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Exploring Aloe *vera*'s Lipid-Lowering Potential: Regulation of HDL-Cholesterol and Associated Proteins

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Abstract

Introduction: Obesity caused by excessive dietary fat intake is associated with abnormal lipid accumulation and metabolic disturbances. This study evaluated the anti-obesity effects of aqueous extracts of *Aloe vera* in female Swiss albino mice.

Methods: Female Swiss albino mice were maintained under controlled laboratory conditions and divided into four groups (n = 6 per group). Group I served as the normal control. Group II received a high-fat diet (35% carbohydrate, 20% protein, and 45% fat) for 60 days to induce obesity. Group III consisted of obese mice treated orally with *Aloe vera* extract (0.3 mL/kg body weight) for 30 days, while Group IV included normal mice treated with *Aloe vera* extract for 30 days. Body weight, adipose tissue lipid deposition (Oil Red O staining), and HDL-associated protein expression (SDS-PAGE) were evaluated.

Results: *Aloe vera* treatment significantly reduced body weight and lipid accumulation in adipose tissue of obese mice. Histological analysis demonstrated reduced intracellular lipid deposition following treatment. In addition, HDL-associated protein expression was enhanced in treated obese mice compared with untreated obese animals, suggesting improved lipid transport and metabolism.

Conclusion: *Aloe vera* administration reduced body weight, decreased adipose tissue lipid accumulation, and enhanced HDL-associated protein expression in diet-induced obese mice. These findings support the potential of *Aloe vera* as a complementary therapeutic agent for improving lipid metabolism and managing obesity-associated metabolic disturbances.

Keywords: Aloe, Mice, Obesity, Adipocytes, Lipid metabolism, Cholesterol

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Introduction

Aloe *vera* is widely incorporated into commercial health products, nutraceutical formulations, and traditional remedies owing to its wide medicinal values (1, 2). It has been reported to exert multiple systemic benefits, including improvements in lipid balance, glycemic regulation, oxidative status, inflammation, skin health, and general metabolic well-being (3-5). Much of the existing research, however, has emphasized its influence on circulating lipid parameters—such as reductions in total cholesterol, triglycerides, and LDL—without fully examining how Aloe *vera* interacts with lipid-handling pathways within specific metabolic tissues (6-8).

Obesity is characterized by excessive fat accumulation resulting from disturbances in adipocyte lipid turnover. Dysregulated lipolysis and lipogenesis in adipose tissue contribute directly to metabolic dysfunction. Although *Aloe vera* has been studied for its hypolipidemic effects, evidence describing its role in modulating lipid breakdown at the adipocyte level is limited. This represents a significant gap, as adipocytes are the primary site for lipid deposition and serve as a key regulator of whole-body metabolic homeostasis through tightly coordinated enzymatic and hormonal pathways (9).

HDL-cholesterol is vital to maintain lipid balance by accepting cholesterol and other lipids from peripheral tissues, including adipose depots, and transporting them to the liver for clearance through reverse cholesterol transport. Increasing attention has shifted from total HDL levels toward the protein composition of HDL, as alterations in its protein cargo can modify its functional properties and cardiometabolic benefits. Despite substantial literature on Aloe *vera's* systemic lipid-lowering effects, very few studies have examined whether it influences HDL at the molecular or protein level (10-12).

To address these understudied areas, the present work

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investigated two mechanistic aspects of *Aloe vera's* metabolic action: (i) its ability to alter lipid mobilization within adipose tissue, assessed through Oil Red O staining as an indicator of changes in lipid content and lipolysis, and (ii) its effect on the expression of HDL-associated proteins, evaluated using SDS–PAGE to detect shifts in protein composition.

By coupling tissue-level assessment of adipocyte lipid breakdown with protein-level analysis of HDL, this study provides a more detailed understanding of how *Aloe vera* may influence lipid metabolism beyond conventional serum lipid measurements. These findings offer preliminary mechanistic insight into *Aloe vera's* potential utility as a complementary intervention for obesity and related metabolic disturbances.

