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Review Article

Pharmacological Review of Anti-Obesity Herbs from *Chakradatta*: Insights and Mechanisms

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ABSTRACT

Background: Obesity has become a major global health crisis, significantly increasing the risk of Type 2 diabetes, cardiovascular diseases, and certain cancers, while placing a considerable economic burden on healthcare systems. Its prevalence has more than tripled between 1975 and 2022. With growing concerns over the side effects of synthetic anti-obesity drugs, traditional medicinal herbs are gaining attention for their safer and multi-targeted therapeutic potential. **Aim:** This review aims to evaluate the efficacy of Ayurvedic herbs in obesity management by examining their roles in modulating key molecular mechanisms associated with lipid metabolism. **Materials and Methods:** A systematic literature review was conducted focusing on anti-obesity herbs mentioned in the "*Sthoulya*" chapter of the classical Ayurvedic text *Chakradatta*. Databases such as PubMed were searched using keywords and MeSH terms including "Ayurvedic herbs" and "anti-obesity." Studies published between 2007 and 2024 were screened for *in vivo* and *in vitro* evidence related to the mechanisms of action of these herbs. Clinical trials, case studies, and articles with methodological limitations were excluded. **Results:** This study explores the impact of 34 Ayurvedic herbs on lipid metabolism and their potential in managing obesity. The herbs evaluated include *Embelia ribes, Hordeum vulgare, Phyllanthus emblica, Cyperus rotundus, Piper longum, Aegle marmelos, Premna Serratifolia, Terminalia bellirica, Terminalia chebula, Plumbago zeylanica, Adhatoda zeylanica, Ziziphus mauritiana, Acorus calamus, Alstonia scholaris, Aconitum heterophyllum, Azadirachta indica, Citrullus colocynthis, Saussurea lappa, Brassica campestris, Zingiber officinale, Curcuma longa, Commiphora wightii, Acacia catechu, Piper nigrum, Cuminum cyminum, Moringa oleifera, Picrorhiza kurroa, Coriandrum sativum, Mangifera indica, Punica granatum, Foeniculum vulgare, Tinospora cordifolia, Elettaria cardamomum, Juniperus communis.* These herbs demonstrate a wide range of mechanisms in lipid metabolism regulation, such as inhibiting lipogenesis, enhancing lipolysis, regulating appetite, promoting thermogenesis, and improving fat oxidation. Their multi-targeted actions highlight their potential as effective natural agents in the management of obesity. **Conclusion:** Ayurvedic herbs offer promising multi-targeted approaches for managing obesity. Further scientific validation and clinical studies are essential to establish their efficacy and integrate them into modern therapeutic frameworks.

Keywords: Obesity, Pathways, *Sthoulya*, Adipose tissues.

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INTRODUCTION

Obesity has emerged as a significant health crisis, particularly in advanced economies, where it poses one of the greatest burdens of illness. It not only increases the risk of Type 2 diabetes, cardiovascular diseases, and cancer, but also places a substantial economic burden on healthcare systems. The global prevalence of obesity has risen dramatically, more than tripling between 1975 and 2022. In 2022, an estimated 2.5 billion adults aged 18 and older were considered overweight, with 890 million of them classified as obese. Among children and adolescents aged 5 to 19 years, over 390 million were found to be overweight, including 160 million who were classified as obese.[1]

The World Health Organization (WHO) defines obesity as an "abnormal or excessive fat accumulation that may impair health," further clarifying that "the fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended.[2] In Ayurveda, *Sthoulya* is primarily caused by vitiation of *Kapha* and *Meda dosha*, stemming from factors such as excessive intake of heavy, unctuous food, sedentary habits, and impaired digestion, factors that align closely with modern risk contributors like overnutrition, lack of physical activity,

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and metabolic dysregulation.[3] The underlying mechanism is summarized in Figure 1. Clinically, *Sthoulya* is characterized by increased fat deposition, lethargy, breathlessness on exertion, and excessive appetite, mirroring the features of obesity.

Traditional herbs, used for centuries, offer a promising alternative to synthetic anti-obesity drugs, which often come with undesirable side effects and high costs.[4] The NFHS-5 (National Family Health Survey) data revealed that abdominal obesity is more prevalent in India among women (40%) than men (12%). Abdominal obesity was defined as a waist circumference greater than 80 cm for women and greater than 94 cm for men.[5]

Adult-onset obesity is predominantly characterized by adipose cell hypertrophy with minimal hyperplasia.[6] Apart from the increased size of normal fat depots such as subcutaneous tissue, omentum, retroperitoneal tissues, and epicardium, adipose tissue in obesity may extend to areas where it is normally absent.[7] The pathogenesis of obesity involves 3 main components: excessive lipid deposition due to increased food intake, hypothalamic lesions, and adipose cell hyperplasia[8] diminished lipid mobilization due to decreased levels of lipolytic hormones and abnormalities in autonomic innervation[9] and diminished lipid utilization resulting from abnormalities in thyroxine and adrenaline, which stimulate the mobilization of unsaturated fatty acids from adipose tissues.[10]

These herbs are thought to contribute to weight management by targeting multiple physiological pathways, including appetite suppression, thermogenesis enhancement, inhibition of fat absorption, and modulation of lipid metabolism.[11] In the contemporary era, extensive research has been undertaken to validate these claims and elucidate the pharmacological actions of various traditional herbs. For instance, dysregulation of proteins like AQP7, which facilitate glycerol transport in adipocytes, has been linked to obesity.[12] Traditional herbs may influence such pathways, thereby aiding in fat metabolism and adipogenesis regulation.[13] In this context, the present review draws upon the "*Chakradatta,"* a key text by *Acharya Chakrapanidatta*. "*Chakradatta,"* originally titled "*Chikitsa Sangraha*," stands as a pivotal work in the history of Ayurvedic therapeutics. Chosen for its historical and practical significance, the *"Chakradatta*" builds on earlier works like *'Siddhayoga'* and is renowned for its comprehensive therapeutic guidelines. The treatise's detailed prescriptions for various herbs and formulations make it a valuable resource for understanding traditional approaches to conditions such as obesity. Its continued relevance and influence in modern Ayurveda underscore its importance for historical study and contemporary practice. This review focuses on the anti-obesity mechanisms of various medicinal plants. By examining their effects on lipid metabolism and related pathways, we aim to emphasize the potential of traditional herbs for preventing and treating obesity. Our objective is to demonstrate how traditional herbs could provide safer and more effective treatment modalities, offering new pharmacological options for improved obesity management through a detailed understanding of their molecular mechanisms.

MATERIALS AND METHODS

Development of Search Strategy: The search strategy was developed to comprehensively explore Ayurvedic herbs associated with combating obesity, focusing particularly on those detailed in the classical text "*Chakradatta*" chapter on obesity, referred to as "*Sthoulya*." Keywords and phrases specific to Ayurvedic herbs mentioned in this chapter were identified and used to formulate a targeted search strategy. MeSH terms related to obesity, ayurvedic herbs were incorporated to ensure a comprehensive search across literature databases.

Identification of Keywords: Keywords such as "Ayurvedic herbs," AND "anti-obesity," OR "*Sthoulya*," and their variations were utilized to identify relevant studies. Specific botanical names and traditional terms associated with the herbs mentioned in "*Chakradatta*" were also included in the search strategy to capture all relevant literature.

