortex

Groups	SOD(U/g)	CAT(U/g)	GPx(U/g)	GSH(U/g)
Conrol	2.13 ± 0.02^{b}	1.86 ± 0.02^{b}	2.26 ± 0.60^{b}	4.10 ± 0.45^{b}
Diabetes	0.06 ± 0.02^{a}	0.024 ± 0.01^{a}	0.66 ± 0.19^{a}	1.24 ± 0.13^{a}
$AlCl_3$	0.04 ± 0.02^{ab}	0.01 ± 0.00^{ab}	0.32 ± 0.17^{a}	0.58 ± 0.09^{ab}
Diabetes+ *P. dactylifera*	2.67 ± 0.21^{ab}	2.49 ± 0.30^{ab}	2.51 ± 0.13^{b}	5.42 ± 0.15^{ab}
$AlCl_3$+ *P. dactylifera*	5.14 ± 0.10^{ab}	4.54 ± 1.40^{ab}	5.52 ± 0.19^{ab}	7.30 ± 0.28^{ab}
Total	10.04	8.924	11.27	18.64
Average	2.008	1.785	2.257	3.728

Key: [a](P≤0.05)-Significantly different compared to control group, [b] (P<0.05)-Significantly different compared to diabetes group. Data represented as Mean ±SEM. P≤0.05 was considered significant

Results showed a significant increase (P≤0.05) in SOD levels in group D (Diabetes+ *P. dactylifera*) and E ($AlCl_3$+ *P. dactylifera*) when compared to the control and diabetes group. A significant decrease (P≤0.05) in SOD levels in Group C (AlCl3) when compared to control and diabetes was also shown in the result. In comparison with control group SOD levels in group B was significantly decreased(P≤0.05).

Results showed a significant increase (P≤0.05) in CAT levels in group A(control), D(diabetes+ *P. dactylifera*) and E($AlCL_3$+ *P. dactylifera*),when compared to the diabetes group. A significant increase(P≤0.05) in CAT levels in group D(diabetes+ *P. dactylifera*) and E($AlCl_3$+ *P. dactylifera*) when compared to the

control group. A significant decrease (P≤0.05) in CAT levels in Group C ($AlCl_3$) when compared to control and diabetes was also shown in the result. In comparison with control group CAT levels in group B, were significantly decreased(P≤0.05).

Results showed a significant increase (P≤0.05) in GPx levels in group D (diabetes+PD) and E($AlCl_3$+ *P. dactylifera*) when compared to the control and diabetes group. A significant decrease (P≤0.05) in GPx levels in Group B (diabetes) and C($AlCl_3$) when compared to control was also shown in the result. In comparison with diabetes group, GPx levels in group C was significantly decreased(P≤0.05).

Results showed a significant increase (P≤0.05) in GSH levels in group D(diabetes+ *P. dactylifera*) and E($AlCl_3$+ *P. dactylifera*) when compared to the control and diabetes group. A significant decrease (P≤0.05) in GSH levels in Group B(diabetes) when compared to control, was also shown in the result. In comparison with diabetes group, GSH levels in group C had no significant change.

Table 8: MDA and PC levels in the cerebral cortex and CK-MM and AChE levels in Blood

Groups	CKMM(U/L)	ACHe(U/L)	MDA (mmol/g)	PC (mmol/g)
Conrol	12.12±0.10[b]	9.40±0.21[b]	1.07±0.10[b]	3.42±0.09[b]
Diabetes	17.60±1.90[a]	5.04±0.30[a]	4.81±0.23[a]	6.42±0.12a
$AlCl_3$	26.60±0.62[ab]	3.10±0.50[ab]	6.02±0.18[ab]	7.90±0.12[ab]
Diabetes+ *P. dactylifera*	8.10±0.0.08[ab]	11.04±0.14[ab]	0.09±0.00[ab]	1.03±0.08[ab]
$AlCl_3$+ *P. dactylifera*	5.84±0.20[ab]	14.64±0.21[ab]	0.04±0.16[ab]	0.69±0.03[ab]
Total	70.26	43.22	12.03	19.46
Average	14.052	8.644	2.406	3.892

