

Original Research Article

Effect of *Odogwu Bitters* on pancreatic health of healthy male Wistar rats

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Abstract

Purpose: To investigate the effect of *Odogwu Bitters* on pancreatic health by assessing histopathological, enzymatic, hormonal, and morphometric parameters in healthy male Wistar rats.

Methods: Twenty male Wistar rats were divided into four groups ($n = 5$). The control group (A) received water, while study groups B, C, and D were administered 0.45, 0.75, and 1.05 mL of *Odogwu Bitters* daily for five weeks, respectively. Rat serum was analyzed for amylase, lipase, and insulin. Pancreatic tissue was processed for histology (H&E staining), and relative organ weight was calculated.

Results: The herbal drink treatment induced a dose-dependent reduction in islet β -cell density, with moderate to severe depletion in groups B and C. Serum amylase and lipase were significantly elevated in the high-dose groups (C & D), while insulin levels were significantly reduced in group B. The relative pancreas weight was significantly lower in groups B and C. Altered feed and water intake and attenuated body weight gain were also observed.

Conclusion: This study provides the first direct evidence that *Odogwu Bitters* induces pancreatic injury, manifesting as endocrine cell loss, exocrine enzyme elevation, and organ atrophy in Wistar rats. These findings identify the pancreas as a new target organ for its toxicity and provide grounds for further preclinical safety studies and caution regarding human consumption.

Keywords: Enzyme activity, Histopathology, Islet of Langerhans, Morphometry, *Odogwu Bitters*, Toxicity, Wistar Rats

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INTRODUCTION

In Nigeria, and across many parts of the world, there is a deep-seated and growing trust in herbal remedies [1]. These products, often passed down through generations or amplified by modern marketing, are perceived as natural and therefore safer alternatives to conventional medicines [2]. Among the most popular in recent years is *Odogwu Bitters*, an alcoholic herbal tonic

aggressively promoted for a sweeping range of benefits – from boosting sexual performance and aiding digestion to acting as a general detoxifier and "body warmer". There is a powerful cultural belief that such herbal drinks are "natural" and, as such, are harmless [2], making this herbal drink a staple in many Nigerian households. This unwavering belief, however, is increasingly challenged by emerging science. While consumers reach for *Odogwu Bitters* to cleanse

or fortify their bodies, a different story is unfolding in research laboratories. Preliminary scientific investigations have begun to document the tangible effects of this mixture on vital organs. Studies on Wistar rats have shown that *Odogwu Bitters* elevate liver enzymes such as alanine transaminase (ALT), aspartate transaminase (AST), and alkaline phosphatase (ALP) [3], elevate inflammatory markers like IL-6 [4], cause structural and biochemical injury to the kidneys [5], provoke adverse changes in cardiac tissue [4], and lead to undesirable alterations in the duodenum [6]. Interestingly, research on the testes indicated an absence of gross structural damage [7], revealing that the effects are not blanket toxicity but are peculiarly selective, targeting specific organs.

Despite this growing body of concerning evidence, a major and concerning gap persists. The pancreas, a master regulator of digestion and blood sugar through its production of enzymes and insulin [8], remains almost entirely unexamined. This oversight is both critical and ironic. The pancreas is fundamental to the digestive process that these bitters claim to aid, and it is acutely vulnerable to alcohol, which is a primary component of the drink [9]. Alcohol is a known risk factor for pancreatitis [9], a painful and dangerous inflammation of the organ. Yet, the potential compounded effect of alcohol mixed with the complex cocktail of phytochemicals in *Odogwu Bitters*, including saponins (0.13 %), alkaloids (0.09 %), terpenoids (0.30 %), flavonoids (0.12 %), carbohydrates, cardiac glycosides, and reducing sugars [10], on pancreatic health is completely unknown.

Therefore, while people drink *Odogwu Bitters* for wellness, there is a pressing and unanswered question behind it: What is the cost to the pancreas? This study directly addresses this urgent public health concern. Moving beyond the examination of the liver, kidney, duodenum, testis and heart, this study turned its focus on the pancreas. Using Wistar rats, this study meticulously investigated the histopathological, hormonal, enzymatic, and morphometric consequences of *Odogwu Bitters* consumption on the pancreas, providing clear, scientific data on its safety profile.

