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Effect of Dietary Addition of 2% *Aloe vera* gel on Serum Lipids and Body Fats Accumulation in Rats

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DEDICATION

I dedicate my dissertation work to my family and my friends.

A special feeling of gratitude to my loving parents, for their continual faithful support during my study.

Thanks and gratitude are also extended, to my brothers, my lovely sisters

I would like to appreciate Kismoun, R., for his kindness and his precious advice as well as his great support.

I also pay thanks to my best friends; Berriche, B., Betchine, A., Beziane, N., and specially to Chebel, B.

Finally, I pray that Allah accept this effort and make it of real benefit to all who read it.

Zeineb

The background of the page is a watercolor-style illustration of pink cherry blossoms. The flowers are in various stages of bloom, with some fully open and others as buds. The colors range from light pink to deep magenta. The blossoms are scattered across the page, with some branches visible. The overall aesthetic is soft and delicate.

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Thanks and gratitude are also extended, to my brothers, Mahdi and Borhane, and to my sister Marwa.

I also pay thankfulness to my best friends, Bahri, N., Chebel, B.

Finally, I pray that Allah accept this effort and make it of real benefit to all who read it.

Safa

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ABREVIATIONS

ADP: Adenosine diphosphate

ATP: Adenosine triphosphate

AP: Aminophenazone

CHE: Cholesterol esters

CHOD: Cholesterol oxidase

CMG: Chios mastic gum

DAP: Dihydroxyacetone phosphate

GAL: Galactomannans

GOD: Glucose oxidase

GPO: Glycerol phosphate dehydrogenase

GK: Glycerol kinase

G3P: Glycerol-3-phosphate

HDL: High-density lipoproteins

H₂O₂: Hydrogen peroxide

LDL: Low density lipoprotein

LPL: Lipoproteinlipase

POD: Peroxidase

GD6P :Glucose dehydrogenase 6 phosphate

FAS : Fatty acid synthase

AE : Aloin-Emodin

VEGF : Vascular endothelial growth factor

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INTRODUCTION

INTRODUCTION

Introduction

Aloe vera is of uncertain origin but cultivated long standing Mediterranean region, North Africa, the Canary Islands and Cape Verde, known since ancient times but mostly used since the first century, *Aloe vera* has been adopted in many traditional medicines warm regions of the world, Europe, the Middle East and North Africa first, then India, China and Asia mostly after the tenth century and America after the seventeenth century. Currently, *Aloe vera* gel comes mainly in the composition of cosmetics or beverages (Bruneton, *et al.*, 2009). Among the 300 varieties of Aloe plants known, there are only 3 or 4 types of Aloe which are used for cosmetic and medicinal purposes. The most powerful of all these varieties is *Aloe Barbadenis Miller*. This is the only deserves the name of *Aloe vera* (Rajeswari,*et al.*, 2012).

Aloe vera is a species of Aloe (Aloe genus) that has been used for thousands of years to heal a variety of conditions, most notably burns, wounds, skin irritations, constipation, ulcers, Diabetes, Cancer, headaches, arthritis, immune-system deficiencies, and many other conditions (Gauri,*et al.*, 2011). It is incredible natural remedy that has internal and external applications, with hundreds of active components that benefit the body; it is grown in subtropical locations. Aloe was one of the most frequently prescribed medicines throughout most of the 18th and 19th centuries (Rajeswari,*et al.*, 2012).

The invaluable oligoelements present in Aloe juice, manganese and selenium, constitute the enzymes superoxide dismutase and glutathione peroxydase, recognized as powerful antioxidants and cellular anti-aging agents (bassetti, *et al.*, 2005). Their high antioxidant properties slow down the aging process. This helps cells to become stronger in combating the negative effects caused by oxygen and the broad spectrum radiation to which human skin is exposed daily. The non essential amino acid, proline, is a constituent of collagen. Whose role is to insure the perfect holding capacity and elasticity of epithelial tissues. This leads to skin becoming smoother, hydrated and more elastic. Protected from free radicals and their degenerative activity resulting in substantial anti-aging effects by constant use of Aloe (Gamboa-Gomez,*et al.*, 2015).

Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health; Obesity is the most prevalent nutritional disease and a growing public health problem worldwide. This disease is a causal component of the metabolic syndrome related with abnormalities, including hyperglycemia, dyslipidemia, hypertension, inflammation, among others (Grundy,*et al.*, 2004). There are anti-obesity drugs, affecting the fundamental

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processes of the weight regulation; however they have shown serious side effects, which outweigh their beneficial effects. Most recent studies on the treatment of obesity and its complications have focused on the potential role of different plants preparation that can exert a positive effect on the mechanisms involved in this pathology (Claudia, *et al.*, 2015).

Aloe vera have been found to be effective in the treatment of cardio vascular disease, improving blood glucose levels in diabetics (Bunyapraphat, *et al.*, 1996), lowering blood lipids in hyperlipidemic patients (Rajasekaran, *et al.*, 2006), acute hepatic diseases (Bottenberg, *et al.*, 2007), high blood pressure (Hossain, *et al.*, 2013), it was also used for its anti-inflammatory, and antitumoral effects (Reynolds, *et al.*, 1999; Chithra, *et al.*, 1998). Obesity is an established risk factor for type 2 diabetes and cardiovascular diseases, where the central and causal component is the metabolic syndrome (Montague, *et al.*, 2000). This is a series of metabolic abnormalities including hyperglycemia, dyslipidemia, hypertension, inflammation, oxidative stress, among others (Grundy, *et al.*, 2004). Although the association of cardio vascular disease, hyperlipidemia, hypertension and diabetes with obesity suggests a possible role of *Aloe vera* in the regulation of serum lipids and obesity. Little is known about the effect of *Aloe vera* gel on body fat accumulation. Taking all in consideration, it is of interest to conduct the effect of *Aloe vera* gel on obesity.

This study was conducted for the first time to elucidate the potential role of *Aloe vera* gel on serum lipids and on body fat accumulation in rats. Here we describe the first evidence that feeding 2% of *Aloe vera* gel causes hypotriglycerimia and low body fat accumulation.

BRIEF REVIEW
ON
ALOE VERA

BRIEF REVIEW ON ALOE VERA

Brief Review on *Aloe vera*

1.1 *Aloe vera*

1.1.1 Definition

Aloe vera is a succulent plant. Succulents are xerophytes, which are adapted to living in areas of low water availability and are characterized by possessing a large water storage tissue (Ni, *et al.*, 2004).

1.1.2 Taxonomical classification of *Aloe vera*

Aloe vera belongs to the family Aloaceae, and it is widely used in the manufacturing of food and drink products, pharmaceuticals, and cosmetics (Reynolds, *et al.*, 1999).

Kingdom : Plantae

Subkingdom : Tracheobionta

Superdivision : Spermatophyta

Division : Magnoliophyta

Classe : Liliopsida

Subclass : Liliidae

Order : Liliales

Family : Aloaceae

Genus : Aloe

Species : *Aloe vera* (L.) Burm. f. (Botanical Journal of the Linnean Society, 2009).

1.1.3 Botanical Description

The botanical name of *Aloe vera* is *Aloe Barbadensis* Miller. It belongs to Asphodelaceae (Aloaceae) family, and is a shrubby or arborescent, perennial, xerophytic, succulent, pea-green color plant. It grows mainly in the dry regions of Africa, Asia, Europe and America. The plant has triangular, fleshy leaves that can reach up to 80 cm long and 10 cm wide at the base in the adult plant with serrated edges, yellow tubular flowers and fruits that contain numerous seeds (Fig. 1-2).

Each leaf is composed of three layers:

BRIEF REVIW ON ALOE VERA

- 1- An inner clear gel that contains 99% water and rest is made of glucomannans, amino acids, lipids, sterols and vitamins.
- 2- The middle layer of latex which is the bitter yellow sap and contains anthraquinones and glycosides.
- 3-The outer thick layer of 15-20 cells called as rind which has protective function and synthesizes carbohydrates and proteins. Inside the rind are vascular bundles responsible for transportation of substances such as water (xylem) and starch (phloem) (Surjushe,*et al.*, 2008).



Figure 1:*Aloe vera Barbadensis Miller* (Michayewicz, 2013).

BRIEF REVIW ON ALOE VERA

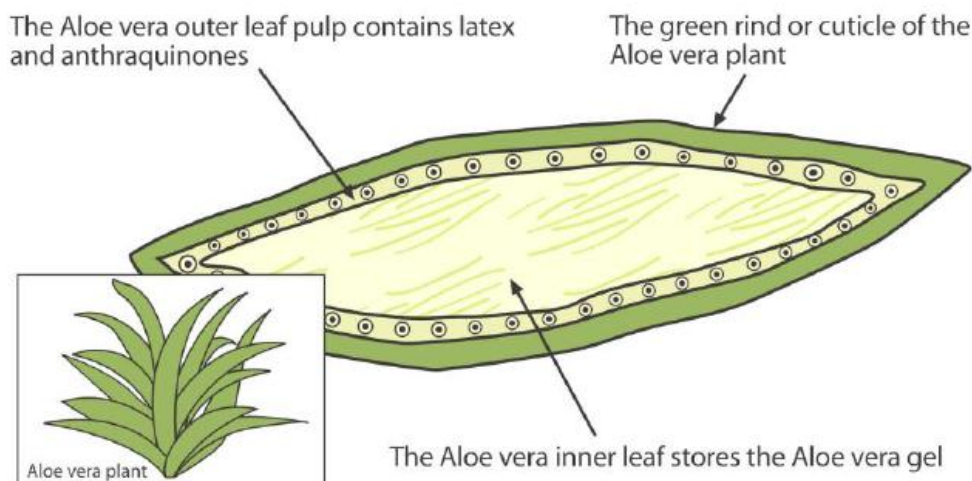


Figure 2: *Aloe vera* leaf cross section (Michayewicz, 2013).

