Effect of Statin use on Patients with Hypertension: A Systematic Review and Meta-analysis

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Abstract

Introduction

This meta-analysis focused on the effects of statin use (either alone or in combination with antihypertensive drugs) on hypertension-related outcome measures, including systolic blood pressure (SBP), diastolic blood pressure (DBP), high-density lipoprotein-cholesterol (LDL-C), low-density lipoprotein-cholesterol (HDL-C), triglyceride (TG), total cholesterol, TG and total cholesterol.

Methods

We searched the PubMed, EMBASE, and Cochrane databases before October1 2023.Studies designed as cohort study or randomized controlled trials and investigating the effects of statin use or combined with antihypertensive therapy versus no statin use or antihypertensive therapy alone were included. Authors extracted the data independently; differences were decided to discussion. we use random-effects models to evaluate the merged outcomes. Due to the high heterogeneity of HDL-C group, we performed subgroup analysis according to the type of statin. We use sensitivity analysis, Egger's test and Funnel plots to evaluate the stability and publication bias of our study.

Results

23 trials were included in this meta-analysis. The primary outcomes revealed that (1) administering statins did not significantly impact the SBP of hypertensive patients (MD,-1.77; 95% CI, -4.82 to 1.27). Subgroup analyses revealed a decrease in SBP in patients who received rosuvastatin (MD,-1.70; 95% CI,-2.75 to -0.65) and pravastatin (MD,-8.00; 95% CI,-10.79 to -5.21); (2) no significant effect of statin treatment on DBP in hypertensive patients (MD,-2.04; 95% CI,-4.11 to 0.02). However, subgroup analyses suggest that simvastatin (MD,-2.49; 95% CI, -4.91 to -0.07) and pravastatin (MD,-5.00; 95% CI, -6.60 to -3.40) significantly reduced DBP in hypertensive patients. The secondary outcomes revealed that (1) the use of statins resulted in a significant reduction in LDL-C in hypertensive patients (MD, -0.95; 95% CI, -1.32 to -0.65), while significantly increasing HDL-C (MD, 0.39; 95% CI, 0.15 to 0.64); (2) statins were shown to significantly reduce TG levels in hypertensive patients (MD -0.14, 95% CI -0.23 to -0.05); (3) statins significantly reduced total cholesterol in those hypertensive patients (MD, -1.75; 95% CI, -2.66 to -0.83); (4) statins significantly reduced the incidence of cardiovascular events (HR, 0.73; 95% CI, 0.62 to 0.85) and mortality (HR, 0.47; 95% CI, 0.33 to 0.60).

Conclusion

Statin use did not modulate SBP and DBP of patients with hypertension, but SBP was decreased in rosuvastatin or pravastatin subgroup and DBP was decreased in simvastatin or pravastatin subgroup. Statin treatment reduced LDL-C, increased HDL-C, reduced TG and total cholesterol, reduced the incidence of cardiovascular events and mortality compared to control groups.

Introduction

Hypertension, also known as high blood pressure, is one of the most common diseases in adults and is considered as the leading risk for death globally. Over the past few decades, the incidence rates of hypertension have been increasing largely in low- and middle-income countries. A meta-analysis in 2020 reported that Europe has the highest prevalence of hypertension(61.09%), and followed by Africa(55.87%) and Asia(54.96%)[1]. Hypertension is the strongest evidence for causes of cardiovascular disease and it also has a high prevalence of exposure for cardiovascular disease[2–4]. Large cohort studies have also demonstrated that hypertension is an important risk factor to contribute to heart failure, chronic kidney disease, atrial fibrillation, heart valve diseases, aortic syndromes, coronary heart disease, dementia and stroke[2]. The definition of hypertension is that SBP values ≥ 140 mmHg and/or DBP values ≥ 90 mmHg[5].In addition to controlling blood pressure through lifestyle, many patients also need to control blood pressure through drug treatment, and there are five major drug classes to lower blood pressure: angiotensin converting enzyme inhibitors, betablockers, calcium channel blockers, angiotensin receptor blockers, and diuretics[5]. However, even with the use of various treatment modalities, it is still hard to control the blood pressure to guideline-recommended target levels[4], and there are possible contraindications for each class of drug[5].