EXPERIMENTAL

Animals

The study used 20 healthy male Wistar rats with an initial weight range of 159 – 205 g sourced from a local farm in Nsukka, Enugu State, Nigeria. A veterinarian confirmed that the rats were healthy when they were brought to our research facility. The rats were allowed to

acclimatize for two weeks with access to feed and water *ad libitum*. The health status of rats was closely monitored throughout the period of the experiment.

Acute toxicity test

This study adopted the published toxicity test result for *Odogwu Bitters* in male Wistar rats since the herbal drinks were of the same batch [5]. The published result for LD₅₀ was 2154.07 mg/kg. Hence, the study groups B, C, and D received 0.45, 0.75, and 1.05 mL of *Odogwu Bitters*, respectively, for low, moderate and high doses.

Protocol

The protocol was approved by the animal research ethics committee of Nnamdi Azikiwe University (NAUAREC) with approval number NAU/AREC/2023/00125, dated 11th June 2023. The animal handling guidelines stipulated by NAUAREC were strictly followed. The protocols adopted in this study were reported in line with ARRIVE guidelines (Animal Research: Reporting of In Vivo Experiments).

The herbal drink was purchased from an authorized distributor. The animal feed (Top Feeds Grower's Mash Super-Deluxe, manufactured by Eastern Premier Feed Mills Ltd., a division of Premier Feeds Mills Co. Ltd., Plateau State, Nigeria) was purchased from a local trader in Nnewi, Nigeria. The analytical reagents used included Dragendorff's reagent, Benedict's reagent, and other high-purity chemicals, which were supplied by Syntrol Bioresearch Inc., USA. All the materials were of analytical grade, ensuring their suitability for the procedures.

The rats were divided into four groups of five rats per group ($n = 5$) and housed in spacious cages with a controlled 12-hour light/dark cycle. The choice of the sample size was based on common practice in similar toxicological studies that used Wistar rats [6], which provides sufficient data for statistical reliability while adhering to 3R principles of animal ethics [11].

Group A served as the control, while groups B, C, and D served as the study groups. Group A received only water and standard feed; Group B received 0.45 mL of *Odogwu Bitters*, Group C received 0.75 mL of *Odogwu Bitters*, and Group D received 1.05 mL of *Odogwu Bitters* daily. The treatments were given orally, and in the morning hours between 6 am and 8 am daily for 5 weeks.

Before the administration of the herbal drinks, the rats were weighed using a 6000 g capacity scale (model WT6000GT, WANT Balance Instrument Company Ltd., China). During both the acclimatization and study periods, all the rats had *ad libitum* access to standard pelleted feed and clean water. The cages and environment were cleaned and disinfected daily.

Animal euthanasia

The rats were euthanized via overdose of inhalant anaesthesia (CO₂ gas). The gas flow rate was set to displace 10 – 30 % of the chamber volume per minute to minimize distress. The animals were closely monitored until unconsciousness was achieved, followed by confirmation of euthanasia through the complete cessation of respiratory and cardiac activity and reflexes, including corneal reflexes. The carcasses of the Wistar rats were disposed of in accordance with institutional biosafety protocols. The euthanasia was conducted in full compliance with the institutional animal welfare regulations and ethical guidelines, which follow established protocols for studies involving Wistar rats [12].

Blood collection and organ harvesting

Blood samples were collected through ocular puncture into plastic tubes without additives. The samples were allowed to clot for 30 min to allow complete clot formation. After clotting, the samples were centrifuged at 2500 rpm for 10 min using an 800D Electric Centrifuge (4000 rpm, 6 x 20 mL rotor capacity). The resulting clear serum samples were carefully aspirated and stored at -20°C until further assay. The procedure for blood collection followed an established protocol for animal studies [13].