1.1.4 *Aloe vera* Composition

Aloe vera is a plant of arid environments that stores water in its leaves. Also, it is the main component of the sheet and represents 98 to 99 % of its weight. The dry material which thus accounts for only 1 to 2%, consists of 60% polysaccharides (Josia, 2008; Emmanuel, 2008). *Aloe vera* leaf contains more than 75 active compounds polysaccharides, phenolic compounds, organic acids as well as 20 minerals, 20 amino acids and 12 vitamins. The main secondary metabolites are phenolic compound of anthrone type and chromone. But despite numerous studies, therapeutic activities have not been well correlated with the compounds.

- 1- The carbohydrate fraction consists of monosaccharides (carbohydrate, xylose ...), polysaccharides reserves (acemannan, aloeride, cellulose ...) stored in the protoplasm of cellules. Acemannan (Emmanuel, 2008).
- 2- The protein fraction is formed by amino acids, glycoproteins (alprogène, aloctin A and B, verectine).
- 3- The lipid fraction (5% of the dry weight of the pulp) is composed of sterols (cholesterol, campesterol, β -sitosterol, phytosterol), triterpenes (lupeol), triglycerides and phospholipids.
- 4- Overriding minerals are potassium, calcium, sodium, magnesium and phosphorus.
- 5- Key vitamins are vitamin C and vitamins B1, B2, B3 and B6.

BRIEF REVIEW ON ALOE VERA

- 6- Organic acids such as malic acid, succinic acid, uric, isovaleric, of phenol- acid such as cinnamic acid, vanillin, citric, férulique1 (Bruneton, *et al.*, 2009).
- 7- Anthraquinone (aloin, isobarbaloin, anthranol, aloe -emodin, emodin etc.). Aloin is located in the outer layer of the sheet and constitutes almost 30% of exudates (Emmanuel,2008).
- 8- Chromones: aloesone, aloeresin.
- 9- Saponins, phthalate esters, growth hormones.

1.1.4.1 Phytosterols

Phytosterols (lophenol and cycloartanol) derived from *Aleo vera* are the plant version of cholesterol. Animals primarily make cholesterol, and plants make a variety of phytosterols and stanols. While humans can make cholesterol or obtain it from the diet, we are unable to make any kind of phytosterol, and thus only get those from dietary sources. There are two main classes: sterols and stanols. Sterols have a double bond in their sterol ring, while stanols do not (Fig. 3).

Studies have found that consuming 1-2 grams each day of phytosterols can lower low-density lipoprotein (LDL) cholesterol by 6 to 10 %, which may reduce risk for coronary heart disease (Katan, *et al.*, 2003; Plat, *et al.*, 2005). Phytosterols work by reducing the amount of cholesterol that is absorbed in the small intestine. This includes cholesterol from dietary sources, as well as cholesterol from bile that would normally be reabsorbed and reused (Ostlund, 2002; Plat, *et al.*, 2005). The decrease in absorbed cholesterol upregulates LDL receptors, which in turn removes more LDL from circulation. It also causes an increase in endogenous production of cholesterol, although not enough to compensate for the increased blood clearance. The end result is lower LDL cholesterol (Ostlund, 2002; Plat, *et al.*, 2005). Administration of Lophenol and Cycloartanol also reduced the serum free fatty acid and triglyceride levels except total cholesterol . and weights of total abdominal fat tissues. (Eriko, M.,*et al.*, 2008)

BRIEF REVIW ON ALOE VERA

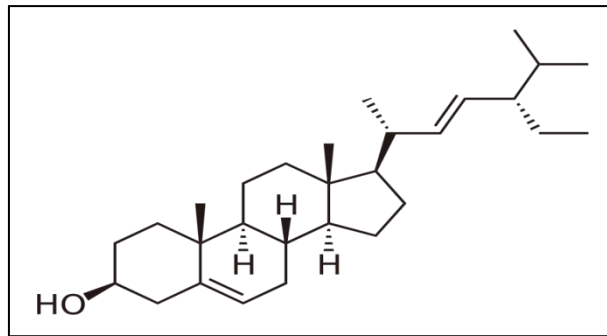


Figure: 3 Phytosterols (jonnes., 2011)

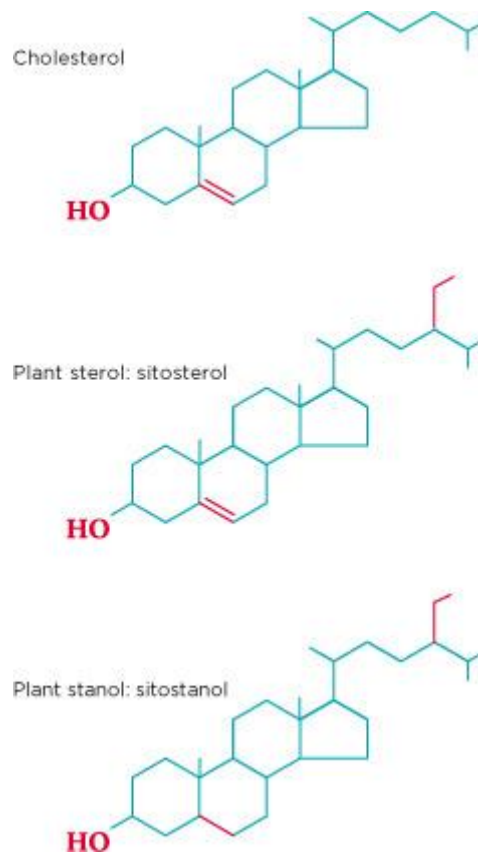


Figure:4 cholesterol, plant sterol, plant stanol

1.2 Pharmacological aspects and therapeutic effects of *Aloe vera* gel

It has been claimed that the polysaccharides in *Aloe vera* gel have therapeutic properties such as immunostimulation, anti-inflammatory effects, wound healing, promotion of radiation damage repair, anti-bacterial, anti-viral, anti-fungal, anti-diabetic and anti-neoplastic activities, and stimulation of hematopoiesis and anti-oxidant effects. (Ni, *et al.*, 2004; Reynolds,*et al.*, 1999). On the other hand, there are a number of clinical reports that have

BRIEF REVIEW ON ALOE VERA

found *Aloe vera* gel not effective in terms of the above mentioned therapeutic activities or even to cause undesirable effects such as retardation of wound healing. As mentioned before, these conflicting results could be due to the use of plants from different locations, with variations in their chemical composition and also because of different isolation techniques that were used to extract compounds from the Aloe leaf pulp (Femenia, *et al.*, 2003).

1.2.1 Anti-diabetic effects

Several pre-clinical and clinical trials showed a blood glucose lowering effect for *Aloe vera* gel preparations in different forms (e.g. juice or as constituents in bread etc.), while other studies indicated that no change in glucose levels could be obtained. The differences in results of these in vivo studies can possibly be explained by differences in the way that the Aloe mucilaginous gel was isolated and separated from the exudates anthraquinones. Furthermore, it is not always clear what constituent of the Aloe leaf was tested in some studies, which makes it difficult to correlate the effect (or lack of effect) with the product tested (Reynolds, *et al.*, 1999). In a study on streptozotocin-induced diabetic rats, oral administration of *Aloe vera* gel (alcoholinsoluble residue extract) significantly reduced the fasting blood glucose, hepatic transaminases, plasma and tissue cholesterol, triglycerides, free fatty acids and phospholipids and in addition also significantly increased plasma insulin levels. The decreased plasma levels of high density lipoprotein cholesterol and increased levels of low density lipoprotein cholesterol in the streptozotocin-induced rats were restored to normal after treatment with gel extract (Rajasekaran, *et al.*, 2006).

1.2.2 Anti-oxidant effects

Aloe vera contains substantial amounts of antioxidants including α -tocopherol (vitamin E), carotenoids, ascorbic acid (vitamin C), flavonoids, and tannins, and it has been suggested that antioxidant action may be an important property of plant medicines used in treatment of various diseases (Hamman, J., 2008). The administration of ethanolic extract of *Aloe vera* gel on tissue antioxidants led to reduction in blood glucose level in diabetic rats, which helps to prevent excessive formation of free radicals through various biochemical pathways and also reduces the potential glycation of the enzymes (Rajasekaran, *et al.*, 2005; Kammoun, *et al.*, 2011). One study determined that the total phenolic content of *Aloe vera* leaf skin extracts is

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significantly correlated with the antioxidant capacity (Kammoun, *et al.*, 2011). The methanol extracts of leaf skins and flowers of *Aloe vera* were also screened for their antioxidant and antimycoplasmic activities, and in vitro both extracts of leaf skin exhibited antioxidant activity, are the most active (Lopez, *et al.*, 2013).

