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A Review of Turmeric (*Curcuma longa*) and Chicory (*Cichorium intybus*) in Metabolic and Hepatic Health

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ABSTRACT

Metabolic syndrome and progressive hepatic dysfunctions -- which can include non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH), chemical hepatotoxicity, and dyslipidemia—have become a major global healthcare challenge caused by systemic low-grade inflammation and oxidative stress. Conventional pharmacological approach frequently face limitations regarding long-term tolerability, variable efficacy, and side-effect profiles, encouraged renewed scientific investigation into bioactive botanical therapeutics. *Curcuma longa* (Turmeric, family Zingiberaceae) and *Cichorium intybus* (Chicory, family Asteraceae) represent two historically prominent medicinal plants documented extensively in traditional Ayurvedic, Unani, and Chinese medical systems. This review synthesizes comparative biochemical profiles, molecular mechanisms, and pharmacological evidence supporting the therapeutic utility of *C. longa* (primarily via curcuminoids) and *C. intybus* (via inulin, sesquiterpene lactones, and polyphenols) in metabolic and hepatic homeostasis. Curcumin effectively attenuates pro-inflammatory signaling (NF-κB, TNF-α, IL-1β, COX-2), promotes phase II antioxidant enzyme transcription via the Nrf2 axis, suppresses hepatic lipid accumulation, and mitigates serum transaminases (AST, ALT) and alkaline phosphatase (ALP). Concurrently, *C. intybus* acts as a potent hepatoprotective and cholagogic agent, replenishing endogenous glutathione pools, counteracting lipid peroxidation, and exerting prebiotic glycemic and lipid-lowering controls through inulin-mediated microbial modulation and hepatic lipogenesis downregulation. Furthermore, the review delineates pharmacokinetics, bioavailability hurdles, toxicity considerations, and the scientific rationale for future synergistic combination strategies of both botanical agents.

Introduction

Metabolic disorders, characterized by impaired insulin sensitivity, atherogenic dyslipidemia, elevated circulating inflammatory cytokines, and non-alcoholic fatty liver disease (NAFLD), represent interconnected clinical pathologies sharing core etiological roots: systemic lipotoxicity and unmitigated oxidative stress. Under sustained cellular metabolic strain, reactive oxygen species (ROS) trigger macromolecular lipid peroxidation and mitochondrial dysfunction, stimulating pro-inflammatory cytokine secretion (e.g., TNF-α, IL-6, IL-1) via the activation of nuclear factor kappa B (NF-κB) cascades. In hepatic tissue, this cascade precipitates hepatocyte ballooning, steatosis, membrane permeability disruption, and leakage of intracellular hepatic enzymes—such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP)—into systemic circulation. Because of the complex etiology of metabolic-hepatic syndromes, multi-target phytochemical interventions have emerged as compelling therapeutic and adjunctive modalities. Botanical agents have attracted significant clinical and experimental attention. Among ancient medicinal herbs, Turmeric (*Curcuma longa* L., Zingiberaceae) and Chicory (*Cichorium intybus* L., Asteraceae) stand out for their verified multi-pathway pharmacological activities.

Curcuma longa, historically acknowledged across Sanskrit medical texts (Ayurvedic Compendia such as *Susruta*) under names such as Haridra, Mehagni (eliminator of fat), and Vishagni (destroyer of poisons), is extensively cultivated across India and Southeast Asia. Its golden rhizome yields non-toxic bioactive curcuminoids that modulate cytokine expression, scavenge free radicals, normalize

serum transaminases, and inhibit lipid accumulation. Concurrently, *Cichorium intybus* (commonly termed Kasni or Chicory), a versatile perennial herb belonging to Asteraceae, has been utilized worldwide as a digestive stimulant, bitter stomachic, diuretic, and classical liver tonic. Rich in prebiotics (inulin), sesquiterpene lactones, caffeic acid derivatives, and flavonoids, chicory demonstrates strong membrane-stabilizing, antihepatotoxic, hypocholesterolemic, and hypoglycemic actions (S. Nandagopal and B. D. Ranjitha Kumari, 2007). While both botanicals have been investigated individually, their converging pathways across metabolic regulation and hepatic cytoprotection present exceptional synergistic opportunities. This review provides a comprehensive comparative synthesis of the bioactive constituents, pharmacological activities, glycemic and lipid – lowering actions, pharmacokinetic considerations, and clinical potentials of *Curcuma longa* and *Cichorium intybus*.