Rats were positioned on a flat board, with their limbs gently secured using optical pins. The abdomen was then carefully dissected using a dissection kit. Each pancreas was excised, rinsed in a plain tube containing phosphate buffer, and fixed in 10 % formalin for histological examination. This procedure followed an established protocol for organ harvesting of rat models [12].

Histology analysis

Each pancreas was processed through fixation, dehydration, clearing, embedding, sectioning, and staining with haematoxylin and eosin (H&E). Photomicrographs were captured using an Amscope 14MP digital camera mounted on a NOVEX compound microscope equipped with Hi-PLAN objectives. Image labelling was performed

with Photoscape v3.7. All histological procedures adhered to established protocols for rat studies [14].

Determination of serum lipase activity

The serum lipase activity was measured via a standard colorimetric assay [15]. Three tubes were prepared for the assay: a blank, a standard, and a sample tube. 1000 µL of the primary lipase reagent (R1) was added to each tube. This was immediately followed by introducing 20 µL of the prepared standard to the standard tube, 20 µL of the serum sample to the sample tube, and 20 µL of distilled water to the blank tube. After mixing, the tubes were incubated at 37°C for 5 minutes. This was followed by adding 250 µL of the secondary reagent (R2) to each tube, which was mixed and returned to the 37°C incubator for 2 minutes.

Absorbance readings were taken at 580 nm. The first reading (A1) was measured 120 seconds after adding R2, followed by a second reading (A2) after another 120 seconds. The lipase concentration in the sample was finally calculated using Eq 1.

$$CL = \left\{ \frac{(A2-A1)B}{(A2-AI)ST} \right\} CST \dots\dots\dots (1)$$

Determination of serum amylase

Serum amylase activity was measured using the classic starch-iodine colorimetric method [17]. Three reaction tubes were set up: a control (C), a test (T), and a blank (B). A 1000 µL starch substrate was added to each tube. The control and test tubes were then incubated at 37 °C for 3 minutes.

Next, 20 µL of control serum was added to tube C, and 20 µL of the test serum was added to tube T. After thorough mixing, the tubes were returned to the 37 °C incubator for exactly 7.5 minutes. The reaction was then stopped by adding 1000 µL of working iodine reagent to all three tubes, including the blank. Each tube was mixed well and diluted to a final volume of 10 mL with distilled water. Absorbance was measured at 660 nm using a spectrophotometer. Amylase concentration was calculated using Eq 2.

$$CA = \left\{ \frac{(Abs\ B - Abs\ \frac{T}{C})}{(Abs\ B)} \right\} 1480 \frac{U}{i} \dots\dots\dots (2)$$

Determination of rat insulin (Ins) levels

Rat insulin levels were measured using a sandwich enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's instructions (Abcam Rat Insulin ELISA Kit) [16]. One hundred microliters of each rat serum sample was dispensed into the wells of a pre-coated 96-well microplate. The plate was sealed and incubated at 37 °C for 90 minutes. After incubation, the liquid was removed from each well, and the wells were washed 3 times with 300 µL of wash buffer. Next, 100 µL of a biotinylated detection antibody was added to each well, and the plate was incubated again at 37 °C for 60 minutes. Following another wash cycle, 100 µL of an avidin–horseradish peroxidase (HRP) conjugate was added, and the plate was incubated for a further 30 minutes at 37 °C.

The wells were washed 5 times, after which 90 µL of TMB substrate solution was added. The plate was incubated in the dark at 37 °C for 15 minutes, allowing colour development. The reaction was stopped by adding 50 µL of stop solution, which changed the solution to yellow. The absorbance of each well was read immediately at 450 nm using a microplate reader. A standard curve was created using known insulin concentrations, and the insulin level in each sample was determined by interpolating the sample absorbance from the standard curve.

