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RESEARCH ARTICLE

Anti-Obesity Properties of Binahong (Anredera Cordifolia) Extract on Hepatic Steatosis in Wistar Rats Model

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ABSTRACT

Obesity, characterized by the accumulation of adipocytes, is associated with complications such as fatty liver and non-alcoholic steatosis, which can be fatal. This research investigates the properties use of *Anredera cordifolia* ethanol extract in reducing steatosis through microscopic analysis of the non-alcoholic fatty liver disease activity scores (NAS) and immunohistochemical examination (IRS) of PPARγ expression in hepatic cells and abdominal fat tissue in obese Wistar rats .Significant differences were observed in overall steatosis scores among groups ($p < 0.005$), with the control group (K1) exhibiting markedly higher scores compared to all treatment groups (P50, P100, P150; $P < 0.005$). Additionally, significant differences in NAS scores were found between the K1 and K2 groups ($P < 0.05$), as well as among the K0 and K1 groups ($P < 0.05$). A significant difference in PPARγ IRS was noted between K0 and K2 groups and all treatment groups (P50, P100, P150; $P < 0.05$). Similarly, a significant difference was observed among the K2 group and all treatment groups ($P < 0.05$), as well as among the K1 and a higher-dose treatment group (P100, P150). Beyond reducing abdominal circumference and adipose tissue in the abdomen, the anti-obesity effect of *A. cordifolia* extract was evident in reducing hepatic steatosis, potentially mediated through the downregulation of PPARγ expression.

Keywords: Anredera cordifolia, NAS score, Obesity, PPARγ, Steatosis

Introduction

The global overweight and obesity are becoming more common, posing a heightened risk of metabolic disease including diabetes type 2, hypertension, high cholesterol and dyslipidemia, and chronic liver disease.[1] The liver had a fundamental role in maintaining systemic balance of lipids through the regulation of lipid influx from the circulatory system, *de novo* lipogenesis, and the production of fats as very-low-density lipoproteins (VLDL) for peripheral tissue distribution.[2]

While the liver regulates lipid flux in normal homeostasis, white fat tissues are believed to represent the primary organ that acts as a reservoir for surplus lipids and plays a pivotal role in maintaining systemic

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lipid balance.[3] However, in conditions like diabetes or obesity, hepatic lipid homeostasis becomes dysregulated. This causes lipid accumulation, which can predispose individuals to greater in severity non-alcoholic fatty liver disease (NAFLD), encompassing non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma.[4]

Non Alcoholic Fatty Liver Disease is associated with macrovesicular alterations, non-inflammatory steatosis, and non-inflammatory lobular inflammation, and results from overconsumption of alcohol.[5] It is distinguished by hepatic steatosis in the absence of hepatocellular injury, characterized by increased hepatic fatty acid inflow as a result to either an adipocyte deficiency for triglyceride repository or an increased manner of adipogenesis driven by upregulated expression of the transcription factor peroxisome proliferated activated regulator γ (PPARγ).[6] This upregulation is induced by lymphocytic and neutrophilic inflammation in the perivenular area, with or without fibrosis.[6] PPARγ is a transcription factor that acts as a crucial mediator of cellular fat formation.[7]

To prevent complications associated with obesity, weight loss is imperative and can be achieved using natural weight-loss therapeutics and interventions. Extract from *Anredera cordifolia*, a plant commonly known as binahong, is a well-established natural therapeutic remedy for obesity with the potential to induce weight loss and decreased fatty liver. This goal of the research is to find the imfact of Binahong (*Anredera c)* extract a weight loss drugs regarding the prevalence of steatosis in the liver in obese Wistar rats. The assessment will include an evaluation of NAFLD activity scores (NAS) and PPARγ expression, key regulators of cellular lipogenesis.

Materials and methods

Materials

The study was performed at the Department of Pharmacology, Medical Faculty, Universitas Sumatera Utara, within Pharmacology and Therapy Laboratory. The ethanol extract of binahong leaves was prepared in Phytopharmaceuticals Laboratory of the Faculty of Pharmacy, Universitas Sumatera Utara. Histopathological examination of the livers and analysis of PPARγ expression were performed at the Histology Laboratory, Universitas Sumatera Utara.