1.2.3 Anti-cancer effects

Aloin, an anthraquinone being a natural compound and the main ingredient of Aloe, has been documented for its remarkable potential therapeutic options in cancer, aloin treatment could inhibit the secretion of VEGF in cancer cells. VEGF is one of the most important proangiogenic cytokines known and well characterized as an inducer of tumor neovascularization. Aloin treatment significantly inhibited in vitro VEGF-induced angiogenic response of human endothelial cells, causing an inhibition of proliferation and migration of endothelial cells (Pan, *et al.*, 2013). Aloe-emodin (AE) is also a subtype of anthraquinone, a natural compound that has traditionally been found to have diverse biological activities including anticancer functions. AE (1, 8-dihydroxy-3-hydroxymethyl-9,10-anthracenedione) is an herbal anthracenedione derivative from *Aloe vera* leaves. Recent reports have shown that AE possesses antiproliferation effects on some types of cancer cells (Lin, *et al.*, 2011; Masaldan, *et al.*, 2014). The inhibitory effect of AE on the activity and gene expression of N-acetyl transferase, which plays an initial role in the metabolism of aryl amine carcinogens, was found in human malignant melanoma cells. (Lin, *et al.*, 2005; Lin, *et al.*, 2006) Recently, (Lin, *et al.*, 2006) demonstrated that AE-induced apoptosis in T24 human bladder. Aloin, derived from *Aloe vera* leaves, has been shown to possess anticancer potential activities, (Lin, *et al.*, 2006) as it inhibits tumor angiogenesis and growth via blocking signal transducer and activator of transcription 3 activation, with the potential of a drug candidate for cancer therapy (Jakson, *et al.*, 2013).

1.2.4 Anti-hyperlipidemic effects

Aloe vera is known for its antihyperlipidemic property wherein it has beneficial effects on the prevention of fatty streak development and may help to reduce the development of atherosclerosis through modification of risk factors (Huseini, *et al.*, 2012). *Aloe vera* leaf gel efficacy has been checked in hyperlipidemic type 2 diabetic patients; a randomized double-blind placebo-controlled clinical trial wherein it reduced total cholesterol and LDL levels

BRIEF REVIEW ON ALOE VERA

significantly (Huseini, *et al.*, 2012). A recent study also demonstrated that administration of phytosterols isolated from *Aloe vera* gel reduces visceral fat mass and improves hyperglycemia in Zucker diabetic fatty rats (Dana, *et al.*, 2012). Dried pulp of *Aloe vera* succotrina leaves produced significant antihyperlipidemic effect in high-fat diet- and fructose-induced hyperlipidemic rats, where it significant decreased serum levels of total cholesterol, total triglycerides, low-density lipoproteine cholesterol, very low-density lipoprotein, and high-density lipoproteine cholesterol (Dhingra, *et al.*, 2014). Previous reports also suggested that *Aloe vera* gel-treated polycystic ovarian syndrome rats exhibited significant reduction in plasma triglyceride and LDL cholesterol levels, with an increase in highdensity lipoproteinecholesterol, polycystic ovarian syndrome condition wherein hyperlipidemia is one of main consequences. The gel treatment also caused reversion of abnormal estrous cyclicity, glucose intolerance, and lipid metabolizing enzyme activities, bringing them to normal. It has phytocomponents with antihyperlipidemic effects and has shown efficacy also in management of polycystic ovarian syndrome but also the associated metabolic complications. (Maharjan, *et al.*, 2010; Desai, *et al.*, 2012).

MATERIALS
AND
METHODS

MATERIALS AND METHODS

2. Materials and Methods

2.1 Animals and Diets

Male rats *Albino Wistar* 4 weeks old weighting (about 45g) were used, animals were housed in individuals plastic cages with free access to deionized water, room temperature was kept at 24 °C on 12-hour light (08:00-20:00) and dark cycle. Poor diet in the form of compresses (tablets) was broken until obtaining a homogeneous powder and then the same amount were daily incorporated into food cups at 09:00 am with or without 2% of *Aloe vera* gel (12 g d1-d3,14g d4-d8,16g d9-21,18g d22-26,20g d27-d30, and 22g for d30-41).

2.2 Experiment

Rats were fed poor diet, in a two-way design, diets with or without containing *Aloe vera* gel. *Aloe vera* gel (Supplements Aromatik Inc., Quebec, Canada) (Fig. 4), was added to the basal diet at the level of 2%. 300mg/kg of dietary *Aloe vera* has been reported to reduce fasting blood glucose, cholesterol, triglycerides, free fatty acids, phospholipids (Subbiah, *et al.*, 2006). After 6 weeks of consuming diet (6 animals per diet group), food was removed from the cages at 08:00 am and the rats were lightly anesthetized with chloroform and killed between 10:00 am and 14:00 pm. Blood was collected and samples were allowed to clot at (-4°C) for 1h. Serum samples were obtained by centrifugation (3000 rpm for 20 min). Abdominal adipose tissues (epididymal and perirenal adipose tissues) and liver were immediately removed, weighted and stored at -50 °C until use.



Figure 4: *Aloe vera* gel (Supplements Aromatik Inc., Quebec, Canada)

MATERIALS AND METHODS

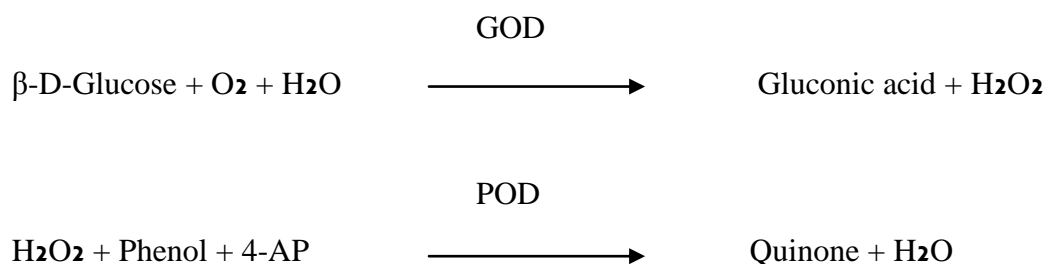
2.3 Analytical Procedure

Serum concentrations of Glucose, total Cholesterol, Triglycerides and Albumin were measured by kits (SPINREACT). HDL-Cholesterol and Creatinine levels were measured using automate (Siemens XRL, laboratory of biochemistry, hospital, Constantine).

2.3.1 Quantitative determination of Glucose

a. Test principle of Glucose

A colorimetric assay following two coupled enzymatic reactions. A specific enzymatic reaction, in which glucose oxidase (GOD), oxidizes glucose present in the sample to gluconic acid and hydrogen peroxide (H_2O_2). It serves as the substrate for the peroxidase (POD) in a coupled reaction and it is detected by a chromogenic oxygen acceptor, phenol, 4-aminophenazone (4-AP) resulting a colored product. The intensity of the color is proportional to the glucose concentration¹. (Kaplan A et al., 1984)



b. Procedure

One milliliter of the reaction mixture containing 92 mM TRIS buffer (pH 7.4), 0.3 mM phenol, 15000 U/L glucose oxidase (GOD), 1000U/L peroxydase (POD) and 2.6 mM 4-aminophenazone (4-AP) was incubated with 10 μ l sample. After incubation at 37°C for 10 Min., optical density of sample and standard (Glucose aqueous primary standard 100 mg/dl) were recorded against blank. At the wavelength 505nm using Spectrophotometer. The concentration of serum glucose was calculated by the difference in absorbance between the standard and the sample (Eq. 1) and (Eq.2).

(A) Sample / (A) Standard X 100 (Standard conc.) = mg/dl glucose in the sample (Eq.1).

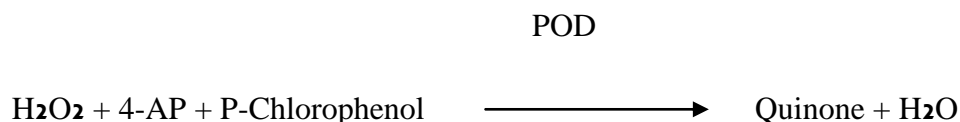
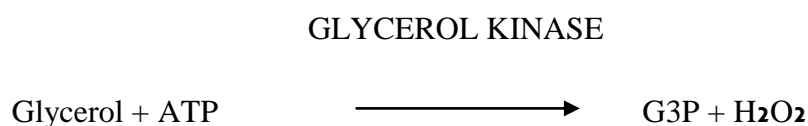
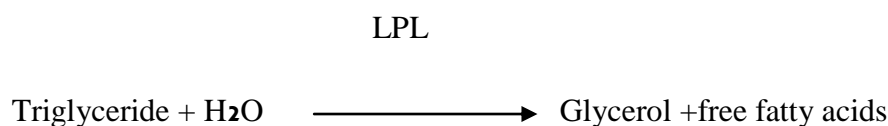
Then mg/dl X 0.0555 (conversion factor) = mmol/L (Eq.2).

