

The Potential Role of Purple Butterfly Pea (*Clitoria Ternatea* L.) in Modulating Visceral Fat, IL-1B-Mediated Inflammation, and Foam Cell Formation: Implications for Atherosclerosis

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Abstract

Atherosclerosis develops through interconnected disturbances in lipid metabolism, vascular inflammation, and immune responses, with visceral adipose tissue dysfunction, interleukin-1 beta (IL-1 β)-mediated inflammation, and macrophage-derived foam cell formation playing important roles. This review aimed to evaluate the potential role of purple butterfly pea (*Clitoria ternatea* L.) in modulating visceral adiposity, IL-1 β -mediated inflammation, and foam cell formation associated with atherosclerosis. A literature review was conducted using PubMed/MEDLINE, Scopus, and Google Scholar, supplemented by manual reference screening. Studies addressing *Clitoria ternatea*, visceral fat or obesity, IL-1 β or NLRP3 signaling, and foam cells or atherosclerosis were narratively synthesized. The evidence indicates that visceral adipose tissue dysfunction can promote oxidative stress and inflammation, even without substantial increases in fat mass. NLRP3-driven IL-1 β production may suppress the GPR109a–ABCA1 pathway, impair macrophage cholesterol efflux, and enhance foam cell formation. *Clitoria ternatea*, which is rich in anthocyanins, flavonoids, and phenolic compounds, has demonstrated antioxidant, anti-inflammatory, lipid-lowering, and metabolic effects in animal and human studies. However, its effects on IL-1 β regulation remain context-dependent, and direct inhibition of foam cell formation has not yet been confirmed. Therefore, *Clitoria ternatea* may function as a multi-target modulator of atherogenic pathways; however, integrated experimental studies examining inflammasome signaling, cholesterol transport, histopathological changes, and dose-response relationships are required to rigorously establish its antiatherosclerotic efficacy in future research.

INTRODUCTION

Atherosclerosis is a progressive arterial disease driven by the interplay of lipid accumulation, vascular inflammation, endothelial dysfunction, and maladaptive immune responses. The process begins with the retention and modification of lipoproteins within the arterial wall, followed by the recruitment of inflammatory cells and the accumulation of lipid-laden macrophages. A pivotal step in the development of atherosclerotic lesions is foam cell formation. Macrophages phagocytose modified lipoproteins—particularly oxidized low-density lipoprotein (oxLDL)—through various scavenger receptors. Under pathological conditions, an imbalance between cholesterol influx into macrophages and cholesterol efflux leads to intracellular lipid accumulation and foam cell formation, thereby contributing to the progression of atherosclerotic plaques (Gui et al., 2022).

Obesity is a major risk factor for cardiovascular disease; however, adipose tissue distribution plays a more critical role than total body fat mass alone. Visceral adipose tissue (VAT) is a metabolically active tissue closely associated with lipid metabolism disorders,

insulin resistance, oxidative stress, and systemic inflammation. VAT expansion may be accompanied by altered adipocyte function and increased immune cell activity, including macrophage activation, creating a pro-inflammatory environment. This condition potentially links visceral obesity with metabolic disturbances and vascular inflammation. Therefore, VAT should be considered not merely an energy storage depot but also a source of metabolic and inflammatory signals that contribute to cardiovascular disease development (Gui et al., 2022).

Interleukin-1 beta (IL-1 β) is a pro-inflammatory cytokine that plays a crucial role in the atherosclerotic process. Inflammasome activation, including activation of the NOD-like receptor family pyrin domain-containing 3 (NLRP3) inflammasome, triggers caspase-1 activation and the maturation of pro-IL-1 β into active IL-1 β . In the context of atherosclerosis, IL-1 β not only amplifies inflammatory responses but also influences cholesterol homeostasis in macrophages. Experimental studies indicate that NLRP3 activation and elevated IL-1 β levels can suppress the GPR109a–ABCA1 pathway, thereby reducing cholesterol efflux from macrophages and promoting intracellular cholesterol accumulation. Ultimately, these conditions can enhance foam cell formation and accelerate atherosclerosis progression (Tumurkhuu et al., 2018).