Key: [a](P≤0.05)-Significantly different compared to control group, [b] (P<0.05)-Significantly different compared to diabetes group. Data represented as Mean ±SEM. P≤0.05 was considered significant

Results showed a significant increase (P≤0.05) in CKMM levels in group C($AlCl_3$) when compared to the control and diabetes group. A significant decrease (P≤0.05) in CKMM levels in Group E ($AlCl_3$+ *P. dactylifera*) and D (diabetes+ *P. dactylifera*) when compared to control and also a significance decrease(P≤0.05) in CKMM levels in Group E($AlCl_3$+ *P. dactylifera*),A(control)and D(diabetes+ *P. dactylifera*) diabetes was also shown in the result.

Results showed a significant increase (P≤0.05) in AChE levels in group D (diabetes+ *P. dactylifera*) and E ($AlCl_3$+ *P. dactylifera*) when compared to the control and diabetes group. Also a significant decrease (P≤0.05) in AChE level was also seen in group B(diabetes) and C($AlCl_3$) when compared to the control. A significant decrease (P≤0.05) in AChE levels in Group C($AlCl_3$) when compared to diabetes was also shown in the result.

Results showed a significant decrease (P≤0.05) in MDA levels in group E ($AlCl_3$+ *P. dactylifera*) and D (diabetes+ *P. dactylifera*) when compared to the control and diabetes group. Also a significant decrease in MDA level was also seen in group A(control) when compared to the diabetes group. A significant increase (P≤0.05) in MDA levels in Group C($AlCl_3$) when compared to control and diabetes, was also shown in the result.

Results showed a significant decrease (P≤0.05) in PC levels in group E($AlCl_3$+PD), A(control) and D (diabetes+ *P. dactylifera*) when compared to the control and diabetes group. Also a significant decrease in PC level was also seen in group E ($AlCl_3$+ *P. dactylifera*) and D (diabetes+ *P. dactylifera*) when compared to the control group. A significant increase (P≤0.05) in PC levels in Group C($AlCl_3$) when compared to diabetes, was also shown in the result.

Table 9: Plasma Electrolyte (Ca^{2+} and Na^{+}) levels and Fasting Blood Glucose (BG1; day 1 and BG2; day 28) level

Groups	Ca^{2+}(mg/dL)	Na^{+} (mg/dL)	BG1	BG2
Conrol	2.10±0.10[b]	0.43±0.01	3.90±0.20	4.10±0.23[b]
Diabetes	0.41±0.01[a]	0.40±0.01	4.20±0.11	6.40±0.30[a]
$AlCl_3$	0.40±0.03[a]	0.40±0.03	4.12±0.15	4.50±0.23[b]
Diabetes+ *P. dactylifera*	0.44±0.02[b]	0.44±0.02	4.10±0.11	3.44±0.15[ab]
$AlCl_3$+ *P. dactylifera*	1.40±0.93[b]	1.40±0.93	4.00±0.90	3.50±0.10[ab]
Total	9.66	3.08	20.32	21.94
Average	1.932	0.616	4.064	4.388

Key: [a](P≤0.05)-Significantly different compared to control group, [b] (P<0.05)-Significantly different compared to diabetes group; BG1; day 1 and BG2; day 28. Data represented as Mean ±SEM. P≤0.05 was considered significant

Results showed a significant increase (P≤0.05) in Ca^{2+} levels in group A(control) when compared to the diabetes group. In comparison with control group, Ca^{2+} levels in group B. C, D and E were significantly decreased (P≤0.05). Results showed a significant decrease (P≤0.05) in BG2 levels in A (control), C ($AlCl_3$), D (diabetes+ *P. dactylifera*) and E ($AlCl_3$+ *P. dactylifera*) when compared to diabetes group and decrease (P≤0.05) in BG2 levels in C(AlCl3), D (diabetes+ *P. dactylifera*) and E($AlCl_3$+PD), when compared to control.