MATERIALS AND METHODS

2.3.2 Quantitative determination of Triglycerides

a. Test principle of Triglycerides

Sample triglycerides incubated with lipoprotein lipase (LPL), liberate glycerol and free fatty acids. Glycerol is converted to glycerol-3-phosphate (G3P) and adenosine-5-diphosphate (ADP) by glycerol kinase and ATP. Glycerol-3-phosphate (G3P) is then converted by glycerol phosphate dehydrogenase (GPO) to dihydroxyacetone phosphate (DAP) and hydrogen peroxide (H_2O_2). In the last reaction, hydrogen peroxide (H_2O_2) reacts with 4-aminophenazone (4-AP) and p-chlorophenol in presence of peroxidase (POD) to give a red colored dye:



The intensity of the color formed is proportional to the triglycerides concentration in the sample. (Buccolo G et al., 1973)

MATERIALS AND METHODS

b. Procedure

One milliliter of working reagent contain 50 mM. GOOD buffer (pH 7.5), 2 mM. 4-chlorophenol, LPL 150000 U/L, GK 500 U/L, GPO 2500 U/L, POD 440 U/L, 4-AP 0.1 mM. and 0.1 mM. ATP was incubated with 10 μ l sample. After incubation at 37°C for 10 min, optical density of sample and standard (Glucose aqueous primary standard 100 mg/dl) were

recorded against blank. At the wavelength 505nm using Spectrophotometer. The concentration of serum triglycerides was calculated by the difference in absorbance between the standard and the sample (Eq. 3) and (Eq.4).

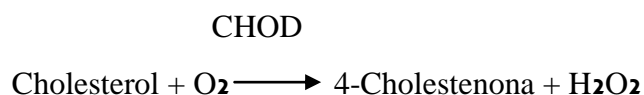
(A) Sample / (A) Standard X 200 (Standard conc.) = mg/dl triglycerides in the sample (Eq.3).

Then mg/dl X 0.0113 (conversion factor) = mmol/L (Eq.4).

2.3.3 Quantitative determination of Cholesterol

a. Test principle of Cholesterol

The cholesterol is determined after enzymatic hydrolysis and oxidation. The quinoneimine indicator is formed by the hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase.



The intensity of the color formed is proportional to the cholesterol concentration in the sample. (Naito, H., K., et al., 1984; Meiattini, F., et al., 1978)

MATERIALS AND METHODS

b.Procedure

One milliliter of the reaction mixture containing 90 mM. PIPES buffer (pH 6.9), 26 mM. phenol, 1000 U/L Cholesterol esterase (CHE), 300U/L Cholesterol oxidase (CHOD) , 650 U/L Peroxidase (POD) and 0.4 mM 4 – Aminophenazone (4-AP) was incubated with 10 µl sample. Each sample had both blank and standard (Cholesterol aqueous primary standard 200 mg/dl). After incubation at 37°C for 10 min, optical density at the wavelength 505 nm was recorded using Spectrophotometer. The concentration of serumcholesterol was calculated from the difference in absorbance between the standard and the sample (Eq. 5) and (Eq. 6).

(A) Sample / (A) Standard X 200 (Standard conc.) = mg/dl cholesterol in the sample (Eq.5).

Then mg/dl X 0.0258 (conversion factor) = mmol/L (Eq. 6).

2.3.4 Quantitative determination of HDL-Cholesterol

a.Test principle of HDL-Cholesterol

The very low density (VLDL) and low density (LDL) lipoproteins from serum or plasma are precipitated by phosphotungstate in the presence of magnesium ions. After centrifugation the supernatant contains high density lipoproteins (HDL). The HDL-cholesterol fraction is determined using the total cholesterol enzymatic reagent. Naito H K. High-density lipoprotein (HDL) cholesterol. (Kaplan, A., et al., 1984; Grove, T., H., 1979).

b.Procedure

Two reagents the first one is R1 for elimination of lipoprotein no-HDL, 300microlitter contain N,N-bis(2-hydroxyethyl)-2-aminoethanesulphonic acid (pH 6.6). 100mM., N-(2-hydroxy-3-sulfopropyl)-3,5-dimethoxyaniline (HDAOS) 0.7 mM., CholesterolEsterase \geq 800 U/L, Cholesterol oxidase \geq 500 U/L, Catalase \geq 300 U/L Ascorbic oxidase \geq 3000 U/L was incubated with 3 µl sample for 5 min at 37°C. The absorbances (A1) of the samples and standard (Lyophilized human serum) were recorded against blank at the wavelength 600 nm.

MATERIALS AND METHODS

2.3.5 Quantitative determination of Creatinine

a. Test principle of Creatinine

The assay is based on the reaction of creatinine with sodium picrate, as described by Jaffé. Creatinine reacts with alkaline picrate forming a red complex (Janovsky complex). The time interval chosen for measurements avoids interferences from other serum constituents, (absorbance at 492 nm). The intensity of the color formed is proportional to the creatinine concentration in the sample. (Kaplan, A., et al., 1984).

b. Procedure

Creatinine Base Reagent containing 0.29 mol/L sodium hydroxide and surfactants and Creatinine Picrate Reagent containing 17.5 mM. picric acid can be used separately on two reagent systems. A working reagent may be prepared by combining one volume of creatinine picrate reagent and four volumes of creatinine base reagent. Mix well before using. Studies using this reagent were performed on an automated analyzer using a kinetic test mode, with a sample to reagent ratio of 1:24, and a wavelength reading of 505 nm. The analyzer automatically calculates the creatinine concentration of each sample.

2.3.6 Quantitative determination of Albumin

a. Test principle of Albumin

Albumin in the presence of bromocresol green at a slightly acid pH, produces a colour change of the indicator from yellow-green to green-blue. The intensity of the color formed is proportional to the albumin concentration in the sample. (Gendler, S., Kaplan, A., et al., 1984).

Ph= 4.20

Albumine + BCG \longrightarrow albumine complex -BCG

MATERIALS AND METHODS

b. Procedure

One milliliter of the reaction mixture containing 0.12 mM. Bromcresol green buffer (pH 4.2) was incubated with 5 μ l sample. Each sample had both blank and standard (Albumin aqueous primary standard 5 g/dl). After incubation for 10 min at room temperature (15-25 $^{\circ}$ C) optical density at the wavelength 630 nm was recorded using Spectrophotometer (JENWAY 6300). The concentration of serum albumin was calculated from the difference in absorbance between the standard and the sample (Eq. 9) and (Eq. 10).

$(A)_{\text{Sample}} / (A)_{\text{Standard}} \times 5 \text{ (Standard conc.)} = \text{g/dl albumin in the sample (Eq. 9)}$.

Then $\text{g/dl} \times 144.9 = \mu\text{mol/L (Eq. 10)}$.

2.4 Statistical analysis

All results were tested for statistical significance by Student's t-test using StatView software.