The relationship between inflammation and lipid accumulation is bidirectional. The accumulation of lipids and modified lipoproteins can trigger inflammatory responses in macrophages, whereas persistent inflammation can disrupt mechanisms regulating cholesterol homeostasis and efflux. Under normal conditions, ABCA1 and ABCG1 play essential roles in transporting cholesterol from macrophages to apolipoprotein A-I (ApoA-I) and high-density lipoprotein (HDL). Disruption of this process can increase intracellular cholesterol accumulation and promote foam cell formation. Thus, the interaction between lipid metabolism and inflammation can establish a pathological cycle that sustains the progression of atherosclerotic lesions (Gui et al., 2022).

In recent years, interest has increased in the use of plant-derived bioactive compounds as potential modulators of metabolic and inflammatory disorders. One plant of particular interest is the purple butterfly pea, *Clitoria ternatea* L. This plant contains various bioactive compounds, including anthocyanins, flavonoids, and other phenolic compounds. These phytochemical constituents have formed the basis of numerous studies evaluating the antioxidant, anti-inflammatory, and metabolic potential of *Clitoria ternatea* L.

Several experimental studies indicate that *Clitoria ternatea* L. has potential effects on obesity-related parameters and lipid metabolism. For example, Sasmana et al. (2024) reported that administration of an aqueous extract of *Clitoria ternatea* L. petals to rats fed a high-fat diet improved body weight, lipid profiles, and aortic tunica thickness. The study demonstrated reductions in total cholesterol, LDL, VLDL, body weight, and aortic tunica thickness in the extract-treated group compared with the high-fat diet group. These findings suggest that *Clitoria ternatea* L. may exert metabolic and vascular effects relevant to the development of atherosclerosis (Sasmana et al., 2024).

Other studies have also highlighted the potential of *Clitoria ternatea* L. in influencing metabolic and inflammatory disorders. Widowati et al. (2024) reported that *Clitoria ternatea* L. flower extract, tested in rat models of diabetes and dyslipidemia, modulated metabolic parameters as well as markers associated with oxidative stress and inflammation. Although

these models do not directly represent atherosclerosis, the findings support the possibility that bioactive compounds in *Clitoria ternatea* L. may influence pathways associated with metabolic and inflammatory disorders (Widowati et al., 2024).

Based on this evidence, *Clitoria ternatea* L. warrants further investigation regarding the interaction between obesity, inflammation, and atherosclerosis. However, a research gap remains concerning the direct relationship between *Clitoria ternatea* L. administration and changes in VAT, IL-1 β regulation, and foam cell formation (Alinia-Ahandani, 2023; Aruwa & Sabiu, 2024; Negm & Fathy, 2025; Tiono et al., 2026). Most existing studies have focused on body weight, obesity indices, lipid profiles, oxidative stress, or specific inflammatory biomarkers, whereas studies simultaneously evaluating these three processes remain limited (Sasmana et al., 2024; Widowati et al., 2024).

Therefore, a biological rationale exists for developing a conceptual framework in which visceral adiposity, IL-1 β -mediated inflammation, and foam cell formation are interconnected processes in atherosclerosis development, while *Clitoria ternatea* L. may influence several pathways within this cascade through its metabolic, antioxidant, and anti-inflammatory activities. However, these relationships require confirmation through studies that directly assess changes in VAT, IL-1 β regulation, inflammasome activation, macrophage cholesterol metabolism, and foam cell formation (Jang et al., 2025; Leleu et al., 2025; Zhang et al., 2021; Zwartjes et al., 2025).

Therefore, this review article aimed to discuss the potential role of *Clitoria ternatea* L. in modulating VAT, IL-1 β -mediated inflammation, and foam cell formation, as well as to evaluate its potential relevance to the development of atherosclerosis.

METHOD

A literature search was conducted using PubMed/MEDLINE, Scopus, and Google Scholar to identify relevant scientific publications. The search strategy focused on four key concepts: *Clitoria ternatea*, visceral adiposity/obesity, IL-1 β -mediated inflammation, and foam cell formation/atherosclerosis. The search terms included combinations of keywords related to *Clitoria ternatea* or butterfly pea flower; visceral adiposity, visceral fat, or obesity; IL-1 β , interleukin-1 beta, or NLRP3; and foam cell formation or atherosclerosis.

