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Ameliorative Influence of Matured Coconut Water on Alloxan-induced Pancreatic Damage in Wistar Rats

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ABSTRACT

Diabetes mellitus is a chronic metabolic disease marked by sustained hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Central to its pathogenesis is damage to pancreatic β-cells, prompting interest in natural agents capable of protecting or restoring pancreatic structure and function. This study evaluated the histological effects of matured coconut water (MCW) on the pancreas of alloxan-induced diabetic Wistar rats. Twenty healthy adult male Wistar rats (150–200 g) were randomly assigned to four groups: normal control, diabetic control, diabetic treated with matured coconut water, and diabetic treated with insulin. Diabetes was induced using a single intraperitoneal injection of alloxan monohydrate at 150 mg/kg body weight. Fresh matured coconut water from coconuts aged seven to eight months was administered freely to the treatment group for three weeks. At the end of the experiment, pancreatic tissues were harvested, fixed in 10% neutral buffered formalin, and processed for histological analysis using Hematoxylin and Eosin and Masson's Trichrome stains. Histological findings revealed severe pancreatic damage in the diabetic control group, including acinar atrophy, cellular degeneration, vacuolization, and disrupted islets of Langerhans. In contrast, rats treated with matured coconut water showed improved cellular architecture, reduced vacuolization, and partial restoration of normal pancreatic organization, comparable to insulin-treated animals. These findings suggest that matured coconut water possesses protective and regenerative effects on pancreatic tissue, likely due to its antioxidant and cytoprotective properties, and may serve as a supportive natural supplement in diabetes management.

Keywords: Diabetes mellitus, alloxan, coconut water, pancreas, beta-cells

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder of global significance, affecting millions of individuals worldwide and contributing substantially to morbidity and mortality[13]. It is characterized by persistent hyperglycemia resulting from impaired insulin secretion, defective insulin action, or a combination of both. Central to the pathogenesis of diabetes is pancreatic dysfunction, particularly the destruction or degeneration of insulin-producing beta (β) cells located within the islets of Langerhans[17,23]. These cells play a pivotal role in maintaining glucose homeostasis by synthesizing and secreting insulin in response to blood glucose fluctuations[7,19,28]. Loss or functional impairment of β-cells disrupts glycemic regulation, leading to sustained hyperglycemia and the development of secondary complications, including cardiovascular disease, nephropathy, neuropathy, and retinopathy[4,14,20]. Despite advances in conventional therapy, including insulin administration and oral hypoglycemic agents, achieving long-term glycemic control remains challenging. Limitations include adverse drug effects, poor patient compliance, and incomplete restoration of pancreatic structure and function. Consequently, there is growing interest in exploring natural products and bioactive compounds as adjunctive or complementary interventions for diabetes management[8,10].

Natural compounds are widely regarded for their accessibility, safety profile, and potential to target multiple pathogenic mechanisms simultaneously, such as oxidative stress, inflammation, and β-cell apoptosis. Coconut water, the clear endosperm liquid obtained from *Cocos nucifera*, has long been consumed in tropical regions for its nutritional and therapeutic properties[30]. It is rich in electrolytes, vitamins, amino acids, and bioactive phytochemicals,

including phenolic compounds, flavonoids, and antioxidants, which collectively confer hydration, cardioprotective, and cytoprotective effects. In the context of diabetes, coconut water has been reported to exhibit hypoglycemic activity, reduce oxidative stress, improve lipid profiles, and support pancreatic β-cell integrity[11,21,24]. These effects are thought to result from both direct antioxidant action—neutralizing reactive oxygen species ROS and limiting lipid peroxidation—and indirect modulation of intracellular signaling pathways that regulate β-cell survival, proliferation, and insulin secretion[16,18].

Importantly, the composition of coconut water varies with fruit maturity, which may influence its biological activity. Tender coconut water is predominantly aqueous, rich in electrolytes, and low in macronutrients, whereas matured coconut water MCW, obtained from coconuts aged beyond seven to eight months, contains higher concentrations of sugars, proteins, lipids, and essential minerals such as potassium, magnesium, and calcium[18,26,27,31]. MCW also exhibits increased levels of bioactive compounds, including phenolics and flavonoids, which enhance its antioxidant and cytoprotective capacity[18,27]. These properties suggest that MCW may be particularly effective in attenuating β-cell damage induced by oxidative stress, reducing inflammation, and improving pancreatic tissue architecture in diabetic conditions[18].