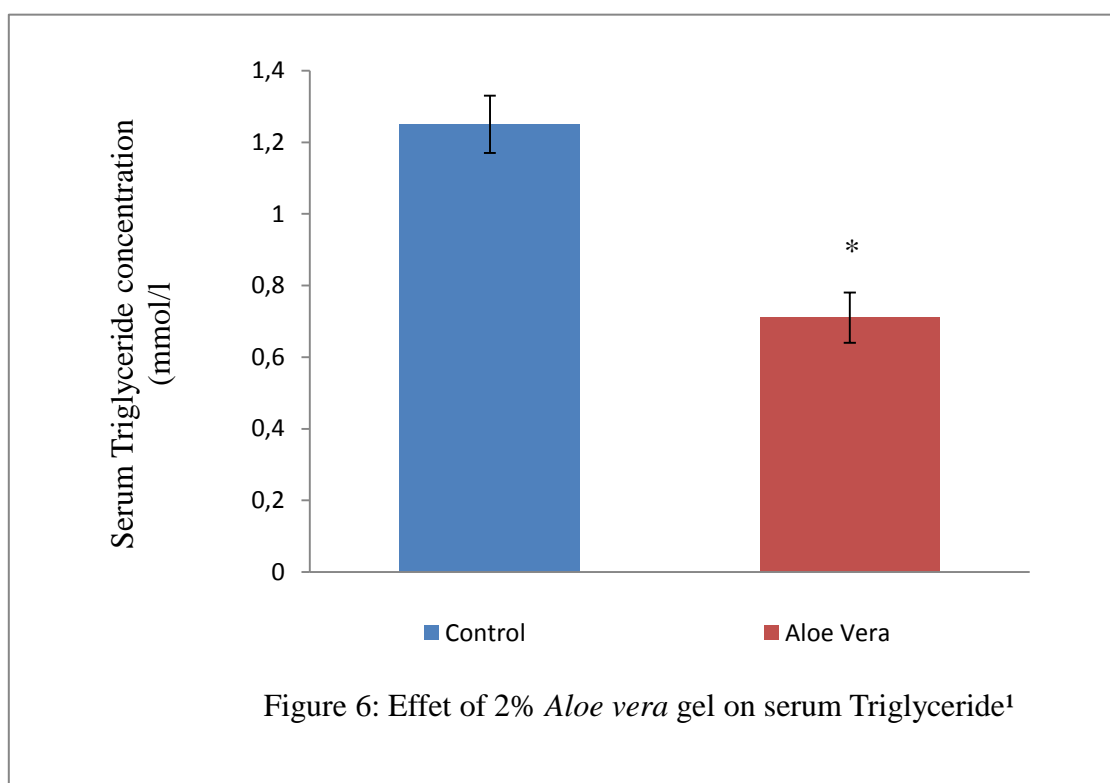
RESULTS
AND
DISCUSSION

RESULTS AND DISCUSSION

3. Results and Discussion

a. Effect of 2% *Aloe vera* gel on serum Triglyceride

Feeding rats 2% *Aloe vera* gel significantly reduced serum triglycerides (Fig. 6). Our results are in agreement with the previous studies (Kwanghee, *et al.*, 2009; Eriko, *et al.*, 2008). (Subbiah, *et al.*, 2006; Nasiff, *et al.*, 1993) found that the levels of free fatty acids were significantly decreased, and therefore we speculate that feeding rats 2% *Aloe vera* might activate hepatic fatty acid oxidation, which leads to high conversion of fatty acids to ketone bodies thereby reducing serum triglyceride. Further study is needed to measure serum free fatty acid, the activity of hepatic carnitine palmitoyltransferase (CPT), as a rate limiting enzyme of mitochondrial fatty acid oxidation and also the activities of enzymes of lipogenesis such as G6PD and FAS.



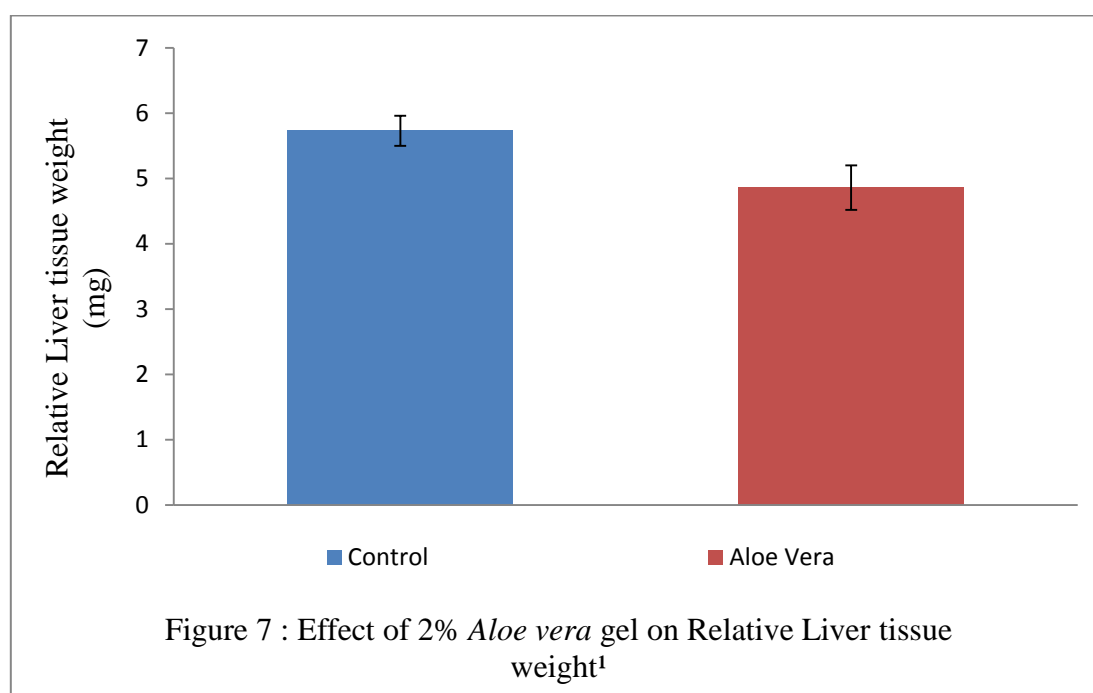
¹ Value are means \pm SE (n= 6) for each group

*Significantly different from control by student's test ($P < 0.05$)

RESULTS AND DISCUSSION

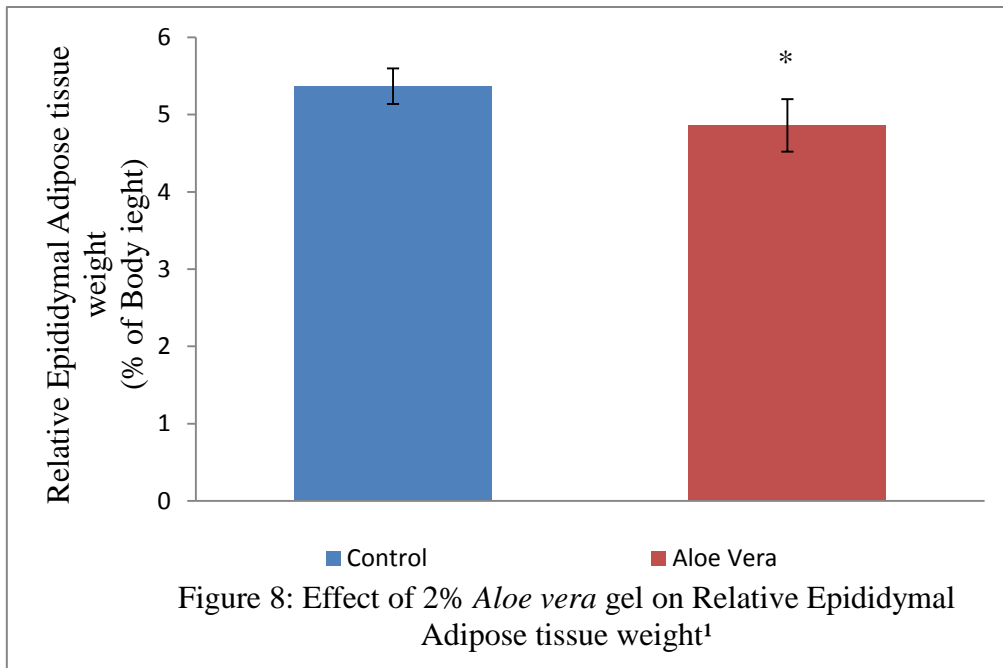
b. Effect of 2% *Aloe vera* gel on Liver, relative Perirenal and Epididymal adipose tissue weight

Weight of liver was unaffected by dietary 2% *Aloe vera* (Fig. 7). This is in agreement with the research done by (Zodap, *et al.*, 2012). Dietary 2% of *Aloe vera* gel caused 43% and 40% reduction in weight of epididymal adipose tissue and perirenal adipose tissue, respectively (Fig. 8, 9). Phytosterols isolated from *Aloe vera* gel has been known to reduce both abdominal fat and subcutaneous fat and prevent obesity (Eriko, *et al.*, 2008). In consistent with Eriko's study, dietary 2% of *Aloe vera* might elevates the activity of hepatic carnitine palmitoyltransferase, the rate-limiting enzyme of mitochondrial fatty acid oxidation, which causes higher fatty acid oxidation. Our results suggest that the lower accumulation of body fat is mediated through phytosterols by a mechanism involving higher fatty acids activity. Further study is needed to explain the phenomena.



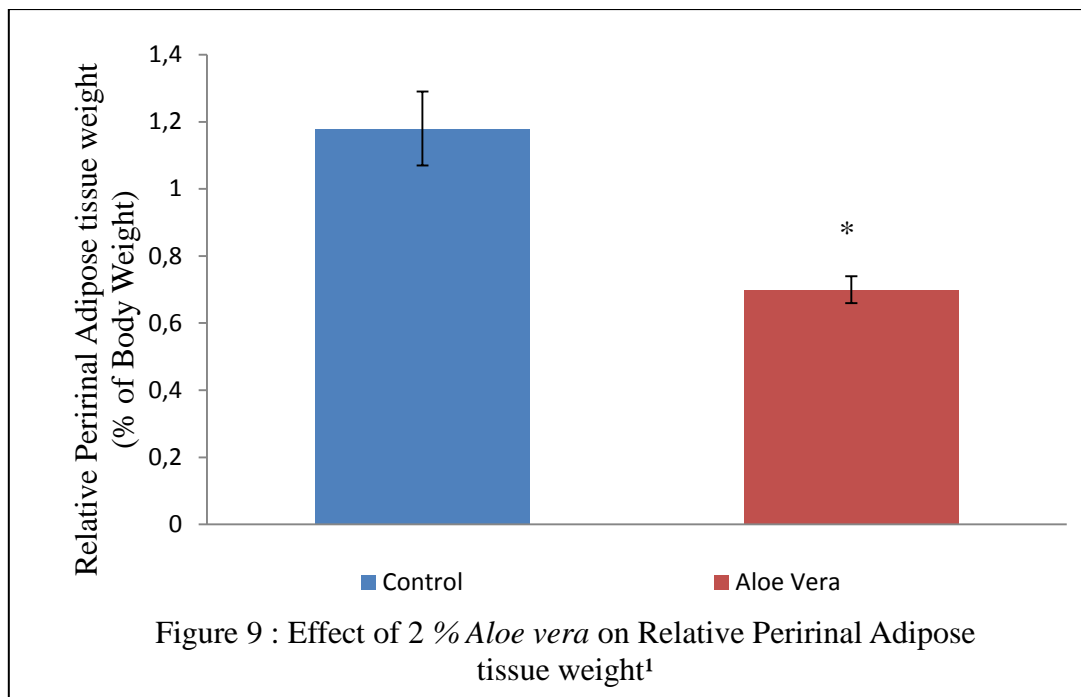
¹ Value are means \pm SE (n= 6) for each group

RESULTS AND DISCUSSION



¹ Value are means \pm SE (n= 6) for each group

*Significantly different from control by student's test (P < 0.05).