Database Search: A thorough search was conducted in an online database, with a primary focus on PubMed, to investigate the mechanisms of action and physiological pathways underlying the potential efficacy of Ayurvedic herbs in combating obesity.

Study Selection

Inclusion Criteria: Articles published between the years 2007 and 2024 were included in this review. Studies included were primarily *in vivo* and *in vitro* investigations that discussed the anti-obesity properties of Ayurvedic herbs. Emphasis was placed on studies providing detailed insights into the mechanisms of action, metabolic pathways, and physiological effects of these herbs.

Exclusion Criteria: Case studies, case series, clinical trials, and review articles were excluded from this review. Additionally, studies with significant methodological flaws, insufficient data, or those not directly related to the anti-obesity effects of Ayurvedic herbs were excluded. Articles not available in English or published before the year 2007 were also excluded to focus on recent advancements and current understanding in the field.

Data Extraction and Analysis: Data extraction focused on summarizing findings related to the mechanisms of action, efficacy, and potential therapeutic applications of each Ayurvedic herb in combating obesity. Data were analyzed and synthesized to gain comprehensive insights into the evidence supporting the use of these herbs as agents against obesity. The flow chart (Figure 2) depicts:

RESULTS

Important plants used in clinical practices

Each medicinal plant or herb contains numerous active compounds that interact synergistically to produce therapeutic benefits. Below, several important plants of medicinal value are highlighted for their anti-obesity action.

Vidanga

Effects of *Vidanga* (*Embelia ribes* Burm.f, *Myrsinaceae*) on obesity are multifaceted and have been demonstrated in experimental studies involving ethanolic extracts of the fruit in albino rat models. It decreases serum leptin levels and lowers hepatic TBARS levels, as evidenced by malondialdehyde levels, thus enhancing hepatic oxidative balance.[15] It blocks Protein Kinase C (PKC) and its subsequent signaling pathway, including GPIIb/IIIa activation and secretin secretion, which helps reduce inflammation. Additionally, embelin inhibits the NF-κB signaling pathways and IL6/STAT3 using *in vitro* ICR mice[16] suppresses adipogenesis, partially agonizes PPARγ, and activates the PI3K/p-Akt pathway, leading to GLUT4 translocation using High-Fat Diet (HFD) fed-streptozotocin induced type 2 diabetic rats.[17]

Yava

Barley ethanolic extract (*Hordeum vulgare* Linn., *Poaceae*) reduced the expression of C/EBPα and PPARγ, along with their downstream target genes FABP4, LPL, and FAS, and increased adiponectin levels as demonstrated in studies conducted on C57BL/6N mice.[18] The extracts caused cell cycle arrest at the G1/S phase, resulting in a decrease in cells in the G2 phase[19,20] increased Short-Chain Fatty Acids (SCFAs) in the cecum, decreased mRNA levels of HMG-CoA reductase, and enhanced oxidative metabolism in the liver and adipose tissue.[20] Barley also raised plasma levels of PYY and GLP-1, hormones known to suppress appetite and enhance glucose-dependent insulin secretion, likely via SCFA receptors GPR41 and GPR43.[21] Additionally, barley β-glucan upregulated NeuroD gene expression, promoting L cell differentiation.[22] LFBE upregulated the expression of PGC-1α, β-AR, UCP1, and COX in 3T3-L1 preadipocytes, particularly the UCP1 gene, crucial for activating BAT via the UCP1 mechanism.[23]

Amalaki

(*Phyllanthus emblica* L., *Euphorbiaceae*) Water Extract of *Phyllanthus emblica* L. fruit (WEPE) and its bioactive compound Gallic Acid (GA) control appetite by reducing the expression of appetite-stimulating neuropeptides (AgRP, NPY, MCH) and increasing the expression of appetite-suppressing neuropeptides (CRH, POMC, CART). WEPE also regulates SOCS3, which is a critical inhibitor of leptin signaling pathways involved in lipid metabolism and enhances energy expenditure via the leptin-AMPK/STAT3 pathway using High-Fat Diet (HFD)-induced obese Sprague-Dawley (SD) rats as the animal model.[24] It improves intestinal homeostasis by increasing the jejunum's mRNA expression of tight junction proteins (occludin, claudin-1, ZO-1, claudin-3). Additionally, it inhibits carbohydrate digestive enzymes and lipase activity, reducing glucose and fat absorption. It activates 5'AMPK, which promotes catabolic processes like glycolysis and inhibits anabolic processes such as fatty acid synthesis, while also increasing adiponectin levels in the liver and perirenal fat.[25] Furthermore, it enhances antioxidant enzyme production (catalase, SOD, GPx) and reduces Malondialdehyde (MDA) production, a marker of oxidative stress. By regulating MG metabolism through increased Glo-1 activity[26] *amalaki* helps manage oxidative stress and inflammation, contributing to effective obesity management.

Musta

(*Cyperus rotundus* Benth, *Cyperaceae*) exhibits significant anti-obesity effects, as demonstrated in studies conducted on obese Zucker rats. The *Cyperus rotundus* extract from rhizomes, which included piceatannol, scirpusin A, and scirpusin B, reduces PPARγ and adipocyte protein 2 (aP2/FABP4) expression, preventing adipogenesis. These compounds act as agonists of the GABAA-benzodiazepine receptor[27] and inhibit α-lipase and amylase. CRE also reduces inflammatory enzyme activities (HAase and MPO), increases pancreatic antioxidant activity by stimulating CAT and SOD, raises reduced glutathione levels, and suppresses TBARS formation.[28]

Pippali

(*Piper longum* L. *Piperaceae*) The Ethyl Acetate Extract (EAP) of *Piper longum* fruit, containing apigenin 7,4'-dimethyl ether and piperine, has been shown to exhibit anti-obesity effects in male Sprague-Dawley (SD) rats. It inhibits α-glucosidase and α-amylase enzymes, controlling postprandial blood glucose levels and reducing fat deposition. *Pippali* also exhibits anti-lipid peroxidative effects and hypo-lipidemic properties, possibly mediated by PPAR upregulation[29] and modulation of calcium channels. Additionally, it influences adipocyte biology by decreasing leptin and increasing adiponectin levels, regulating appetite and insulin sensitivity. Pippali's inhibition of FAS and enhancement of LCAT activity further contribute to its anti-obesity effects.[30]

Bilva

(*Aegle marmelos* (L.) Correa, *Rutaceae*) has shown promising anti-obesity effects in male C57BL/6J mice; its dichloro methane extract DCM extract, halfordinol, aegeline, and esculetin help reduce white adipose tissue by inhibiting adipocyte hypertrophy and proliferation.[31] Umbelliferone and esculetin have been shown to prevent adipogenesis, enhance lipolysis, and inhibit pancreatic lipase, reducing fat accumulation.[32] Halfordinol, isolated from the leaves of *A. marmelos*, acts as a β3-adrenergic

receptor agonist, regulating lipid metabolism and increasing GLUT4 expression, which improves glucose uptake in adipose tissue.[33]