Previous studies have demonstrated that antioxidant-rich interventions, such as grape seed proanthocyanidins and medicinal plant extracts, can protect β-cells from apoptosis, restore insulin secretion, and improve pancreatic histology in chemically induced diabetic models[5,9,20]. Similarly, coconut water has shown regenerative effects on pancreatic tissue, though most research has focused on tender coconut water rather than MCW[21,22,24]. Given the distinct biochemical profile of MCW, it is plausible that its administration could confer superior protective effects on the pancreas compared to tender coconut water, yet histological evidence remains limited. Given the rising burden of diabetes and limited histological evidence on MCW, this study investigates its effects on pancreatic architecture in alloxan-induced diabetic Wistar rats, addressing a critical gap in experimental diabetes research.

MATERIALS AND METHODS

Experimental design

This experimental study adopted a controlled laboratory animal design to evaluate the histological effects of matured coconut water on alloxan-induced diabetic Wistar rats. Twenty adult male rats were randomly assigned to four experimental groups (Table 1). Diabetes was chemically induced, followed by treatment with matured coconut water or insulin for a period of 21 days.

Procurement and Extraction of Matured Coconut Water

Fresh matured coconuts were purchased from Orie Agu Nsu daily market in Owerri, Imo State, to ensure freshness and consistency. The coconuts were authenticated based on physical characteristics of maturity such as weight, size, shell, water, liquid content, physical density, and sound, etc.

Matured coconut water (MCW) was extracted from the coconuts each day under hygienic laboratory conditions. The extraction process was done using a local method, which involves manually opening mature coconuts and collecting the liquid without any additional processing (6). This method preserves the natural nutrient composition and minimizes the risk of microbial contamination. The extracted coconut water is then poured into a sealed container and stored in a refrigerator at 4 °C until use.

Drug procurement

Alloxan monohydrate and insulin were purchased from Emmak pharmaceutical suppliers with batch numbers E038609 and PG866347. All chemicals and drugs used in the study were of analytical grade and handled according to manufacturer recommendations.

Animal procurement

Twenty (20) healthy adult male Wistar rats, weighing between 150–200 g, were obtained from the animal house of the Department of Anatomy, Federal University of Technology, Owerri. Only male rats were used to eliminate hormonal variations associated with the female reproductive cycle.

Animal housing

Animals were housed in standard polypropylene cages fitted with stainless steel wire mesh covers. Non-toxic wood shavings were used as bedding and replaced every 48 hours. Rats were kept under controlled environmental conditions (temperature: 22 ± 2°C; relative humidity: 60–70%; 12-hour light/dark cycle) and were fed standard rat pellets with unrestricted access to clean tap water.

Ethical approval

All experimental procedures involving animals were approved by the Institutional Animal Ethics Committee (IAEC) of the Department of Anatomy, Federal University of Technology, Owerri, Imo State, reference number FUTO/REC/MPC/2742/36.

Animal treatment and administration

Induction of diabetes

Following a 14-day acclimatization period, diabetes mellitus was induced in the appropriate groups using a single intraperitoneal injection of alloxan monohydrate at a dose of 150 mg/kg body weight[29]. Animals were fasted for 30 hours before alloxan administration. Fasting blood glucose levels were measured 48–72 hours post-injection using a

glucometer. Rats with blood glucose levels ≥11 mmol/L ≥200 mg/dL were considered diabetic[1].

Treatment regimen

Treatment commenced after confirmation of diabetes and lasted for 21 days. Matured coconut water was administered orally as the sole drinking fluid, while insulin was administered at a standard therapeutic dose of 3 IU/kg body weight per day.