Animals and ethics

The study utilized obese white Wistar rats weighing between 100–150 grams. The sample size was determined utilizing the Federer formula, forming six groups, each initially consisting of 24 rats. To account for potential dropouts, loss to follow-up, and the need for repetition, the sample size was adjusted to 6 rats per group, resulting in a total of 36 rats across all test groups, divided equally among control and treatment groups.

The control group consisted of three subgroups: a standard diet sham group (K0), a diet heavy in fat absent treatment positive control (K1), and a diet heavy in fat with orlistat intervention Negative control (K2). The treatment group was given a diet heavy in fat in addition to intervention with *A. cordifolia* extract at various doses: P50, P100 and P150 mg/kg BW, respectively, administered once daily orally through a gastric tube. The rats were fed according to their experimental groups for eight weeks, followed by *A. cordifolia* extract treatment for 4 weeks, experimental design displayed in Fig. 1.

Preparation of A. Cordifolia extract

A sample of *A. cordifolia* was collected from the Tiganderket highland in the Kabanjahe district of Sumatra Utara, Indonesia. The leaves were washed, sorted, and fully dried in a cabinet under a lamp, and then ground into a powder. Subsequently, 50 g of the powdered leaves were macerated with 250 ml of 70% ethanol solvent for a duration of one day. That solution was then refined to extract the filter out from the pulp. The remaining pulp was then serially macerated for additional extraction with 150 ml and then 100 ml of ethanol for 24 hours each. The resulting extracts were pooled together and completed to a total volume of 500 ml with ethanol. The pooled solution was allowed to sit for 24 hours before a final filtration. A rotary vacuum evaporator was employed to evaporate the effluent and dried using the freeze dryer.[8] Finally, the evaluation for phytochemicals test was performed to analyze the molecules of metabolites present with in the *A. cordifolia* extract.

Examination procedure for abdominal adipose tissue

After 4 weeks of treatment, animals were euthanized using a Ketamine dose of 75 mg/kg BW administered intraperitoneally. The abdominal cavity was opened to observe and isolate the total abdominal fat, which was then placed in a Petri dish[9] and examined for PPARγ expression using immunohistochemistry.

Liver microscopic examination procedure

The liver was examined histologically by fixing samples in a solution of 10 % neutrally buffered formalin and embedding them in paraffin at room