Agnimantha

(*Premna Serratifolia* Linn., *Lamiaceae*), Chloroform: Methanol extract of *P. integrifolia* root (CMPI) enhances fecal bile acid excretion, which decreases cholesterol levels and reduces the atherogenic index as demonstrated in female Swiss albino mice. Additionally, it inhibits catabolizing enzymes such as Lecithin Cholesterol Acyltransferase (LCAT), and Lipoprotein Lipase (LPL), thereby inhibiting peripheral lipolysis. These actions collectively contribute to regulating lipid metabolism and support weight management.[34]

Bibhitaki

(*Terminalia bellirica* (Gaertn.) Roxb., *Combretaceae*) The hot water extract of *Terminalia bellirica* fruit, which contains gallic acid, has been shown to exhibit anti-obesity and insulin-sensitizing effects in male TSOD and TSNO mice. It promotes adipocyte differentiation through a PPARγ-independent histone methylation and acetylation mechanism. It also demonstrates insulin-mimetic activity by improving glucose uptake in 3T3-L1 adipocytes and reducing HOMA-IR, thereby improving insulin sensitivity and metabolic function.[35]

Haritaki

(*Terminalia chebula* Retz., *Combretaceae*) Chebulinic acid suppresses adipogenesis in 3T3-L1 adipocytes by significantly reducing C/EBPα and PPARγ levels. It also decreases the expression of adipocyte-specific markers such as aP2 and adiponectin. Additionally, *Haritaki* inhibits protein tyrosine phosphatases PTPN11 and PTPN9, further contributing to its anti-adipogenic effects.[36]

Chitraka

(*Plumbago zeylanica* L., *Plumbaginaceae*) combats obesity through its active compound, Plumbagin (PLB), a Vitamin K3 analogue which increases AMPK phosphorylation and inhibits TNF-α and GM-CSF expression, as a study was conducted on four-week-old male C57BL6/J mice.[37] It reduces SREBP-1c mRNA, suppressing lipogenesis and stimulating fat oxidation. PLB also inhibits pancreatic lipase, increasing fecal cholesterol content, lowers serum fasting glucose, insulin, and HOMA-IR levels, enhancing metabolic health as demonstrated in experimental models of fructose-fed rats.[38]

Vasa

(*Adhatoda zeylanica* Medik., *Acanthaceae*) Antihyperlipidemic effect was assessed in a high-fat diet-induced hyperlipidemic model in Wistar albino rats. Aqueous extracts of leaves contain bioactive compounds like phenols and flavonoids, which exhibit anti-inflammatory and antioxidant properties, which can help inhibit lipid peroxidation. They decrease liver MDA formation and increase activities of -SH content, cytochrome b5, NADH-cytochrome b5 reductase, NADPH-cytochrome P450 reductase, cytochrome P450, DTD, GST, CAT, SOD, GPx, and GR as demonstrated by using *invitro* assays.[39]

Badara

(*Ziziphus mauritiana* Lam., *Rhamnaceae*) The Ziziphus jujuba Mill. Leaf extract contains betulinic acid that suppresses the PI3K/AKT signaling pathway, leading to reduced PI3KCA mRNA levels and decreased PPARγ and C/EBPα expression. As a result, this leads to elevated plasma levels of adiponectin and activation of AMPK, offering an anti-steatogenic effect using human adipocyte cell cultures.[40] Additionally, the extract inhibits PTB1 activity and induces GLUT4 expression, as the study was conducted in rat L6 myotubes.[41]

Vacha

(*Acorus calamus* Linn., *Acoraceae*) The anti-obesity action of ethanol extracts of *Vacha* has been demonstrated both in C57BL/Ks db/db mice and using the 3T3-L1 adipocyte differentiation model. Its primary mechanism involves inhibiting pancreatic lipase activity, which increases fat excretion in the feces. This process reduces fat absorption and adipose tissue accumulation, contributing to weight loss and lower levels of adiponectin.[42]

Saptaparna

(*Alstonia scholaris* Linn.) R.Br., *Apocynaceae*) Both alkaloid and saponin fractions of the *A. boonei* have demonstrated anti-obesity effects in the 3T3-L1 adipocytes model. Its mode of action involves the inhibition of pancreatic lipase, which reduces fat absorption in the intestine, and suppressing adipogenesis, which is the formation of new fat cells. It decreases overall lipid content in the body and promotes lipolysis, the breakdown of fats into free fatty acids and glycerol, for energy use.[43]

Ativisha

(*Aconitum heterophyllum* Wall., *Ranunculaceae*) Root samples of *A. heterophyllum* have been shown to reduce the enzymatic activity of HMG-CoA Reductase (HMGR), and increase Lecithin-Cholesterol Acyltransferase (LCAT) activity as demonstrated in an experimental model of Sprague Dawley (SD) strain rats[44] and inhibition of amylase and lipase activities by using *in vitro* assays.[45]

Nimbha

(*Azadirachta indica* A. Juss., *Meliaceae*) The ethanolic extract of *A. indica* contained the highest phenolic and flavonoid content, and has demonstrated anti-obesity effects in male Swiss albino rats. It involves inhibiting pancreatic lipase, reducing lipid and protein absorption, decreasing calorie intake, suppressing appetite,

impairing adipogenesis, activating AMP-activated protein kinase in key tissues, and improving systemic inflammation.[46] It has been shown to inhibit Dipeptidyl Peptidase-IV (DPP-IV) activity both *in vitro* and *in vivo* in High-Fat Fed (HFF) obese-diabetic rats.[47]

Indravaruni

(*Citrullus colocynthis* (L.) Schrad *Cucurbitaceae*) involves inhibiting STAT3 and PKB phosphorylation, exhibiting insulinotropic activity, and increasing AMPK activity using 3T3-L1 adipocytes as a cell model.[48] In the High-Fat Diet (HFD) induced obese rats model, it reduces the absorption of dietary lipids, boosts adiponectin levels, potentially enhances leptin signaling, lowers TNF-α and IL-6 levels as demonstrated by using, and suppresses pancreatic lipase activity.[49]

Kustha

(*Saussurea lappa* (Decne.) C.B.Clarke, *Asteraceae*) The aqueous extract of flowers of *Saussurea lappa* has demonstrated significant anti-obesity effects in *in vitro* assays. Its mechanism of action involves aiding the enzymatic hydrolysis of complex carbohydrate molecules. It inhibits amylase and lipase.[50] Acacetin, a flavone isolated from the plant studied on High-Fat Diet (HFD) induced obese mice suppresses aP2, FAS, and LPL protein expression while increasing AQP7 expression.[51]

Sarshapa

(*Brassica campestris* Linn., *Brassicaceae*) *Brassica juncea* L. leaf extract combats obesity by reducing lipid synthesis enzymes while enhancing β-oxidation through PPARα activation as demonstrated in Male Sprague-Dawley rats experimental models.[52] It suppresses adipogenesis by decreasing C/EBP-α and aP2 expression and inhibits lipid condensation via p-ACC/ACC regulation, as the experiment was conducted in both 3T3-L1 Preadipocytes and High-Fat Diet-Induced Obese C57BL/6J Mice.[53] Glucoraphanin present in the plant promotes fatty acid oxidation by modulating CPT-1, UCP1, and PGC-1α and inhibits adipogenesis and lipogenesis by increasing p-AMPK levels.[54] Erucic acid, present in the studied plant, was shown to act as a PPAR-γ agonist, regulating lipid metabolism and countering obesity in obese/diabetic KK-Ay mice and *in vitro* assays.[55]