Statins are the inhibitors of the hydroxymethylglutaryl-CoA (HMG-CoA) reductase enzyme. Statins effectively lower cholesterol by inhibiting a key step in the sterol biosynthetic pathway and have made a great achievements to the prevention of cardiovascular disease[6]. In addition to lowering blood lipids, statins can also improve endothelial function by retaining endothelial nitric oxide synthase, leading to vasodilation, and then prevent artery disease. The effect of statins on blood vessels may lead to improved cardiovascular outcomes after Percutaneous transluminal coronary angioplasty[6]. A study has shown that statins can prevent the progression of arterial stiffness when used in combination with recommended antihypertensive treatment[7]. Previous studies have reported a reduction in blood pressure in hypertensive patients also treated with statins[8, 9].

Among the meta-analyses now published, there was a meta-analysis that studied the effects of combination statin use on the cardiology cardiovascular risk[10], and our study focused on the effects of statin use (either alone or in combination with antihypertensive drugs) on hypertension-related outcome measures, including SBP, DBP, LDL-C, HDL-C, TG, total cholesterol, cardiovascular event and mortality,.

Methods

This study was conducted according to the 2020 preferred reporting items for systematic reviews and meta-analysis (PRISMA) guidelines.

Search strategy and inclusion criteria

We searched the PubMed, EMBASE, and Cochrane databases for keywords: (rosuvastatin OR simvastatin OR atorvastatin OR pitavastatin OR pravastatin OR fluvastatin OR lovastatin OR cerivastatin OR "statin therapy" OR statins OR statin) AND (hypertension or high blood pressure). English articles and studies on humans were adopted. We searched the studies before October 1 2023.

The original studies were included according to the following criteria: (1) involved adult patients with confirmed hypertension; (2) comparative group containing no statin use; (4) assessed the relationship between statins and hypertension-related outcome measures; (5) provide one or more of the following data: SBP, DBP, LDL-C, HDL-C,, TG, total cholesterol, cardiovascular event and mortality provide 95% confidence interval (CI) including (cardiovascular and mortality), standard deviation including (SBP, DBP, LDL-C, HDL-C, TG and total cholesterol) to assess outcomes; (6) were cohort study or randomized controlled design. (Study search flowchart for searching to Oct 2023 in Fig. 1)

Data extraction

Relevant information was collected from qualified studies, including the first author and year of publication, study design, Single/Multi Center, Study period, sample size, Mean Age or y, Female/ Male of Patients, experimental interventions, and outcomes. Authors extracted the data independently; differences were decided to discussion.

Statistical analysis

Meta-analysis data were analyzed by using STATA 12.0 software. We use the Q test and I2 to assess the heterogeneities among studies. When P < 0.05 and I2 > 50%, it will be defined as high heterogeneity. Therefore, we use random-effects models to evaluate the merged outcomes. Due to the high heterogeneity of HDL-C group, we performed subgroup analysis according to the type of statin. We use sensitivity analysis, Egger's test and Funnel plots to evaluate the stability and publication bias of our study.

Results

Characteristics of Eligible Studies

A total of 30,212 records were initially retrieved from the database, with 23,091 duplicates being eliminated. Further, 22,990 records were excluded by screening the abstracts and titles, leaving only 101 pieces of literature for initial eligibility screening. Subsequently, 78 articles were excluded from these 101 documents, and eventually, 23 trials were included in this meta-analysis. There is a detailed overview of the characteristics of these 23 trials in **Table 1**. Among the reviewed literature, 15 studies[11-25] and 14 studies[11-23, 25] assessed the impact of statins on the SBP and DBP of hypertensive patients respectively. whereas 11 studies[11, 13, 15, 18-21, 25-28] examined the effect of statins on HDL-C levels in hypertensive patients. Additionally, 8 studies[11, 12, 15, 19, 26-29] investigated the effect of statins on LDL-C levels in hypertensive patients, 8 studies[11, 15, 18-20, 25, 26, 28] examined the effect of statins on TG levels in hypertensive patients, 7 studies[18-21, 25, 26, 29] evaluated the effect of statins on total cholesterol levels in hypertensive patients, while 2 studies reported mortality outcomes and another 2 studies focused on cardiovascular events. Five types of statins, namely Pravastatin, Simvastatin, Fluvastatin, Rosuvastatin, and Atorvastatin, were administered at a maximum dose of 80 mg per day in these trials.

