ORIGINAL ARTICLE

Efficacy of Statins on Low-Density Lipoprotein Cholesterol Level Reduction in Thai Patients

Thanatcha Lertnimittham, MD[1], Thinnakrit Sasiprapha, MD[1]

[1] Division of Cardiology, Department of Internal Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

Objective: To evaluate the efficacy of different types and doses of statins on low-density lipoprotein cholesterol (LDL-c) reduction in Thai individuals.

Materials and Methods: The present study was a real-world, retrospective study conducted at Ramathibodi Hospital in Bangkok, Thailand. The authors reviewed medical records and databases for the period between January 1, 2021 and December 31, 2022. Eligible participants were adults aged 18 years and older who initiated statin therapy during this period. The primary outcome was the percentage reduction in LDL-c levels from baseline to the first follow-up visit, categorized by statin intensity.

Results: Four thousand three hundred ninety-four patients were included in the analysis. Simvastatin was the most frequently prescribed statin at 46.9%. The mean percentage reductions in LDL-c were 32.5±14.0% for the low-intensity group, 38.7±16.4% for the moderate-intensity group, and 43.8±18.1% for the high-intensity group. The highest LDL-c reduction within each category was observed with simvastatin 10 mg at 32.9% for low-intensity, rosuvastatin 5 mg at 47.8% for moderate-intensity, and atorvastatin 80 mg at 61.2% for high-intensity. However, the result for atorvastatin 80 mg should be interpreted with caution due to a very small sample size.

Conclusion: In the Thai population, low- and moderate-intensity statins achieved LDL-c reductions consistent with guideline recommendations. However, high-intensity statins demonstrated mean reductions below the guideline target of more than 50%.

Keywords: Dyslipidemia; Statin; LDL-cholesterol reduction

Received 3 April 2025 | Revised 9 October 2025 | Accepted 24 October 2025

J Med Assoc Thai 2026;109(2):88-94

Website: http://www.jmatonline.com

Cardiovascular diseases (CVDs) remain the leading cause of mortality globally, imposing a significant burden on public health systems worldwide. Among the various modifiable risk factors for CVD, dyslipidemia, particularly elevated low-density lipoprotein cholesterol (LDL-c), is recognized as a primary causal factor in the development of atherosclerosis[1]. Consequently, effective management of LDL-c is a cornerstone of both primary and secondary prevention of cardiovascular events[2-6]. Meta-analyses[7] indicate that a one mmol/L reduction in LDL-c is correlated with an approximately 22% reduction in the risk of atherosclerotic cardiovascular disease (ASCVD). Therefore, recommendations suggest maintaining LDL-c levels below 100 mg/dL (2.6 mmol/L) in low-risk patients and lower than 70 mg/dL (1.8 mmol/L)[8] or even 55 mg/dL (1.4 mmol/L)[9] in high-risk patients have been shown to significantly reduce the incidence of ASCVD[10-14].

For decades, HMG-CoA reductase inhibitors, commonly known as statins, have been the first-line pharmacological treatment for dyslipidemia. Large-scale, landmark clinical trials have consistently demonstrated that statin therapy significantly reduces the risk of major adverse cardiovascular events (MACE), including myocardial infarction, stroke, and cardiovascular death. Based on this robust evidence, international clinical practice guidelines, such as those from the American College of Cardiology/American Heart Association (ACC/AHA)[8], have established a framework for statin therapy based on intensity. This framework categorizes statins into high-intensity, which is expected LDL-c reduction of 50% or more, moderate-intensity, which is expected LDL-c reduction of 30% to 49%, and low-intensity, which is expected LDL-c reduction of less than 30% therapies to guide clinical decision-making.

However, the majority of these pivotal trials

Correspondence to:

Lertnimittham T.

Division of Cardiology, Department of Internal Medicine, Faculty of Medicine Ramathibodi hospital, Mahidol University, Bangkok 10400, Thailand.

Phone: +66-2-2012426

Email: Thanatcha.l@hotmail.com

How to cite this article:

Lertnimittham T, Sasiprapha T. Efficacy of Statins on Low-Density Lipoprotein Cholesterol Level Reduction in Thai Patients. J Med Assoc Thai 2026;109:88-94.

