



Potential of Turmeric and Mangosteen in Combating High-Fat Diet-Induced Inflammation and Gut Dysbiosis: A Review

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ABSTRACT

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Due to excessive fat intake, obesity has become a major global health concern with substantial social and economic repercussions. Phytochemicals found in herbal plants, such as curcumin from turmeric and xanthones, especially α -mangostin from mangosteen pericarp, have shown promise in reducing oxidative stress and altering the composition of the gut microbiota, which is brought on by high-fat diets. This narrative review was conducted using original articles from 2014 to 2024 obtained via Google Scholar and PubMed with the keywords “turmeric,” “mangosteen,” “mangostin,” “high fat diet,” “gut dysbiosis,” and “gut microbiota.” Inclusion criteria were full-text original articles in English with detailed methods; exclusion criteria were articles in other languages, lacking full text, or missing methodological details. From a total of twelve studies, curcumin has been shown to promote beneficial gut microbiota while reducing pro-inflammatory taxa associated with hepatic steatosis, neurodegenerative disease, and oxidative liver injury, while xanthones (particularly α -mangostin), possess strong anti-inflammatory activities, although their effects on gut microbial communities in high-fat diet models require further investigation. Emerging evidence suggests that both compounds may influence gut homeostasis, reduce systemic inflammation, and attenuate obesity-related complications. This review highlights the potential hormetic effects of high doses of these phytochemicals, particularly α -mangostin, and emphasizes the need for dose optimization in therapeutic applications. The synergistic modulation of gut microbiota and inflammatory pathways by curcumin and mangosteen may represent a promising complementary strategy for combating obesity and its metabolic consequences. However, additional mechanistic studies are warranted to clarify these interactions and optimize their clinical application.

Keywords: Curcuma, Gut Microbiota, Inflammation, Obesity, Xanthones

Introduction

Obesity, a complex and multifactorial metabolic disorder, appears as an excessive accumulation of body fat that reaches unhealthy levels.¹ The worldwide obesity epidemic is significantly influenced by its connection to lifestyle-related or degenerative conditions such as heart disease, type 2 diabetes, various cancers, and issues affecting the musculoskeletal and nervous systems.¹⁻³ The rise in obesity cases underscores the necessity for effective treatment strategies that extend beyond traditional pharmacological approaches, given the substantial efforts to mitigate the obesity crisis in reality.⁴ While lifestyle changes, including diet and exercise, are beneficial, they are often difficult to maintain over time.^{4,5} As a response to this issue, there is a growing interest in exploring natural compounds and plant extracts as alternative therapeutic options that can positively influence the physiological processes linked to the metabolic challenges of obesity.

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Notably, extracts from *Garcinia mangostana* (mangosteen) and *Curcuma longa* (curcumin), whether used alone or in combination, have been extensively researched for their health benefits, including antioxidant, anti-inflammatory, and metabolic regulatory effects.⁶⁻⁸ Traditionally, both mangosteen and curcumin have been utilized in various cultures for their healing properties, and recent studies have investigated their potential in addressing obesity and related metabolic disturbances.⁹⁻¹¹ Mangosteen, primarily found in Asia, contains a variety of bioactive compounds, such as xanthones and α -mangostin, which exhibit anti-inflammatory effects. Recent research suggests that curcumin from turmeric can alter gut microbiota composition, enhancing beneficial bacterial species while inhibiting pro-inflammatory taxa associated with conditions such as hepatic steatosis, Alzheimer's disease, and liver oxidative injury.¹²⁻¹⁵

The gut environment's modulation may serve as a crucial mechanism by which mangosteen and turmeric deliver their anti-obesity and anti-inflammatory effects.^{16,17} Understanding how these herbal medicines influence inflammatory pathways associated with the gut microbiome in high-fat diet models can offer valuable insights into their therapeutic potential against obesity. This review aims to evaluate the existing evidence concerning turmeric and mangosteen's effectiveness in addressing inflammatory responses and microbial imbalance triggered by excessive fat intake, emphasizing the interactions between these natural compounds, inflammatory markers, and gut microbial communities. What makes this review distinctive is its integrative approach to exploring the potential synergistic effects of curcumin and xanthones, especially α -mangostin, in regulating inflammation and gut

microbiota within the context of obesity. The methodology involves analyzing of recent and relevant studies to create a contextual synthesis of the evidence, focusing on mechanistic insights and therapeutic implications.