Determination of relative organ weight

The relative organ weight was determined to assess organ size in proportion to the animal's overall body size. The body weight (BW) of the rats was measured on the last day of the experiment. The pancreas of each rat was carefully dissected, rinsed and weighed to record their individual organ weight (OW). The relative weight for each organ was calculated using Eq 3.

$$\text{ROW} = (\text{OW}/\text{BW})100 \dots\dots\dots (3)$$

This calculation expresses the organ weight as a percentage of the animal's body weight, providing a standardized measure for comparison across subjects.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics software (version 25). The data are presented as mean ± standard error of the mean (SEM). A t-test was used to compare the differences between the initial and final body weights of the animals. One-way analysis of

variance (ANOVA) was used to compare results across the control and study groups. The homogeneity of variances was first confirmed using Levene's test.

When the ANOVA indicated a statistically significant result, post hoc analyses were conducted to determine which specific groups differed from each other. A *p*-value less than 0.05 was considered statistically significant for all tests.

RESULTS

Histological analysis

The pancreatic histology of the control group (Group A) showed preserved lobular architecture with normal distribution of Islets of Langerhans. The islets displayed a characteristic peripheral arrangement of Alpha-cells and abundant central Beta-cells. Acinar cells exhibited typical morphology with apical granular eosinophilic cytoplasm and basally located nuclei, while interlobular ducts were surrounded by dense fibrous tissue (Figure 1 a).

Study groups exhibited a dose-dependent reduction in islet Beta-cell density. Group B showed moderate central Beta-cell loss compared to control (Figure 1 b), while Group C exhibited moderate-to-severe depletion (Figure 1 c). In contrast, Group D presented only mild B-cell reduction (Figure 1 d). The peripheral localization of Alpha-cells remained intact across all groups. Acinar cell morphology and peri-ductal fibrous tissue appeared consistent with the control in all treated specimens.

Serum amylase, lipase and insulin levels

Administration of *Odogwu Bitters* significantly altered key pancreatic serum biomarkers in a dose-dependent manner (Table 1).

Serum amylase and lipase levels followed a similar pattern. Compared to the control group (A), Group B showed a decrease in both amylase and lipase, which is statistically significant (*p* < 0.001 and *p* = 0.015). Groups C and D exhibited significant elevations in these pancreatic enzymes (Amylase: *p* = 0.031 and *p* < 0.001; Lipase: both *p* < 0.001; Table 1).

Insulin levels presented a distinct profile. A significant reduction was observed in Group B (*p* < 0.001) compared to control. In contrast, insulin levels in Groups C (*p* = 0.269) and D (*p* = 1.000) were not significantly altered, remaining comparable to those in Group A.

Morphometric findings

Body weight changes and relative pancreas weights are presented in Table 3. Rats in the control group (A) gained significant weight over the study period ($p = 0.008$). In contrast, weight gain in all treatment groups (B, C, and D) was not statistically significant ($p = 0.375$, $p = 0.291$, and $p = 0.279$, respectively).

Body weight changes between the control and study groups showed no significant differences. However, analysis of relative pancreas weight (organ-to-body weight ratio) revealed a significant decrease for groups B ($p = 0.027$) and C ($p = 0.002$) when compared to the control group. The reduction observed in group D was not significant ($p = 0.740$).