DOI: 10.35755/jmedassocthai.2026.2.02687

were conducted in Western populations. It is now increasingly understood that the pharmacokinetic and pharmacodynamic responses to many drugs, including statins, can vary significantly across different ethnic groups. Factors such as genetic polymorphisms in drug-metabolizing enzymes and transporters can influence drug exposure and efficacy[15-17]. Specifically for statins, studies[18-22] had indicated that Asian populations may achieve a greater LDL-c reduction at lower doses compared to their Caucasian counterparts and may also be at a higher risk for dose-related adverse effects[23,24].

Despite the widespread use of statins in Thailand, there is a scarcity of large-scale, real-world data evaluating their effectiveness specifically within the Thai population. Clinical practice in the region relies on extrapolations from international guidelines, which may not be perfectly optimized for Thai patients. Therefore, generating local evidence is crucial for validating and potentially refining treatment strategies. The present study was conducted to address this knowledge gap by evaluating the real-world efficacy of different types, doses, and intensities of statins on LDL-c reduction in a large cohort of Thai patients.

Materials and Methods

Study population and design

Trial design was a real-world study conducted at Ramathibodi Hospital, Bangkok, Thailand. The data were collected by reviewing medical records and the database between January 1, 2021 and December 31, 2022. Eligible patients were 18 years or older with the diagnosis of dyslipidemia and initiated statin in the study period. Exclusion criteria were patients who previously received statin medication before entering the study, patients without pre-statin initiation LDL-c level results, patients with lack of follow-up LDL-c level, patients concurrently receiving lipid-lowering medications other than statins, and patients who exhibited an increase in LDL-c levels after initiating statin therapy were excluded from the analysis. This criterion was established to control for potential confounders such as laboratory measurement error or patient non-adherence.

The authors collected demographic data, including age, gender, body mass index (BMI), underlying diseases, history of stroke, history of coronary artery disease (CAD), renal function, baseline LDL-c level, and follow-up LDL-c level.

The primary outcome was the percentage of reduction in LDL-c level after receiving statins of various types and doses according to intensity of statin at the first follow-up visit.

Statistical analysis

The authors performed the descriptive analysis of the data. The continuous data were presented as mean ± standard deviation (SD). The categorical data was presented in percentage. Continuous variables were compared using one-way analysis of variance (ANOVA) for the three statin intensity groups. Post-hoc comparisons were performed using the Bonferroni test to identify differences between specific groups. A p-value of less than 0.05 was considered statistically significant. All data analyses were performed using IBM SPSS Statistics, version 28.0 (IBM Corp., Armonk, NY, USA).

Ethical approval

The present study protocol was reviewed and approved by the Human Research Ethics Committee, Faculty of Medicine Ramathibodi Hospital, Mahidol University (MURA2023/588).

Results

The patient dispositions are shown in Figure 1. Of the 7610 patients who met the inclusion criteria, the medical records were reviewed to identify exclusion criteria. There were 3,216 patients excluded, and 4,394 patients were used for analyses. Baseline characteristic data classified by intensity of statin are shown in Table 1. There was no difference in age, prevalence of diabetes mellitus (DM) and hypertension (HT) among the groups. History of CAD and stroke were highest in high intensity statin group, at 9.4% and 5.9%, respectively. In addition, the high intensity statin group had the highest baseline LDL-c level.

Primary outcome is shown in Table 2. The mean baseline LDL-c and follow-up LDL-c level were different in each group. The high intensity statin group had the highest mean baseline LDL-c level at 177.0±42.2 mg/dL and the lowest mean follow-up LDL-c level at 97.5±34.3 mg/dL. The mean percent change of LDL-c level in low- and moderate-intensity statin group was 32.5±14% and 38.7±16.4%, respectively, meanwhile in high intensity statin group had the highest mean percent change of LDL-c level at 43.8±18.1%. Post-hoc analysis using the Bonferroni test demonstrated that the reduction in the high-intensity group was significantly greater than in the low- and moderate-intensity groups ($p<0.001$).