2. Botanical Profiles and Bioactive Phytochemical Diversity

2.1 *Curcuma longa* (Turmeric)

Curcuma longa is a sterile, herbaceous perennial characterized by thick, ringed, aromatic underground rhizomes that serve as the main repository of medicinal metabolites. Proximate nutritional analysis of dried rhizome meal indicates carbohydrates (69.4%), moisture (13.1%), crude protein (6.3%), lipids/fats (5.1%), and total minerals/ash (3.5%) (Prasad *et al.*, 2014). The primary pharmacological properties of turmeric are driven by two major fractions: volatile essential oils (3.0%–7.0%, containing turmerones, atlantone, and zingiberene) and non-volatile polyphenolic curcuminoids.

Curcuminoids constitute roughly 3%–8% of dried turmeric by mass (varying according to seasonal agro-climatic parameters) and comprise three primary distinct analogs:

1. **Curcumin (Curcumin I):** Diferuloylmethane, chemical formula " $C_{21}H_{20}O_6$, molecular weight 368.38" g/mol", representing approximately 70%–77% of the curcuminoid pool.
2. **Demethoxycurcumin (DMC, Curcumin II):** Formula $C_{20}H_{18}O_5$, composing roughly 15%–20%.
3. **Bisdemethoxycurcumin (BDMC, Curcumin III):** Formula " $C_{19}H_{16}O_4$, constituting 3%–10% (Goel *et al.*, 2014).

Structurally, curcumin (1,7-bis-(4-hydroxy-3-methoxyphenyl)-hepta-1,6-diene-3,5-dione) or diphenylmethane (Deogade and Ghate, 2015) exists in keto-enol tautomeric equilibrium. Its phenolic hydroxyl domains, coupled with the central β -diketone conjugated diene moiety, dictate its exceptional capacity to trap and neutralize free reactive oxygen and nitrogen radicals (OH $\cdot$, O $_2^{\cdot-}$, NO $\cdot$, NO $_2$, and singlet oxygen $^1O_2$ (Negi, 1999).

Phytochemical Category	Key Identified Compounds	Major Biological Implication	References
Curcuminoids	Curcumin (diferuloylmethane), Demethoxycurcumin, Bisdemethoxycurcumin	NF-κB suppression, lipid-lowering, radical scavenging, Nrf2 activation	Goel <i>et al.</i> , 2008; Prasad <i>et al.</i> , 2014
Sesquiterpene Volatiles (volatile oils)	α -Turmerone, β -Turmerone, Atlantone, Curlone, Zingiberene	Antimicrobial, anti-inflammatory, membrane fluidity modifier, bioavailability enhancement	Chandra <i>et al.</i> , 1972; Negi, 1999
Soluble Phenolics & Pigments	Anthocyanins (18.9–37.0 μ g/g), Tannins (0.32%–0.76%), Phenols (0.15%–0.62%)	Interfacial antioxidant defense, inhibition of microsomal lipid peroxidation	Niranjan <i>et al.</i> , 2003; Sarangthem & Haokip, 2010

2.2 Cichorium intybus (Chicory): *Cichorium intybus* is an erect biennial or perennial taproot herb (from Asteraceae family) producing rosette leaves, stout fibrous roots, and distinct bright lavender-blue composite flower heads (Street *et al.*, 2013). Historically celebrated in ethnomedicine as a cholagogue, liver purifier, and febrifuge, chicory roots contain distinct phytochemical complexes differing profoundly from ginger-family botanicals:

Fructan Polysaccharides (Inulin): The tuberous roots contain extensive reserves of inulin (40%–60% of dry weight), a linear β -(2" $\rightarrow$ 1") linked fructosyl polymer with a terminal glucose unit. Inulin resists upper gastrointestinal hydrolysis, functioning as a non-digestible prebiotic fiber that fuels beneficial probiotic fermentation in the lower gut (Pool – Zobel *et al.*, 2005).