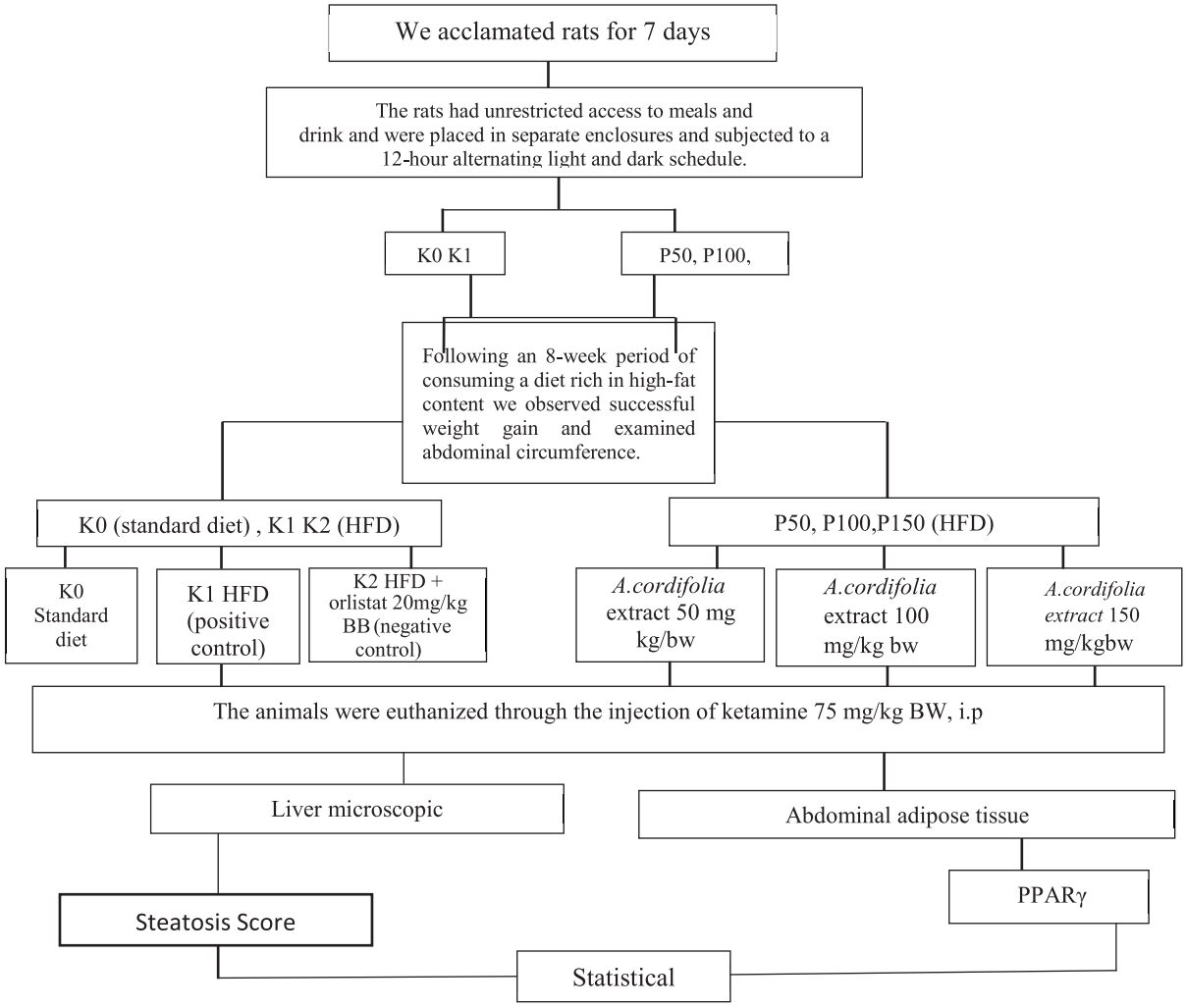

Fig. 1. Timeline of the study.

temperature (25°C). A thin section (4 μm) of the paraffin block was prepared for histology and hematoxylin-eosin staining using routine techniques at the Department of Histology, College of Medicine, Universitas Sumatera Utara. The histopathologists used the NAS scoring system to assess the tissue.

Statistical analysis

SPSS 20.0 was utilized to statistically analyze the data the one-way analysis of variance (ANOVA), Post Hoc Least Significant Difference (LSD), Kruskal-Wallis, Mann-Whitney, and Wilcoxon tests. *P*-values were used to find significance in statistics were < 0.05.

Result and discussion

An examination of phytochemicals of the extract *A. cordifolia* revealed the presence of flavonoid compounds and saponins, with no detectable alkaloids, tannins, or steroids/triterpenoids, consistent with findings from a previous study.[9] The results of the phytochemical analysis of *A. cordifolia* ethanol extract are presented in Table 1.

Table 1. Phytochemical screening test of ethanol extract of *A.cordifolia*.

No	Secondary Metabolite	Reagents	Result
1	Alkaloid	Mayer Bouchardat Dragendorf	–
2	Flavonoid	Mg powder + HCl p + amyl alcohol	+
3	Glycoside	Molish + H_2SO_4	+
4	Saponin	hot distilled water/shake	+
5	Tanin	$FeCl_3$	–
6	Steroid/terpenoid	Lieberman-Bourchat	–

The study demonstrated that the ethanol extract of *A.cordifolia* leads to a decrease in body weight in obese rat groups across all dosages (50, 100, and 150

mg/kg BW) following four-week intervention period. This investigation revealed significant differences in body weight among the K1 and the K2 groups ($P < 0.05$). The data indicates a reduction in abdominal circumference (AC) in the rats were administered extract from *A. cordifolia* using fifty mg/kg BW (P50) dosage and one hundred mg/kg BW (P100), That proved to be of statistical significance ($p < 0.05$; Table 2.). However, at 150 mg/kg BW dosage, the reduction in AC was not of statistical significance ($p > 0.05$).