For multivariable analysis, a multiple linear

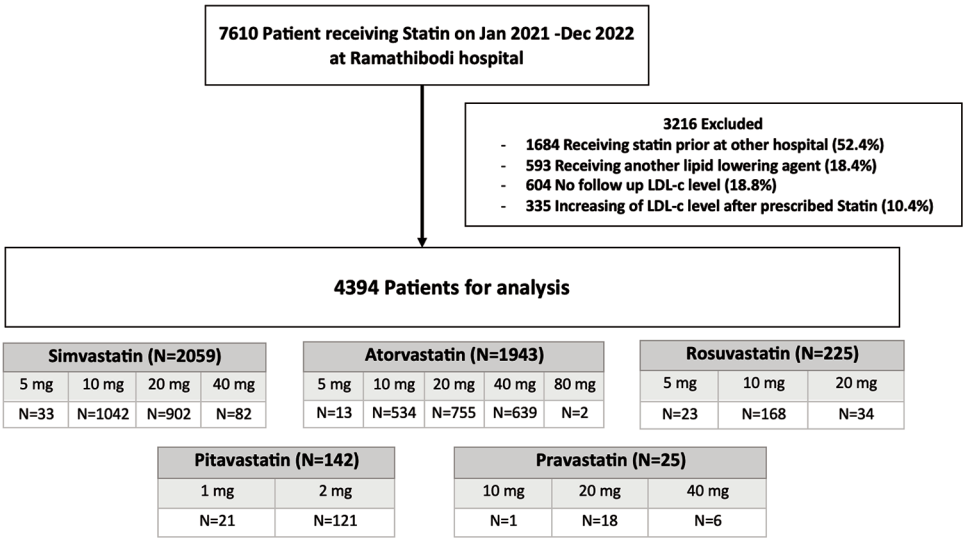

Figure 1. Patient disposition.

Table 1. Baseline patient characteristic classified by intensity of statin

	Low intensity statin	Moderate intensity statin	High intensity statin	p-value
Number of patients; n (%)	1,115 (25.4)	2,606 (59.3)	673 (15.3)	
Age (years); mean±SD	55.3±13.8	56.0±13.2	55.6±14.2	0.257
Sex: male; n (%)	344 (30.9)	1003 (38.5)	282 (41.9)	<0.001
BMI (kg/m^2); mean±SD	25.2±5.2	25.5±6.7	26.0±6.4	0.030
DM; n (%)	272 (24.4)	632 (24.3)	173 (25.7)	0.733
HT; n (%)	485 (43.5)	1124 (43.1)	307 (45.6)	0.509
History of CAD; n (%)	14 (1.3)	55 (2.1)	63 (9.4)	<0.001
History of stroke; n (%)	9 (0.8)	37 (1.4)	40 (5.9)	<0.001
eGFR (mL/min/1.73 cm^2); mean±SD	92.0±19.3	91.1±19.0	90.4±21.9	0.221
Baseline LDL-c (mg/dL); mean±SD	165.5±27.6	171.6±33.3	177.0±42.2	<0.001

BMI=body mass index; DM=diabetes mellitus; HT=hypertension; CAD=coronary artery disease; eGFR=estimated glomerular filtration rate; LDL-c=low density lipoprotein cholesterol

Low intensity statin: Simvastatin 5 to 10 mg, Pitavastatin 1 mg, Pravastatin 20 mg; Moderate intensity statin: Simvastatin 20 to 40 mg, Atorvastatin 5 to 20 mg, Rosuvastatin 5 to 10 mg, Pitavastatin 2 mg, Pravastatin 40 mg; High intensity statin: Atorvastatin 40 to 80 mg, Rosuvastatin 20 mg

Table 2. Baseline and percentage change from baseline in LDL-cholesterol categorized by intensity of statin

	Low intensity statin (n=1,115) mean±SD	Moderate intensity statin (n=2,606) mean±SD	High intensity statin (n=673) mean±SD	p-value
Baseline LDL-c (mg/dL)	165.5±27.6	171.6±33.3	177.0±42.2	<0.001
Follow up LDL-c (mg/dL)	111.0±27.6	104.2±31.8	97.5±34.3	<0.001
% Change	32.5±14.0	38.7±16.4	43.8±18.1	<0.001

LDL-c=low density lipoprotein cholesterol; SD=standard deviation

regression analysis was performed to adjust for potential confounders including age, gender, BMI, and history of CAD and stroke. The analysis confirmed that statin intensity remained a significant predictor of LDL-c reduction ($p<0.001$) after adjusting for these factors.