Methods

Experimental Animals and Study Design

This study was carried out in 3-month-old female Swiss albino mice procured from registered breeders. Animals were housed under standard laboratory conditions in propylene cages at a temperature of 25°C and 55% humidity. Standard pellet feed and water were supplied freely throughout the experiment ad libitum.

After one week of acclimatization, animals were classified into 4 groups containing 6 mice each. Group I consists of control animals under regular laboratory diet. Group II animals were induced with obesity through a high-fat diet containing 35% carbohydrate, 20% protein, and 45% fat for 60 days. Group III consisted of obese mice that subsequently received oral Aloe vera administration at a dose of 0.3 mL/kg body weight for 30 days. Group IV included non-obese mice maintained on a standard diet and treated with the same dosage of the preparation.

Body weight was recorded periodically during the experimental period to monitor obesity progression and treatment-associated changes. At the end of the study, animals underwent overnight fasting for approximately 12 h before sample collection. Anaesthesia was induced using isoflurane (5 mg/kg body weight), and blood samples were collected; the serum was collected and stored at −80°C until further analysis.

This study was carried out after obtaining approval from the Institutional Animal Ethics Committee, Coimbatore Medical College and Hospital, in accordance with the CPCSEA recommendations (Ethical Approval Code: CMCH/PH/004/2017).

Aloe vera Extract

Fresh mature Aloe vera leaves were washed thoroughly using distilled water. The outer rind was carefully removed, and the inner gel was collected. The gel material was homogenized and subjected to aqueous extraction for 48 h at room temperature under continuous stirring. The extract was filtered through Whatman No.1 filter paper and concentrated using a rotary evaporator maintained at 40–45°C to obtain a semi-solid preparation. The final extract was stored at 4°C until use.

The extract contains several naturally occurring phytochemicals including flavonoids, polysaccharides, anthraquinones, and plant sterols such as β-sitosterol. Earlier reports have associated these phytochemicals with wide anti-obesity properties (13,14).

Analysis of HDL-Associated Protein Expression by SDS-PAGE

Bradford assay was done to estimate the serum protein concentration in the samples under study. 20 µg of protein was then run on SDS-polyacrylamide gel electrophoresis using a 10% resolving gel at 50 V. 70 kDa molecular weight marker obtained from Genei Laboratories, Bangalore, served as a protein ladder. Following electrophoretic separation, gels were stained using Coomassie Brilliant Blue R-250 and destained with a methanol-acetic acid-water solution. Protein bands were visualized and documented for comparative evaluation of expression patterns.

Oil Red O Staining of Adipose Tissue

White adipose tissue samples were excised, washed in chilled phosphate-buffered saline (PBS), and rapidly preserved in liquid nitrogen. The frozen tissues were subsequently sectioned in a cryostat with 10 µm thickness and carefully mounted onto clean glass slides. Fixation was done using cold acetone for 10 min, washed with water, and treated with propylene glycol in two successive steps. Slides were stained with Oil Red O for 7 min and rinsed using 85% propylene glycol followed by distilled water. Hematoxylin counterstaining was carried out for 1 min before mounting with glycerin jelly. Tissue sections were examined microscopically to evaluate intracellular lipid deposition.

All samples were processed simultaneously using identical staining reagents and uniform handling conditions to minimize technical variability.

Statistical Analysis

Statistical analysis was carried out using GraphPad Prism software. Data obtained from baseline and final observation periods were expressed as mean ± standard deviation (SD). Since the measurements were recorded from the same experimental animals at two different time intervals, a paired Student's t-test was applied to determine the statistical significance within each group. Differences were considered statistically significant at $P < 0.05$.

