Biomedical Journal of Indonesia
Journal Homepage: https://bji-fk.ejournal.unsri.ac.id

Hypolipidemic Effect of Galoba Fruit (*Hornstedtia* sp.) Extract on LDL-Cholesterol in Hypercholesterolemic Mice

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ARTICLE INFO

Keywords:
Hypercholesterolemia
Hornstedtia sp.,
LDL-cholesterol
High-Fat diet

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.32539/BJI.v12i1.289

Received 8 August 2025;
Accepted 6 September 2025

ABSTRACT

Introduction. Elevated low-density lipoprotein cholesterol (LDL-C), particularly in its oxidized form, contributes to atherosclerosis and increases the risk of cardiovascular disease. Galoba fruit (*Hornstedtia* sp.) contains antioxidant phytochemicals that may support lipid regulation. This study evaluated the hypolipidemic effect of *Hornstedtia* sp. extract in hypercholesterolemic mice. **Methods.** Twenty-five male mice were randomly assigned to five groups: normal control (KN), negative control (K–, high-fat diet only), positive control (K+, simvastatin 10 mg/kg BW), and two treatment groups receiving Galoba fruit extract at doses of 200 mg/kg BW (P1) and 400 mg/kg BW (P2). Hypercholesterolemia was induced with a high-fat diet, and the extract was prepared by ethanol maceration. Because of limited serum volume, LDL-C levels were estimated indirectly using the Anandaraja equation based on total cholesterol and triglyceride concentrations. **Results.** LDL-C levels decreased in the extract-treated groups compared with the negative control. The Kruskal–Wallis test showed significant differences among groups ($p < 0.05$; overall $p = 0.000$). The greatest reduction was observed in P1 (11.90 mg/dL), followed by P2 (16.60 mg/dL), although neither exceeded the effect of K+ (7.90 mg/dL). **Conclusion.** *Hornstedtia* sp. extract demonstrated potential in lowering LDL-C in hypercholesterolemic mice, suggesting its role as a natural adjunct for lipid control. However, the use of indirect LDL-C estimation and the small sample size warrant cautious interpretation. Further studies with direct lipid profiling and mechanistic exploration are recommended before translational application.

1. Introduction

Hypercholesterolemia is defined as a condition in which blood cholesterol levels exceed the normal limit (>200 mg/dL). It can also be diagnosed by measuring specific lipid fractions, including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides. If left untreated, hypercholesterolemia may lead to atherosclerosis, thereby increasing the risk of coronary heart disease and stroke.[1,2] According to recent global data, the prevalence of hypercholesterolemia is highest in Western Europe (54%), followed by the United States (48%) and Southeast Asia (30%).[3,4] In Indonesia, the 2018 Basic Health Research (Riskesdas) reported that the prevalence of hypercholesterolemia was 39.4% among individuals aged 15–34 years, increasing to 52.9% in those aged 35–59 years.[5] At the regional level, the prevalence in Ambon City, Baguala District, was 34% in 2021, while in Leihitu District, Central Maluku Regency, it reached 58%.[6,7] These findings indicate that hypercholesterolemia remains a major public health concern, underscoring the importance of early prevention and effective treatment.

The increase in cholesterol levels is mainly associated with elevated low-density lipoprotein (LDL) concentrations. LDL particles carry approximately 70% of circulating cholesterol and about 22% protein by mass, making them the primary atherogenic lipoproteins that easily adhere to arterial walls.[8,9] Optimal LDL levels should remain below 100 mg/dL to prevent plaque formation in blood vessels.

Plaque develops as a result of cholesterol deposition in the vascular intima, which promotes clot formation and subsequent obstruction of blood flow, ultimately triggering atherosclerosis. Lowering blood cholesterol is therefore an essential strategy to reduce the risk of cardiovascular damage. Therapeutic approaches may include both non-pharmacological and pharmacological interventions.[8]

Current pharmacological treatment for hypercholesterolemia generally begins with the use of simvastatin as the initial therapy. However, simvastatin can cause side effects such as indigestion, muscle pain, liver damage, gallstone formation, and kidney damage, especially in long-term use.[10] Therefore, non-pharmacological therapy can be a companion therapy to reduce the risk of pharmacological side effects and as an effort to overcome hypercholesterolemia. One of them comes from herbal plants that contain antioxidants to fight excess free radicals in the body by inhibiting the absorption of fat in the digestive system.[11]