This study also found a notable increase in the AC in standard diet control (K0) and the high-fat diet (HFD; K1) groups also ($p < 0.05$). Conversely, K2 control group, intervention with orlistat, did not exhibit a decreased significantly in AC ($P > 0.05$).

In both the standard diet control (K0) and HFD control no treatment group (K1), there was a s notable rise in AC ($P < 0.05$), while the control group with orlistat treatment (K2) showed no statistically significant reduction in abdominal size ($P > 0.05$). This indicates that a HFD leads to greater weight gain compared to a typical diet, aligning with previous research.[10] The use of high-fat diets is a common method utilized to induce obesity in experimental animal models over an 8-week period.[11,12] Such diets result in harmful effects, underscoring the significant role nutrition plays in the obesity epidemic.[13,14] These data are summarized in Table 2.

The data showed the average visceral abdominal fat in the control groups as follows: K0 (untreated standard diet) had 2.52 ± 0.44 grams, K1 (untreated HFD) had 6.04 ± 3.36 grams, and K2 (HFD with orlistat treatment) 3.65 ± 2.60 grams. In the treatment groups, the average visceral abdominal fat weights were: P50 (50 mg/kg BW) at 3.12 ± 0.88 grams, P100 (100 mg/kg BW) at 3.40 ± 0.44 grams, and P150 (150 mg/kg BW) at 3.27 ± 0.69 grams.

Based on the one-way ANOVA test, our research demonstrated a noteworthy disparity in the abdominal visceral fat weight among the groups ($P < 0.05$). To identify specific group differences, the LSD one-way ANOVA post hoc test was performed, revealing significant differences in abdominal visceral fat weight between untreated HFD rats (K1) and all the rats were conducted with *Anredera c* extract across all doses ($P < 0.05$; Table 3.) as opposed to orlistat intervention. Consequently, it may be said that the use of *Anredera c* extract aids in shedding pounds and may decrease fats contained in the abdominal cavity. This is evidenced by the notable decline in abdominal fat weight in all treatment groups (P50, P100, P150) in contrast to the HFD group (K1; $P < 0.05$), indicating the extract can reduce abdominal fat weight at any of the tested doses. Central obese is defined as the excess buildup of adipose tissue in the body excess of subcutaneous and visceral fat, which can pose significant health risks, often resulting from an energy imbalance between nutritional intake and insufficient physical activity.[15]

Assessment of hepatic steatosis was based on the total steatosis score, which includes evaluation of fatty liver, lobular inflammation, and number of ballooning hepatocytes. Liver fibrosis was not assessed due to the absence of Masson's trichrome-stained smear preparation. Non-alcoholic hepatic steatosis (NASH) comprises several components, each with a specific scoring range: steatosis (0–3), lobular inflammation (0–3), and hepatocyte ballooning (0–2).[16] Based on an interpretation of the NAS score, a score from 0–2 was not considered to be a diagnosis of NASH, a score from 3–4 was borderline for NASH, and a score from 5–8 fulfilled the diagnostic criteria for NASH.

The K1 group had a median NAS score of 2.5, ranging from 1–5, indicating that the group was borderline for NASH. A group was fed a diet rich in fats and un-treatment (K2), had a higher median NAS score of 7 (range 5–7), indicating NASH. The K3 control group, treated with orlistat, had a median NAS score of 4 (range 3–5) which is borderline for NASH. The treatment groups had the following median NAS Scores: P1 (50 mg/kg BW) was 3 (range 2–4), P2 was 2.5 (range 1–4), and P3 was 4 (range 3–6). All treatment groups (P1, P2, P3) were borderline for NASH.

In this study, the Kruskal-Wallis test revealed a considerable disparity in the total steatosis scores among

Table 2. Characteristic sample body weight (BW), abdominal circumference (AC) before and after treatment.