Table 1: Experimental animal grouping (N= 5 per group)

Group	Description	Treatment/ Administered Substance
A	Normal Control	Standard rat chow + water
B	Diabetic Control	Alloxan + water (untreated)
C	Diabetic + Mature Coconut water	Alloxan + matured coconut water
D	Diabetic + Insulin	Alloxan + water + insulin

Animal sacrifice

At the end of the experimental period, animals were humanely euthanized by cervical dislocation. A midline abdominal incision was made, and the pancreas was carefully excised, cleaned of surrounding tissues, and immediately fixed in 10% neutral buffered formalin for histological analysis.

Histological procedures

Pancreatic tissues were fixed in 10% neutral buffered formalin for 48 hours, dehydrated through graded ethanol concentrations, cleared in xylene, and embedded in paraffin wax. Sections of 5 µm thickness were obtained using a rotary microtome and mounted on positively charged glass slides. Sections were stained with Hematoxylin and Eosin (H&E) for general histological architecture and Masson's Trichrome stain for evaluation of fibrosis. Selected sections were further processed for immunohistochemical staining to assess insulin- and glucagon-producing cells. Histological assessments were performed using a light microscope by a blinded observer.

Statistical analysis

All quantitative data were expressed as mean ± standard error of the mean (SEM). Group comparisons were conducted using one-way analysis of variance (ANOVA), followed by Tukey's post-hoc test for multiple comparisons. A p-value less than 0.05 ($p < 0.05$) was considered statistically significant. Statistical analyses were performed using GraphPad Prism version 10.0 and SPSS software 26.0.

RESULTS

Microscopic findings

Acinar cells (A) are arranged in compact clusters with uniform eosinophilic cytoplasm and clearly defined nuclei. The interlobular duct (ILd) appears as a distinct, clear-lumened structure lined by intact epithelium, while blood vessels (BV) are patent with thin walls. No cellular distortion, inflammation, edema, or other histological abnormalities were observed, indicating normal pancreatic morphology in Group A. Group B shows pronounced histopathological changes, including marked widening of the interstitial spaces consistent with edema and scattered inflammatory cell infiltration. The staining pattern appears uneven with pale cytoplasm and hyperchromatic nuclei, while both interlobular and intralobular ducts exhibit irregular luminal outlines containing cellular debris. Blood vessels (BV) appear congested with thickened walls, indicating vascular compromise. The diabetic + MCW pancreas (Group C) shows moderate alterations characterized by cytoplasmic vacuolization and mild-to-moderate acinar atrophy with moderately widened interstitial spaces. Conversely, Group D demonstrates well-preserved pancreatic architecture comparable to the normal control, with intact ducts and absence of vacuolization, edema, or inflammatory infiltration following insulin treatment.

Semi-quantitative histological scoring of pancreatic tissue

Semi-quantitative scoring showed normal pancreatic histology in the control and insulin-treated groups, with vacuolization, acinar atrophy, and edema scores of 0.0 ± 0.0. The diabetic control group exhibited severe damage, with scores of 3.0 ± 0.2, 3.0 ± 0.3, and 3.0 ± 0.2, respectively. MCW treatment reduced these changes to moderate levels, with corresponding scores of 2.0 ± 0.3, 2.0 ± 0.4, and 2.0 ± 0.3, indicating partial pancreatic protection (Table 2).

Statistical analysis of post-administration glucose

The one-way ANOVA resulted in an F-value of 27.12 ($df = 3, 8$), indicating significant differences in post-administration glucose levels across groups ($p < 0.001$). Tukey's HSD post-hoc test confirmed significantly higher glucose levels in the Diabetic control ($p < 0.01$), Diabetic + MCW ($p < 0.01$), and Diabetic + Insulin ($p < 0.01$) groups compared to the normal control. No significant differences were observed among the diabetic groups ($p > 0.05$), suggesting that neither MCW nor insulin fully normalized glucose levels (Table 3).