One of the fruits that is an endemic plant from Maluku Province, namely Galoba fruit (*Hornstedtia* sp.), has a content of chemical compounds including flavonoids, saponins, alkaloids, tannins, and polyphenols which are antioxidants, anticoagulants, antihypertensive, and anti-inflammatory.[12] Flavonoids are a diverse group of polyphenolic compounds with antioxidant properties that have been reported to influence lipid metabolism. Certain flavonoid subclasses may reduce cholesterol levels by modulating 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase activity, a key enzyme in the initial stage of cholesterol synthesis. Inhibition of this pathway can enhance LDL receptor expression and promote LDL catabolism, thereby lowering plasma LDL concentrations.[13] However, the specific mechanisms vary considerably among flavonoid subclasses, and further evidence is needed to confirm these effects.

2. Methods

Study Design and Animals

This study employed an experimental post-test-only control group design. A total of 25 healthy male BALB/c inbred mice (*Mus musculus*), aged 8–10 weeks and weighing 25–30 g, were used. The animals were acclimatized for 7 days under standard laboratory conditions (temperature 22–25 °C, humidity 55–70%, 12-hour light/dark cycle) with ad libitum access to food and water.[14,15]

Ethical Approval

All procedures involving animals were conducted in accordance with the guidelines of the Institutional Animal Care and Use Committee (IACUC) and were approved by the Animal Ethics Committee of Universitas Pattimura, with ethical clearance number 054/FK-KOM.ETIK/VIII/2024, dated 15 Mei 2024.

Animals Group and Treatment

The mice were randomly divided into five groups (n = 5 per group) as follows:

1. KN (Normal Control): standard diet without treatment
2. K^- (Negative Control): High-Fat diet (HFD) without treatment
3. K^+ (Positive Control): HFD + simvastatin 10 mg/kg BW
4. P1: HFD + *Hornstedtia* sp. extract 200 mg/kg BW
5. P2: HFD + *Hornstedtia* sp. extract 400 mg/kg BW

Hypercholesterolemia was induced in all groups except KN by administering a high-fat diet for 14 days. Simvastatin and *Hornstedtia* sp. extract were then administered orally once daily for 14 days via gavage. The extract doses (200 and 400 mg/kg BW) were chosen based on precedent from similar preclinical plant-extract studies, such as *Vernonia amygdalina* (100–200 mg/kg) demonstrating lipid-lowering effects.[16] Moreover, preliminary data on *Hornstedtia* sp. suggest significant antioxidant activity.[17] However, to our knowledge, no specific pharmacokinetic data or dose-finding studies for *Hornstedtia* sp. in hypercholesterolemia models are available, and thus this remains a limitation.

Preparation of Galoba fruit Extract

Fresh fruits of *Hornstedtia* sp. were collected, washed, sliced, and dried at room temperature. The dried materials were ground into powder and macerated in 96% ethanol (1:10 w/v) for 72 hours with occasional stirring. The mixture was filtered, and the filtrate was concentrated using a rotary evaporator at 40–50 °C to obtain a thick ethanolic extract. The extract yield was 10% (w/w) relative to the dried starting material. Standardization of the extract was performed through preliminary phytochemical screening, and antioxidant activity was confirmed using the DPPH radical scavenging assay, with an IC_{50} value of 1.2935 µg/mL.

Blood Collection and LDL Analysis

On day 29, the mice were fasted overnight, anesthetized with ketamine (100 mg/kg BW) and xylazine (10 mg/kg BW), and sacrificed by cervical dislocation. Blood samples were obtained via cardiac puncture and centrifuged at 3000 rpm for 15 minutes to separate serum. Serum LDL-cholesterol levels were estimated indirectly using the Anandaraja formula, which has been validated primarily in human studies but not specifically in murine models, thus representing a methodological limitation in this study.[18,19]

LDL-cholesterol levels were calculated using the Anandaraja formula (LDL = Total Cholesterol − [0.9 × Triglycerides / 5] − 28), which does not require HDL values. Although this equation has been validated in human clinical populations, its application in murine models lacks species-specific validation and therefore represents a methodological limitation of the present study.[18,20] The biochemical parameters (Total Cholesterol, HDL, Triglycerides) were measured

spectrophotometrically using commercial diagnostic kits.

Statistical Analysis

Data were analyzed using SPSS version 23. Since LDL values were only measured post-treatment, between-group comparisons were performed using the Kruskal–Wallis test. When the global test was significant, pairwise post hoc comparisons were conducted using Dunn's test with Bonferroni correction to control for Type I error. A p-value < 0.05 was considered statistically significant.