In addition to database searches, the reference lists of relevant publications were manually screened to identify additional studies. This approach enhanced the comprehensiveness of the literature identification process.

Table 1. Summary of Studies on the Metabolic and Cardiovascular Effects of *Clitoria ternatea* Extract

Researcher, Year	Study Model	Intervention	Parameters Assessed	Main Results
Sasmana et al., 2024	Wistar rats, HFD	Aqueous extract of <i>C. ternatea</i> petals; 250, 500, 750 mg/kgBW	Body weight, lipid profile, aortic tunica thickness	Reduced body weight, cholesterol, LDL, VLDL, and aortic tunica thickness, particularly at the 750 mg/kgBW dose

Wang et al., 2022	C57BL/6 mice, high-fat + fructose diet	Aqueous extract of blue C. ternatea petals; several concentrations	Body weight, lipids, leptin, FFA, inflammation, oxidative stress	Improved obesity, insulin resistance, lipid profile, oxidative stress, and inflammation
Thilavech et al., 2021	Overweight humans	C. ternatea flower extract administered with a high-fat meal	TG, FFA, antioxidant capacity, IL-1 β , IL-6, TNF- α	Reduced postprandial TG and FFA responses and improved antioxidant status; IL-1 β , IL-6, and TNF- α did not change significantly
Widowati et al., 2024	Diabetic + dyslipidemic rats	C. ternatea flower extract; 28 days	IL-1 β , CRP, MDA, metabolic parameters	Reduced IL-1 β , CRP, MDA and improved several metabolic parameters
Wihandani et al., 2026	Wistar rats, HFD	Aqueous extract of C. ternatea; 250, 500, 750 mg/kgBW	Perivascular adipose tissue (PVAT) and tissue morphology	Improved PVAT dysfunction and reduced WAT-like PVAT characteristics
Damayanti et al., 2026	Wistar rats, UV-B inflammation model	C. ternatea extract cream; 2.5-30%	IL-1 β and inflammation	Doses of 2.5% and 5% reduced IL-1 β compared with control
Tumurkhuu et al., 2018	In vitro + atherosclerosis mouse model	NLRP3/IL-1 β activation	IL-1 β , ABCA1, cholesterol efflux, foam cell	IL-1 β reduced ABCA1 and cholesterol efflux and increased foam cell formation
Gui et al., 2022	Mechanistic review	Foam cell biology	Lipid uptake, cholesterol efflux, macrophage	Explains the mechanisms of foam cell formation and progression

RESULTS AND DISCUSSION

Visceral Fat as a Trigger for Inflammation

Visceral fat, or visceral adipose tissue (VAT), serves not merely as an energy storage site but also as an active metabolic and immunological organ. An increase in visceral fat can lead to adipocyte hypertrophy, increased release of free fatty acids, altered adipokine secretion, oxidative stress, and activation of inflammatory responses. These conditions can create an environment conducive to endothelial dysfunction and the development of atherosclerosis. Thus, an increase in VAT reflects not only fat accumulation but also alterations in adipose tissue function that can contribute to systemic and vascular inflammation (Kawai et al., 2021; Neeland et al., 2019).

However, the amount of visceral fat does not always correlate directly with the level of adipose tissue inflammation. VAT mass is a quantitative measure, whereas inflammation reflects functional and immunometabolic changes within the tissue. Adipose tissue can undergo changes—such as altered adipokine secretion, oxidative stress, immune cell infiltration, and activation of inflammatory pathways—without necessarily exhibiting a substantial increase in mass. Consequently, inflammatory changes and changes in the amount of adipose tissue may occur at different times or to varying degrees (Kawai et al., 2021; Chait & den Hartigh, 2020).

Experimental studies have shown that while visceral fat mass may not always undergo significant changes, levels of IL-1 β and foam cell formation show significant alterations following the induction of a high-fat diet. These findings indicate that inflammatory and atherogenic changes can proceed without significant quantitative changes in visceral fat depots.