Bitter Sesquiterpene Lactones: Concentrated predominantly in root laticifers, these include guaianolides such as lactucin, lactucopicrin, and 8-deoxylactucin, as well as the phytoalexin cichorelaxin. These bitter principles drive hepatobiliary secretion, enhance appetite/digestion, and exhibit marked antimicrobial and anti-inflammatory properties.

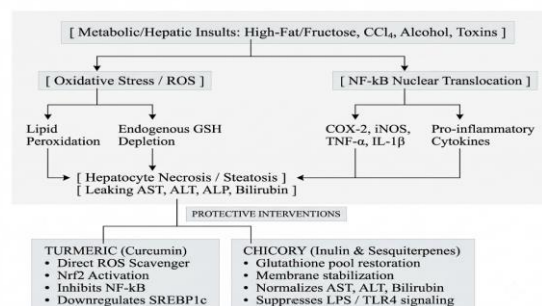
Caffeic Acid Derivatives and Polyphenols: Chicory root tissues are enriched with hydroxycinnamic acids (chicoric acid, chlorogenic

acid, caffeic acid, isochlorogenic acid), coumarins (esculin, cichoriin, umbelliferone), and flavonoids (quercetin, luteolin, apigenin glycosides) and tannins as secondary metabolites.

Aliphatic and Triterpene Lipids: Solvent extraction confirms the presence of essential volatile components, plant fatty acids, and pentacyclic triterpenes extracted in hexane and non-polar phases.

3. Mechanisms in Hepatic Protection and Cellular Defense

The liver serves as the central orchestration site for intermediate nutrient turnover, xenobiotic biotransformation, and antioxidant reserve maintenance. Hepatic insults—induced by alcohol consumption, toxic chemicals (e.g., carbon tetrachloride, acetaminophen), high-fat diets, or industrial chemicals—converge on endoplasmic reticulum stress, cellular glutathione (GSH) depletion, and mitochondrial membrane damage in hepatic cells. Both turmeric and chicory intercept these injurious cascades through complementary antioxidant and immunomodulatory pathways.



3.1 Curcumin-Mediated Hepatoprotection

The hepatoprotective capacity of *C. longa* is largely governed by curcumin's ability to boost endogenous redox capacity and shut down chronic inflammatory cascades:

Serum Biomarker Normalization: In experimental models of alcohol-induced liver toxicity and chemical insult, curcumin administration markedly reduces pathologically elevated serum aspartate transaminase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP). It halts necrotic hepatocyte breakdown, preserving structural membrane integrity and containing transaminases within intracellular compartments.

Suppression of Pro-Inflammatory Mediators: Curcumin directly blocks the activation and nuclear translocation of NF- κ B, leading to the direct transcriptional downregulation of pro-inflammatory cytokines, specifically tumor necrosis factor- α (TNF- α), interleukin-1 beta (IL-1 β), interleukin-6 (IL-6), as well as inducible enzymes such as cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) (Rahman and Biswas, 2009).

Induction of Phase II Detoxification via the Nrf2 Axis: Curcumin disrupts the Keap1-Nrf2 inhibitory complex, enabling the nuclear entry of nuclear factor erythroid 2-related factor 2 (Nrf2). This activates antioxidant response elements (ARE), upregulating transcription of protective enzymes: superoxide dismutase ("SOD"), catalase (CAT), glutathione peroxidase (GPx), and glucose-6-phosphate dehydrogenase (G6PD), concurrently replenishing depleted cellular reduced glutathione (GSH).