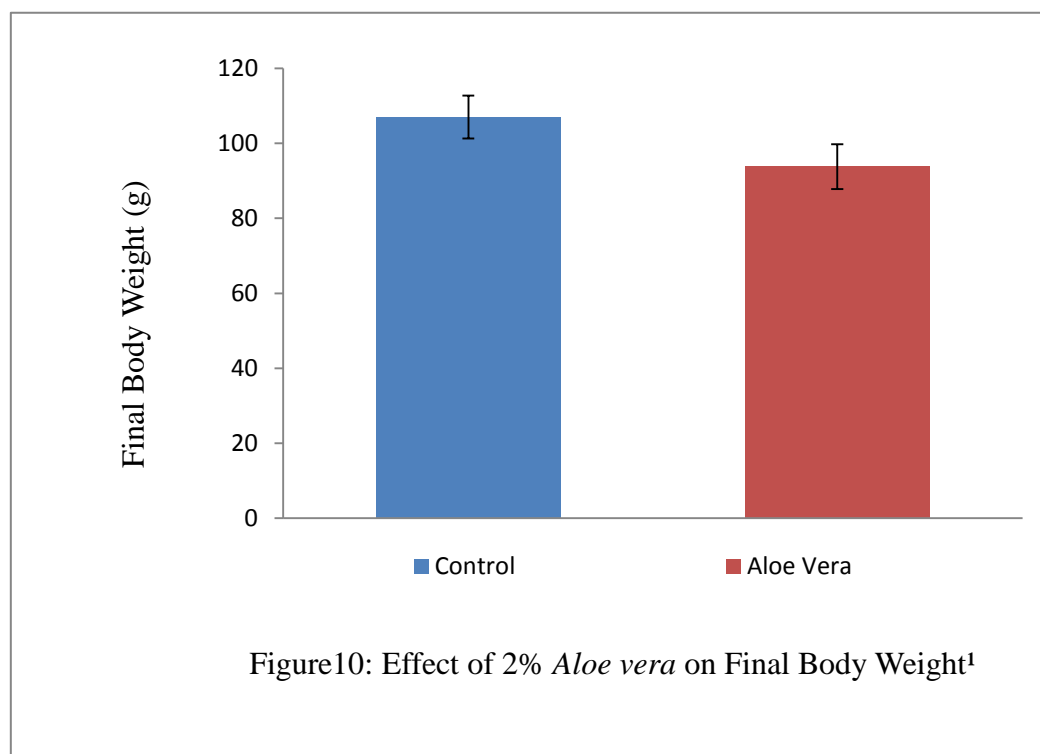
¹ Value are means \pm SE (n= 6) for each group

*Significantly different from control by student's test (P < 0.05).

RESULTS AND DISCUSSION

c. Effect of 2% *Aloe vera* gel on Final Body Weight

Feeding 2% *Aloe vera* gel slightly reduced gain body weight, but not significant (Fig. 10). This is in agreement with (Manoj *et al.*, 2013; Kwanghee *et al.*, 2009)

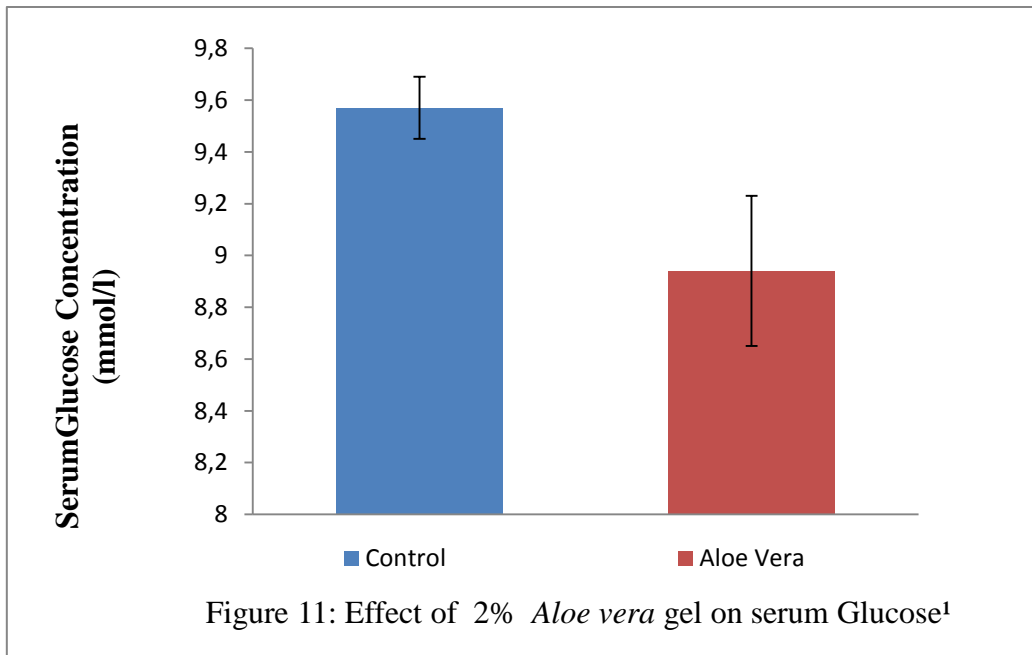


¹Value are means \pm SE (n= 6) for each group

d. Effect of 2% *Aloe vera* gel on serum Glucose

Serum glucose concentration was slightly reduced by 2% *Aloe vera* gel treatment (Fig. 11). Our results are in agreement with the previous studies. It has been shown that *Aloe vera* gel feeding caused lower serum concentration of glucose (Kwanghee, *et al.*, 2009; Manoj, *et al.*, 2013).