Figure 2 showed the percentage of LDL-c change classified by intensity of statin. The highest percentage of LDL-c reduction was simvastatin 10 mg at 32.9% from low intensity statin group, rosuvastatin 5 mg at 47.8% from moderate intensity statin group, and atorvastatin 80 mg at 61.2% from high intensity statin group. The percentage of LDL-c change classified by type of statin is shown

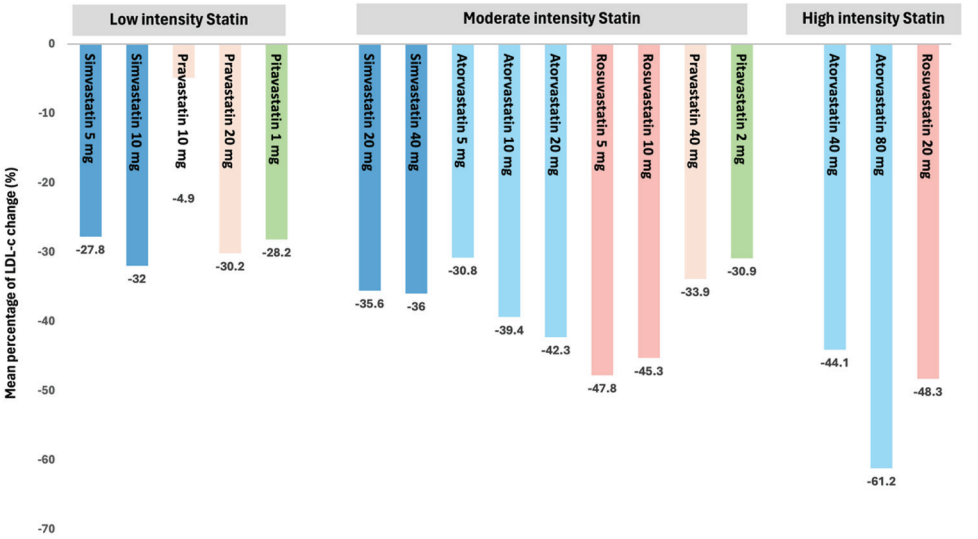

Figure 2. Mean percentage of LDL-c change classified by intensity of statin.