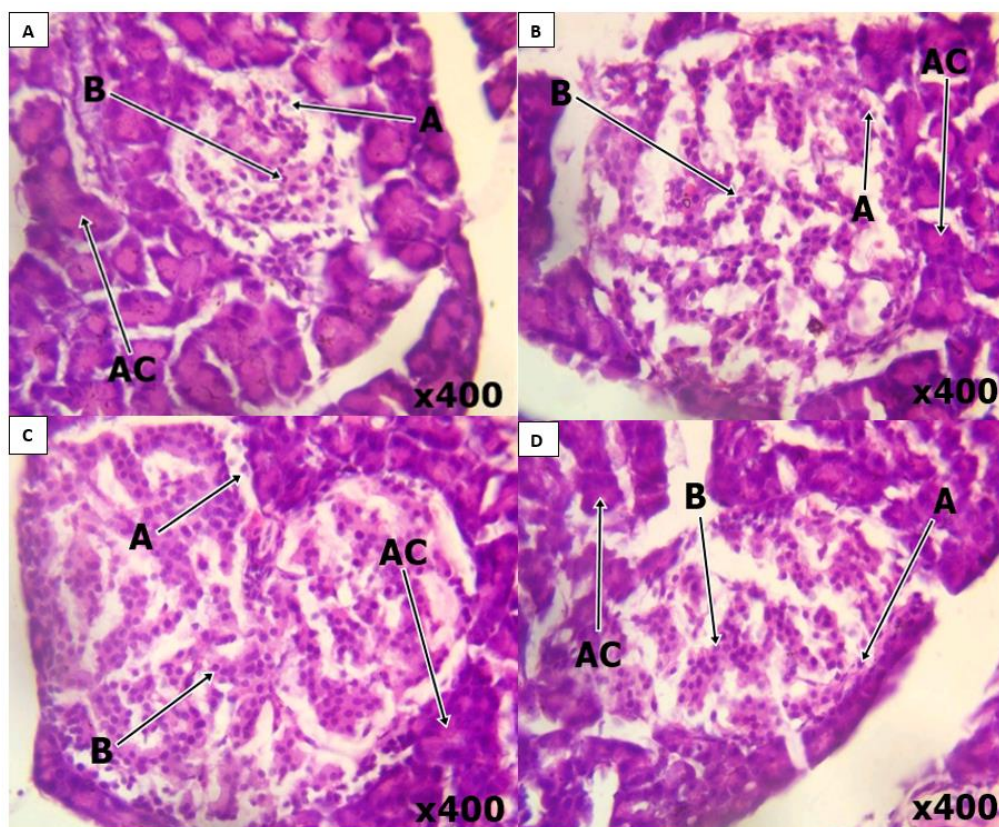


Figure 1: (a – d) Representative photomicrograph of the pancreas of rats in Groups A – D, respectively. AC=Acinar cells, A= α -cell, B= β -cell; 400x magnification; H&E stain

Table 1: Effect of *Odogwu Bitters* on serum amylase, lipase, and insulin of the rats

Group	Amylase (U/L)	p-value	Lipase (U/L)	p-value	Insulin (mIU/L)	P-value
A	225.7 $\pm$ 0.00		56.11 $\pm$ 0.00		4.25 $\pm$ 0.00	
B	132.7 $\pm$ 0.00*	0.000	44.89 $\pm$ 0.00*	0.015	3.92 $\pm$ 0.00*	0.001
C	252.2 $\pm$ 0.00*	0.031	112.22 $\pm$ 0.00*	0.000	4.14 $\pm$ 0.00#	0.269
D	376.1 $\pm$ 0.00*	0.000	112.22 $\pm$ 0.00*	0.000	4.25 $\pm$ 0.00#	1.000

Values are Mean $\pm$ SEM. * $P \leq 0.05$ vs Group A; # $P \geq 0.05$ vs Group A

Table 2: Results of the initial and final body weights of the rats

Group	Body weight	Values	P-value
A	Initial	171.19 $\pm$ 32.5	0.008*
	Final	221.82 $\pm$ 5.52	
B	Initial	177.35 $\pm$ 5.64	0.375#
	Final	192.25 $\pm$ 12.91	
C	Initial	185.77 $\pm$ 8.31	0.291#
	Final	205.00 $\pm$ 3.67	
D	Initial	159.13 $\pm$ 6.36	0.279#
	Final body weight	171.00 $\pm$ 13.05	

Values are Mean $\pm$ SEM. * $P \leq 0.05$ vs initial body weight for Group A; # $P \geq 0.05$ vs final body weight for Group A

Table 3: Result of the effect of *Odogwu Bitters* on body weight and relative pancreas weight

Group	Body weight (g)	P-value	Relative pancreas weight (g)	P-value
A	171.19±11.27		0.38±0.02	
B	177.35±1.86 [#]	1.000	0.32±0.02*	0.027
C	185.77±2.73 [#]	0.516	0.29±0.01*	0.002
D	159.13±2.09 [#]	0.928	0.37±0.02 [#]	0.740