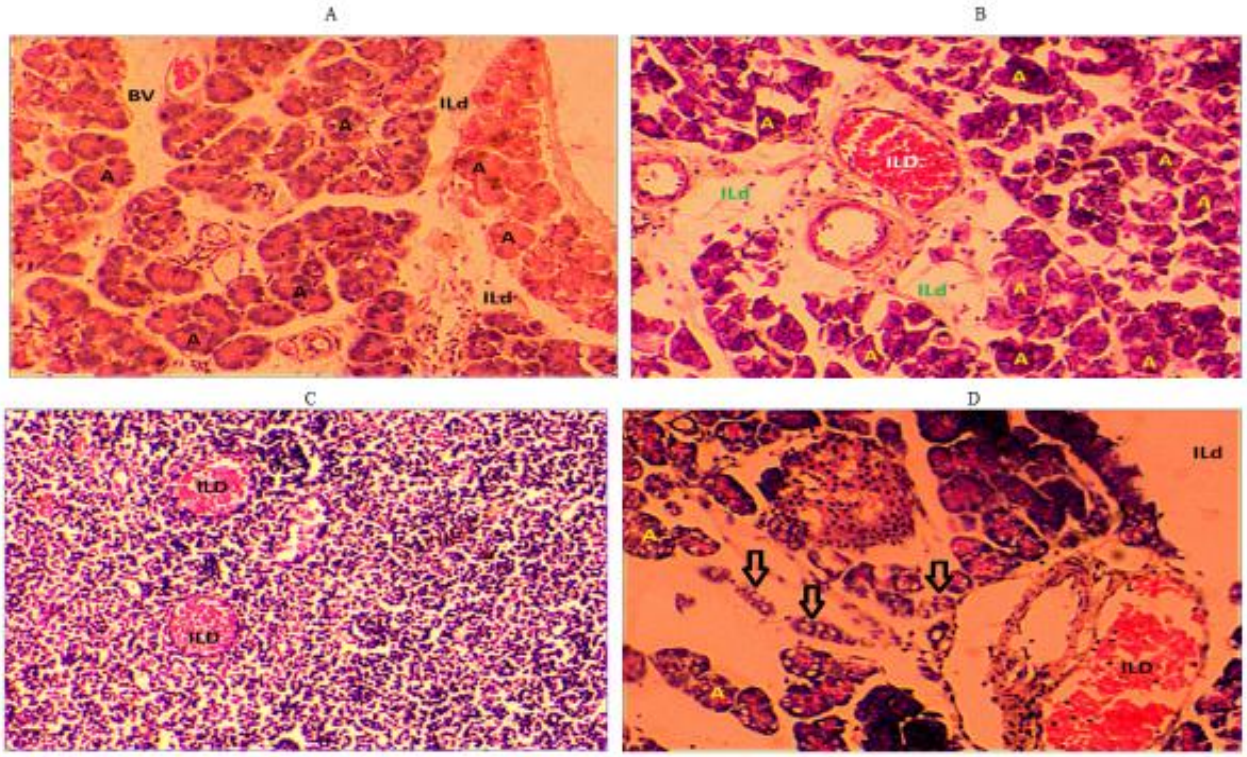

Figure 1: Photomicrographs of pancreatic tissue from Groups A–D at ×100 magnification. Group A is the negative control, group B is the positive control, group C is diabetic + mature coconut water, and group D is diabetic + insulin.

Table 2: Semi-Quantitative Histological Scoring of Pancreatic Tissue

Feature	Normal Control (Mean± SD)	Diabetic Control (Mean± SD)	Diabetic+ MCW (Mean± SD)	Diabetic+ Insulin (Mean± SD)
Vacuolization	0.0 ± 0.0	3.0 ± 0.2	2.0 ± 0.3	0.0 ± 0.0
Acinar atrophy	0.0 ± 0.0	3.0 ± 0.3	2.0 ± 0.4	0.0 ± 0.0
Edema	0.0 ± 0.0	3.0 ± 0.2	2.0 ± 0.3	0.0 ± 0.0

Table 3: Statistical Analysis of Post-Administration Glucose

Group	Mean ± SD	Sample Size (n)	ANOVA F-value (df)	p-value	Tukey's HSD p-values (vs. Normal Control)
Normal Control	169.3 ± 7.0	5	F (5,8) = 3.45	0.071	-
Diabetic Control	154.7 ± 11.0	5			0.121
Diabetic + MCW	153.3 ± 6.1	5			0.098
Diabetic + Insulin	162.3 ± 19.7	5			0.573