Materials and Methods

This study employed a narrative literature review method by sourcing original articles from search engines like Google Scholar and PubMed with keywords such as “turmeric,” “mangosteen,” “mangostin,” “high fat diet,” “gut dysbiosis,” and “gut microbiota.” The inclusion criteria comprised original articles published between 2014 and 2024, available in English, and with complete texts accessible, while articles were excluded if they were in other languages, had incomplete texts, or lacked detailed methodologies for the experiments. This review incorporates eleven studies, with their specific details outlined in Table 1.

Results and Discussion

Mangosteen and Turmeric

Garcinia mangostana (mangosteen) and *Curcuma longa* (turmeric) are two medicinal plants recognized for their ability to reduce inflammation and combat free radicals.^{6,11,18,19} Animal studies involving mangosteen have provided evidence of its anti-inflammatory benefits in conditions such as osteoarthritis, obese-type 2 diabetes, ulcerative colitis, and cardiovascular remodeling.^{11,20–22} In a systematic review, Setiawan et al. (2023) analyzed 24 studies and found that xanthones and flavonoids from mangosteen showed consistent anti-inflammatory effects across a wide range of conditions, including obesity, asthma, arthritis, and liver toxicity. These compounds were shown to reduce inflammatory cytokines and modulate immune responses.²³ Elmund and Hartrianti (2020) also conducted a systematic review of in vivo and clinical studies, confirming that mangosteen extract increases antioxidant defenses, such as superoxide dismutase, catalase, and glutathione peroxidase, while lowering oxidative stress markers like malondialdehyde. However, the authors noted that more clinical studies are needed to confirm these benefits in humans.²⁴ Similarly, turmeric, particularly its active ingredient curcumin, has shown potential by influencing gut microbial diversity and enhancing intestinal barrier function, which collectively helps to diminish systemic inflammation.^{16,25} This review highlights the individual and collaborative roles of mangosteen and turmeric in regulating inflammation and gut microbial balance, with the results summarized in Table 1.

The Anti-Inflammatory Effects of Mangosteen in Relation to Gut Microbiota

Nurrahma et al. investigated the ability of mangosteen pericarp (MP) to neutralize reactive oxygen species (ROS) and reduce inflammation in a rat model of Parkinson's disease induced by 6-hydroxydopamine (OHDA). Eight-week-old male Sprague Dawley rats were assigned to four experimental groups: normal control (NC), untreated Parkinson's disease control, and groups receiving either low-dose MP (LMP) or high-dose MP (HMP). Parkinsonian symptoms were induced by administering a unilateral injection of 6-OHDA into the brain, and the model was confirmed six weeks later using an apomorphine-induced rotation test. The rats then received oral supplementation of LMP (30 mg/kgBW) or HMP (60 mg/kgBW) for eight weeks. The researchers assessed anti-inflammatory and antioxidant effects, as well as gut microbiota composition. After MP supplementation, there was a significant decrease in ROS levels, along with increased expression of important antioxidant genes (SOD, CAT, GPx) in both the brain and muscles. This was coupled with a reduction in circulating inflammatory cytokines, including TNF- α , IL-6, and IL-1 β . Furthermore, MP influenced the gut microbiome by promoting the growth of beneficial bacterial populations, such as *Lactobacillus*, while reducing pathogenic genera like *Prevotella* and *Sutterella*.²⁶

In the study conducted by Orozco et al (2014), α -mangostin (α -MG) sourced from mangosteen was supplemented at a dosage of 900 mg/kg in the diets of mice, which is equivalent to 112 mg/kgBW each day (for

mice) and 547 mg each day (for humans). Colitis was induced in 10-week-old female C57BL/6J mice using dextran sulfate sodium (DSS), a chemical that triggers colonic inflammation similar to that found in human ulcerative colitis. Mice were grouped into four categories, with the control group as a baseline, and other groups as follows: α -MG, DSS, and DSS + α -MG. The DSS treatment was administered through the drinking water over four days, while α -MG was given one week before DSS and two weeks after. Evaluation of disease severity involved measuring changes in body weight, occurrence of diarrhea, and rectal hemorrhage, alongside further investigations into immune cell infiltration and microbiota profiles in the colon and cecum.