Groups	BW before treatment (gram)	BW after treatment (gram)	*P* value	AC before treatment(cm)	AC after treatment(cm)	*P* value
K0 (n = 6)	180 (171–181)	212.5 (208–215)	0.027*	12.8 (12–12.8)	13.6 (13.4–13.8)	0.026*
K1 (n = 6)	231 (195–242)	261 (209–290)	0.043*	14 (13–14.5)	16.5 (15–17)	0.041*
K2 (n = 6)	220 (198–248)	198 (189–235)	0.068	14 (13.5–14.6)	14 (13.2–14)	0.063
P50 (n = 6)	215 (191–241)	217 (190–223)	0.686	14 (13.5–14.5)	13.7 (13.3–14.4)	0.041*
P100 (n = 6)	210 (200–260)	199.50 (198–248)	0.075	13.7 (13.5–15)	13.35 (13.2–13.5)	0.043*
P150 (n = 6)	211.5(180–231)	207.50 (190–228)	0.528	13.85 (12.5–14.3)	13.4 (13–14)	0.345

Wilcoxon test, significant $p < 0.05$ data are Median (min-max).

Table 3. Abdominal fat weight in animal obesity model groups.

Groups	Adipose tissue weight (gram)	*P* value	Groups	Average weight of adipose tissue (gram)	*P* value
K0	2.52 ± 0.44				
K1	6.04 ± 3.36	0.045*	K0VS K1	3.52	0.002*
K2	3.65 ± 2.60		K1VS K2	2.39	0.036*
P50	3.12 ± 0.88		K1VSP50	2.92	0.012*
P100	3.40 ± 0.44		K1VSP100	2.64	0.017*
P150	3.27 ± 0.69		K1VS P150	2.77	0.013

One way anova test, LSD analysis data are mean± SD.

Table 4. The microscopic examination for an occurrence the steatosis in the group of experimental animals Wistar male rats with obesity model after 4 weeks of treatment.

Groups	Steatosis score	*P* value	Inflamation Lobular	*P* value	Hepatocyte Balloning	*P* value
K0	2.5 (1–5)		2 (1–3)		0.5(0–1)	
K1	6.4 (5–7)		3 (2–3)		2 (2)	
K2	4 (3–5)	0.015*	2 (1–2)	0.037*	1 (0–2)	0.007*
P50	3.5 (2–4)		2 (1–2)		0 (0–1)	
P100	2.5 (1–6)		1.5(1–2)		0.5 (0–1)	
P150	4 (3–5)		2 (2–3)		1 (0–2)	

Kruskal Wallis test.

groups ($P < 0.005$). Subsequent analysis tests with the Mann-Whitney test found a significantly different steatosis score in the K1 control group as compared to all groups treated with *A. cordifolia* (P50, P100, P150; $P < 0.005$). Additionally, this study we found a notable distinction in NAS scores among the K0 and the HFD groups without treatment (K1; $P < 0.05$), and the notable distinction in NAS scores among the K1 and the K2 groups ($P < 0.05$).

This treatment of an ethanol extract of binahong leaves at all doses (P50, P100, P150) was found to reduce the severity of NASH to borderline levels based on NAS score compared to the HFD group without treatment (K1). Among the three treatment groups, the lowest NAS score after treatment was noticed in the groupings intervention by using 100 mg/kg BW (P100) dosage with *Anredera c* (Table 4, Fig. 2).

Immunohistochemical assessment of PPARγ expression in adipose tissue utilized the wider range, employing the Immunoreactive Score (IRS) category assessment. This assessment includes two indicators: the percentile score of cell distribution and the staining intensity score. The final IRS assessment is obtained by multiplying the percentage score of cell distribution with the staining intensity score.[17] The IRS category assessment is divided according to the absence of protein expression (0), low protein expression (0–3), medium protein expression (4–8), and high protein expression (9–12).

The Kruskal-Wallis test revealed the noticeable distinction in IRS for PPARγ immunohistochemical staining results among groups ($p < 0.05$). Subsequent analysis using the Mann-Whitney test identified the noticeable distinction in PPARγ IRS among the standard diet groups (K0) and the orlistat-treated HFD control group (K2) as well as intervention with all doses (50,100, 150; $p < 0.05$). The noticeable distinction in PPARγ IRS was also identified when comparing the untreated HFD groups (K1) with all intervention with all doses (50, 100,150; $p < 0.05$). Furthermore, the study found a noticeable distinction in PPARγ IRS among the K2 control groups and the higher-dose at the groups receiving treatment (P100, P150). Finally, significant differences were found among the groups with using 50 mg/kg BW dosage and both using dose 100 mg/kgBW dosage and 150 mg/kg BW ($p < 0.05$).