Table 4: One-Way ANOVA and Post-hoc Analysis of Histopathological Scores across Experimental Groups

Histological Feature	Group	Mean ± SD	ANOVA F-value (df)	p-value	Tukey's HSD (vs Normal Control)
Vacuolization	Normal Control	0.0 ± 0.0	F(3,8) = 135.67	< 0.001	–
	Diabetic Control	3.0 ± 0.2			< 0.001
	Diabetic + MCW	2.0 ± 0.3			< 0.01
	Diabetic + Insulin	0.0 ± 0.0			1.000
Acinar atrophy	Normal Control	0.0 ± 0.0	F(3,8) = 140.23	< 0.001	–
	Diabetic Control	3.0 ± 0.3			< 0.001
	Diabetic + MCW	2.0 ± 0.4			< 0.01
	Diabetic + Insulin	0.0 ± 0.0			1.000
Edema	Normal Control	0.0 ± 0.0	F(3,8) = 132.89	< 0.001	–
	Diabetic Control	3.0 ± 0.2			< 0.001
	Diabetic + MCW	2.0 ± 0.3			< 0.01
	Diabetic + Insulin	0.0 ± 0.0			1.000

P < 0.001

Statistical analysis of histopathological data

The ANOVA revealed significant differences in vacuolization, acinar atrophy, and edema among the groups ($p < 0.001$). The diabetic control group showed severe histopathological alterations, while treatment with matured coconut water significantly reduced these changes to moderate levels ($p < 0.01$). No significant differences were observed between the normal control and insulin-treated groups ($p = 1.000$), indicating near-complete restoration of pancreatic tissue architecture following insulin therapy (Table 4).

DISCUSSION

The present study demonstrated distinct differences in pancreatic histology and physiological parameters among the experimental groups. Normal control rats group A exhibited well-preserved pancreatic architecture, with compact acinar cells, intact interlobular and intralobular ducts, and patent blood vessels. These findings confirm normal structural integrity, consistent with previous reports of preserved pancreatic morphology in non-diabetic Wistar rats[15].

However, diabetic control rats showed severe histopathological alterations, including extensive acinar vacuolization, cytoplasmic atrophy, interstitial edema, and infiltration of inflammatory cells. These changes reflect alloxan-induced oxidative stress, which selectively damages pancreatic β-cells, leading to reduced insulin secretion and hyperglycemia. The observed structural damage aligns with prior studies reporting β-cell destruction and acinar degeneration following alloxan or streptozotocin

administration[5,9,25]. The reduction in HOMA-B indices further supports impaired β-cell function in diabetic rats, consistent with previous findings[3,9].

Moreover, treatment with matured coconut water MCW produced moderate improvement in pancreatic histology. Acinar cell vacuolization, atrophy, and edema were reduced, and the islets of Langerhans became more discernible, indicating partial protection. These results corroborate reports of regenerative effects of coconut water on diabetic pancreatic tissue[21]. The antioxidant content of MCW likely mitigates ROS-induced β-cell damage[11]. However, residual moderate lesions suggest that MCW alone may not fully restore tissue architecture, contrasting with studies reporting near-complete recovery under higher doses or prolonged treatment[22,24].

Finally, insulin-treated diabetic rats demonstrated near-normal pancreatic histology, with densely packed acinar cells, intact ducts, and minimal vacuolization or edema. These findings align with previous reports highlighting insulin's protective effects on β-cells, including prevention of apoptosis and maintenance of acinar cell integrity[2,3,9,12]. However, glucose levels in insulin-treated rats remained elevated, indicating that histological restoration does not automatically translate to full glycemic normalization, possibly due to suboptimal dosing or individual metabolic variability.

Therefore, the present study demonstrates that MCW provides moderate protection against alloxan-induced pancreatic injury, while insulin offers superior structural recovery. Combining natural antioxidants such as MCW with conventional therapy may enhance pancreatic preservation and support β-cell function in diabetes, although further studies are needed to optimize dosage, treatment duration, and synergistic effects[11,15,22,24].