Therefore, the impact of a high-fat diet on atherogenesis is likely determined not only by the amount of visceral fat but also by changes in adipose tissue metabolic function, oxidative stress, vascular inflammation, and tissue lipid metabolism (Chait & den Hartigh, 2020; Libby, 2021).

Interleukin-1 Beta in Atherosclerosis

One key mechanism linking metabolic disturbances to vascular inflammation is the activation of the NLRP3 inflammasome. Inflammasome activation leads to the activation of caspase-1 and the conversion of pro-IL-1 β into mature IL-1 β . Interleukin-1 beta (IL-1 β) subsequently acts as an inflammatory mediator that can amplify the inflammatory response in the vessel wall and contribute to the development of atherosclerotic lesions. Inflammasome activation is also triggered by various stimuli associated with atherosclerosis, including cholesterol crystals, reactive oxygen species (ROS), and disruptions in cellular homeostasis (Duewell et al., 2018; Tall & Yvan-Charvet, 2015).

The role of IL-1 β in atherosclerosis becomes increasingly significant when linked to macrophage lipid metabolism. Tumurkhuu et al. (2018) demonstrated that IL-1 β can inhibit pathways associated with G protein-coupled receptor 109a (GPR109a) and ATP-binding cassette transporter subfamily A member 1 (ABCA1), thereby reducing the capacity of macrophages for cholesterol efflux. This reduction in cholesterol efflux leads to intracellular cholesterol accumulation and increases the likelihood of macrophages transforming into foam cells. Thus, IL-1 β functions not only as an inflammatory mediator but also as a link between inflammation and the disruption of macrophage cholesterol homeostasis (Tumurkhuu et al., 2018).

This relationship provides a fundamental mechanism for understanding why elevated IL-1 β levels often coincide with increased foam cell formation under high-fat diet conditions. Although the simultaneous measurement of IL-1 β and foam cells cannot establish direct causality, the observed pattern is consistent with the mechanism by which inflammation impairs cholesterol efflux and accelerates lipid accumulation within macrophages (Tumurkhuu et al., 2018; Tall & Yvan-Charvet, 2015).

Potential Metabolic Effects of *Clitoria ternatea* L.

Purple butterfly pea *Clitoria ternatea* L. flowers contain various bioactive compounds—including anthocyanins, flavonoids, and phenolic compounds—that possess antioxidant activity and the potential to influence lipid metabolism and inflammation. Experimental evidence indicates that *Clitoria ternatea* L. extract can modulate several metabolic parameters associated with obesity and cardiometabolic diseases. In a mouse model fed a high-fat, high-fructose diet, administration of *Clitoria ternatea* L. extract improved body weight, lipid profiles, free fatty acid levels, leptin levels, insulin resistance, oxidative stress, and various inflammatory markers (Wang et al., 2022).

These effects are further supported by research involving human subjects. Thilavech et al. ...demonstrated that the administration of purple butterfly pea *Clitoria ternatea* L. flower extract alongside a high-fat meal could reduce postprandial triglyceride and free fatty acid responses while improving antioxidant status in overweight and obese individuals. However, the study did not find significant changes in IL-1 β , IL-6, or TNF- α levels. These findings indicate that the biological effects of *Clitoria ternatea* L. do not necessarily result in a reduction of all inflammatory markers and are likely influenced by factors such as the type of extract,

dosage, duration of intervention, and the metabolic characteristics of the subjects (Thilavech et al., 2021).

In animal models of diabetes and dyslipidemia, the administration of *Clitoria ternatea* L. extract has also been reported to lower levels of IL-1 β , C-reactive protein, and malondialdehyde, as well as improve various metabolic parameters. These results support the possibility that *Clitoria ternatea* L. possesses anti-inflammatory effects linked to the amelioration of oxidative stress and metabolic disturbances. However, since these research models were not specifically designed to study atherosclerosis, the findings are best viewed as supporting evidence for anti-inflammatory activity rather than direct proof that *Clitoria ternatea* L. inhibits atherogenesis (Widowati et al., 2024).