The results indicated that α -MG aggravated DSS-induced colitis, leading to increased disease activity, colonic inflammation, and epithelial cell proliferation. Mice fed α -MG without DSS-induced colitis also showed less stool consistency and changes in their gut microbiome, characterized by a reduction in the abundance of *Firmicutes*, *Bacteroidetes*, and *Lactobacillus*, along with an increase in *Enterobacteriaceae* and *Proteobacteria* populations. These effects were similar to those observed in human ulcerative colitis, indicating that α -MG may trigger inflammatory responses even in the absence of obvious colonic inflammation. The research concluded that the hormetic effects of α -mangostin should be considered, and its use for treating inflammatory bowel disorders or in healthy individuals should be approached with caution.²⁷

In 2015, Orozco et al. conducted another study using four different mouse strains—C57BL/6J, C3H, Balb/c, and Nude FoxN1nu—over a four-week period. The subjects were given a standard chow diet or a diet supplemented with 0.1% α -mangostin (α -MG). Various parameters, including the enhancement of colonic epithelial cell proliferation, immune response infiltration, and changes in the gut microbiome, were assessed using histology, immunohistochemistry, and pyrosequencing techniques. All four mouse strains exhibited significant changes in gut microbiota composition due to α -MG. There was a decrease in the relative abundance of *Firmicutes* (including *Lachnospiraceae*, *Ruminococcaceae*, and *Lactobacillaceae*) alongside an increase in *Proteobacteria*, which are associated with *Enterobacteriaceae*, *Akkermansia*, and *Alistipes*. Supplementation with α -MG promoted the growth of colonic epithelial layers and increased immune cell infiltration in all strains, indicating an upregulation of inflammatory processes. Despite the genetic differences among the strains, the response to α -MG was consistent, suggesting a strain-independent effect.²⁸ Both studies suggest that α -MG, despite its claimed anti-inflammatory properties, may negatively affect gut microbiome composition and aggravate colonic inflammation, particularly at high dose (112 mg/kg body weight per day for mice, which is equivalent to 547 mg per day for humans).

In contrast, lower doses of α -mangostin (30 mg/kg/day) and mangosteen ethanolic extract (40, 200, 1000 mg/kg/day) have been shown to reduce the severity of DSS-induced ulcerative colitis through antioxidant effects (reducing ROS, NO, MDA, and increasing catalase, SOD) and anti-inflammatory effects (reducing TNF α , TLR2, ICAM1, VCAM1, MCP1).³⁷

Another xanthone compound from mangosteen pericarp, γ -mangostin, has been reported to have an anti-obesity effect that may be even stronger than that of α -mangostin.^{38,39} Some studies have confirmed its potential effects on adipogenesis cell models³⁸, reducing fatty acid-induced accumulation in hepatocyte cells via the SIRT1/LKB1/AMPK pathway,³⁹ and demonstrating glucose-lowering properties in synergy with insulin through the AMPK pathway.^{40,41} Unfortunately, research confirming its anti-inflammatory effects related to changes in gut microbiota in high-fat diet-induced models remains limited.

The Effects of Turmeric on Gut Microbiota

The low physiological and solubility uptake of turmeric may limit its effectiveness as agent for reducing ROS and inflammation.⁴² Therefore, it is hypothesized that turmeric can alter gut microbiota, leading to indirect effects that produce significant health benefits related to various diseases.^{25,30}

Table 1: Research on the Effects of Turmeric and Mangosteen on Gut Microbiota Modulation