Sunthi

(*Zingiber officinale* Roscoe., *Zingiberaceae*) Research indicates that ginger containing chrysin and galangin influences lipid and glucose metabolism in male Wistar rat models. In the liver, ginger upregulates ACOX1, CPT1, and PGC1α, decreases triacylglycerol content[56] and reduces lipid peroxidation. It inhibits hepatic gluconeogenesis and increases cholesterol 7α-hydroxylase activity[57] potentially blocking gut cholesterol absorption.[58] In adipose tissue, ginger enhances CPT1 levels, reduces MCP1 gene expression[59] and prevents Foxa2 inactivation[60] leading to reduced lipid accumulation via AMPK activation.[61] It blocks PPAR-γ[62] suppresses miR-375 and VAMP7[63] and decreases FASN expression[64] while increasing Pnpla2 (Atgl) and Lpl, enhancing lipid oxidation.[65] Ginger also inhibits adipogenesis through Nrf2 in 3T3-L1 cells and adiposity in diet-induced obese mice[66] and Akt/GSK3β pathways.[67] Ginger inhibits dietary lipid absorption[68] and pancreatic lipase activity[69] upregulates TGR5 in the intestine, and increases fecal lipid content.[70] It modulates metabolic pathways by activating Wnt/β-catenin, inhibiting JAK1-STAT3[71] controlling mTOR through AMPK[72] and increasing mitochondrial biogenesis via PI3K/Akt[73] Hormonal and enzyme effects[74] include increased ghrelin[75] reduced leptin, and inhibition of iNOS, HMG-CoA reductase[76] glucosidase[77] and amylase activity.[78] Overall, ginger enhances lipid metabolism, reduces lipid accumulation, and protects against hepatic steatosis[79] through multiple pathways.

Haridra

The study highlights the metabolic benefits of *Haridra* (Curcuma longa Linn., *Zingiberaceae*) contains curcumin, in C57BL/6J mice. It elevates the IRE1α sulfonation-SIRT1 degradation axis, improving adiponectin and reducing leptin levels. Haridra lowers,[80] PPAR-γ, C/EBPα, SREBP1and FAS expressions activate adrenergic receptors,[81] upregulates UCP-1, and enhances Brown Adipose Tissue (BAT) activity.[82] It reduces adipogenic differentiation[83] via Wnt/β-Catenin signaling[84] and boosts lipolysis and β-oxidation by upregulating ATGL, HSL, adiponectin, and AMPK phosphorylation.[85] It downregulates adipocyte protein 2, fatty acid synthase, acetyl-CoA carboxylase, and lipoprotein lipase. while upregulating β-oxidation enzymes.[86] and inhibiting NF-κB signaling.[87] Haridra also decreases leptin secretion, reducing appetite, and regulates immune markers by reducing CD11c and increasing CD206.[88] It enhances glucose uptake by upregulating GLUT4, GLUT2, and GLUT3, and inhibits PTP1B[89] and pancreatic lipase[90] Additionally, *haridra* improves mitochondrial respiratory capacity, induces browning of white adipocytes[91] through PPARγ, regulates AMPK, and NF-κB, activates AMPKα phosphorylation, reducing ACC[92] and FAS, decreases MAPK activity, and increases nuclear beta-catenin levels and blocking adipogenesis. Overall, *haridra* significantly improves lipid and glucose metabolism and enhances adipose tissue function.

Guggulu

(*Commiphora wightii* (Arn.) Bhandari, *Burseraceae*) *Commiphora myrrha* resin ethanolic extract contains guggulsterone that enhances the biogenesis of Peroxisome Proliferator-Activated Receptor-γ (PPARγ), peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC1α) demonstrated in the experimental model of male Wistar rats. It also boosts the expression of markers characteristic of the beige adipocyte phenotype, such as UCP1, T-box transcription factor 1 (TBX1),

UCP1 and β-3 Adrenergic Receptor (β-3AR).[93] Additionally, guggulsterone blocks NF-κB activation and inhibits IκBα kinase activation.[94] It also downregulates Fabp5 and Scd1, leading to decreased leptin levels and increased adiponectin levels.[95]

Khadir

(*Acacia catechu* (L.F.) Willd, *Leguminosae*) known for its active compound proanthocyanidins, exerts several beneficial effects through specific mechanisms, activates AMPK, and inhibits DPP-4, regulating glucose metabolism. It preserves CPT1 and UCP3,[96] suppresses pancreatic lipase, reduces oxidative stress markers (MDA) while enhancing antioxidant enzymes (SOD, GSH, catalase). Khadir modulates NF-κB and MAPK pathways,[97] increases PPARα/δ expression, boosts adiponectin, and reduces TNF-α secretion in adipocytes. It enhances GLUT4 in muscle,[28] Inhibits alpha-glucosidase/lipase and reduces glucose absorption[98] via SGLT1.[99] Khadir promotes bile acid excretion, lowers cholesterol, and suppresses GPR43 in fat, mitigating inflammation. These effects were observed in male KKAy mice.[100]

Maricha

(*Piper nigrum* L., *Piperaceae*) combats obesity through precise mechanisms: it inhibits pancreatic lipase, as a study was conducted by using *in vitro* assays.[101] Studies conducted in male Wistar rats showed enhanced lipid metabolism by promoting lipolysis and activating lipoprotein lipase.[102] and AMPK,[103] acts as a PPARγ agonist to prevent lipid accumulation, and inhibits α-glucosidase and α-amylase using *in vitro* assays.[104] enzymes and DGAT enzyme activity[105] to reduce carbohydrate absorption. It lowers liver cholesterol, triglycerides, phospholipids, and FFAs, boosts antioxidant enzymes (SOD, CAT, GPx),[106] and normalizes key metabolic and inflammatory markers (TNF-α, PPARγ, Fab-4, FAS, UCP-2, SREBP-1c. It increases thermogenesis via UCP-2 expression,[107] enhances plasma LCAT activity,[108] and improves adipogenesis-related transcription factors (PPARγ, SREBP-1c, C/EBPβ) mRNA expression using male Wistar Albino rats model[109] collectively aiding in obesity management.

Swetjiraka

The administration of *jiraka* (*Cuminum cyminum* Linn., *Apiaceae*), which contains cumin, significantly prevented mesenteric and epididymal fat deposition as the study was conducted in male Wistar rats. In 3T3-L1 preadipocytes, it reduced oxidative stress-related biomarkers such as Thiobarbituric Acid Reactive Substances (TBARS), Nitric Oxide (NO), and Advanced Oxidation Protein Products (AOPP) as demonstrated in 3T3-L1 preadipocytes.[110] Jeera also exhibited higher lipase, amylase, and glucosidase inhibitory activities,[111] contributing to its effectiveness in managing obesity.