3.2 Cichorium intybus-Mediated Hepatoprotection and Cholagogue Action

Ethnobotanical literature designates chicory as an exceptional "liver tonic" (Yakrit pleeha hara) capable of resolving splenomegaly, hepatomegaly, and bilious jaundice.

Antitoxic and Membrane Stabilization: Preclinical screening of *C. intybus* extracts in chemically stressed animal models (e.g., carbon tetrachloride, acetaminophen, and thioacetamide toxicity) demonstrates that chicory extracts significantly suppress serum transaminase elevation (ALT, AST) and total bilirubin accumulation. The active phytochemicals prevent carbon tetrachloride-induced cytochrome P450 activation to free radical trichloromethyl radicals (CCl $_3$), directly preventing widespread lipid peroxidative lysis of hepatocyte membranes.

Restoration of Hepatic Redox Reserves: Chicory root extracts enhance hepatic SOD, catalase, and non-protein sulfhydryl (GSH) concentrations, protecting mitochondrial structural integrity against oxidative damage.

Choleretic and Cholagogic Modulation: Chicory sesquiterpene lactones (lactucin, lactucopicrin) act as bitter principles that stimulate hepatocyte bile acid secretion (choleretic activity) and stimulate gallbladder contraction (cholagogic action), accelerating the biliary clearance of conjugated toxins and eliminating hepatic congestion.

4. Regulation of Metabolic Syndrome, Glycemia, and Lipid Profiles

Metabolic syndrome is defined by coexisting visceral obesity, dyslipidemia (high triacylglycerols, elevated low-density lipoprotein cholesterol [LDL-C], decreased high-density lipoprotein cholesterol [HDL-C]), peripheral insulin resistance, and impaired glucose tolerance. Both *C. longa* and *C. intybus* exert broad regulatory control across this metabolic cluster.

Metabolic Feature	<i>Curcuma longa</i> (Curcuminoids)	<i>Cichorium intybus</i> (Inulin/Lactones)
Glycemic Control & Insulin Sensitivity	<ul style="list-style-type: none"> Enhances insulin receptor tyrosine phosphorylation Stimulates postprandial insulin response (OGTT) Reduces insulin resistance 	<ul style="list-style-type: none"> Inulin delays glucose uptake in the intestinal lumen Enhances pancreatic β-cell protective status Acts as low-calorie substrate
Hepatic Lipogenesis & Lipid Accumulation	<ul style="list-style-type: none"> Downregulates SREBP-1c and FAS gene transcription Activates AMPK-driven lipid β-oxidation 	<ul style="list-style-type: none"> Suppresses SREBP-1c and fatty acid synthesis pathways Inulin decreases intestinal free fatty acid absorption
Gut Microbiota Modulation	<ul style="list-style-type: none"> Low systemic bioavailability modulates intestinal microbiome composition and diversity Preserves gut barrier integrity and attenuates metabolic endotoxemia 	<ul style="list-style-type: none"> Core prebiotic: selective growth of <i>Bifidobacterium</i> and <i>Lactobacillus</i> Fermentation yields SCFAs (acetate, propionate, butyrate) attenuating inflammation

4.1 Anti-Diabetic and Hypoglycemic Activities

Turmeric: Curcumin enhances whole-body glucose disposal by activating adenosine monophosphate-activated protein kinase (AMPK) and attenuating cellular insulin resistance. Clinical studies (Wickenberg *et al.*, 2010) confirmed that acute oral intake of turmeric significantly amplifies the postprandial insulin release profile in oral glucose tolerance testing (OGTT), compensating for peripheral insulin insensitivity and helping prevent the transition from prediabetes to overt Type-2 diabetes (Ghorbani *et al.*, 2014).