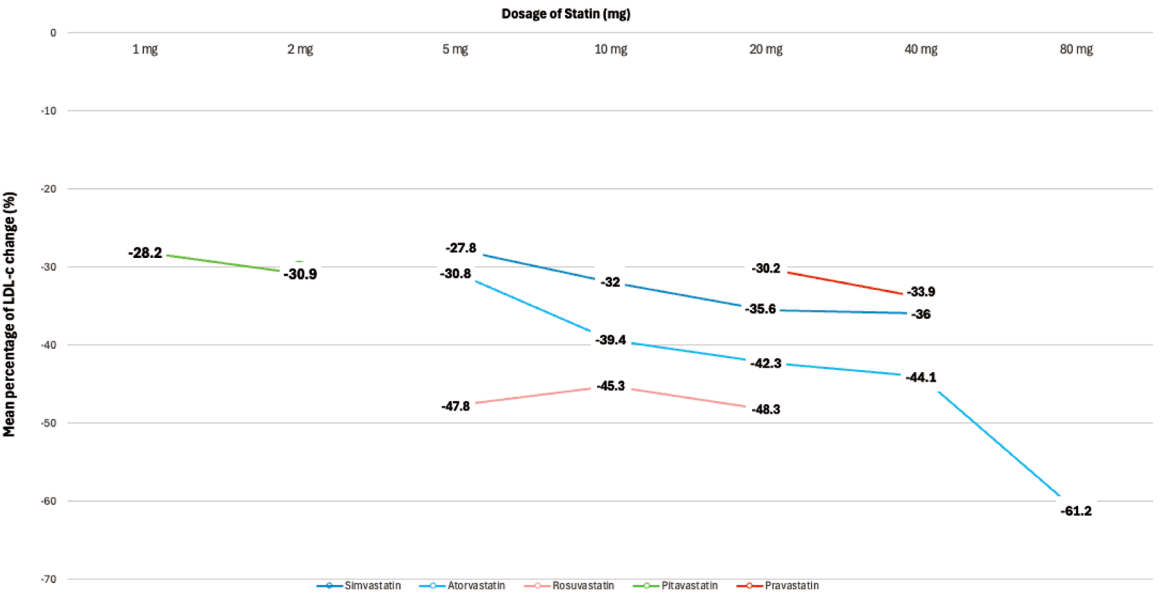

Figure 3. Mean percentage of LDL-c change classified by type of statin.

in Figure 3. There was a gradient of reduction of LDL-c when increasing the dose of statin, except for rosuvastatin, which the percentage of LDL-c reduction was not much different across the dose 5 to 20 mg.

Discussion

This real-world study provides valuable insights into the efficacy of statin therapy in a large Thai cohort, highlighting both consistencies with and deviations from established clinical guidelines. The present study's primary findings demonstrate that low- and moderate-intensity statins achieved mean LDL-c reductions of 32.5% and 38.7%, respectively, which are consistent with the therapeutic targets recommended by the ACC/AHA guidelines. This suggests that for a majority of patients requiring less aggressive lipid-lowering, standard statin regimens are effective in the Thai population.

The most significant finding of this study is the performance of high-intensity statins. As a group, they failed to reach the guideline-recommended target

of a 50% or greater reduction, achieving a mean reduction of only 43.8%. While the high-intensity group started with the highest baseline LDL-c and achieved the lowest absolute follow-up levels, indicating treatment of a high-risk population, the percentage reduction fell short. This observation may be multifactorial. It could reflect "clinical inertia", where physicians might be hesitant to use the highest doses in clinical practice, or it may be influenced by patient adherence. Crucially, the exceptionally high efficacy reported for atorvastatin 80 mg at 61.2% was derived from a sample size of only two patients, which is insufficient for a reliable conclusion and skewed the overall average for this group. Therefore, the true real-world efficacy of high-intensity statins in this population warrants further investigation with larger sample sizes.

The observed variations in LDL-c reduction across different statin intensities are consistent with the previous studies demonstrating the dose-dependent effect of statins on lipid lowering[7,8].

In comparison to the STELLAR study[25], the present research provides additional insights into the efficacy of statins, particularly in the context of Thai patients with dyslipidemia. While the STELLAR study primarily focused on Western populations, the present study specifically targeted Thai individuals. It is important to note that the magnitude of LDL-c reduction observed in the present study may differ from that reported in the STELLAR study due to potential differences in genetic, metabolic, and environmental factors between Thai and Western populations.

One notable difference between the present study and the STELLAR study is the response to rosuvastatin across different doses. While the STELLAR study demonstrated a dose-dependent effect of rosuvastatin on LDL-c reduction, the present study found that lower doses of rosuvastatin provided comparable LDL-c reduction to higher doses. The lack of a significant difference in LDL-c reduction between the 10 mg (45.3%) and 20 mg (48.3%) doses suggests that for Thai patients, escalating the dose may not confer additional lipid-lowering benefits. This could be related to pharmacogenetic variations prevalent in Asian populations, which can lead to higher systemic drug exposure and a more pronounced response at lower doses. This finding has important clinical and economic implications, suggesting that rosuvastatin 10 mg may be an optimal starting dose for achieving significant LDL-c reduction in this population, reserving higher doses for select cases. In contrast, simvastatin, the most prescribed statin in the present study cohort, with 46.9%, performed as expected, providing reliable moderate-intensity efficacy, which explains its role as a workhorse medication in this clinical setting, driven by both cost-effectiveness and physician familiarity.

In addition, it is important to note that not all patients achieved the target LDL-c reduction levels recommended by guidelines, despite receiving high-intensity statin therapy. This suggests that factors beyond the dose of statins may influence the response to treatment. In clinical practice, the use of an initial combination of lipid-lowering therapies, such as ezetimibe, could be considered for high-risk patients who aim to achieve an LDL-c reduction greater than 50%.