Shigru

(*Moringa oleifera* Lam., *Moringaceae*) Isothiocyanate-rich moringa enhances lipolysis and the ratio of fat to carbohydrate oxidation, inhibits liver gluconeogenesis, and down-regulates GcK as the study was conducted using C57BL/6L mice fed a Very High Fat Diet (VHFD)[112] It regulates the expression of iNOS and NQO1 genes, improves glucose tolerance and lipid metabolism,[113] reduces Visceral Adipose Tissue (VAT) accumulation, and significantly decreases early adipogenesis markers, including C/EBPβ and C/EBPδ.[114]

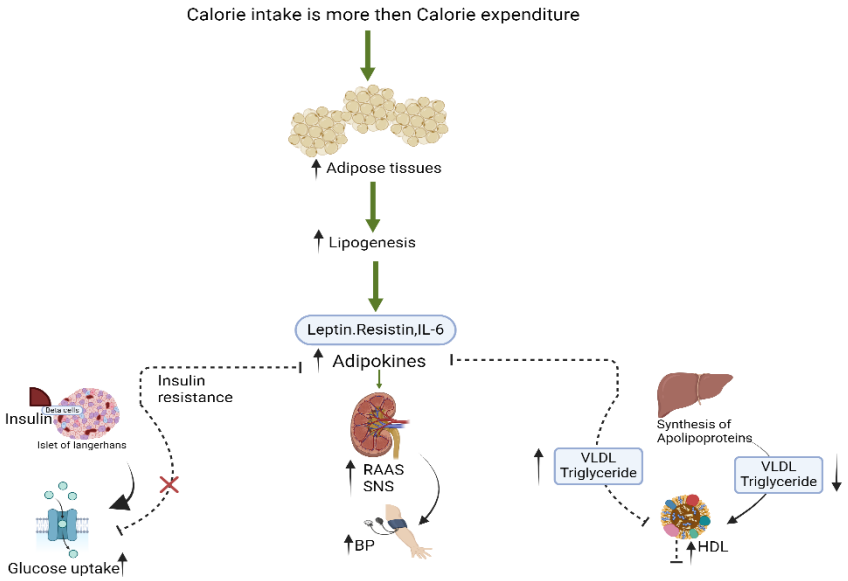

Figure 1: Obesity-induced Metabolic disruption.

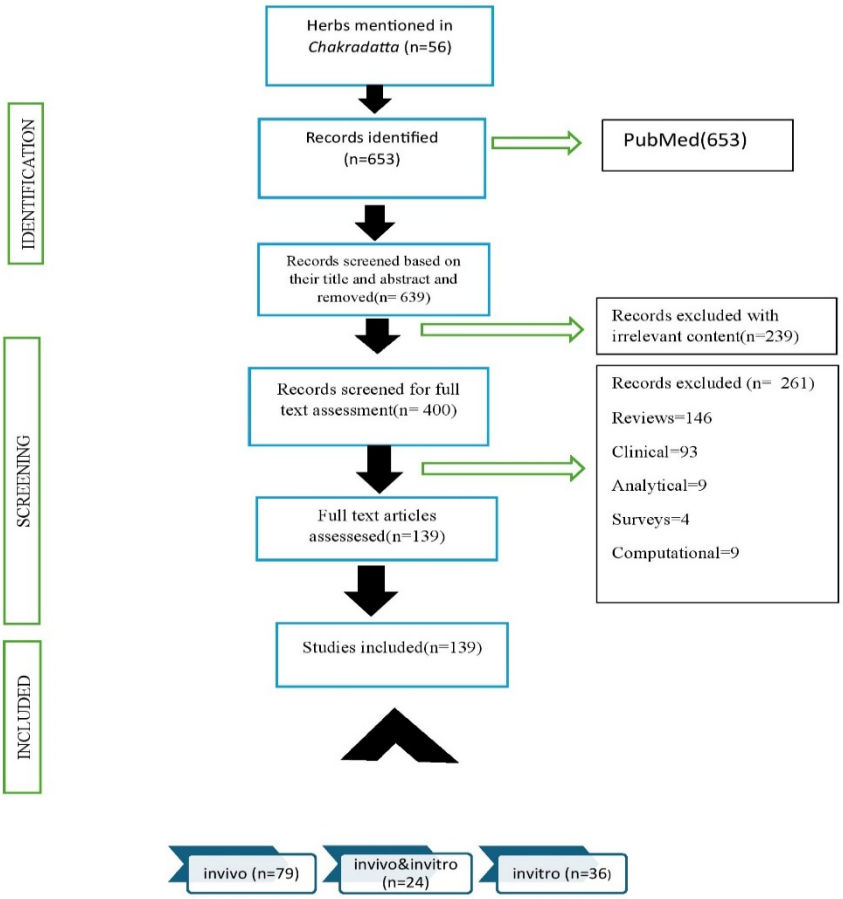

Figure 2: PRISMA chart: Process of identification and selection studies.

Kutki

(*Picrorhiza kurroa* Royle ex Benth, *Scrophulariaceae*) Apocynin present in *Picrorhiza kurroa* extract exhibits anti-adipogenic activity by downregulating key markers such as PPARα, adiponectin, and SREBP1. It also replicated the effects of insulin in the insulin-signaling pathway. These effects were observed *in vitro* studies using 3T3-L1 adipocytes and L6 myotubes.[115]

Dhanyaka

(*Coriandrum sativum* Linn., *Apiaceae*) expressed the Uncoupling Protein (UCP1) and β3-adrenergic receptors (β3AR) that mediate thermogenesis, both of which play a role in mediating thermogenesis in 3T3-L1 adipocytes.[116]

Amra

(*Mangifera indica* Linn., *Anacardiaceae*) Mango peel and mango seed aqueous extract both reduced the expression of PPARγ downstream and promoted the browning of adipocytes by activating AMPK. This led to a decrease in adipocyte Fatty-Acid Binding Protein (FABP4/aP2) and SREBP-1c levels. These effects were observed *in vitro* studies using 3T3-L1 adipocytes.[117] inhibiting lipogenesis and promoting fatty acid oxidation. The inhibition of α-glucosidase and PPAR-α activation prevented inflammation in adipose tissue and enhanced adiponectin action, ameliorating obesity-induced insulin resistance.[118] It also reduced collagen deposition in the liver, preventing fibrosis, and acted as a strong inhibitor of α-glucosidase.[119] The reduction in perilipin-2 levels activated lipolysis in adipocytes, counteracting ROS production and oxidative stress, as the study was conducted in a high-fat diet-induced obese mouse model of Four groups of Swiss albino mice.[120] This process upregulated mitochondrial bioenergetics, downregulated lipogenesis, and reduced Acac and Scd1 levels.[121] Additionally, it improved insulin secretion, possibly through incretin action, and enhanced anti-inflammatory cytokine production in the gut.[122] It increased relative mRNA levels of Adipor1, Irs1, and Slc2a4, with a tendency to also increase relative mRNA levels of Leprb, Insr, and Pfkfb3.[123]

Dadima

(*Punica granatum* Linn., *Lythraceae*) effectively inhibits pancreatic lipase and alpha-glucosidase[124] blocks adipocyte differentiation and reduces lipid accumulation by suppressing PPARγ and C/EBPα expression, as the study was conducted by using *in vitro* antioxidant, anti-diabetic, and anti-obesity inhibition traits of *Punica granatum* fruits peel extract. It also inhibits PI3K/Akt and TLR4/NF-κB pathways, altering cytokine

levels and promoting anti-inflammatory effects.[125] Activation of β-adrenergic receptors enhances thermogenesis and adipose tissue browning.[126] Uro-A and Uro-B treatments downregulate LXRα and SREBP1c[127] reducing lipid synthesis and increasing fatty acid oxidation.[128] Pomegranate Extract (PE) containing punicalagin sufficiently prevented High-Fat Diet (HFD)-induced obesity by AMPK activation that boosts mitochondrial function and ATP production while suppressing lipogenesis[129] MCP-1 inhibition[130] decreases MUFA levels and suppresses delta-9 desaturation[131] further aiding in lipid management.