Potential Role of *Clitoria ternatea* L. in Modulating Interleukin-1 β

The ability of *Clitoria ternatea* L. to modulate IL-1 β is a relevant aspect regarding the development of atherosclerosis, yet direct evidence concerning this mechanism remains limited. In several experimental models, the administration of *Clitoria ternatea* L. has been associated with reductions in inflammatory markers and oxidative stress. These effects suggest that the flavonoid and anthocyanin content of the plant may influence the IL-1 β inflammatory pathway, although a direct link to NLRP3 activation has not yet been fully established (Wang et al., 2022; Widowati et al., 2024).

Interestingly, a human study by Thilavech et al. showed that *Clitoria ternatea* did not induce significant changes in IL-1 β levels following the consumption of a high-fat meal, despite improvements in postprandial lipid responses and antioxidant status. These results suggest that the effects of *Clitoria ternatea* L. on inflammation may be context-dependent— Influenced by factors such as metabolic conditions, baseline inflammation levels, dosage, extract form, and duration of administration. Therefore, a reduction in IL-1 β should not be considered a universal effect of *Clitoria ternatea* L. across all research models (Thilavech et al., 2021).

Based on this evidence, a more plausible mechanism is that *Clitoria ternatea* L. has the potential to modulate the metabolic and inflammatory environment, thereby influencing IL-1 β . To substantiate this mechanism, future studies need to simultaneously measure NLRP3, ASC, caspase-1, and IL-1 β , while also evaluating the relationship between changes in these markers and alterations in vascular morphology or function (Wang et al., 2022; Tumurkhuu et al., 2018).

Potential Role of *Clitoria ternatea* L. in Foam Cell Formation

Direct evidence regarding the effect of *Clitoria ternatea* L. on foam cell formation remains limited. Most available studies have evaluated parameters upstream of, or associated with, foam cell formation—such as lipid profiles, free fatty acids, oxidative stress, inflammation, and vascular function. Consequently, based on current evidence, it cannot be definitively stated that *Clitoria ternatea* L. directly inhibits foam cell formation within atherosclerotic lesions (Wang et al., 2022; Sasmana et al., 2024).

Nevertheless, there is a rationale supporting this possibility. Improvements in lipid profiles and cholesterol metabolism could reduce the availability of atherogenic lipids, while the reduction of oxidative stress and inflammation might create an environment more conducive to maintaining macrophage cholesterol homeostasis. Since IL-1 β can inhibit

ABCA1 and cholesterol efflux, theoretically, reducing IL-1 β -mediated inflammation could help maintain cholesterol efflux and decrease cholesterol accumulation in macrophages (Tumurkhuu et al., 2018; Wang et al., 2022).

Integrated Perspective: Visceral Fat, IL-1 β , and Foam Cell Formation

The relationship between visceral fat, IL-1 β , and foam cell formation should be viewed as a network of metabolic and inflammatory interactions rather than a linear pathway that always proceeds sequentially. Visceral fat can increase inflammatory risk through altered adipocyte function, the release of free fatty acids, oxidative stress, and immune cell activation. However, changes in adipose mass and alterations in adipose tissue inflammatory function do not always occur simultaneously. Therefore, the absence of significant changes in visceral fat mass does not rule out the possibility of inflammatory activation or atherogenic processes (Chait & den Hartigh, 2020; Kawai et al., 2021). This pattern suggests that, within a given study period, changes in inflammation and vascular lipid metabolism may occur earlier or via mechanisms that do not rely entirely on an increase in visceral fat mass. Such an interpretation reinforces the importance of distinguishing between adipose tissue quantity and adipose tissue dysfunction when evaluating the link between obesity and atherosclerosis (Chait & den Hartigh, 2020). Recent research indicates that an aqueous extract of *Clitoria ternatea* L. can ameliorate changes in perivascular adipose tissue (PVAT) in rats fed a high-fat diet, suggesting that the plant's vascular effects may be mediated by alterations in the function of adipose tissue adjacent to blood vessels (Wihandani et al., 2026).