In the standard diet group (K0), all samples exhibited an IRS value of 9, indicating high PPARγ protein expression. In the K1 group, an average IRS value of 6 (range 6–9) was found, revealing that PPARγ expression in these samples is moderate to high. The HFD control group treated with orlistat (K2) showed protein expression levels with scores ranging from 3–6, indicating that the samples in this group express low to moderate levels of PPARγ. Across all groups treated with A. *cordifolia* extract (P50, P100, P150), low PPARγ protein expression was detected in Table 5.

A significant difference in total steatosis score was observed between the control and treatment groups.

Discussion

The utilization of ethanol extract from binahong leaves (*Anredera c*) as an anti-obesity agent has demonstrated efficacy in reducing body weight

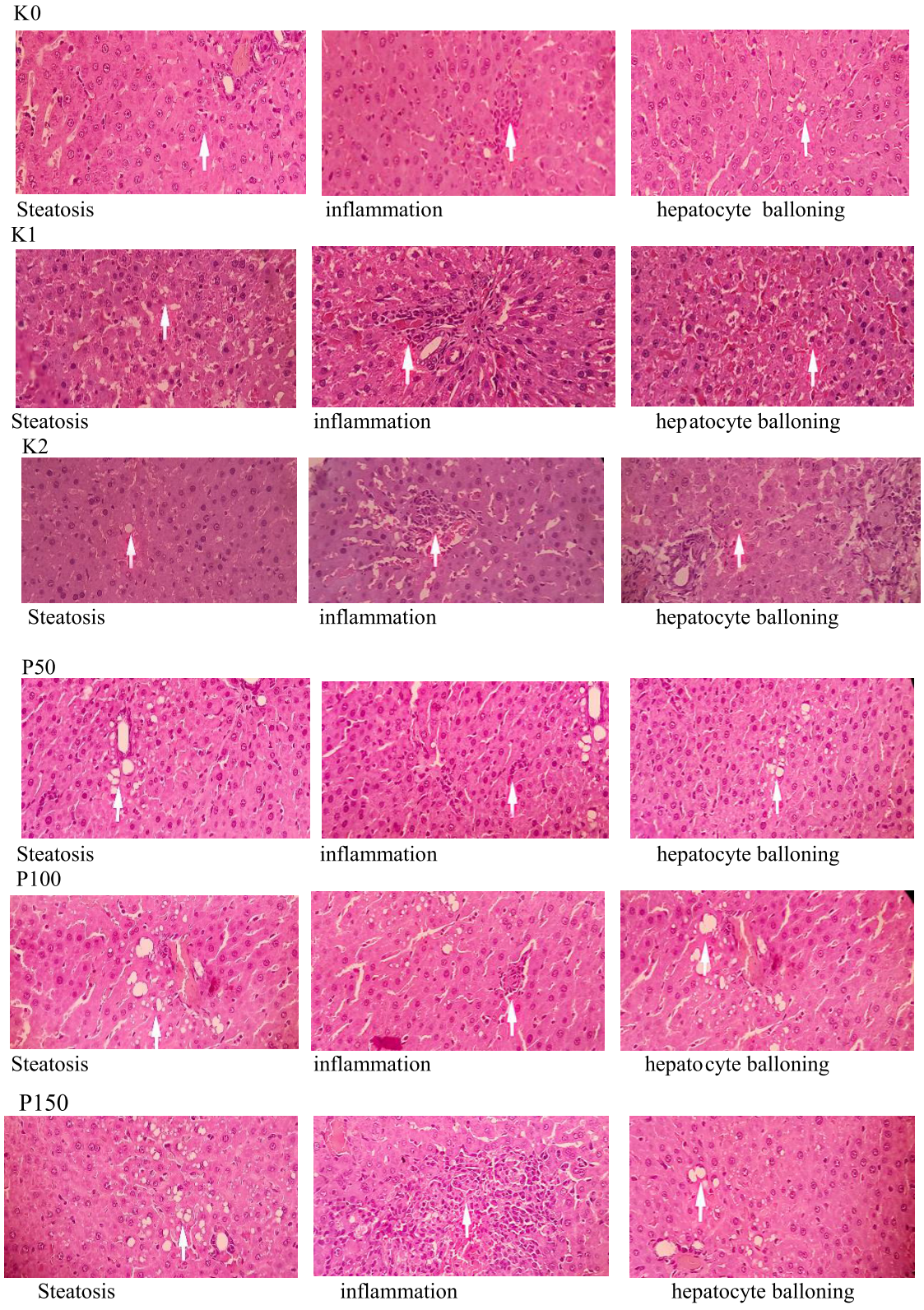

Fig. 2. The image of liver cells with Hematoxylin-Eosin staining shows steatosis, inflammation and hepatocyte ballooning in the control and treatment groups.