Chicory: Due to its non-digestible β -(2 $\rightarrow$ 1) glycosidic linkages, inulin possesses minimal glycemic impact, making chicory extracts safe and beneficial in diabetic nutritional interventions. Inulin slows gastric emptying, delays postprandial glucose absorption across intestinal enterocytes, improves insulin sensitivity, and preserves functional pancreatic β -cell architecture (Ozturk *et al.*, 2025).

4.2 Hypolipidemic and Anti-Steatotic Activities

Turmeric: Preclinical investigations validate that curcumin administration substantially decreases circulating triglycerides, total cholesterol, phospholipids, and free fatty acids in experimentally hypercholesterolemic animal subjects. At the molecular level, curcumin inhibits sterol regulatory element-binding protein-1c (SREBP-1c) and fatty acid synthase (FAS), while upregulating peroxisome proliferator-activated receptor- α (PPAR- α) to stimulate hepatic fatty acid β -oxidation. In randomized, double-blind, placebo-controlled clinical trials, oral administration of bioavailable curcumin led to significant reductions in liver lipid content in patients diagnosed with non-alcoholic fatty liver disease (NAFLD).

Chicory: Dietary integration of chicory root meal downregulates systemic cholesterol and triglyceride accumulation. Inulin acts as a dietary sequestrant, binding bile salts in the small intestine, increasing their fecal excretion, and obligating the liver to divert excess intrahepatic cholesterol pools toward de novo bile acid synthesis.

4.3 Gut Microbiome Modulation and the Gut-Liver Axis

Emerging research identifies the "gut-liver axis" as a vital regulatory hub in metabolic inflammation. Translocation of bacterial lipopolysaccharides from an altered intestinal lumen through a permeable gut barrier stimulates hepatic Kupffer cells via Toll-like Receptor 4 (TLR4), driving progressive liver inflammation and hepatic steatosis.

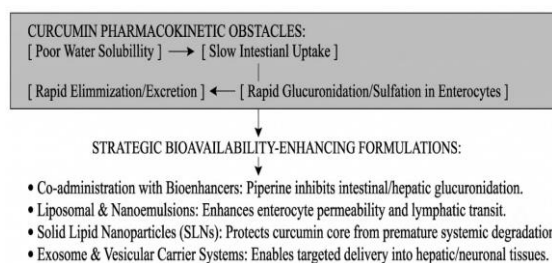
Prebiotic Chicory Inulin: Chicory inulin resists enzymatic digestion until it reaches the colon, where it is selectively metabolized by beneficial taxa (*Bifidobacterium*, *Lactobacillus*). This prebiotic fermentation generates immunomodulatory short-chain fatty acids (SCFAs: acetate, propionate, and butyrate). SCFAs reinforce tight junctions in the gut epithelium, lower mucosal luminal pH, and suppress circulating LPS levels, mitigating endotoxin-induced hepatic damage.

Curcumin's Non-Absorbed Fraction: Although systemic absorption of oral curcumin is limited, unabsorbed curcumin remains at high concentrations within intestinal lumen. There, it directly regulates the richness, diversity, and taxonomic balance of the gut microbiota, suppressing pathogenic pro-inflammatory enterobacteria and collaborate with prebiotic fermentation to reduce metabolic endotoxemia.

5. Curcumin Bioavailability Hurdles and Delivery Technologies

Despite compelling in vitro and preclinical efficacy, the clinical translation of native crystalline curcumin has long been challenged by four major pharmacokinetic barriers:

1. Extremely poor aqueous solubility at neutral and acidic physiological pH.
2. Sub-optimal gastrointestinal absorption across mucosal epithelial cells.
3. Rapid biotransformation, undergoing extensive glucuronidation and sulfation in intestinal and hepatic enterocytes to less-active metabolites.
4. High systemic clearance and rapid physiological excretion.