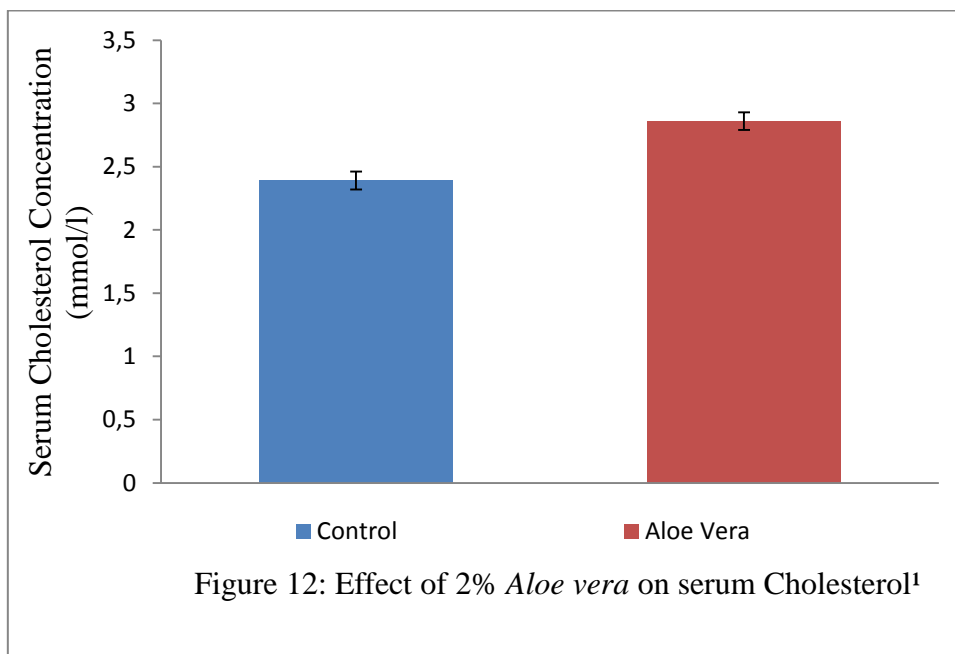
RESULTS AND DISCUSSION



¹ Value are means \pm SE (n= 6) for each group

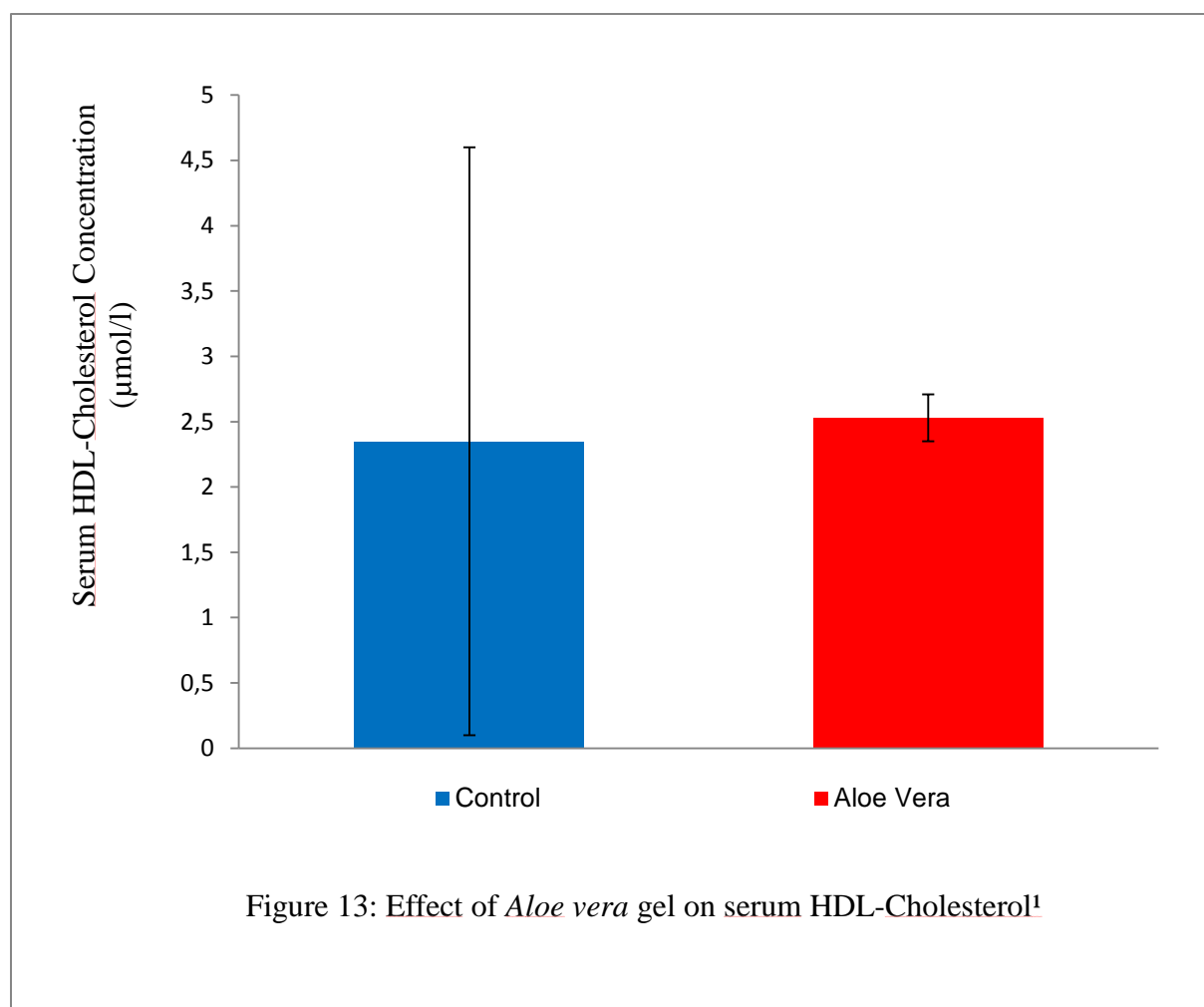
e. Effect of 2% *Aloe vera* gel on serum Cholesterol and HDL-Cholesterol

2% *Aloe vera* gel has no significant effect on the serum total cholesterol and HDL-cholesterol (Fig. 12, 13). Our study is in agreement with previous studies (Eriko, *et al.*, 2008).



¹ Value are means \pm SE (n= 6) for each group

RESULTS AND DISCUSSION

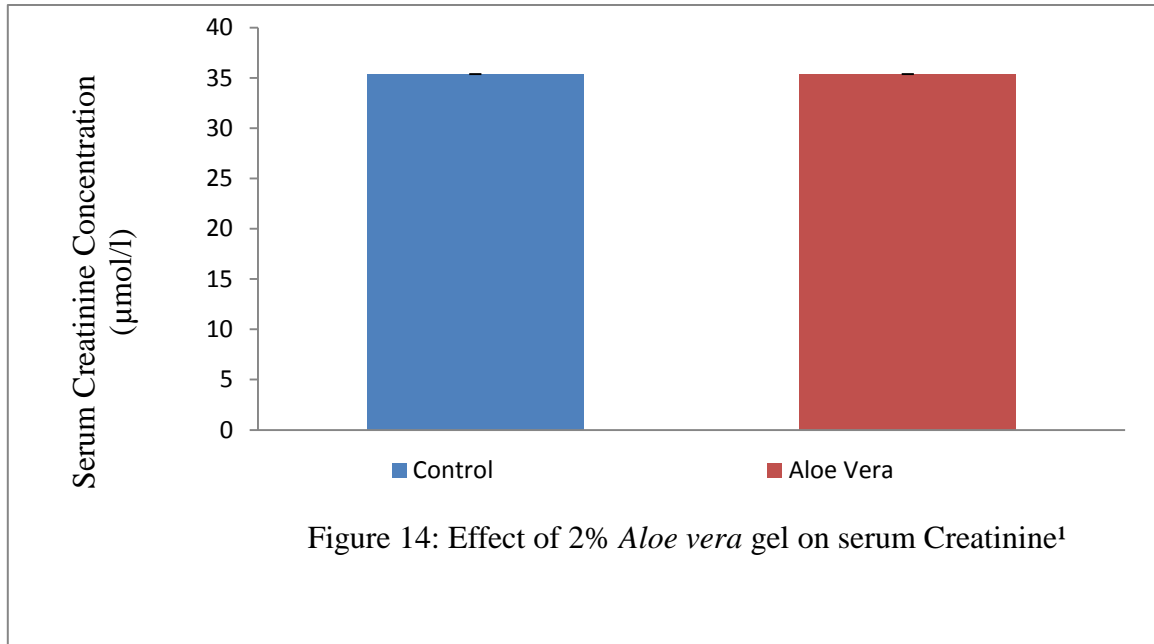


¹Value are means \pm SE (n= 6) for each group

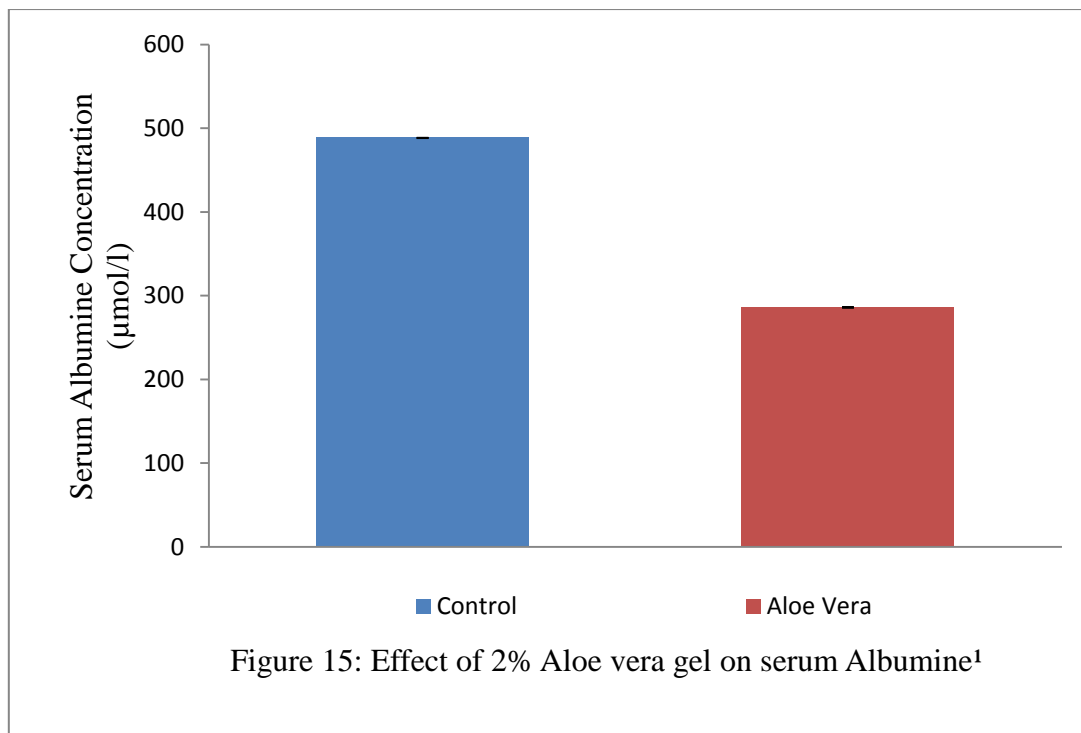
f. Effect of 2% *Aloe vera* gel on serum Creatinine and Albumine

Addition of 2% *Aloe vera* gel significantly decreased serum Albumin without affecting serum Creatinine (Fig. 14, 15). Our results are in agreement with previous studies (Zubic, 2001). The hypoalbuminemic effect of *Aloe vera* gel is due to liver disease, acute infection (Guyton, *et al.*, 2006), Or to the low protein content of *Aloe vera* (Nuzhat, *et al.*, 2013).