The present study has notable strengths, including its large sample size and real-world design, which enhance the generalizability of the present study findings to routine clinical practice in Thailand. However, limitations must be acknowledged. First, its retrospective nature makes it susceptible to confounding variables. Although the authors described baseline differences between groups, a multivariate analysis was not performed to adjust for factors like age, comorbidities, or baseline LDL-c, which could influence the outcome. Second, the timing of the first follow-up visit was not standardized, which may have introduced variability into the results.

Finally, the exclusion of patients with an observed increase in LDL-c levels represents a significant limitation. This paradoxical outcome is not consistent with the pharmacological action of statins and was therefore attributed to external factors, laboratory measurement error, or non-adherence to prescribed therapy. While excluding these individuals was methodologically necessary to analyze the true efficacy of the medication, it introduces a potential selection bias by focusing the analysis primarily on patients who responded to the treatment. This should be taken into consideration when interpreting the overall efficacy rates reported.

Conclusion

The present study confirms the effectiveness of low- and moderate-intensity statins in Thai patients. However, it raises important questions about the real-world application and efficacy of high-intensity therapy. The present study findings underscore the necessity of population-specific

data. Future prospective studies are needed to confirm these findings, and further research into the pharmacogenomics of statin response in the Thai population could help optimize lipid management and guide more personalized treatment strategies.

What is already known about this topic?

Dyslipidemia, characterized by elevated LDL-c levels, is a major risk factor for ASCVD like coronary artery disease and stroke. Reducing LDL-c has been shown to lower the risk of ASCVD, with statins being the primary treatment. Statins work by inhibiting HMG-CoA reductase, reducing cholesterol synthesis, and increasing LDL-c clearance, thereby lowering LDL-c levels and cardiovascular risk.

The 2013 ACC/AHA guidelines classify statins into low, moderate, and high intensity based on their ability to lower LDL-c. High-intensity statins are recommended for high-risk patients to achieve more than a 50% LDL-c reduction. However, responses to statins may differ by population. Studies in Asian populations suggest that lower doses of statins can achieve similar LDL-c reductions compared to Western populations, due to genetic and metabolic differences.

What does this study add?

This study provides valuable insights into the efficacy of different types and doses of statins in reducing LDL-c levels among Thai patients with dyslipidemia. Unlike Western studies, which primarily inform current guidelines, this research specifically addresses the response of the Thai population to statin therapy.

These findings suggest that even low and moderate-intensity statins achieve significant LDL-c reductions in this population, aligning with guideline recommendations. Notably, the response to rosuvastatin was found to be dose-independent, differing from the dose-dependent effects observed in Western cohorts. This highlights the potential for lower doses to achieve optimal LDL-c lowering, minimizing the risk of side effects associated with higher doses.

This study also identifies a gap in the achievement of target LDL-c reductions with high-intensity statins, underlining the need for further investigation into factors influencing statin response in diverse populations.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004;364:937-52.
2. The Lipid Research Clinics Coronary Primary Prevention Trial results. I. Reduction in incidence of coronary heart disease. Jama 1984;251:351-64.
3. Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: final report of the pooling project. The pooling project research group. J Chronic Dis 1978;31:201-306.
4. Anderson KM, Castelli WP, Levy D. Cholesterol and mortality. 30 years of follow-up from the Framingham study. Jama 1987;257:2176-80.
5. Raymond C, Cho L, Rocco M, Hazen SL. New cholesterol guidelines: worth the wait? Cleve Clin J Med 2014;81:11-9.
6. Ference BA, Majeed F, Penumetcha R, Flack JM, Brook RD. Effect of naturally random allocation to lower low-density lipoprotein cholesterol on the risk of coronary heart disease mediated by polymorphisms in NPC1L1, HMGCR, or both: a 2 × 2 factorial Mendelian randomization study. J Am Coll Cardiol 2015;65:1552-61.
7. Baigent C, Blackwell L, Emberson J, Holland LE, Reith C, Bhala N, et al. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170,000 participants in 26 randomised trials. Lancet 2010;376:1670-81.
8. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;129(25 Suppl 2):S1-45.
9. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J 2020;41:111-88.
10. Boekholdt SM, Arsenault BJ, Mora S, Pedersen TR, LaRosa JC, Nestel PJ, et al. Association of LDL cholesterol, non-HDL cholesterol, and apolipoprotein B levels with risk of cardiovascular events among patients treated with statins: a meta-analysis. Jama 2012;307:1302-9.
11. Naci H, Brugts JJ, Fleurence R, Tsoi B, Toor H, Ades AE. Comparative benefits of statins in the primary and secondary prevention of major coronary events and all-cause mortality: a network meta-analysis of placebo-controlled and active-comparator trials. Eur J Prev Cardiol 2013;20:641-57.
12. Law MR, Wald NJ, Rudnicka AR. Quantifying effect