3. Results

Cholesterol Levels

At baseline (Day 0), all groups showed cholesterol levels below the detectable limit of the POCT device (<100 mg/dL). Therefore, quantitative comparisons of baseline values among groups could not be performed. By day 15, cholesterol levels increased markedly in all groups receiving a high-fat diet (K−, K+, P1, and P2), with values exceeding 130 mg/dL, confirming successful induction of hypercholesterolemia. In contrast, the normal control group (KN) maintained cholesterol levels within the normal range. After 14 days of treatment (Day 29), cholesterol levels decreased significantly in the simvastatin group (K+) and in both Hornstedtia sp. extract groups (P1 and P2), reaching levels <130 mg/dL, indicating a lipid-lowering effect.

LDL Cholesterol Levels After Treatment

LDL cholesterol was not measured directly, but rather estimated using the Anandaraja formula (LDL = Total Cholesterol − [0.9 × Triglycerides / 5] − 28), as shown in Table 2. According to this approach, the negative control group (K−) exhibited a median LDL of 247 mg/dL (Table 3). However, such extraordinarily high LDL values are atypical for mouse models and likely reflect calculation artifacts or inappropriate application of a human-derived formula to murine data, highlighting a significant methodological limitation. As a more accurate alternative, direct LDL measurement via homogeneous enzymatic assays or validated equations tailored to the species, such as Vujovic's, Martin-Hopkins, or Sampson's formulas, should be considered for future studies.[6,21,22]

The Kruskal–Wallis test showed a significant difference in LDL cholesterol levels between groups ($p < 0.001$). Based on these results, a post-hoc analysis was carried out using the Dunn test with Bonferroni correction. The results showed that the negative control group (K−) had significantly higher LDL cholesterol levels compared to the normal control (KN), positive control (K+), and treatment groups (P1 and P2) ($p < 0.05$). Conversely, no significant differences were found between the positive control, treatment group, and normal control (all $p > 0.05$). These results indicate that administration of Hornstedtia sp. extract at doses of 200 and 400 mg/kg BW effectively reduces LDL cholesterol levels to values comparable to simvastatin and normal controls, thus supporting the research objective to demonstrate the potential hypolipidemic effects of the extract.

Table 1. Cholesterol levels on days 0, 15, and 29

Group	Day-0	Day-15	Day-29
KN	<100	101.00 ± 1.30	101.20 ± 1.30
K-	<100	132.20 ± 1.92	139.80 ± 5.07
K+	<100	134.80 ± 2.05	103.80 ± 3.03
P1	<100	134.20 ± 3.96	106.60 ± 9.24
P2	<100	135.20 ± 3.49	113.60 ± 7.60

Table 2. Total cholesterol levels and triglyceride levels

Group	Total Cholesterol Levels (mg/dL)	Triglyceride Levels (mg/dL)
KN	50.60 ± 26.07	99.40 ± 7.06
K-	242.00 ± 34.83	188.40 ± 65.10
K+	58.40 ± 44.29	146.80 ± 43.18
P1	94.40 ± 67.65	120.00 ± 22.25
P2	128.00 ± 77.48	159.00 ± 62.53

Table 3. LDL-C levels after treatment

Group	Average LDL-C Levels (mg/dL)	*P Value*
KN	41 (30-44)	
K-	247 (189-283)	
K+	43 (27-135)	P<0.001
P1	51 (42-178)	
P2	102 (58-216)	

4. Discussion

This study demonstrated that a high-fat diet (HFD) comprising quail egg yolks, lard, and repeatedly heated cooking oil effectively induces hypercholesterolemia in mice, as reflected in increased levels of LDL cholesterol. Similar findings have been reported by Anggriani et al. (2021), although there were differences in baseline values and study duration. In hypercholesterolemic conditions, LDL cholesterol concentrations increase in the body. Elevated LDL is susceptible to precipitation and oxidation by free radicals, which can lead to plaque formation in blood vessels and increase the risk of serious disease. Therefore, adequate therapy to lower LDL cholesterol is necessary [23,24]

Simvastatin, a standard HMG-CoA reductase inhibitor, significantly lowered LDL cholesterol, consistent with its established mechanism of inhibiting cholesterol synthesis and increasing LDL clearance. [25] Treatment with *Hornstedtia* sp. fruit extract at both tested doses also reduced LDL cholesterol compared to the untreated HFD group. Specifically, the 200 mg/kg BW dose produced a stronger effect than the 400 mg/kg BW dose. Although this pattern may indicate a non-linear dose-response behavior, strong conclusions about a biphasic response cannot be drawn from just two dose levels. Further research with a wider dose range is needed.