RESULTS AND DISCUSSION



¹ Value are means \pm SE (n= 6) for each group



¹ Value are means \pm SE (n= 6) for each group

*Significantly different from control by student's test ($P < 0.05$)

CONCLUSION
AND
PERSPECTIVS

CONCLUSION AND PERSPECTIVS

Conclusion

Obesity is now the most prevalent nutritional disease and a growing public health problem worldwide. The disease has acquired epidemic proportions. World wide obesity has more than doubled since 1980. In 2015, more than 2.3 billion adults were overweight and 700 million adults were obese (Malik *et al.*, 2013).

Studies were reported that *Aloe vera* and its active compounds such as phytosterols have an ability to improve the hyperglycemia and decrease lipogenesis (Eriko *et al.*, 2008). To control blood glucose levels (Bunyapraphat, *et al.*, 1996), to lower blood lipids in hyperlipidemic (Rajasekaran, *et al.*, 2006), it was also used for its anti-inflammatory, and antitumoral effects (Reynolds, *et al.*, 1999; Chithra, *et al.*, 1998).

This study provided the first evidence that feeding rats 2% *Aloe vera* gel caused not only hypotriglyceridemia but also lower body fat. The results suggest that 2% *Aloe vera* gel activates fatty acids oxidation, which leads high conversion of fatty acids to ketone bodies, thereby reducing serum triglyceride. This phenomena is one of the mechanisms responsible for the lower accumulation of body fat.

In summary, this study has demonstrated lower body fat and hypotriglyceridemia caused by feeding rats 2% *Aloe vera* gel and suggest that the reduction in body fat is mediated by the activation of the hepatic fatty acid oxidation.

Further study is needed to measure serum free fatty acid, the activity of hepatic carnitine palmitoyltransferase (CPT), as a rate limiting enzyme of mitochondrial fatty acid oxidation and also the activities of enzymes of lipogenesis such as G6PD and FAS.

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SUMMARY

Summary

Aloe vera is a species of Aloe (Aloe genus) that has been used for thousands of years to heal a variety of conditions. It contains phyosterols. These compound make it an effective medicinal herb in the treatment of cardio vascular disease, improvs diabetics, and cancer.

Obesity is defined as an excessive fat accumulation that may impair healt. Obesity is the most prevalent nutritional disease and a growing public health problem worldwide. This disease is a causal component of the metabolic syndrome related with abnormalities, including hyperglycemia, dyslipidemia, hypertension, inflammation, among others, obesity and *Aloe vera* seems to be associated with the same diseases such as cancer, diabetes and suggest a possible effect of *Aloe vera* on obesity.

This study was conducted to define the effect of 2% *Aloe vera* gel on serum lipid and body fat accumulation. Rats were fed for 42 days diets with or without 2% *Aloe vera* gel. Feeding a diet containing 2% *Aloe vera* gel caused reduction in relative epididymal and perirenal adipose tissue weight, decreasing in serum triglyceride and serum glucose concentration.

This study provided the first evidence suggesting that 2% *Aloe vera* lower body fat accumulation. *Aloe vera* appears to have anti-obesity effect by inhibiting adipocyte differentiation through a mechanism involving its active compounds such as phyosterols.

Keywords: *Aloe vera*, Hypolipidemia, Hypotriglyceridemia, Obesity.

SUMMARY

Résumé

Aloe vera est une espèce d'Aloès (*Aloe* genre) qui a été utilisé depuis des milliers d'années pour guérir une variété de maladies. Elle contient des phytostérols. Ces composés font une plante médicinale efficace dans le traitement des pathologies cardio-vasculaires, diabète, et le cancer.

L'obésité est définie comme une accumulation excessive de graisse qui peuvent nuire la santé, l'obésité est la maladie nutritionnelle la plus répandue et un problème croissant de santé publique dans le monde entier. Cette maladie est un élément causal du syndrome métabolique lié à des anomalies, y compris l'hyperglycémie, la dyslipidémie, l'hypertension, l'inflammation, entre autres, l'obésité et l'*Aloe vera* semble être associée aux mêmes maladies telles que le cancer, le diabète et suggèrent un effet possible d'*Aloe vera* sur l'obésité.

Cette étude a été menée pour définir l'effet de 2 % de gel d'*Aloe vera* sur les lipides sériques et accumulation des graisses dans le corps. Les rats ont été nourris pendant 42 jours un régime alimentaire avec ou sans 2 % de gel d'*Aloe vera*. Le régime alimentaire contenant 2 % de gel d'*Aloe vera* a provoqué une réduction du poids relatif des tissus adipeux épидидymial et péri-rénal, la diminution des triglycérides dans le sérum et la concentration du glucose sérique.

Cette étude a fourni la première preuve suggérant que 2 % d'*Aloe vera* diminue l'accumulation de graisse corporelle. *Aloe vera* semble avoir un effet anti-obésité en inhibant la différenciation des adipocytes par un mécanisme impliquant ses composés actifs tels que les phytostérols.

Mots-Clés : *Aloe vera*, Hypolipidémie, Hypotriglycéridémie, L'obésité.

SUMMARY

المخلص

الصبر الحقيقي أو الألوّة الحقيقية أو ألوي فيرا هو نوع من الصبار التي استخدمت منذ آلاف السنين لعلاج مجموعة متنوعة من الأمراض . يحتوي على الفايوتوستيرول . هذه المركبات فعالة لعلاج أمراض القلب والأوعية الدموية، السكري والسرطان.

يتم تعريف السمنة على أنها تراكم المفرط للدهون التي يمكن ان تضر بالصحة ، والسمنة هي مرض الغذائية الأكثر انتشارا و مشكلة متنامية للصحة العامة في جميع أنحاء العالم . هذا المرض هو العنصر المسبب لمتلازمة الأيض المرتبطة ، بارتفاع السكر في الدم ، اضطراب شحوم الدم وارتفاع ضغط الدم ، والتهاب ، من بين أمور أخرى ، ويبدو الألوّة فيرا تترافق مع نفس الأمراض مثل السرطان والسكري و تشير إلى التأثير المحتمل للألوّة الحقيقية على السمنة.

وقد أجريت هذه الدراسة لتحديد تأثير 2 ٪ هلام الألوّة الحقيقية على نسبة الدهون في الدم و تراكم الدهون في الجسم . تم تغذية الفئران لمدة 42 يوما نظام غذائي مع أو دون 2 ٪ هلام الألوّة الحقيقية. اتباع نظام غذائي يحتوي على 2 ٪ هلام الألوّة الحقيقية سبب انخفاض في الوزن النسبي للأنسجة الدهنية البطنية والانسجة الدهنية القرب كلوية ، وانخفاض الدهون الثلاثية في الدم و تركيز الجلوكوز في مصل الدم.

قدمت هذه الدراسة أول دليل يشير إلى أن 2 ٪ من الألوّة الحقيقية يقلل من تراكم الدهون في الجسم . يبدو ان الألوّة الحقيقية لها تأثير مضاد للسمنة عن طريق تثبيط تمايز الخلايا الشحمية من خلال آلية تنطوي على احد مركباتها مثل الفايوتوستيرول.

الكلمات المفتاحية : الألوّة الحقيقية ،انخفاض نسبة الدهون، انخفاض نسبة الجليسيريدات الثلاثية ، السمنة.

Effect of Dietary Addition of 2% *Aloe vera* gel on Serum Lipids and Body Fats Accumulation in Rats

Diplôme: To Get a Diploma of Master in Biochemistry
Option: Proteomic Analysis and Health

Résumé :

Aloe vera est une espèce d'Aloès (Aloe genre) qui a été utilisé depuis des milliers d'années pour guérir une variété de maladies. Elle contient des phytostéroles. Ces composés font une plante médicinale efficace dans le traitement des pathologies cardio-vasculaires, diabète, et le cancer.

L'obésité est définie comme une accumulation excessive de graisse qui peuvent nuire la santé, l'obésité est la maladie nutritionnelle la plus répandue et un problème croissant de santé publique dans le monde entier. Cette maladie est un élément causal du syndrome métabolique lié à des anomalies, y compris l'hyperglycémie, la dyslipidémie, l'hypertension, l'inflammation, entre autres, l'obésité et l'*Aloe vera* semble être associée aux mêmes maladies telles que le cancer, le diabète et suggèrent un effet possible d'*Aloe vera* sur l'obésité.

Cette étude a été menée pour définir l'effet de 2 % de gel d'*Aloe vera* sur les lipides sériques et accumulation des graisses dans le corps. Les rats ont été nourris pendant 42 jours un régime alimentaire avec ou sans 2 % de gel d'*Aloe vera*. Le régime alimentaire contenant 2 % de gel d'*Aloe vera* a provoqué une réduction du poids relatif des tissu adipeux épидidymial et péri-rénal, la diminution des triglycérides dans le sérum et la concentration du glucose sérique.

Cette étude a fourni la première preuve suggérant que 2 % d'*Aloe vera* diminue l'accumulation de graisse corporelle. *Aloe vera* semble avoir un effet anti-obésité en inhibant la différenciation des adipocytes par un mécanisme impliquant ses composés actifs tels que les phytostéroles.

Keywords: *Aloe vera*, Hypolipidemia, Hypotriglyceridemia, Obesity.

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