Misreya

(*Foeniculum vulgare* Mill., *Apiaceae*) increase leptin receptor expression and inhibit adipogenesis by reducing intracellular triglyceride levels. This is achieved primarily through down-regulating the expression of transcription factors involved in adipogenesis, such as leptin, C/EBPα, and PPARγ. The study was conducted using adult male BALB/c mice[132] Fennel seeds also act as inhibitors of amylase, lipase, and glucosidase, as studied *in vitro* assays.[133]

Guduchi

(*Tinospora cordifolia* (Willd.) Miers *ex* Hook.f. & Thomson, *Menispermaceae*). The hydroalcoholic extract of *Tinospora cordifolia* administered to Swiss albino female mice was found to reduce HOMA-IR levels and downregulate PPAR-γ expression, while causing a slight upregulation in SREBP-1c expression.[134]

Suksmaila

(*Elettaria cardamomum* (L.) Maton, *Zingiberaceae*) Cardamom Seeds reduce the MDA concentration in plasma and liver, and decrease in APOP concentration in both plasma, as observed in male Wistar rats.[135] and liver, nitric oxide normalized plasma free fatty acids concentrations, liver enzyme activity[136] increase in HSL phosphorylation, suggesting increased lipolytic activity. increases mitochondrial activity by activating a mechanism associated with increased AMPK content, demonstrated in C57BL/6 mice[137] downregulated the TNF-α and NF-κB while upregulated the expression of Nrf-2.

Hapusha

(*Juniperus communis* Thunb., *Cupressaceae*) *Juniperus chinensis* hot water extract was shown to down-regulate genes in epididymal adipose tissue linked to adipogenesis, including PPARg2, aP2, SREBP1c, FAS, HMGR, UCP2, and UCP3, and enhance AMPK protein expression and phosphorylation in visceral adipose tissue.[138] The study was conducted in a rodent model with HFD-induced obesity.

DISCUSSION

The frequencies of traditional herbs mentioned in the *Chakradatta* in Figure 3 provide additional insights into their potential significance. Herbs frequently listed, such as *Amalaki* (*Phyllanthus emblica*), *Haritaki* (*Terminalia chebula*), and *Bhibhitaki* (*Terminalia bellirica*), are highlighted for their prominent roles in traditional obesity management. This high frequency underscores their established importance and potential effectiveness in targeting multiple metabolic pathways. The consistent mention of these herbs aligns with their broad pharmacological activities observed in this study, reinforcing their potential as key therapeutic agents in comprehensive obesity management strategies. Further investigation into the specific

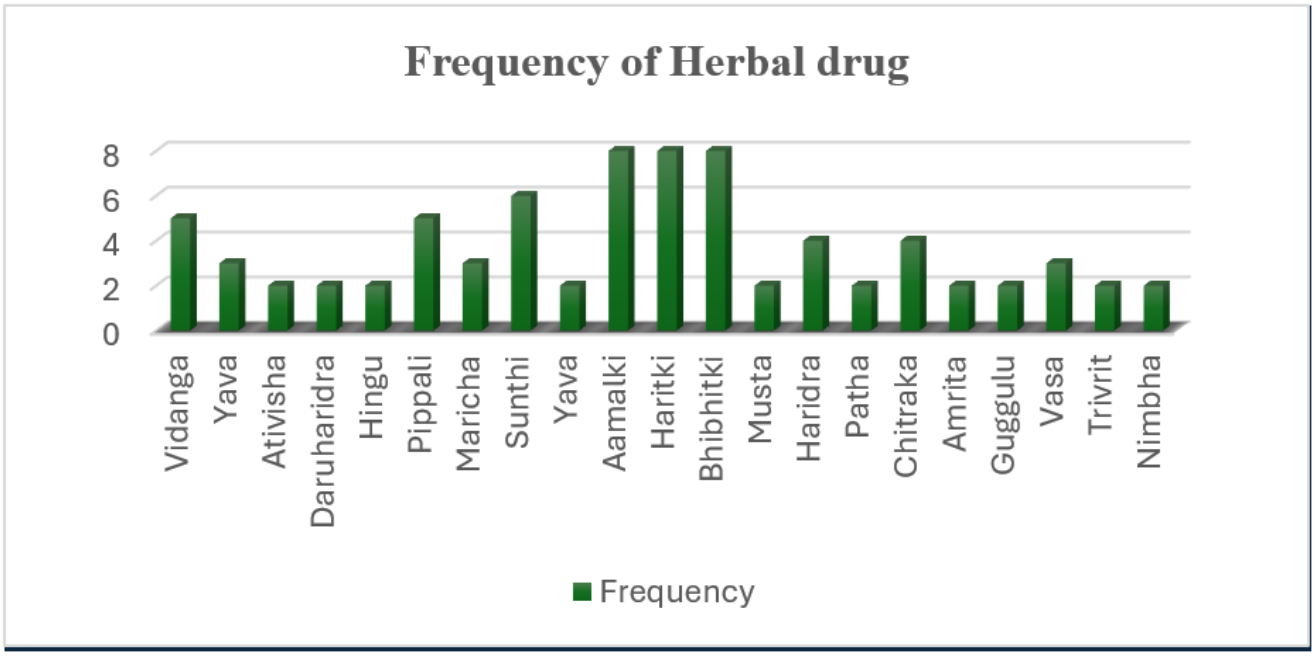

Figure 3: Summarizing the frequency of each herbal drug mentioned in the chapter on Obesity (*Sthoulya*) in *Chakradatta*.[14]

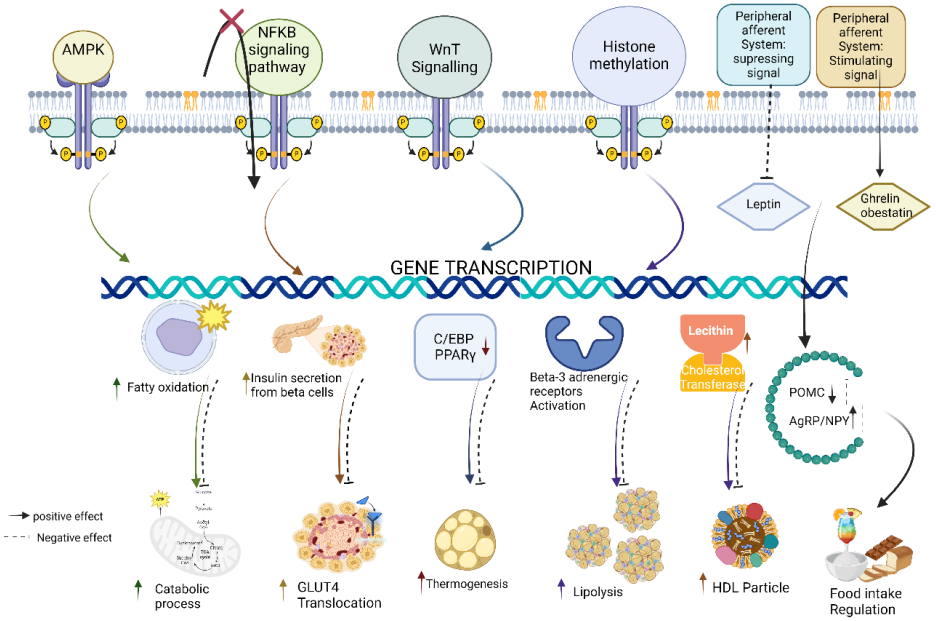

Figure 4: Gene Transcription Pathways Involved in Anti-Obesity Effects.