of statins on low density lipoprotein cholesterol, ischaemic heart disease, and stroke: systematic review and meta-analysis. BMJ 2003;326:1423. doi: 10.1136/bmj.326.7404.1423.
13. Cannon CP, Blazing MA, Giugliano RP, McCagg A, White JA, Theroux P, et al. Ezetimibe added to statin therapy after acute coronary syndromes. N Engl J Med 2015;372:2387-97.
14. Byrne P, Demasi M, Jones M, Smith SM, O'Brien KK, DuBroff R. Evaluating the association between low-density lipoprotein cholesterol reduction and relative and absolute effects of statin treatment: A systematic review and meta-analysis. JAMA Intern Med 2022;182:474-81.
15. Kim K, Johnson JA, Derendorf H. Differences in drug pharmacokinetics between East Asians and Caucasians and the role of genetic polymorphisms. J Clin Pharmacol 2004;44:1083-105.
16. Wang A, Yu BN, Luo CH, Tan ZR, Zhou G, Wang LS, et al. Ile118Val genetic polymorphism of CYP3A4 and its effects on lipid-lowering efficacy of simvastatin in Chinese hyperlipidemic patients. Eur J Clin Pharmacol 2005;60:843-8.
17. Liao JK. Safety and efficacy of statins in Asians. Am J Cardiol 2007;99:410-4.
18. Wu CC, Sy R, Tanphaichitr V, Hin AT, Suyono S, Lee YT. Comparing the efficacy and safety of atorvastatin and simvastatin in Asians with elevated low-density lipoprotein-cholesterol--a multinational, multicenter, double-blind study. J Formos Med Assoc 2002;101:478-87.
19. Matsuzawa Y, Kita T, Mabuchi H, Matsuzaki M, Nakaya N, Oikawa S, et al. Sustained reduction of serum cholesterol in low-dose 6-year simvastatin treatment with minimum side effects in 51,321 Japanese hypercholesterolemic patients. Circ J 2003;67:287-94.
20. Patel JV, Gupta S, Lie F, Hughes EA. Efficacy and safety of atorvastatin in South Asian patients with dyslipidemia: an open label noncomparative pilot study. Vasc Health Risk Manag 2005;1:351-6.
21. Nakamura H, Arakawa K, Itakura H, Kitabatake A, Goto Y, Toyota T, et al. Primary prevention of cardiovascular disease with pravastatin in Japan (MEGA Study): a prospective randomised controlled trial. Lancet 2006;368:1155-63.
22. Li YF, Feng QZ, Gao WQ, Zhang XJ, Huang Y, Chen YD. The difference between Asian and Western in the effect of LDL-C lowering therapy on coronary atherosclerotic plaque: a meta-analysis report. BMC Cardiovasc Disord 2015;15:6. doi: 10.1186/1471-2261-15-6.
23. Cannon CP, Braunwald E, McCabe CH, Rader DJ, Rouleau JL, Belder R, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med 2004;350:1495-504.
24. LaRosa JC, Grundy SM, Waters DD, Shear C, Barter P, Fruchart JC, et al. Intensive lipid lowering with atorvastatin in patients with stable coronary disease. N Engl J Med 2005;352:1425-35.
25. Jones PH, Davidson MH, Stein EA, Bays HE, McKenney JM, Miller E, et al. Comparison of the efficacy and safety of rosuvastatin versus atorvastatin, simvastatin, and pravastatin across doses (STELLAR* Trial). Am J Cardiol 2003;92:152-60.