The LDL-lowering effect of *Hornstedtia* sp. extract is likely related to its phytochemical content. Previous studies have shown that flavonoids and terpenoids can modulate lipid metabolism and oxidative stress.[26,27] Flavonoids have been reported to downregulate HMG-CoA reductase and increase LDL receptor expression, thereby promoting LDL clearance.[28] Tannins can reduce cholesterol absorption through intestinal interactions.[29] These antioxidants function to scavenge peroxyl radicals, thus protecting LDL from oxidative damage, increasing VLDL absorption, and increasing VLDL excretion from plasma. Higher levels of antioxidants in the blood are associated with lower LDL and triglyceride levels.[30,31] However, this mechanism remains speculative in this context, as our study did not directly investigate the molecular target.

The antioxidant capacity of *Hornstedtia* sp. has been previously reported, with DPPH assay results classifying its activity as very potent ($IC_{50} \approx 1.29$ ppm).[32] Antioxidants can reduce LDL oxidation, a crucial step in atherosclerotic plaque formation.[24] Therefore, the observed LDL-lowering effect may be partly due to the antioxidant properties of *Hornstedtia* sp. extract. Importantly, the efficacy of this extract did not surpass simvastatin, consistent with previous findings that herbal preparations are often best used as adjunctive rather than replacement therapy.[11] Given the side effects of long-term statin use, natural extracts with antioxidant and lipid-lowering potential may provide additional benefits.

In summary, *Hornstedtia* sp. extract. demonstrated significant LDL-lowering activity, with the most pronounced effect at a dose of 200 mg/kg body weight. At higher doses (400 mg/kg body weight), *Hornstedtia* sp. extract was less effective due to its high active ingredient content, which can accumulate in the digestive tract. These active compounds may not act optimally in excess, as overly high concentrations of exogenous antioxidants can disrupt endogenous antioxidant enzyme regulation, interfere with redox signaling, and potentially inhibit downstream metabolic pathways phenomena encapsulated in the concept of "antioxidative stress" or the "antioxidant paradox.[33] Thus, excessive supplementation may impair enzymatic function and metabolic homeostasis, rather than enhance it. This reaction causes the antioxidant compounds in *Hornstedtia* sp. extract, such as flavonoids, tannins, and others, to not work optimally to lower LDL cholesterol levels.[34] These findings support its potential as an adjuvant in the management of dyslipidemia. However, further research is needed to establish its pharmacokinetics, long-term safety, and synergistic interactions with conventional lipid-lowering agents.

5. Conclusion

Hornstedtia sp. (Galoba fruit) extract was associated with reductions in LDL cholesterol levels in hypercholesterolemic mice, with the 200 mg/kg BW dose showing greater activity than the 400 mg/kg BW dose. While these effects may relate to the extract's antioxidant constituents, the response was less pronounced than simvastatin. Interpretation of these findings should be made cautiously, as LDL was not directly measured but estimated using a formula not specifically validated in mice, and the study applied a post-test only design without baseline LDL data. Given these limitations and the absence of safety evaluation, further mechanistic studies and controlled clinical trials are needed before therapeutic implications can be drawn.

6. Author Contribution

N.F.R and R.D.A. carried out the experiment. N.F.R. wrote the manuscript with support from R.D.A. R.L. V.Z.L and S. N.F.R collected the Cholesterol, Total Cholesterol, and LDL-C data sample. R.D.A and R.L. helped supervise the project. V.Z.L. and S. conceived the original idea. R.D.A. supervised the project.

7. Acknowledgements

The authors would like to express their sincere gratitude to the Faculty of Medicine, Universitas Pattimura, for providing financial support through the 2024 PNBP Research Grant (Grant No. 1180/UN13/SK/2024). This research was also made

possible with the institutional support and research facilities provided by Universitas Pattimura.