bioactive compounds from these families is crucial for developing targeted interventions for obesity and metabolic syndrome. This study aimed to identify and analyze the pathways involved in anti-obesity action. Research has led to the identification of 14 distinct pathways mentioned in Figures 4 and 5 that contribute to preventing and reducing obesity. The complex mechanisms by which these herbs exert their effects offer valuable insights into their potential as therapeutic agents for managing obesity. The study explores a variety of biochemical pathways, their mechanisms of action, outcomes, and the herbs that influence these processes. PPARγ agonists, which include herbs such as *Vidanga* (*Embelia ribes*), *Amalaki* (*Phyllanthus emblica*), *Musta* (*Cyperus rotundus*), *Haritaki* (*Terminalia chebula*), *and Sarshapa* (*Brassica campestris*), activate AMPK to increase fatty acid oxidation, leading to decreased leptin levels and increased adiponectin levels, thereby stimulating catabolic processes. HMG-CO reductase inhibitors, influenced by herbs like *Bhibhitaki* (*Terminalia bellirica*), *Chitraka* (*Plumbago zeylanica*), *Vasa* (*Adhatoda zeylanica*), and *Guduchi* (*Tinospora cordifolia*), reduce mevalonic acid formation, activating SREBP-2 and increasing cholesterol biosynthesis. UCP1 activity, stimulated by *Yava* (*Hordeum vulgare*) and *Dhanyaka* (*Coriandrum sativum*), activates Brown Adipose Tissue (BAT), promoting non-shivering thermogenesis and higher energy expenditure. Lipoprotein lipase inhibitors, associated with herbs such as *Vacha* (*Acorus calamus*), *Saptaparna* (*Alstonia scholaris*), *Nimbha* (*Azadirachta indica*), *Kustha* (*Saussurea lappa*), *Khadir* (*Acacia catechu*), *Haridra* (*Curcuma longa*), *Sunthi* (*Zingiber officinale*), and *Shigru* (*Moringa oleifera*), reduce triglyceride breakdown, resulting in decreased fat storage and accumulation. Alpha-glucosidase inhibitors, such as *Maricha* (*Piper nigrum*), *Amra* (*Mangifera indica*), and *Misreya* (*Foeniculum vulgare*), increase GLP1 levels, slow digestion, enhance insulin sensitivity, and improve glucose uptake, ultimately reducing food intake.

Bile acid sequestrants like *Agnimantha* (*Premna Serratifolia*) bind bile acids in the intestine, disrupt enterohepatic circulation, increase bile acid synthesis in the liver, upregulate LDL receptors, and decrease blood LDL cholesterol levels. Increased LCAT activity, promoted by *Ativisha* (*Aconitum heterophyllum*) and *Pippali* (*Piper longum*), enhances cholesterol esterification, leading to the formation of HDL particles. P13K/AKT pathway inhibitors, influenced by *Sunthi* (*Zingiber officinale*), *Amra* (*Mangifera indica*), and *Hapusha* (*Juniperus communis*), activate AMPK to increase SIRT1, modulating transcription factors and preventing macrophage infiltration and inflammation in adipose tissue. Insulinotropic agents such as *Indravaruni* (*Citrullus colocynthis*) and *Kutki* (*Picrorhiza kurroa*) enhance insulin secretion from pancreatic beta cells, facilitating glucose uptake and reducing postprandial blood glucose levels. Activation of beta-3 adrenergic receptors, regulated by *Bilva* (*Aegle marmelos*), *Guggulu* (*Commiphora wightii*), and *Dhanyaka* (*Coriandrum sativum*), promotes lipolysis and thermogenesis, increasing lipid metabolism and energy expenditure.

Cholesterol 7-alpha hydroxylase, activated by *Sunthi* (*Zingiber officinale*), converts cholesterol into 7-alpha-hydroxycholesterol, initiating primary bile acid synthesis and promoting cholesterol elimination through bile. Canonical Wnt signaling, influenced by *Sunthi* (*Zingiber officinale*) and *Haridra* (*Curcuma longa*), inhibits adipocyte differentiation by suppressing adipogenic transcription

Figure 5: Herbal Modulation of 14 Key Pathways in Anti-Obesity Action.

factors (C/EBP and PPARγ), potentially promoting adipose tissue browning. Histone methylation and acetylation activators like *Bhibhitaki* (*Terminalia bellirica*) promote preadipocyte differentiation into mature adipocytes, enhancing fat storage capacity and regulating adipogenesis. Lastly, NFKB signaling pathway inhibitors, such as *Vidanga* (*Embelia ribes*) and *Guggulu* (*Commiphora wightii*), reduce pro-inflammatory mediators and promote genes involved in adipogenesis and lipid storage, thus reducing inflammation and improving insulin sensitivity in peripheral tissues like adipose tissue, liver, and muscle.

This integration of historical and modern perspectives highlights the enduring relevance of these traditional remedies and their potential to complement contemporary approaches in obesity treatment.

Limitations of the Study

The difference in activity between the crude raw drug and its extract highlights certain inherent limitations of the present study. Most of the cited research predominantly emphasizes isolated bioactive compounds, often overlooking the complex synergistic interactions present in the whole plant or formulation. As a result, such studies serve primarily as a foundation for identifying potential pharmacological leads rather than establishing definitive biochemical pathways. While animal studies provided valuable insights into biological mechanisms, the differences in metabolism and physiology between animal models and humans may limit the direct applicability of these findings to human obesity. *In vitro* experiments, while useful for clarifying molecular interactions, cannot fully replicate the complex interplay of genetic, environmental, and behavioural factors that contribute to obesity in humans. Additionally, a thorough literature search using databases like PubMed revealed a significant gap in recent scientific research specifically investigating the anti-obesity effects of herbs documented in ancient texts such as *Chakradatta Samhita*. Herbs like *Patha* (*Cissampelos pareira* L.,) *Ushira* (*Vetiveria zizanioides* Nash inSmall), *Kutaja* (*Holarrhena antidysenterica* (L.)Wall.), *Alambusha* (*Biophytum sensitivum* (L.)DC), *Hingu* (*Ferula narthex* Boiss.), *Yavakshara* (*ash of Hordeum vulgare*), *Chandan* (*Santalum album* L.), *Bala* (*Sida cordifolia* L.), *Kantkari* (*Solanum virginianum* L.), *Daruharidra* (*Berberis aristata* DC.), *Salparni* (*Desmodium gangeticum* (L.) DC.), *Talmuli* (*Curculigo orchioides* Gaertn.), *Trivrita* (*Operculina turpethum* (L.)Silva Manso), *Snuhi* (*Euphorbia neriifolia* L.), *Sati* (*Hedychium spicatum* G.Lodd.), *Aaragbadh* (*Cassia fistula* L.), *Lamajjaka* (*Cymbopogon jwarancusa subsp.* olivieri (Boiss.)Soenarko), *Nagkesar* (*Mesua ferrea* L.), *Lodhra* (*Symplocos racemosa* Roxb.), *Kevuka* (*Costus speciosus* (J.Koenig)Sm.), *Nirgundi* (*Vitex negundo* L.), *Dhanyaka* (*Coriandrum sativum* L.), *Saptaparna* (*Alstonia scholaris* (L.) R.Br), *Ativisha* (*Aconitum heterophyllum* Wall.), *Kutki* (*Picrorhiza kurroa* Royle exBenth.), and *Guduchi* (*Tinospora cordifolia* (Willd.)Miers exHook.f. &Thomson) lack substantial modern scientific validation. This highlights the need for rigorous studies, including phytochemical analyses and clinical trials, to explore their potential therapeutic benefits, effectiveness, safety, and mechanisms of action in managing obesity. Therefore, future research must prioritize these areas to establish these herbs' efficacy, safety, and optimal usage in human populations.