Results

Evaluation of Aloe vera Treatment on Body Weight in Obese Mice

Body weight variations recorded during the experimental period are presented in Table 1. Animals maintained on the normal laboratory diet (Group I) showed no significant alteration in body mass throughout the study. Similarly, administration of the plant preparation to non-obese mice (Group IV) did not produce measurable changes in weight.

Mice receiving the high-fat diet exhibited a marked

Table 1. Weight-reducing potential of Aloe vera across the study groups

Body weight (in grams)	GROUP			
	I	II	III	IV
Baseline Observation	26.7 ± 1.8	30.2 ± 1.4	33.1 ± 0.8	29.1 ± 1.5
Final Observation	27.5 ± 0.09	34.2 ± 1.2	29.6 ± 1.6	28.2±0.9
P value	0.5^{NS}	0.02^{*}	0.03^{*}	0.4^{NS}

increase in body mass, confirming successful induction of obesity. A significant rise in weight was observed in Group II animals ($P = 0.02$). In contrast, obese mice under Aloe vera treatment showed a reduction in body weight following intervention. The significant decrease from 33.1 ± 0.8 to 29.6 ± 1.6 g indicates that the treatment may counter excessive weight gain associated with high-fat feeding ($P = 0.03$).

The absence of major weight reduction in normal animals suggests that the treatment primarily influences obesity-associated metabolic alterations rather than normal physiological growth.

Values are presented as mean ± standard deviation (SD) (n = 6 animals per group). Statistical analysis was performed using the paired Student›s t-test to compare baseline and final body weights within each group. A P value < 0.05 was considered statistically significant. Abbreviations: NS, not significant; SD, standard deviation.

Analysis of HDL-Associated Protein:

Serum protein patterns obtained from different groups were analyzed using SDS-PAGE. Figure 1 illustrates the electrophoretic profile of serum samples from all experimental groups. Lane 1 corresponds to the protein ladder, whereas lanes 2–5 represent Groups I–IV, respectively.

Band intensity varied among the groups, reflecting differences in the HDL-associated protein expression. Obese animals receiving the high-fat diet demonstrated altered protein profiles when compared with control mice. Following treatment, improved protein expression patterns were observed in obese animals, indicating a possible beneficial effect on lipid transport and lipoprotein regulation.

Effect on Adipose Tissue Lipid Deposition

Oil Red O staining revealed distinct differences in adipose tissue lipid storage among the groups. Tissue sections from normal mice (Figure 2A) displayed normal cellular appearance with minimal lipid accumulation and relatively lighter staining.

In obese animals receiving the high-fat diet (Figure 2B), enlarged adipocytes with intense staining were evident, indicating increased intracellular lipid storage. Peripheral displacement of nuclei was also observed, consistent with adipocyte hypertrophy.

Sections obtained from treated obese mice (Figure 2C) exhibited comparatively lighter staining than untreated obese animals, suggesting reduced lipid deposition following treatment. Cellular morphology remained largely similar, although intracellular lipid content appeared diminished.

Non-obese treated mice (Figure 2D) also demonstrated reduced staining intensity relative to normal controls. Smaller adipocytes were more frequently observed in this group, indicating reduced lipid storage and possible suppression of adipocyte maturation.

Overall, the histological findings indicate reduced lipid accumulation following treatment. Since all tissue sections were processed under identical experimental conditions, differences in staining intensity are considered to represent true biological variation.

Discussion

The present study evaluated the influence of Aloe vera administration on body weight regulation, adipose tissue lipid storage, and HDL-associated protein expression in mice with diet-induced obesity. The findings demonstrated that treatment decreased body weight, reduced lipid deposition in adipocytes, and enhanced HDL expression in obese animals. These observations suggest that the plant-derived preparation may beneficially influence lipid metabolism under obese conditions (15).

The high-fat diet increased the body weight of group II mice and enhanced adipocyte lipid accumulation, confirming successful induction of obesity in the experimental model. Enlarged adipocytes and intense Oil Red O staining observed in untreated obese animals indicate excessive intracellular lipid storage, which is characteristic of obesity-associated metabolic imbalance. Following treatment, obese mice exhibited reduced staining intensity and lower body weight, indicating diminished lipid deposition and possible enhancement of lipid mobilization.