Table 5. Immunoreactive PPPAR γ expression score from immunohistochemical staining.

			Post hoc				
Groups	Total Score IRS PPARγ	*P* value	K1	K2	P50	P100	P150
K0	9		0.126	0.004**	0.004**	0.002**	0.002**
K1	6 (6–9)			0.056	0.008**	0.004**	0.004**
K2	3 (3–6)	0.000*			0.310	0.009**	0.017**
P50	3					0.017**	0.004**
P100	2 (2–3)						0.699
P150	2						

Kruskal wallis test, mann whitney analysis.

across all dosages (50 mg/kg, 100 mg/kg, and 150 mg/kg BW) over a 4-week treatment period. However, previous research suggests that the use of a dose of 100 mg/kg BW is particularly effective in reducing body weight.[18]

The research also discovered that the ethanol extract of *Anredera c* at 50 mg/kg BW dosage and 100 mg/kg BW created a decrease significantly in AC ($P < 0.05$). In contrast, 150 mg/kg BW dosage showed no significant reduce statistically in AC ($P > 0.05$), and the K2 control group did not exhibit a notable reduction in AC ($P > 0.05$). The administration of *Anredera. c* extract reduced body weight while also efficiently decreasing AC, as evidenced by statistically significant results. This indicates that anthropometric measurements of AC strongly correlate with fat deposition.[19,20]

Previous research indicates that flavonoids reduce free fatty acid levels and increase glucose consumption in a dose-dependent manner.[18] Furthermore, flavonoid therapy has been explored in the context of NAFLD.[21] This study did not examine the secondary metabolite vitexin, which has a demonstrated role in weight loss and significantly inhibits fat accumulation in 3T3-L1 adipocytes, potentially preventing obesity or adipogenesis induced by HFD.[20] The percentage of vitexin content in *A. cordifolia* ethanol extract varies depending on the extraction method used, with the partial least squares method yielding a smaller vitexin content (5.603%) compared to the principal component regression method (6.917%).[21]

In obesity, fatty liver is induced by the accumulating of fat contained in tissues of fat, and the occurrence of fatty liver (steatosis) can be stimulated by eating habits in rich-fat.[22,23] This research found that the untreated HFD control group (K2) met the diagnostic requirements for NASH based on the NAS score. The standard diet group showed a lower NAS score, which is in accordance with prior research that suggests that the percentage of hepatic cells experiencing fatty liver in rats' obesity being triggered by a high-fat diet is significantly higher compare those induced by a standard diet.[24] The utilization of *Anredera. c* extract in the treatment group reduced the NAS score or fatty liver (steatosis) across all doses (50 mg/kg BW, 100 mg/kg BW, and 150 mg/kg BW), although treatment using by 100 mg/kg BW showed the lowest NAS score compared to the other doses. The decrease in body weight was also associated with a decrease in the percentage of steatosis.[25]

Peroxisome proliferated activated regulator γ is a transcription factor that is crucial in the process of adipogenesis and adipocyte gene expression.[25,26] PPARγ is present in an abundance of tissues, but its expression is particularly high in fat tissue.[27] PPARγ facilitates the conversion of preadipocytes in adipose tissue into adipocyte cells and promotes the migration of circulating progenitor cells from bone marrow into white adipose tissue, where they subsequently separate into adipocyte cells.[28] Therefore, it significantly affects a range of metabolic imbalances.[28,29] PPARγ contributed to the growth of NASH by promoting processes such as adipogenesis, insulin resistance, inflammation, oxidative stress, endoplasmic reticulum stress, and fibrosis.[30]