CONCLUSION

This review critically explored traditional Ayurvedic herbs mentioned in *Chakradatta*, a classical text authored by *Acharya Chakrapanidatta*, renowned for its practical and therapeutic insights. Recognized for its historical significance and clinical applicability, *Chakradatta* presents detailed herbal formulations for various conditions, including obesity, making it a key reference for integrating traditional knowledge with modern Ayurvedic practice.

The pharmacological potential of these herbs was highlighted through evidence from *in vitro* and *in vivo* studies, revealing their capacity to act on multiple metabolic pathways. These include modulation of fatty acid oxidation, enhancement of thermogenesis, regulation of insulin sensitivity, and inhibition of key enzymes involved in lipid accumulation. Such a multifaceted mechanism of action provides a more comprehensive and integrated approach to obesity management than conventional single-target therapies.

The active phytoconstituents present in these herbs show considerable promise for pharmaceutical development. Their ability to target obesity at the molecular level offers safer, more natural, and potentially sustainable alternatives to current anti-obesity drugs. However, to fully harness their therapeutic potential, further research is essential particularly in validating their mechanisms, assessing long-term safety and efficacy, and optimizing formulations for clinical use.

ABBREVIATIONS

NFHS-5: National family health survey 5; **TBARS:** Thiobarbituric acid reactive substances; **PKC:** Protein kinase C; **GP11b/111a:** Glycoproteins; **IL6:** Interleukin-6**; STAT3:** Signal trasducer and activator of transcription 3; **NFKB:** Nuclear factor kappa B; **PPAR y:** Peroximase proliferator-activated receptor; **PI3K/AKT:** Phosphoinositide 3 kinase/protein kinase B; **GLUT-4:** Glucose tranporter type 4; **C/EBP Alpha:** CAAT/enhancer binding protein alpha; **FAB4:** Fatty acid binding protein 4; **LPL:** Lipoprotein lipase; **HMG-COA:** Hydroxymethylglutaryl CoA; **SCFA:** Short chain fatty acids; **PGC-1 Alpha:** Peroxisome proliferator activated receptor gamma coactivator; **B-AR:** Beta adrenergic receptor agonist; **POMC:** Propiomelanocortin; **CART:** Cocaine and amphetamine regulated transcript; **CRH:** Corticotrophic releasing hormone (normalized protein expression); **AgRP:** Agouti-related hormone; **MC4:** Melanocortin 4 receptor; **GLp:** Glucagon like-peptide1; **HOMA-IR:** Homeostatic Model Assessment of insulin resistance; **PTPN11:** Tyrosine-protein phosphatase non-receptor type 11; **TNF Alpha:** Tumour necrosis factor alpha; **DPPIV:** Dipeptidyl peptidase-4; **PKB:** Protein kinase B; **STAT3:** Signal transducer and activator of transcription3; **SREBP1C:** Sterol regulatory element binding protein 1c; **ACC:** Acetyl Coenzyme A Carboxylase 1; **AMPK:** Activated protein kinase; **ACOX1:** acyl-CoA oxidase 1; **CPT1:** Carnitine palmitoyltransferase 1; **PGCIAlpha:** Peroxisome proliferator activated receptor gamma coactivator I-alpha; **MCP1:** Monocyte chemoattractant protein-1; **VAMP1:**Vesicle-associated membrane protein-1; **FASN:** Fatty acid synthase; **NRF2:** Nuclear factor (erythroid-derived 2)-like 2; **AKT/GSK3Beta:** Protein kinase/Glycogen synthase kinase-3 beta; **TGR5:** Takeda G protein-coupled receptor 5; **WnT:** Wingless related integration site; **JAK/STAT:** Janus Kinase/Signal transducer and activators of transcription; **DGAT:** Diacylglycerol acyltransferases; **GPR41:** Free fatty acid receptor 3; **GPR43:** Free fatty acid receptor 2; **PYY:** Pancreatic peptide YY; **SOD:** Superoxide dismutase; **CAT:** Catalase; **BAT:** Brown adipose tissues; **LCAT:** Lecithin cholesterol acyl transferase; **AQP7:** Aquaprotein 7; **TBX1:** T-box transcription factor; **NO:** Nitric oxide; **AOPP:** Advanced oxidation protein products; **GcK:** Glucokinase; **Inos:** Indicible nitric oxide synthetase; **NQO1:** NADPH dehydrogenase quinone1; **VAT:** Visceral adipose tissue; **UCP-1:** Uncoupling protein 1; **AP2:** adipocyte lipid chaperone; **ROS:** Reactive oxygen species; **Acac:** Acetyl CO-A Carboxylase 1; **ScD1:** Stearoyal CO-A desaturase; **IRS1:** Insulin receptor substrate 1; **SLC2a4:** Solute carrier famile 2 member 4; **Leprb:** Leptin receptor long isoform; **InSR:** Heterozygous insulin receptor; **PfKfB:** 6-Phosphofructo-2-kinase; **TLRG:** Toll like receptor-4; **UroA:** Urolithiase A; **UroB:** urolithiase B; **LXRalpha:** Liver x receptors; **ATP:** Adenosine triphosphate; **MUFA:** Monounsaturated fatty acids; **MDA:** Melondialdehyde; **APOP:** Amyloid precursor protein; **HSL:** Hormone sensitive lipase; **NrF-2:** Nuclear factor erythroid-2; **Alpha P2:** Adipocyte p2; **HMGR:** 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHORS' CONTRIBUTION

Arushi Saroch: Conceptualization, Methodology/ Study design, Formal analysis, Investigation, Resources, Data curation, Writing – original draft, Writing – review and editing, Visualization, Project administration. Giramalla Patil, Swapnil Y Chaudhari, Pramod Yadav, Galib Ruknuddin, Pradeep Kumar Prajapati: Conceptualization, Methodology / Study design, Validation, Formal analysis, Writing – review and editing, Visualization, Supervision, Project administration.

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