Previous investigations involving *Aloe vera* have mainly focused on its potential to regulate serum lipid profile. The present study extends these findings by examining HDL-associated protein expression at the molecular level. Increased HDL-related protein expression observed after treatment may indicate improved lipoprotein metabolism and enhanced lipid transport mechanisms (16-20).

The biological activity observed in this study may be associated with phytoconstituents naturally present in *Aloe vera*, including flavonoids, plant sterols, anthraquinones, and polysaccharides. These phytochemicals have been reported to possess antioxidant, anti-inflammatory, and lipid-regulating properties (21). Plant sterols such as β-sitosterol may facilitate cholesterol handling and support HDL-mediated transport, whereas flavonoids may protect lipoproteins from oxidative damage and preserve their

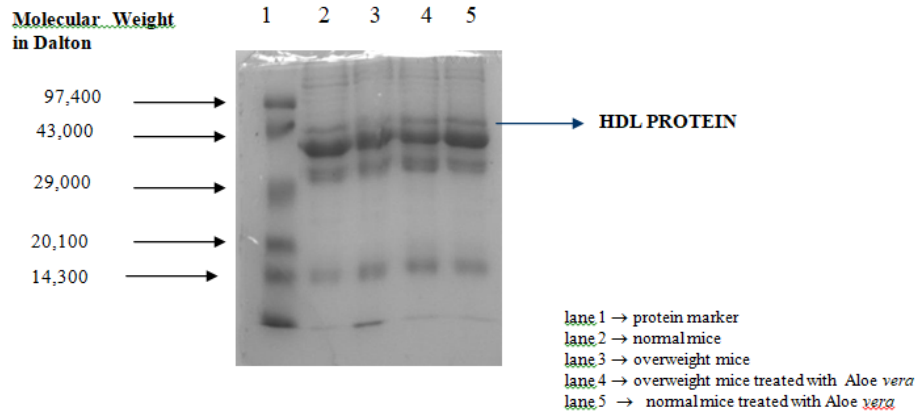


Figure 1. HDL expression in mice under study

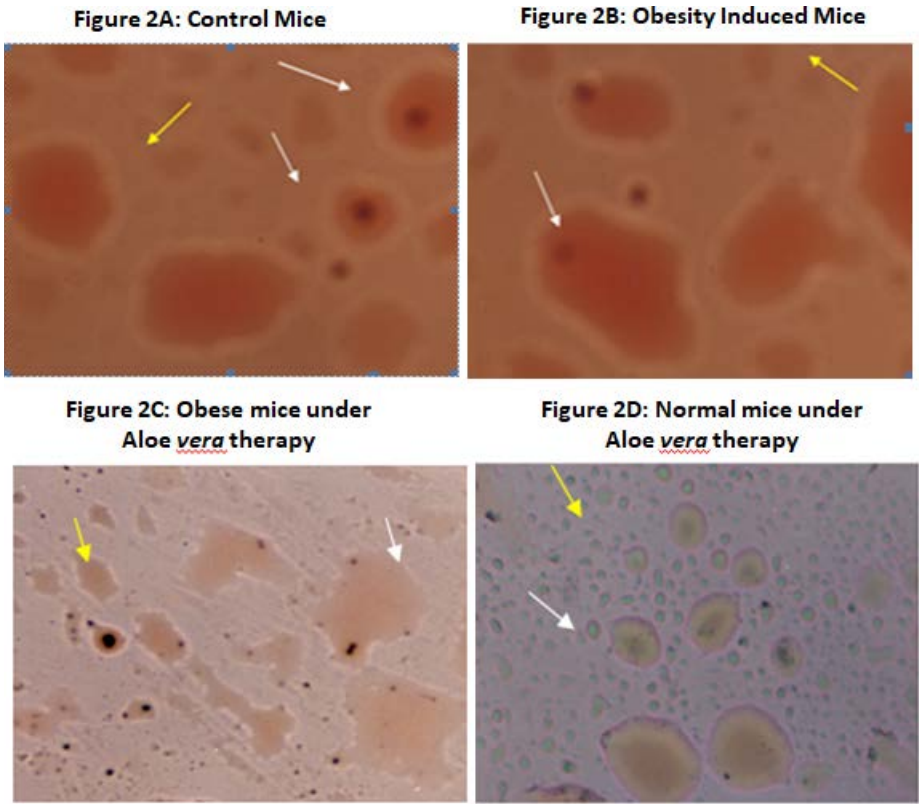


Figure 2 A-D. Microscopic visualization of lipid accumulation in adipose tissue sections following Oil Red O staining in experimental and treatment groups. Yellow arrows indicate mature adipocytes, whereas white arrows indicate immature adipocytes.

structural integrity.

Several mechanisms may explain the observed effects. Bioactive constituents present in the extract may influence hepatic lipid metabolism and contribute to improved apolipoprotein synthesis. In addition, enhanced mobilization of lipids from adipose tissue may promote reverse cholesterol transport pathways. Antioxidant activity may also help preserve HDL-associated proteins from oxidative modification, thereby maintaining their functional properties.

The adipose tissue findings further support the anti-obesity potential of the treatment. Reduced staining intensity in treated groups indicates decreased intracellular lipid accumulation. The presence of smaller adipocytes in treated animals may additionally suggest suppression of adipocyte enlargement and altered lipid storage dynamics.

The present findings may hold importance in the context of obesity-associated dyslipidaemia and cardiovascular complications. Since HDL functionality is closely associated with lipid transport and cardiometabolic health, enhancement of HDL-associated protein expression may provide additional protective effects beyond conventional lipid lowering.