Author	Phytochemicals	Subjects	Methods	Key Findings
Nurrahma et al, 2022 ²⁶	Mangosteen pericarp	8-week-old Sprague Dawley male rats	Four groups: normal control (NC), PD control (untreated), LMP 30 mg/kg, and HMP 60 mg/kg, oral, 8 weeks. PD-like symptoms were induced by unilateral 6-OHDA injection into the brain. Motor function (apomorphine-induced rotation test and rotarod test), dopamine transporter (DAT) imaging, ROS levels, antioxidant gene expression, inflammatory cytokines, mitochondrial function, and gut microbiota composition were analyzed.	The study suggests that MP extract offers neuroprotection in a PD-like model by enhancing antioxidant defenses, reducing oxidative stress, and altering gut microbiota composition. MP's ability to improve mitochondrial function and reduce systemic inflammation may contribute to its beneficial effects on motor deficits.
Orozco et al, 2014 ²⁷	α -mangostin	C57BL/6J mice	Colitis was induced using dextran sulfate sodium (DSS) in C57BL/6J mice. Mice were fed a control diet or a diet with 0.1% α -MG. Disease severity, colonic inflammation, immune cell infiltration, and changes in gut microbiota were measured.	α -MG worsened DSS-induced colitis, leading to increased inflammation and immune cell infiltration. It also altered the gut microbiota, reducing beneficial bacterial species and increasing pathogenic bacteria.
Orozco, 2015 ²⁸	α -mangostin	Different strains of mice	Four strains of mice (C57BL/6J, Balb/c, C3H, and Athymic FoxN1nu) were fed either a control diet or a diet containing 0.1% α -MG for 4 weeks. Analysis of colonic epithelial cell proliferation, immune cell infiltration, and gut microbiota composition was conducted.	α -MG altered gut microbiota composition in all strains, increasing pathogenic bacteria (e.g., <i>Proteobacteria</i>) and reducing beneficial bacteria (e.g., <i>Firmicutes</i> , <i>Lachnospiraceae</i>). It also increased colonic cell proliferation and immune cell infiltration.
Peterson et al, 2018 ²⁹	Turmeric and curcumin	Healthy adult human subjects, 19-58 years old	A double-blind, randomized, placebo-controlled pilot study on 30 subjects. Groups were given turmeric, curcumin (both with Bioperine), or a placebo for 8 weeks. Gut microbiota changes were analyzed using 16S rDNA sequencing.	Curcumin group: 69% increase in detected species. Turmeric group: 7% increase in detected species. Placebo group: 15% reduction in species. Subjects showed personalized responses to treatment. Common microbial changes included increases in <i>Clostridium</i> , <i>Bacteroides</i> , and <i>Parabacteroides</i> species and decreases in <i>Blautia</i> and <i>Ruminococcus</i> species. Fermented turmeric showed better anti-obesity effects than unfermented turmeric in modulating gut microbiota composition. The study found an increase in beneficial bacteria: <i>Akkermansia muciniphila</i> , <i>Desulfovibrio</i> , <i>Muribaculum intestinale</i> , and <i>Deltaproteobacteria</i> ; and a decrease in pathogenic bacteria: <i>E. coprostanoligenes</i> and <i>R. faecis</i> .
Lin et al, 2024 ³⁰	Turmeric and fermented turmeric	Five-week-old C57BL/6J male mice	Normal diet (ND), high-fat diet (HFD), HFD + unfermented turmeric (UT), and HFD + fermented turmeric (FT). UT and FT were administered for 16 weeks. Changes in gut microbiota analyzed using next-generation sequencing and gut microbiota compositional analysis	Fermented turmeric showed better anti-obesity effects than unfermented turmeric in modulating gut microbiota composition. The study found an increase in beneficial bacteria: <i>Akkermansia muciniphila</i> , <i>Desulfovibrio</i> , <i>Muribaculum intestinale</i> , and <i>Deltaproteobacteria</i> ; and a decrease in pathogenic bacteria: <i>E. coprostanoligenes</i> and <i>R. faecis</i> .
Lee at al, 2023 ³¹	Calebin A (non-curcuminoid compound of turmeric)	Five-week-old C57BL/6J male mice	C57BL/6J mice fed a high-fat diet were supplemented with Calebin-A for 12 weeks. Gut microbiota changes were measured using 16S rDNA sequencing and analysis.	Calebin-A decreased the diversity of microbiota (ACE estimator), had a similar composition to the HFD group (PCA), and increased the abundance of beneficial bacteria like <i>Akkermansia</i> and <i>Butyrivibrio</i> in gut microbiota.
Lamichhan e et al, 2024 ³²	Curcumin	7-month-old young female (3xTg-AD) and B6129SF2/J mice (control)	3xTg-AD and control mice fed with a high-fat high-sugar diet (HDHSD) with or without curcumin. Gut microbiome assessed using 16S sequencing and analysis	Curcumin modulated gut microbiota with an increase in beneficial bacteria like <i>Oscillospiraceae</i> and <i>Rikenellaceae</i> (family level), and <i>Oscillibacter</i> , <i>Alistipes</i> , <i>Pseudoflavonifractor</i> , <i>Duncaniella</i> , and <i>Flintibacter</i> (genus level)