To overcome these barriers, diverse pharmaceutical strategies have been developed:

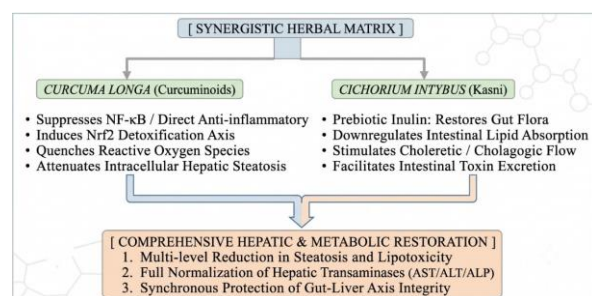
• **Glucuronidation Inhibitors:** Co-administration with piperine (from *Piper nigrum*) competitively inhibits hepatic and intestinal glucuronidation, dramatically boosting circulating plasma concentrations.

• **Novel Nanocarriers:** Encapsulation within solid lipid nanoparticles, liposomes, cyclodextrin complexes, and self-emulsifying nano-delivery systems markedly enhances dispersion, enterocyte uptake, and bioavailability.

• **Exosomal and Vesicular Delivery Systems:** Exosome formulations provide targeted tissue delivery, improving cellular uptake within inflamed hepatocytes and across the blood-brain barrier.

6. Strategic Rationale for a Combined Herbal Formulation

Given the complex, multi-organ pathophysiology of metabolic syndrome, NAFLD, and progressive hepatic steatohepatitis, single-target synthetic pharmacotherapies frequently prove insufficient. A combined *Curcuma longa* and *Cichorium intybus* botanical formulation offers a well-grounded multi-target approach:



Dual Action on the Gut-Liver Axis: Chicory's non-digestible inulin drives the selective proliferation of beneficial gut microbes, producing short-chain fatty acids (SCFAs) that reinforce intestinal barrier integrity. Concurrently, unabsorbed curcumin directly optimizes the intestinal microbiome landscape while downregulating local gut inflammation. Together, they significantly reduce the systemic translocation of circulating LPS into the portal vein, relieving downstream hepatic Kupffer cell activation.

Complementary Hepatoprotective Cascades: Once intrahepatic stress is triggered, curcumin suppresses NF- κ B-dependent transcription of TNF- α , IL-1 β , and COX-2 while upregulating Nrf2-dependent antioxidant enzymes (SOD, catalase, GPx). Simultaneously, chicory sesquiterpenes promote choleresis and bile clearance, preserving parenchymal membrane architecture and lowering serum transaminases and bilirubin levels.

Bimodal Lipid & Glycemic Management: Chicory inulin delays intestinal carbohydrate and fatty acid uptake, lowering circulating free fatty acid influx to the liver. In parallel, curcumin promotes insulin sensitivity and activates hepatic AMPK, suppressing de novo lipogenesis via the downregulation of SREBP-1c.

7. Current Research Gaps and Future Research Directions

To translate the synergistic potential of *Curcuma longa* and *Cichorium intybus* into mainstream medical and clinical practices, several key research gaps must be resolved:

Bioavailability Optimization: While novel nanocarriers, phytosomes, and bioenhancers have improved curcumin's pharmacokinetics, studies must evaluate how co-administering inulin fibers influences systemic curcuminoid absorption and transit times.

Detailed Preclinical Synergistic In Vivo Studies: Rigorous animal trials (e.g., using high-fat/fructose-induced metabolic syndrome or carbon tetrachloride/alcohol hepatotoxicity models) using isobolographic analysis are needed to confirm true pharmacological synergy (CI<1.0) between chicory extracts and curcumin.

Standardization and Phytochemical Profiling: Strict quality benchmarks using HPLC, LC-MS/MS, and NMR are necessary to define standardized extracts—quantifying both curcuminoid percentages (Curcumin, DMC, BDMC) and chicory bitter principles (lactucin, lactucopicrin) alongside inulin chain-length distribution.

Longitudinal Clinical Trials: Well-powered, randomized, double-blind, placebo-controlled human trials are required to establish optimal dosing regimens, monitor long-term safety profiles, and validate objective endpoints in patients with NAFLD, NASH, and metabolic syndrome.