Research Gaps and Future Research Directions

Future research should integrate metabolic, inflammatory, and histopathological parameters into a single experimental design. Measurements of visceral fat weight or indices should be complemented by markers of adipose tissue function, while assessments of IL-1 β could be combined with analyses of NLRP3, ASC, and caspase-1. At the vascular level, the evaluation of foam cells could be strengthened by examining macrophage markers, ABCA1/ABCG1, cholesterol efflux, and lipid accumulation. Such a multidimensional approach would help determine whether changes in foam cells following *Clitoria ternatea* L. administration are truly linked to the modulation of the IL-1 β inflammatory pathway or occur via other metabolic mechanisms (Tumurkhuu et al., 2018; Chistiakov et al., 2017).

Furthermore, dose-response relationships require special attention. The effects of plant bioactive compounds do not always follow a linear dose-response pattern; thus, increasing the dosage does not necessarily yield a greater biological effect. Variations in response based on dosage, extract type, active compound bioavailability, and duration of administration need to be considered in future *Clitoria ternatea* L. studies to allow for a more rational determination of optimal dosages (Thilavech et al., 2021; Wang et al., 2022).

Overall, *Clitoria ternatea* L. can be positioned as a potential multi-target modulator within the network of processes linking metabolic disorders to atherosclerosis. Conceptually, a high-fat diet can induce adipose tissue dysfunction and increased oxidative stress, subsequently contributing to inflammasome activation and IL-1 β production. IL-1 β can then disrupt macrophage cholesterol homeostasis by reducing cholesterol efflux, thereby promoting lipid accumulation and foam cell formation. *Clitoria ternatea* L. has the potential to influence these points through the improvement of lipid metabolism, antioxidant activity, and the modulation

of inflammation; however, its direct effect on the NLRP3–IL-1 β –ABCA1–foam cell pathway still requires experimental verification (Wang et al., 2022; Tumurkhuu et al., 2018; Wihandani et al., 2026).

CONCLUSION

This review indicates that visceral adipose tissue (VAT), IL-1 β -mediated inflammation, and foam cell formation constitute an interconnected metabolic-inflammatory network rather than a strictly linear pathway in the development of atherosclerosis. This occurs because visceral fat mass and adipose tissue inflammation do not always change concurrently, and inflammatory or atherogenic processes may progress independently of quantitative changes in fat depots. Within this network, NLRP3 inflammasome activation and the subsequent increase in IL-1 β play pivotal roles by suppressing the GPR109a–ABCA1 pathway, thereby reducing macrophage cholesterol efflux and promoting foam cell formation.

Clitoria ternatea L. (purple butterfly pea), through its anthocyanin, flavonoid, and phenolic compounds, has demonstrated antioxidant, anti-inflammatory, and lipid- and metabolism-modulating effects in various animal and human studies. These effects include improvements in body weight, lipid profiles, oxidative stress, and several inflammatory markers, as well as potential benefits for perivascular adipose tissue function. However, its effects on IL-1 β regulation remain context-dependent and are not consistently significant. Furthermore, direct evidence of its inhibitory effects on foam cell formation within atherosclerotic lesions remains unavailable, as existing studies have primarily examined upstream parameters or biomarkers associated with foam cell formation rather than directly evaluating foam cell formation itself.

Overall, *Clitoria ternatea* L. can be conceptually positioned as a potential multi-target modulator that may influence several points within the pathway linking visceral obesity, IL-1 β -mediated inflammation, and foam cell formation in atherosclerosis through improvements in lipid metabolism, antioxidant activity, and inflammatory modulation. Nevertheless, its direct effects on the NLRP3–IL-1 β –ABCA1–foam cell pathway have not been experimentally established and require further confirmation. Future research should integrate metabolic, inflammatory parameters (including NLRP3, ASC, and caspase-1), and histopathological assessments (including macrophage markers, ABCA1/ABCG1 expression, and cholesterol efflux) within a unified experimental design. Studies should also examine dose-response relationships across different extract types and administration durations to determine more definitively the role of *Clitoria ternatea* L. in modulating the atherosclerotic process.

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