8. References

1. Mach F, Baigent C, Catapano AL, et al. *2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk*. *Eur Heart J*. 2020;41(1):111-188.
2. Perkumpulan Endokrinologi Indonesia (PERKENI). *Panduan Pengelolaan Dislipidemia di Indonesia*. Jakarta: PERKENI; 2021.
3. NCD Risk Factor Collaboration (NCD-RisC). *Repositioning of the global epicentre of non-optimal cholesterol*. *Nature*. 2020;582(7810):73–77.
4. World Health Organization. *Global Health Observatory: Cholesterol, HDL, mean (crude estimate)*. Geneva: WHO; 2021. https://www.who.int/data/gho/indicator-metadata-registry/imr-details/2384
5. Badan Penelitian dan Pengembangan Kesehatan, Kementerian Kesehatan RI. *Laporan Nasional Riskesdas 2018*. Jakarta: Kemenkes RI; 2019.
6. Nadilla S, Widiarti R, Sabono FN, et al. *Skrining penyakit tidak menular pada masyarakat Desa Negeri Lama, Kecamatan Baguala, Kota Ambon*. *Pattimura Mengabdi*. 2023;1(1):95–105.
7. Leiwakabessy, A. Y., Ode, W., Zawawi, M., & Anmama, A. I. (n.d.). *Skrining penyakit tidak menular (glukosa darah sewaktu, kolesterol, asam urat) di Negeri Ureng Kecamatan Leihitu, Kabupaten Maluku Tengah Tahun 2021.* *Pattimura Mengabdi*. 2023;1(1):76–82.
8. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report*. *Circulation*. 2002;106(25):3143-3421.
9. Nelson DL, Cox MM. *Lehninger Principles of Biochemistry*. 7th ed. New York: W.H. Freeman; 2017.
10. Hariadini AL, Sidharta B, Ebtavanny TG, Minanga EP. *Hubungan Tingkat Pengetahuan dan Ketepatan Penggunaan Obat Simvastatin pada Pasien Hiperkolesterolemia di Apotek Kota Malang*. *Pharmacic Jurnal Indonesia*. 2020;5(2). p.91–6.
11. Sari H, Nurmaulia N, Wahyudi W, Fahdi F.*Uji Efektivitas Antikolesterol Kombinasi Ekstrak Etanol Daun Afrika (Gymnanthemum Amygdalina Del.) Dengan Ekstrak Etanol Daun Kelapa Sawit (Elaeis guineensis Jacq.) Pada Tikus Hiperkolesterol*. *Jurnal Penelitian Farmasi Herbal*.2020;2(2). p.1–7.
12. Sule ME-S, Astuty E, Tahitu R. *In Vitro Antibacterial Activity and Phytochemical Screening of Galoba (Hornstedtia alliacea) Seeds Extract*. Bioactivities. 2023;1(2):917–947
13. Matulatuwa FR, Taihuttu Y, Rahawarin H. *Gambaran Histopatologi Hepar Mencit (Mus musculus) Hiperglikemia Pasca Pemberian Ekstrak Etanol Buah Galoba fruit (Hornstedtia sp.)*. *Molucca Medica*, 2023;16(2), p.128.
14. International Committee on Standardized Genetic Nomenclature for Mice. *Guidelines for Nomenclature of Mouse and Rat Strains*. Bar Harbor (ME): The Jackson Laboratory; 2013.
15. Festing MFW. Inbred strains of mice. In: Hedrich HJ, editor. *The Laboratory Mouse*. 2nd ed. London: Academic Press; 2012. p. 73–96.
16. Adaramoye OA, Akintayo O, Achem J, Fafunso MA. *Lipid-lowering effects of methanolic extract of Vernonia amygdalina leaves in rats fed on high cholesterol diet*. *Vasc Health Risk Manag*. 2008;4(1):235-41.
17. Hemalatha S, Wahi AK, Singh PN, Chansouria JP. *Hypolipidemic activity of aqueous extract of Withania coagulans Dunal in albino rats*. *Phytother Res*. 2006 Jul;20(7):614-7.
18. Anandaraja S, Narang R, Godeswar R, Laksmy R, Talwar KK. *Low-density lipoprotein cholesterol estimation by a new formula in Indian population*. *Int J Cardiol*. 2005;102(1):117–20.
19. *Mouse LDL-Cholesterol Assay Kit*. Assay range 4.5–245 mg/dL; sample volume 3 µL; Crystal Chem.
20. Khovidhunkit W, Kim MS, Memon RA, Shigenaga JK, Moser AH, Feingold KR, et al. *Effects of infection and inflammation on lipid and lipoprotein metabolism: mechanisms and consequences to the host*. *J Lipid Res*. 2004;45(7):1169–96.
21. Alpdemir M, Alpdemir MF, Şeneş M. *Comparison of Friedewald, Martin/Hopkins, and Sampson formulae with direct LDL measurement in hyperlipidaemic and normolipidaemic adults in a Turkish population.* *J Med Biochem*. 2024 Sep 6;43(5):671-680. doi: 10.5937/jomb0-46549. PMID: 39712505; PMCID: PMC11662950.
22. Choi H, Shim JS, Lee MH, Yoon YM, Choi DP, Kim HC. *Comparison of Formulas for Calculating Low-density Lipoprotein Cholesterol in General Population and High-risk Patients with Cardiovascular Disease.* Korean Circ J. 2016 Sep;46(5):688-698. doi: 10.4070/kcj.2016.46.5.688. Epub 2016 Sep 28. PMID: 27721861; PMCID: PMC5054182.
23. Ference BA, Ginsberg HN, Graham I, et al. *Low-density lipoproteins cause atherosclerotic*

cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement from the European Atherosclerosis Society Consensus Panel. *Eur Heart J*. 2017;38(32):2459-2472.

24. Steinberg, D. *The LDL modification hypothesis of atherogenesis: An update. Journal of Lipid Research,* 2009;50(Suppl):S376–S381.
25. Maron, D. J., Fazio, S., Macrae, ;, & Linton, F. (2000). Current Perspectives on Statins. *Cardiovascular Drugs*. http://www.circulationaha.org
26. Yuan Ivani R, Indrawanti K, Rachmawati Dwi A. The *Effect Of Galoba fruit (Hornstedtia Sp.) Fruit Extract On Malondialdehyde Serum Level Of Hyperglycemic Mice (Mus Musculus) Streptozotocin-Induced*. 2024;13(2):72-77.
27. Swargiary A, Daimari A, Daimari M, Basumatary N, Narzary E. *Phytochemicals, antioxidant, and anthelmintic activity of selected traditional wild edible plants of lower Assam.* Indian J Pharmacol. 2016 Jul-Aug;48(4):418-423. doi: 10.4103/0253-7613.186212. PMID: 27756954; PMCID: PMC4980931.
28. Mulvihill EE, Burke AC, Huff MW. *Citrus Flavonoids as Regulators of Lipoprotein Metabolism and Atherosclerosis.* Annu Rev Nutr. 2016 Jul 17;36:275-99. doi: 10.1146/annurev-nutr-071715-050718. Epub 2016 May 4. PMID: 27146015.
29. Nahar, L., Habibi, E., Khuniad, C., Charoensup, R., Kalieva, K., Wang, D., Arabnozari, H., Phuneerub, P., Chaiwut, P., Sangthong, S., Theansungnoen, T., Nath, R., Talukdar, A. das, & Sarker, S. D. (2025). *Bioactive phytochemicals, pharmacological, and therapeutic potential of Dillenia indica: A comprehensive review of current research.* In *Chinese Herbal Medicines*. Elsevier B.V. https://doi.org/10.1016/j.chmed.2025.09.001
30. Gupta, S., Sodhi, S., & Mahajan, V. (2009). Correlation of antioxidants with lipid peroxidation and lipid profile in patients suffering from coronary artery disease. *Expert Opinion on Therapeutic Targets*, *13*(8), 889–894. https://doi.org/10.1517/14728220903099668
31. Kumar, R., Salwe, K. J., & Kumarappan, M. (2017). Evaluation of antioxidant, hypolipidemic, and antiatherogenic property of lycopene and astaxanthin in atherosclerosis-induced rats. *Pharmacognosy Research*, *9*(2), 161–167. https://doi.org/10.4103/0974-8490.204654
32. Gustaman, F., Trisna Wulandari, W., Nurviana, V., Idacahyati STIKes Bakti Tunas Husada, K., Cilolohan No, J., & Korespondensi, T. (2019). *Jurnal Ilmiah Farmako Bahari. Antioxidant Activity Of Pining (Hornstedtia Alliacea) By Using Dpph Method*.
33. Halliwell B. *The antioxidant paradox: less paradoxical now?*. *Br J Clin Pharmacol.* 2013 Mar;75(3):637-644
34. Skibola, C. F., & Smith, M. T. (2000). Potential Health Impacts Of Excessive Flavonoid Intake. *Lars Ernster Commemorative Issue*, *29*, 375–383.