Values are Mean ± SEM. * $P \leq 0.05$ vs Group A; [#] $P \geq 0.05$ vs Group A

Table 4: Result of the effect of *Odogwu Bitters* on feed and water intake

Group	Feed intake (g)	P-value	Water intake (mL)	P-value
A	1101.04±14.63		78.19±1.69	
B	1128.79±19.56	1.000 [#]	82.45±1.63	0.367 [#]
C	1202.54±23.07	0.000*	85.28±1.88	0.012*
D	1268.11±10.33	0.000*	81.79±0.82	1.000 [#]

Values are Mean ± SEM. * $P \leq 0.05$ vs Group A; [#] $P \geq 0.05$ vs Group A

Effect of *Odogwu Bitters* on feed and water consumption

Treatment with *Odogwu Bitters* was associated with increased consumption of both feed and water across study groups (B–D) relative to the control (Group A).

Feed intake was significantly elevated in Groups C ($p < 0.001$) and D ($p < 0.001$). The increase observed in Group B was not statistically significant ($p = 1.000$). Similarly, water intake was significantly higher in Group C ($p = 0.012$). The increases noted in Groups B ($p = 0.367$) and D ($p = 1.000$) did not reach statistical significance.

DISCUSSION

This study provides the first comprehensive evidence of pancreatic injury in Wistar rats following consumption of *Odogwu Bitters*, a popular herbal-alcoholic mixture. Findings from this study revealed a complex, dose-dependent disruption of pancreatic physiology, spanning its exocrine, endocrine, and structural integrity.

The most striking histological alteration was the dose-dependent reduction in insulin-producing β -cell density within the Islets of Langerhans, particularly in Groups B and C. This finding is critical, as β -cell mass is directly linked to insulin secretory capacity [17]. The significant reduction in serum insulin observed in Group B aligns with this histopathological loss, suggesting an early compromise of endocrine function. The preservation of insulin levels in Group D, despite receiving the highest dose, presents a paradox. This may reflect a compensatory hyperinsulinemia response to developing insulin resistance—a common precursor to metabolic dysfunction often associated with chronic herbal-alcohol exposure [18] or an initial adaptive hormetic response that may fail with prolonged

exposure [19]. This non-linear dose-response underscores the complexity of the phytochemical-alcohol interaction on endocrine tissue.

Simultaneously, the exocrine pancreas exhibited significant stress. The pronounced elevation of serum amylase and lipase in Groups C and D is a classic serological hallmark of pancreatic acinar cell injury and pancreatitis [20]. However, the significant decrease in amylase and lipase levels in Group B is more enigmatic but may indicate early pancreatic dysfunction or a suppressant effect of specific phytochemicals on enzyme synthesis prior to overt cellular necrosis and leakage [21]. This biphasic enzymatic response (suppression at a lower dose and marked elevation at higher doses) mirrors patterns seen in toxicant-induced pancreatic damage where cellular homeostasis is disrupted before outright inflammatory destruction [22].

The morphometric data further support the pathological impact. The significant reduction in pancreas weight in Groups B and C suggests organ atrophy or the loss of cellular mass, consistent with the observed β -cell depletion and acinar cell damage. The absence of significant weight change in Group D, coupled with milder histological damage, again points to a divergent adaptive or toxicodynamic pathway at the highest dose that warrants further investigation. Importantly, the stunted body weight gain across all treatment groups, despite increased feed intake in Groups C and D, indicates a state of metabolic inefficiency or malabsorption. This is a recognized consequence of pancreatic exocrine insufficiency, where deficient digestive enzyme secretion impairs nutrient assimilation, even with adequate caloric consumption [23].

The observed increase in water and feed consumption, particularly in Group C, likely

stems from multiple interacting factors. While it may represent a behavioural compensatory response to suboptimal nutrient absorption from pancreatic dysfunction, a direct appetitive effect of the alcohol and phytochemicals of the beverage is also probable. Furthermore, the animals in Group C had the highest final body weight among the treated groups. This larger body mass inherently creates greater metabolic demands, which would be expected to drive increased consumption of both feed and water. Therefore, the elevated intake likely results from a combination of direct pharmacological stimulation, compensatory behaviour, and the basic physiological requirements of a larger body mass.