Conclusion

Metabolic syndrome and progressive liver diseases remain pressing global health challenges. Both *Curcuma longa* and *Cichorium intybus* demonstrate verified, multi-target pharmacological efficacy in experimental and clinical settings. Through complementary antioxidant, anti-inflammatory, hypolipidemic, and gut-modulating mechanisms, their combined use offers a strong scientific foundation for botanical therapeutics. Integrating advanced nanoformulation systems with standardized co-extracts could overcome historical bioavailability hurdles, paving the way for safe, scalable, and effective phytotherapeutic strategies for metabolic and hepatic disorders.

References

- Adlia A, Aslan CC, Safitri L, Adnyana IK. Turmeric-black pepper-honey nanoemulsion formulation and antiulcerogenic effect evaluation against ethanol-induced gastric ulcers in rats. *PLoS ONE*, 2025; 20(1): e0317899.
- Allegra A, Innao V, Russo S, Gerace D, Alonci A, Musolino C. Anticancer Activity of Curcumin and Its Analogues: Preclinical and Clinical Studies. *Cancer Invest*, 2017; 35(1): 1–22.
- Apisariyakul A, Vanittanakom N, Buddhasukh D. Antifungal activity of turmeric oil extracted from *Curcuma longa* (Zingiberaceae). *J Ethnopharmacol*, 1995; 49: 163–169.
- Araujo CAC, Leon LL. Biological activities of *Curcuma longa* L. *Mem Inst Oswaldo Cruz*, 2001; 96: 723–728.
- Bischoff TA, Nguyen-Dinh P, Arefi AG, Laurantos M, Kelley CJ, Karchesy Y. Antimalarial activity of Lactucin and Lactucopicrin: sesquiterpene lactones isolated from *Cichorium intybus* L. *J Ethnopharmacol*, 2004; 95: 455–457.
- Brindha P, Sasikala K, Purushoth K. Preliminary Phytochemical Studies in higher Plants. *Ethnobot*, 1977; 3: 84–96.
- Chandra D, Gupta SS. Antiinflammatory and antiarthritic activity of volatile oil of *Curcuma longa* (Haldi). *Indian J Med Res*, 1972; 60: 138–142.
- Chattopadhyay I, Biswas K, Bandyopadhyay U, Banerjee RK. Turmeric and curcumin: biological actions and medicinal applications. *Curr Sci India*, 2004; 87: 44–53.
- Das, Sneha & Vasudeva, Neeru & Sharma, Sunil. (2016). *Cichorium intybus*: A concise report on its ethnomedicinal, botanical, and phytopharmacological aspects. *Drug Development and Therapeutics*. 7. 1. 10.4103/2394-6555.180157.
- El-Saadony MT, Yang T, Korma SA, Sitohy M, Abd El-Mageed TA, Selim S, et al. Impacts of turmeric and its principal bioactive curcumin on human health: Pharmaceutical, medicinal, and food applications. *Front Nutr*, 2023; 9: 1040259.
- Goel A, Kunnumakkara AB, Aggarwal BB. Curcumin as "Curecumin": From kitchen to clinic. *Biochem Pharmacol*, 2008; 75(4): 787–809.
- Hazra B, Sarkar R, Bhattacharyya S, Roy P. Tumour inhibitory activity of chicory root extract against Ehrlich ascites carcinoma in mice. *Fitoterapia*, 2002; 73: 730–733.
- Khattak S, Saeed-ur-Rehman, Ullah Shah H, Ahmad W, Ahmad M. Biological evaluation of *Curcuma longa* Linn. *Fitoterapia*, 2005; 76(2): 254–257.
- Kim M, Shin HK. The water-soluble extract of chicory influences serum and liver lipid concentrations, cecal short-chain fatty acid concentrations and fecal lipid excretion in rats. *J Nutr* 1998; 128: 1731–6.
- Milala J, Grzelak K, Krol B, Juskiewicz J, Zdunczyk Z. Composition and properties of chicory extracts rich in fructans and polyphenols. *Polish Journal of Food and Nutrition Sciences* 2009; 59: 35–43.
- Nandagopal S, Ranjitha Kumari BD. Phytochemical and Antibacterial Studies of Chicory (*Cichorium intybus* L.) - A Multipurpose Medicinal Plant. *Adv Biol Res*, 2007; 1(1-2): 17–21.
- Nasri H, Sahinfard N, Rafieian M, Rafieian S, Shirzad M, Rafieian-Kopaei M. Turmeric: A spice with multifunctional medicinal properties. *J HerbMed Pharmacol*, 2014; 3(1): 5–8.
- Negi PS. Antibacterial activity of turmeric oil: a byproduct from curcumin manufacture. *J Agric Food Chem*, 1999; 47: 4297–4300.
- Niranjan A, Dhan P, Tewari SK, Pandey A, Pushpangadan P, Prakash D. Chemistry of *Curcuma* spp. Cultivated on Sodic soil. *J Med Aromat Plant Sci*, 2003; 25: 69–75.
- Ozturk K, Kocamuftuoglu GO, Karatepe M, et al. Protective effects of turmeric (*Curcuma longa* L.) supplementation on oxidative stress and metabolic parameters in rats with high-fructose diet-induced metabolic syndrome. *Nutrire*, 2025; 50: 10.
- Patil TN, Srinivasan M. Hypocholesteremic effect of curcumin in induced-hypercholesteremic rats. *Indian J Exp Biol*, 1971; 9: 167–169.
- Prasad S, Gupta SC, Tyagi AK, Aggarwal BB. Curcumin, a component of golden spice: from bedside to bench and back. *Biotechnol Adv*, 2014; 32(6): 1053–1064.
- Rahmani S, Asgary S, Askari G, Keshvari M, Hatamipour M, Feizi A, et al. Treatment of Non-alcoholic Fatty Liver Disease with Curcumin: A Randomized Placebo-controlled Trial. *Phytother Res*, 2016; 30(9): 1540–1548.
- Rajakrishnan V, Viswanathan P, Rajakrishnan KV, Menon VP. Neuroprotective role of curcumin from *Curcuma longa* on ethanol-induced toxicity in rats. *Phytother Res*, 1999; 13(7): 571–574.