Certain limitations should be considered while interpreting the results. The study evaluated overall HDL-associated protein expression using SDS-PAGE without quantification of individual apolipoproteins. Serum HDL-cholesterol concentration was also not directly measured. Furthermore, the phytochemical constituents of the extract were not quantitatively characterized. Functional studies evaluating HDL activity were not performed, and the observations were limited to an experimental animal

model.

Future studies should focus on detailed phytochemical profiling, quantitative analysis of specific HDL-associated proteins, and assessment of HDL functional activity. Application of advanced molecular techniques such as Western blotting or ELISA may provide greater specificity regarding individual protein alterations. Clinical investigations may further clarify the translational relevance of these findings in obesity and metabolic disorders.

Conclusion

The present study demonstrated that Aloe vera administration decreased body weight, reduced adipose tissue lipid deposition, and enhanced HDL-associated protein expression in obesity induced mice. These observations indicate that the treatment may beneficially influence lipid metabolism and adipose tissue lipid handling. The findings support the potential role of Aloe vera as a complementary therapeutic candidate in obesity-associated metabolic disturbances and warrant further mechanistic and clinical evaluation.

Authors' Contribution

Conceptualization: Nalini Devarajan, Ramalingam Mathangi
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Project Administration: Nalini Devarajan
Resources: Karthick Rajendran, E. Susithra, Ramalingam Mathangi
Software: Karthick Rajendran
Supervision: Ramalingam Mathangi
Validation: Nalini Devarajan, Ramalingam Mathangi
Visualization: Nalini Devarajan, Pavithra Amritkumar
Writing–Original Draft: Nalini Devarajan
Writing–Review & Editing: Nalini Devarajan, Ramalingam Mathangi, Karthick Rajendran, E. Susithra, Pavithra Amritkumar

Competing Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethical Approval

The study was approved by the Ethics Committee of Coimbatore Medical College and Hospital (Ethical code: CMCH/PH/004/2017).

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