When contextualized within the broader literature, our findings reveal a significant and concerning pattern. Studies prior to this investigation have established that *Odogwu Bitters* induces hepatorenal toxicity [5], cardiac inflammation [4], and duodenal injury [6]. The present study definitively adds the pancreas to this spectrum of target organs. The mechanism of injury is likely multifactorial, stemming from the synergistic toxicity of its ethanol content and its mixture of phytochemicals (including saponins, alkaloids, terpenoids, and flavonoids) [10], which may provoke oxidative stress, metabolic overload, and inflammatory cascades within pancreatic tissue. A critical implication is that the common consumption pattern, regular, moderate intake for perceived wellness, constitutes a classic low-grade chronic exposure model, precisely the condition under which such progressive organ damage unfolds. Collectively, this evidence fundamentally challenges the "natural equals safe" paradigm surrounding this beverage and underscores an urgent need for targeted public health awareness and regulatory scrutiny.

Overall, the findings indicate that consumption of *Odogwu Bitters* triggers subclinical pancreatitis, impairs insulin secretion at lower doses, and induces paradoxical metabolic and histological responses that challenge the assumption of its safety.

Limitations of the study

While the findings of this study provide clear evidence of pancreatic injury, there are several constraints inherent in the study design. First, this study on Wistar rats, though a robust model, requires cautiousness when considering human implications; metabolic and physiological differences between species mean the precise human equivalent of the observed effects

remains uncertain. Second, the five-week exposure window captures a significant sub-acute phase but cannot reflect the potential consequences of the years of habitual consumption often seen in human populations. It can be recognized from this study that by using only male rats, there are important unanswered questions about whether female physiology might respond differently to this mixture. Furthermore, the controlled dosing method, while precise, does not mirror the variable and social patterns of human drinking. It should be noted that as a commercial herbal product, natural variation between batches could influence the consistency of its effects. Perhaps most importantly, this study maps the *what* and the *where* of the damage but stops short of revealing the precise *how*—the specific molecular cascade triggered by the blend of alcohol and herbs. Finally, this study did not investigate whether the pancreas could recover after stopping exposure, a critical question for understanding the reversibility of the damage. These gaps naturally point the way for future, more detailed investigations.

CONCLUSION

This study establishes, for the first time, that consumption of *Odogwu Bitters* induces significant harm to the pancreas in a mammalian model. It documented a triad of injury: dose-dependent loss of insulin-producing β -cells, elevated serum enzymes indicative of acinar cell damage, and a reduction in relative pancreatic mass. These effects culminated in a state of metabolic inefficiency, where increased feed intake did not translate to healthy weight gain.

Collectively, these findings move the safety profile of this popular herbal-alcoholic beverage from the realm of anecdote into evidence-based concern. The pancreas joins the liver, kidney, and heart as a target organ for its toxicity. Therefore, the widespread perception of this mixture as a harmless "natural" tonic may be untenable. There should be a reconsideration of its unregulated promotion and recommendation for immediate public health education on its potential risks. Additional studies to elucidate the exact mechanisms of this toxicity and investigate the long-term clinical consequences for human consumers should be carried out.

DECLARATIONS

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Conflict of interest

No conflicts of interest are associated with this study.

Contribution of authors

We declare that this work was done by the authors named in this article, and all liabilities pertaining to claims relating to the content of this article will be borne by the authors. All authors read and approved the final manuscript. DNO conceptualized, designed, and wrote the study protocol, supervised the experiment, carried out the data analysis, wrote the first draft of the manuscript, sourced the funding for the study, and assisted POA in managing the literature searches. IFO assisted POA in managing the animals, carried out the assay protocol and reviewed the manuscript. NMU carried out the histology analysis and reviewed the manuscript. POA managed the literature searches, managed the animals and curated the data. ECO assisted POA in curating the data and assisted DNO in sourcing funding.

Availability of data and materials

The datasets generated during and/or analysed during the current study are available within the text.

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