This study demonstrated a notable disparity in the IRS of PPARγ expression between the treatment groups and the control groups ($P < 0.05$). There was a decrease in PPARγ expression in using all doses (50,100, 150) mg/kgBW and an increase in PPARγ expression in the untreated K0 and K1 groups. This indicates that the utilization of the extract of *Anredera c* can decrease the expression of PPARγ, one crucial element implicated in the differentiation and proliferation of adipose tissue.[31,32] Further, the untreated HFD group (K1) displayed the highest levels of steatosis, lobular inflammation, and hepatocyte ballooning scores compared to other control groups, demonstrating that high-fat diets significantly contributes to the occurrence of NAFLD. Although obesity can be induced by a standard diet, the use of binahong leaves effectively reduces fatty liver at all doses, with 100 mg/kg BW showing the least steatosis score. Therefore, this study suggests that binahong leaves can reduce steatosis, followed by a decreased of the PPARγ expression.

Conclusion

The anti-obesity effect of *A. cordifolia* ethanol extract contributes to reducing abdominal circumference and adipose tissue in the abdomen of Wistar rats, and significantly reduces fatty liver (steatosis), potentially by decreasing PPARγ expression. Future pre-clinical investigation into mechanisms underlying the effect of *Anredera cordifolia* ethanol extract on obesity and eventual clinical studies may provide evidence for its efficacy as a therapeutic agent for combatting obesity-related conditions in humans.

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Author's declaration

- No conflicts of interest.
- We are pleased to inform you that all of the figures and tables in the manuscript are our own. Furthermore, the manuscript contains any figures and images that are not our own, and the requisite permission for re-publication is included.
- The author has endorsed an animal welfare declaration.
- Ethical Clearance: The project was approved by the local ethical committee at Medical Faculty, Universitas Sumatera Utara approved the experimental protocols of this study (approval No. 726/KEP/USU/2023).

Authors' contribution

The manuscript was done by the cooperation by the seven authors. Conceptualization: R R; Design: T. W; acquisition of data analysis: D. K. S; Interpretation: S. SW; Drafting MS: R. R; Revision and proof reading: R. R and DRA.

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الخواص المضادة للسمنة لمستخلص نبات البينهونج (Anredera cordifolia) على الكبد الدهني في نموذج فئران ويستار

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الخلاصة

ترتبط السمنة، التي تتميز بتراكم الخلايا الدهنية، بمضاعفات مثل الكبد الدهني والتكلس غير الكحولي، والتي يمكن أن تكون قاتلة. يبحث هذا البحث في خصائص استخدام مستخلص الإيثانول من نبات البينهونج في تقليل التكلس من خلال التحليل المجهري لدرجات نشاط مرض الكبد الدهني غير الكحولي NASوالفحص المناعي الكيميائي IRSلتعبير PPARγ في الخلايا الكبدية والأنسجة الدهنية البطنية في فئران ويستار السمينة. لوحظت فروق كبيرة في درجات التكلس الكلي بين المجموعات (p < 0.005)، مع إظهار المجموعة الضابطة K1درجات أعلى بشكل ملحوظ مقارنة بجميع مجموعات العلاج (P50، P100، P150؛ P < 0.005). بالإضافة إلى ذلك، تم العثور على فروق كبيرة في درجات NAS بين مجموعتي K1 و K2 (P < 0.05)، وكذلك بين مجموعتي K0 و K1 (P < 0.05). لوحظ اختلاف كبير في IRS PPARγ بين مجموعتي K0 وK2 وجميع مجموعات العلاج (P50، P100، P150؛ P < 0.05). وبالمثل، لوحظ اختلاف كبير بين مجموعة K2 وجميع مجموعات العلاج ((P < 0.05، وكذلك بين مجموعة K1 ومجموعة العلاج بجرعات أعلى (P100، P150). وبخلاف تقليل محيط البطن والأنسجة الدهنية في البطن، كان التأثير المضاد للسمنة لمستخلص نبات البينهونج واضحًا في تقليل دهون الكبد، والذي قد يحدث من خلال التنظيم السلبي لتعبير PPARγ

الكلمات المفتاحية : نبات البينهونج Anredera cordifolia ، درجة NAS، السمنة، PPAR γ، دهون الكبد