- Rotimi D, Iroaganachi A, Odeyemi I, Adeyanju A, Akanji M, Adeyemi O. The Double Sides of Curcumin and its Therapeutic Prospects. *Open Med Chem J*, 2024; 18: e18741045349977.
- Sarangthem K, Haokip MJ. Bioactive component in *Curcuma caesia* Roxb. Grown in Manipur. *The Bioscan*, 2010; 5: 113–115.
- Soleimani V, Sahebkar A, Hosseinzadeh H. Turmeric (*Curcuma longa*) and its major constituent (curcumin) as nontoxic and safe substances: Review. *Phytother Res*, 2018; 32(6): 985–995.
- Street RA, Sidana J, Prinsloo G. *Cichoriumintybus*: Traditional Uses, Phytochemistry, Pharmacology, and Toxicology. *Evid Based Complement Alternat Med* 2013; 2013: 579319.
- Unnikrishnan MK, Rao MNA. Inhibition of nitric-induced oxidation of hemoglobin by curcuminoids. *Pharmazie*, 1995; 50: 490–492.
- Verma RK, Kumari P, Maurya RK, Kumar V, Verma RB, Singh RK. Medicinal properties of turmeric (*Curcuma longa* L.): A review. *Int J Chem Stud*, 2018; 6(4): 1354–1357.
- Wickenberg J, Ingemansson SL, Hlebowicz J. Effects of turmeric on postprandial plasma glucose and insulin in healthy subjects. *Nutr J*, 2010; 9: 43.