The effects of turmeric on humans were examined by Peterson et al. A total of thirty-two healthy adults, aged 19 to 58, were selected based on specific inclusion and exclusion criteria (such as smoking, diabetes, cardiovascular disease, metabolic syndrome, the use of angiotensin receptor blockers, pregnancy, breastfeeding, or recent use of systemic antibiotics) and then randomized into three groups: one control group receiving placebo and two treatment groups receiving turmeric or curcumin tablets. Each turmeric tablet contained 1000 mg of turmeric root (*Curcuma longa*) and 1.25 mg of BioPerine black pepper, while the curcumin tablet provided 1000 mg of curcumin along with 1.25 mg of

BioPerine. Participants were instructed to take two tablets three times a day with meals for eight weeks. Ultimately, only 3 participants in the placebo group, six in the turmeric group, and five in the curcumin group were included in the analysis at the end of the study, as some subjects had withdrawn, could not be contacted or were following a different diet, such as a vegan diet.²⁹

Gut microbiome samples were analyzed from fecal samples at the beginning, again at 4 weeks, and finally at eight weeks after treatments using 16S rDNA sequencing. The placebo group showed a 15% decline in bacterial species from a total of 325, while the turmeric group

experienced a 7% increase and the curcumin group saw a 69% increase. Although no significant differences were found among the groups, the curcumin group exhibited higher alpha diversity. In the placebo group, the relative abundance of 89 taxa declined, compared to 71 in the turmeric group and 56 in the curcumin group.

The findings suggest that the responses to turmeric and curcumin vary widely among individuals, ranging from 'responsive' to 'unresponsive'. In the responsive subjects, increases in *Clostridium*, *Bacteroides*, *Enterobacter*, *Enterococcus*, *Parabacteroides*, *Bifidobacterium*, and *Alistipes* were observed. The breakdown of polysaccharides into oligosaccharides, disaccharides, and monosaccharides was noted in the turmeric group, providing energy for fermentative bacteria (*Clostridium*) and resulting in short-chain fatty acids (SCFA) and hydrogen, which promoted the growth of hydrogen-consuming bacteria like *Blautia* and *Desulfovibrio*. In non-responders, more taxa remained unaffected by the intervention or showed lower abundance.²⁹ Turmeric exhibits prebiotic effects, while curcumin has prebiotic-like effects, as it does not directly nourish microbiota but may influence the host's physiology (such as barrier function and the selective survival of bacteria).^{29,43} The authors hypothesized that patients with good absorption of turmeric and curcumin in the small intestine may show fewer prebiotic effects in the colon, whereas those with poor absorption might exhibit greater prebiotic effects in the colon. However, further research is needed to clarify this hypothesis.

The Effects of Turmeric on Gut Microbiota in High-Fat Diet-Induced Models

In their study, Lin and colleagues investigated the impact of turmeric fermented with *Lactobacillus paracasei* (FT) on gut bacterial communities in obesity-induced mice. The research involved 40 five-week-old male C57BL/6J mice, which were divided into four groups: normal diet (ND), high-fat diet (HFD), HFD with 5% unfermented turmeric (UT), and HFD with 5% fermented turmeric (FT), with eight mice in each group. The treatment lasted for 16 weeks. The study observed an increase in *Akkermansia muciniphila*, *Desulfovibrio*, *Muribaculum intestinale*, and *Deltaproteobacteria*, along with a decrease in pathogenic bacteria such as *E. coprostanoligenes* and *R. faecis*. The rise in *Akkermansia muciniphila*, which is negatively correlated with diabetes and obesity, suggests a beneficial effect of FT. Curcumin's ability to remove free radicals and lipoteichoic acid from *L. paracasei* contributed to increased mucin secretion, fostering an environment conducive to the growth of *Akkermansia muciniphila*.^{44,45}