CONCLUSION

This study demonstrated that matured coconut water (MCW) provided moderate protection against alloxan-induced pancreatic damage and partial glycemic improvement, whereas insulin restored pancreatic architecture to near normal but did not fully normalize blood glucose. However, significant histopathological differences were observed among groups, supporting insulin's efficacy and highlighting MCW's potential as an adjunct therapy in diabetes management despite limitations such as small sample size.

Conflict of interest

The authors guarantee responsibility for all the data published in this manuscript. The authors confirm the absence of a conflict of interest and the absence of their financial interest in conducting this research and articulating this manuscript. This manuscript is extracted and written from an original research work and has never been published, nor is it under consideration for publication elsewhere.

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REFERENCES

1. Abboud AM. A modified protocol of the alloxan technique for the induction of diabetes mellitus in Wistar rats. Med Vet UFRPE. 2020;14(4):315–318.
2. Abunasef SK, Amin HA, Abdel-Hamid GA. A histological and immunohistochemical study of beta cells in streptozotocin diabetic rats treated with caffeine. Folia Histochem Cytobiol. 2014;52(1):42–50.
3. Amin A, Lotfy M, Mahmoud-Ghoneim D, Adeghate E, Al-Akhras MA, Al-Saadi M, *et al*. Pancreas-protective effects of chlorella in STZ-induced diabetic animal model: insights into the mechanism. J Diabetes Mellitus. 2011;1:36–45.
4. American Diabetes Association. Standards of medical care in diabetes—2024. Diabetes Care. 2024;47(Suppl 1):S1–S210.
5. Bahar E, Akter KM, Lee GH, Lee HY, Rashid HO, Choi MK, *et al*. β-Cell protection and antidiabetic activities of Crassocephalum crepidioides (Asteraceae) Benth. S. Moore extract against alloxan-induced oxidative stress via regulation of apoptosis and reactive oxygen species (ROS). Biomed Pharmacother. 2017;92:110–120.
6. Bourdeix R, Konan JL, N'Cho YP. Coconut: a guide to traditional and improved varieties. Montpellier, France: Éd. Diversiflora; 2005.
7. Cerf ME. Beta cell dysfunction and insulin resistance. Front Endocrinol Lausanne. 2013;4:37.
8. Das RR, Rahman MA, Al-Araby SQ, Islam MS, Rashid MM, Babteen NA, *et al*. The antioxidative role of natural compounds from a green coconut mesocarp undeniably contributes to control diabetic complications, as evidenced by the associated genes and biochemical indices. Oxid Med Cell Longev. 2021;2021:9711176.
9. Ding Y, Zhang Z, Dai X, Jiang Y, Bao L, Li Y, *et al*. Grape seed proanthocyanidins ameliorate pancreatic beta-cell dysfunction and death in low-dose streptozotocin- and high-carbohydrate/high-fat diet-induced diabetic rats partially by regulating