DISCUSSION

Phoenix dactylifera, commonly known as the date palm, is a plant with a rich profile of bioactive compounds, contributing to its therapeutic properties. The phytochemical analysis of Phoenix dactylifera revealed significant concentrations of phenols, anthocyanins, flavanols, and flavanones. These compounds are known for their antioxidant properties, which play a crucial role in mitigating oxidative stress and inflammation. (Phenols)Phenolic compounds in *P. dactylifera* have potent antioxidant activities, helping to scavenge free radicals and protect cells from oxidative damage. Phenols are also associated with anti-inflammatory and antidiabetic effects [31] [32]. (Anthocyanins) These pigments possess strong antioxidant properties, contributing to the prevention of lipid peroxidation and DNA damage. Anthocyanins also exhibit anti-inflammatory and neuroprotective effects [33]. (Flavanols and Flavanones) These flavonoids enhance the antioxidant defence system by increasing the activity of endogenous antioxidant enzymes. They also have antidiabetic properties, improving insulin sensitivity and glucose metabolism [34]. Oxidative stress plays an important role in the pathogenesis of diabetes and neurotoxicity. It results from an imbalance between the production of reactive oxygen species (ROS) and the body's ability to detoxify these reactive intermediates. *P. dactylifera* exhibited a serious impact on the various oxidative stress markers, including superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH). SOD is an enzyme that catalyzes the dismutation of superoxide radicals into oxygen and hydrogen peroxide. The study showed that SOD levels were significantly higher in the diabetes + *P. dactylifera* and $AlCl_3$ + PD groups compared to the control and diabetes groups, indicating that Phoenix dactylifera enhances the antioxidant defense system [35]. CAT decomposes hydrogen peroxide into water and oxygen, preventing the formation of harmful hydroxyl radicals. Elevated CAT levels in the treatment groups suggest that *P. dactylifera* helps mitigate oxidative stress by enhancing hydrogen peroxide breakdown [36]. GPx reduces lipid hydroperoxides to their corresponding alcohols and free hydrogen peroxide to water. GSH acts as a substrate for GPx and as an antioxidant itself. The study found increased levels of GPx and GSH in the

treatment groups, indicating improved detoxification of peroxides and enhanced cellular protection [37]. MDA is a marker of lipid peroxidation, while PC indicates protein oxidation. The treated groups exhibited significantly lower levels of MDA and PC, demonstrating the antioxidant efficacy of *P. dactylifera* in reducing oxidative damage to lipids and proteins [38]. Diabetes is characterized by hyperglycaemia resulting from impaired insulin secretion or action. In this present study, the administration of Phoenix dactylifera in alloxan-induced diabetic rats significantly reduced blood glucose levels. This hypoglycaemic effect can be attributed to several mechanisms: Bioactive compounds in *Phoenix dactylifera* may stimulate pancreatic β-cells to secrete more insulin, thereby lowering blood glucose levels [39]. The antioxidants present in Phoenix dactylifera can reduce oxidative stress, which is known to impair insulin signaling. Improved insulin sensitivity facilitates better glucose uptake by tissues [40]. The data demonstrated a significant reduction in glucose levels in the diabetes + PD group compared to the untreated diabetic group, underscoring the antidiabetic potential of *P. dactylifera*.

CONCLUSION

The study revealed that *Phoenix dactylifera* has potential antidiabetic and neuroprotective properties due to its rich phytochemical composition. The administration of *Phoenix dactylifera* in alloxan-induced diabetic and aluminium-induced neurotoxicity showed ameliorative tendencies. These shows that *Phoenix dactylifera* can be an effective natural remedy and an alternative medicinal agent for managing diabetes and neurotoxicity, primarily due to its hypoglycemic and antioxidant properties.

Declaration by Authors

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