Desulfovibrio is an H₂-utilizing microorganism that increased due to the enzymatic breakdown of polysaccharides into oligosaccharides, leading to microbial fermentation.²⁹

Other beneficial bacteria, including *E. sinensis* JCM 14551 and *R. hominis* A2-183, also increased in the FT group, indicating a link to reduced inflammation levels in the intestine.⁴⁶ In contrast, pathogenic bacteria like *E. coprostanoligenes*, commonly found in type II diabetes patients, and *R. faecis*, which are associated with obesity,⁴⁷ exhibited elevation under HFD conditions but declined following FT treatment. The study demonstrated that fermented turmeric is more effective than unfermented turmeric as a strategy to combat obesity and modulate gut microbiota.³⁰

Calebin A, a compound sourced from turmeric (*Curcuma longa* L.) that is not classified as a curcuminoid, has been shown to be an effective anti-obesity agent by promoting thermogenesis and altering the gut microbiome in mice consuming a high-fat dietary intake. Throughout a 12-week study, 36 young male C57BL/6 mice (4 weeks old) were grouped into four categories, including a normal diet (ND), high-fat diet (HFD), HFD with 0.1% Calebin-A, and HFD with 0.5% Calebin-A. Fresh fecal samples were analyzed using 16S rDNA sequencing. Although no significant increase in the Firmicutes to Bacteroidetes (F/B) ratio was observed in the HFD groups, Calebin A was found to decrease the F/B ratio. Additionally, Calebin A increased the proportions of the *Verrucomicrobia* phylum and *Proteobacteria*. The Abundance-based Coverage Estimator (ACE) indicated a reduction in gut microbiota richness, while diversity, as measured by the Shannon index, remained relatively stable. The Principal Component Analysis (PCA) diagram revealed that compared to the ND group, the Calebin A group exhibited a gut microbial community more comparable to that of

the HFD group. The results indicated that the levels of *Akkermansia*, *Butyrivibrio*, *Ruminiclostridium*, and unidentified *Ruminococcaceae* increased following Calebin A treatment, with the 0.5% concentration demonstrating greater effectiveness compared to the 0.1% concentration.³¹

The research conducted by Lamichhane et al. investigated the gut microbiome in mouse model of Alzheimer's disease that was fed a calorie-dense diet high in fat and sugar. Using 7-month-old young female transgenic (3xTg-AD) mice and B6129SF2/J mice as controls, the study divided the subjects into four groups: Normal Control Diet (NCD), NCD with 4 g/kg curcumin (NCD+CUR), high-fat high-sugar diet (HFHSD), and HFHSD with curcumin (HFHSD+CUR). Fecal samples were analyzed using 16S rRNA sequencing. Mice receiving curcumin (NCD+CUR) exhibited a reduction in *Verrucomicrobia*, but specific beneficial gut microbiota increased at the family and genus levels in the curcumin-supplemented groups (NCD+CUR and HFHSD+CUR). Notably, the families *Oscillospiraceae* and *Rikenellaceae* showed an increase in abundance, with a corresponding rise in *Oscillibacter*, *Alistipes*, *Pseudoflavonifractor*, *Duncaniella*, and *Flintibacter*.³² Previous studies have established a link between these microbiomes with improvements in metabolic syndrome, liver and cardiovascular health, as well as reduced inflammation.⁴⁸⁻⁵⁰

In a separate study by Feng et al., it was reported that curcumin reduced hepatic steatosis by modulating gut microbiota. Male Sprague Dawley rats, aged 4 weeks, were divided into three groups: a chow diet with a vehicle, high-fat diet (HFD) with curcumin (200 mg/kg), or an HFD with a vehicle. The rats were fed the HFD for 12 weeks, followed by 4 weeks of curcumin administered via gastric gavage. The samples were processed for 16S rRNA sequencing using MiSeq technology and analyzed through bioinformatics. Curcumin altered the structure of gut microbiota, decreased its diversity, and affected its composition across various taxonomic levels. Of the 110 operational taxonomic units (OTUs) influenced by curcumin, 36 out of the 47 functionally relevant OTUs associated with hepatic steatosis were reduced.³³