endoplasmic reticulum stress. Nutr Metab Lond. 2013;10:51.
10. Erukainure OL, Chukwuma CI. Coconut (Cocos nucifera L.) water improves glucose uptake with concomitant modulation of antioxidant and purinergic activities in isolated rat psoas muscles. Plants Basel. 2024;13(5):665.
11. Fuziy RF, Fávero RLV, de Paula FL. Antioxidant-rich fruit extracts as a strategy to mitigate pancreatic injury and improve glycemic control in experimental models of type 1 diabetes. Food Funct. 2022;13(9):4810–4825.
12. Ghanbari E, Nejati V, Khazaei M. Improvement in serum biochemical alterations and oxidative stress of liver and pancreas following use of royal jelly in streptozotocin-induced diabetic rats. Cell J. 2016;18(3):362–370.
13. Huang X, Wu Y, Ni Y, Xu H, He Y. Global, regional, and national burden of type 2 diabetes mellitus caused by high BMI from 1990 to 2021, and forecasts to 2045: analysis from the Global Burden of Disease Study 2021. Front Public Health. 2025;13:1515797.
14. Iftikhar A, Nausheen R, Khurshid M, Iqbal RK, Muzaffar H, Malik A, *et al*. Pancreatic regenerative potential of manuka honey evidenced through pancreatic histology and levels of transcription factors in a diabetic rat model. Heliyon. 2023;9(9):e20017.
15. Jahan S, Nasrin S, Jahan MK. Histomorphometric analysis of the pancreas in normal and experimentally diabetic Wistar rats. Exp Toxicol Pathol. 2023;75(1):47–58.
16. Krawczyk M, Burzynska-Pedziwiatr I, Wozniak LA, Bukowiecka-Matusiak M. Impact of polyphenols on inflammatory and oxidative stress factors in diabetes mellitus: nutritional antioxidants and their application in improving antidiabetic therapy. Biomolecules. 2023;13(9):1402.
17. Iida H, Watada H. Pancreatic β-cell dysfunction and diabetes. Juntendo Med J. 2025;71(3):158–165.
18. Manful CF, Fordjour E, Subramaniam D, Sey AA, Abbey L, Thomas R. Antioxidants and reactive oxygen species: shaping human health and disease outcomes. Int J Mol Sci. 2025;26(15):7520.
19. Młynarska E, Czarnik W, Dzieża N, Jędraszak W, Majchrowicz G, Prusinowski F, *et al*. Type 2 diabetes mellitus: new pathogenetic mechanisms, treatment, and the most important complications. Int J Mol Sci. 2025;26(3):1094.
20. Nurdiana S, Goh YM, Ahmad H, Dom SM, Syimal'ain Azmi N, Noor Mohamad Zin NS, *et al*. Changes in pancreatic histology, insulin secretion, and oxidative status in diabetic rats following treatment with Ficus deltoidea and vitexin. BMC Complement Altern Med. 2017;17(1):290.
21. Nwangwa EK, Aloamaka CP. Regenerative effects of coconut water and coconut milk on the pancreatic β-cells and cyto-architecture in alloxan-induced diabetic Wistar albino rats. Am J Trop Med Public Health. 2011;1(3):137–146.
22. Overi D, Carpino G, Moretti M, Franchitto A, Nevi L, Onori P, *et al*. Islet regeneration and pancreatic duct glands in human and experimental diabetes. Front Cell Dev Biol. 2022;10:814165.
23. Khin PP, Lee JH, Jun HS. Pancreatic beta-cell dysfunction in type 2 diabetes. Eur J Inflamm. 2023;21(2):1721727X231154152.
24. Rao SS, Najam R. Coconut water of different maturity stages ameliorates inflammatory processes in a model of inflammation. J Intercult Ethnopharmacol. 2016;5(3):244–249.
25. Saeed AA, Al-Musawi AM, Al-Quraishi AM. Alloxan-induced diabetes in Wistar rats: biochemical, histological, and molecular changes. Int J Exp Pathol. 2021;102(3):155–164.
26. Selvaraju VP, Gayathri R, Ranpatabendi T, Fiore A. Matured coconut water: nutritional profile and preservation methods. In: Pandiselvam R, Rathnakumar K, Kothakota A, editors. Coconut-based beverages and products. Cambridge: Elsevier; 2025. p. 55–78.
27. Shi S, Wang W, Wang F, Yang P, Yang H, He X, *et al*. Research progress in coconut water: a review of nutritional composition, biological activities, and novel processing technologies. Foods. 2025;14(9):1503.
28. Tsabang Nolé. Anti-diabetic plants used in Cameroon have a potential for endogenous pancreatic β-cell renewal important in the management of diabetes. Interv Obes Diabetes. 2021;4(4):IOD.000595.
29. Tucker LA, Widholm JJ. Animal models in toxicological and behavioral neuroscience research. Neurotoxicology. 2021;87:123–131.
30. Tuyekar RN, Deshmukh AA, Kumbhar BK. Coconut water: production, nutritional properties and health benefits. ResearchGate. 2021.
31. Zhang G, Chen W, Chen W, Chen H. Improving the quality of matured coconut (Cocos nucifera Linn.) water by low alcoholic fermentation with Saccharomyces cerevisiae: antioxidant and volatile profiles. J Food Sci Technol. 2018;55(3):964–976