Zhong et al. demonstrated that curcumin improves insulin sensitivity in mice on a high-fat diet by modifying the gut microbiota. Eight-week-old male C57BL/6J mice, housed in a specific pathogen-free (SPF) environment, were divided in two groups: one group received a high-fat diet (HFD) with curcumin dissolved in 0.5% carboxymethylcellulose, while the control group received just the 0.5% carboxymethylcellulose. The treatment was administered via intragastric lavage daily for 4 weeks. During this period, antibiotics were given every two days to deplete the gut microbiota, along with daily curcumin (100 mg/kg body weight) and vehicle. The study continued for an additional 4 weeks, during which fresh fecal microbiota transplantation (FMT) was provided for both groups via intragastric gavage. The researchers evaluated phenotypic expressions of lipid and glucose levels, as well as FGF15 (a gut-derived hormone) every 4 weeks, to determine if curcumin's ability to mitigate disturbances in glucose and lipid metabolism related to the high-fat diet depended on gut microbiota modulation.³⁴

Curcumin has low oral bioavailability and accumulates significantly in the intestine, exerting its effect through changes in gut microbiota.^{33,51} The study by Zhong et al. showed that depleting endogenous gut microbiota negated curcumin's impact on markers of glucose and lipid metabolic disorders, while fecal microbiota transplantation (FMT) restored the beneficial effects of curcumin on these markers.³⁴

Zhou et al. examined the combined effects of turmeric extract, *Sargassum fusiforme*, and pomegranate peel extract on obesity in C57BL/6J mice fed a high-fat diet. The study revealed that this combination significantly reduced obesity by enhancing lipid metabolism and altering gut microbiota composition. They observed an increase in beneficial bacteria such as *Lactobacillus* and *Bifidobacterium* species, which are associated with improved metabolic health, and a decrease in harmful bacteria like *Desulfovibrio* and *Clostridium* species, which are linked to gut dysbiosis and inflammation related to obesity. The results indicate a synergistic effect of these phytochemicals in preventing obesity through metabolic regulation and modulation of gut microbiota.⁵²

The Effects of Turmeric and Mangosteen Pericarp Extracts on Systemic Inflammation and Gut Microbiota in High-Fat Diet-Induced Models

Labban et al. (2021) investigated the individual and combined effects of turmeric (*Curcuma*) and mangosteen extracts on microbiota composition in both regular and high-fat diets. The study involved eight groups of four-week-old Wistar albino rats. Group 1 acted as the control, Groups 2-4 were given a regular diet, and Groups 5-8 were fed a high-fat diet. Lean and obese rats received 400 mg/kg/day of mangosteen extract, 80 mg/kg/day of turmeric, or a combination of both for a duration of 10 weeks. Fecal samples were collected every two weeks to analyze changes in gut bacterial composition. During the experiment, the *Firmicutes* to *Bacteroides* ratio increased. In Group 1-4, there was an increase in Gram-positive cocci and *Enterobacteriaceae*, while *Clostridium* and *Bacteroidetes* decreased. In Groups 5-8, Gram-positive cocci and *Enterobacteriaceae* also increased by week 10, whereas *Clostridium* and *Bacteroides*, which had increased by week 2, decreased and were absent by week 10 of the experiment. These findings suggested that mangosteen and curcumin influence gut microbiota in both lean and obese rats by promoting beneficial bacteria and inhibiting harmful bacteria.³⁵

Another article by Labban et al. (2022) validated the synergistic effects of mangosteen pericarp and turmeric (*Curcuma*) extract in reducing systemic inflammatory activity in a high-fat diet model involving rats. The study included four groups: Group I served as the control, obese rats fed a high-fat diet (HFD); Group II consisted of HFD-fed rats that received a single dose of 400 mg/kg/day of mangosteen pericarp extract; Group III comprised HFD-fed rats given a single dose of 80 mg/kg/day of turmeric; and Group IV comprised HFD-fed rats that received a combination of both extracts over a period of 6 weeks. The results showed that both mangosteen pericarp and turmeric, individually or in combination, significantly reduced serum levels of IL-6, IL-12, and leptin, indicating a decrease in systemic inflammation following high-fat diet induction. Additionally, the study noted a neuroprotective effect.³⁶

This narrative review highlights the complementary roles of mangosteen and turmeric in modulating gut microbiota composition and systemic inflammation, particularly in high-fat diet-induced models of obesity. Curcumin, the active component of turmeric, has been shown to enhance gut microbial diversity, increase beneficial taxa (including *Akkermansia muciniphila*, *Oscillospira*, and *Rikenellaceae*), and improve intestinal barrier function, leading to reductions in systemic inflammation and metabolic disturbances such as hepatic steatosis and insulin resistance.^{29,30,32} In particular, fermented turmeric was found to be more effective than unfermented forms in promoting the growth of short-chain fatty acid-producing bacteria and reducing pro-inflammatory pathogens such as *E. coprostanoligenes* and *R. faecis*, thereby contributing to improved anthropometric measures and lipid profiles.^{30,52}

Conversely, α -mangostin, a xanthone found in mangosteen pericarp, has demonstrated anti-inflammatory effects and potential to regulate metabolic health, yet its impact on gut microbiota appears dose-dependent and sometimes detrimental in colitis models.^{27,28} High doses of α -mangostin aggravated DSS-induced colitis, increasing pathogenic bacteria such as *Proteobacteria* and reducing beneficial taxa like *Lachnospiraceae* and *Ruminococcaceae*. However, lower doses of mangosteen extract showed protective effects, including antioxidant and anti-inflammatory activity, reduction of pro-inflammatory cytokines, and improvement in gut microbial balance.²⁶ Notably, the synergistic administration of turmeric and mangosteen was more effective than individual treatments in reducing systemic inflammatory markers (IL-6, IL-12, leptin) and promoting gut microbiota homeostasis.^{35,36} The individual and combined effects of turmeric and mangosteen are illustrated in Figure 1.

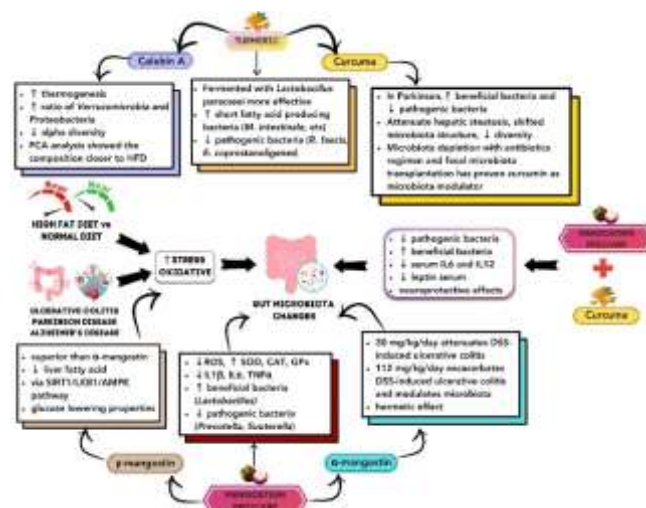


Figure 1: Potential of Turmeric and Mangosteen pericarp in Combating High-Fat Diet Induced Inflammation and Gut Dysbiosis

Conclusion

Curcumin from turmeric and xanthones from mangosteen pericarp have significant potential for altering gut microbiota in models induced by a high-fat diet. The combination of these two herbal plants or their compounds may produce synergistic effects, promoting beneficial bacteria while reducing pathogenic bacteria. While curcumin's role as a modulator of gut microbiota has been demonstrated in numerous studies, research on the role of mangosteen remains limited. Further investigation is necessary to validate the individual and combined effects of turmeric and mangosteen pericarp on the inflammatory response triggered by a high-fat diet and to determine whether their combination offers greater beneficial effects.

Conflict of Interest

The author's declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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