Table 1. Characteristic of the Included Studies in the Meta-analysis of Statin Vs Placebo or Standard Supportive Care in Adults With hypertension

Study	Study type	Single/Multi Center	Study period	Total Patients / Patients in Statins No.	Mean Age, y	Female/ Male of Patients No.	Experimental Intervention	Reported Outcomes
AATeixeira, et al (2011)	RCT	SC	NA	39/19	Fluvastatin:51 PC:51	31/8	Fluvastatin (20mg)	HDL-C; LDL-C; TG
Ichihara, et al (2005)	RCT	SC	NA	85/65	Pravastatin:60 Simvastatin:60 Fluvastatin:60 PC:60	57/28	Pravastatin (10mg/d); Simvastatin (5mg/d); Fluvastatin (10mg/d);	DBP; SBP
Beck,et al (2012)	RCT	SC	NA	54/26	Statin:54.2 PC:54	26/28	Simvastatin (26±2mg/d)	DBP; SBP; HDL-C; LDL-C; TG
Williams, et al (2009)	RCT	SC	NA	891/434	Atorvastatin:62.6 PC: 62.8	128/763	Atorvastatin (10mg/d)	SBP

Table 1. (Continued)

Study	Study type	Single/Multi Center	Study period	Total Patients / Patients in Statins No.	Mean Age, y	Female/ Male of Patients No.	Experimental Intervention	Reported Outcomes
Broghi, et al (2000)	RCT	SC	NA	85/41	Statins:61.5 PC:59.1	24/61	Fluvastatin (10-40mg/d) / Simvastatin (10-40mg/d)	DBP; SBP; HDL-C
Chen, et al (2020)	Retrospective Cohort Study	MC	01/2002-12/2017	2650/1325	Statin:72.64 PC:72.67	1144/1506	Statin	Mortality;
Borghi,et al (2004)	RCT	SC	1998-1993	1356/264	Statin:56.3 Lipid-lowering therapy:56.6 Lipid-lowering diet:53.8	580/776	Simvastatin (20-40mg/d)/ Gemfibrozil(400-800mg/d)/ cholestyramine(4-24g/d)	DBP; SBP; LDL-C

Table 1. (Continued)

Study	Study type	Single/Multi Center	Study period	Total Patients / Patients in Statins No.	Mean Age, y	Female/Male of Patients No.	Experimental Intervention	Reported Outcomes
Dong, et al (2021)	RCT	SC	08/2008-11/2010	1133/581	Rosuvastatins:77.05 PC:77.13	560/573	Rosuvastatins (10mg/d)	DBP; SBP; LDL-C; HDL-C;TG; Cardiovascular events; Mortality
Kanaki, et al (2012)	RCT	SC	NA	50/25	Atorvastatin:59.7PC:58.8	26/24	Atorvastatin (10mg/d)	HDL-C; LDL-C;TG;Total Cholesterol
Koh, et al (2015)	RCT	SC	NA	102/51	Simvastatin:57 PC:57	48/54	Simvastatin 20mg/d	DBP; SBP; HDL-C; TG; Total Cholesterol
V Correa,et al (2014)	RCT	SC	NA	79/39	Simvastatin:57.7 PC:55.9	61/18	Simvastatin (40mg/d)	DBP; SBP;

Table 1. (Continued)

Study	Study type	Single/Multi Center	Study period	Total Patients / Patients in Statins No.	Mean Age, y	Female/Male of Patients No.	Experimental Intervention	Reported Outcomes
Messerli, et al (2006)	RCT	MC	NA	439/200	Atorvastatins:55.1 PC:55.3	177/262	Atorvastatins (10mg/d)	HDL-C; LDL-C
Glorioso, et al (1999)	RCT	SC	NA	49/25	Pravastatin:53 PC:53	14/11	Pravastatin (20mg/d)	DBP; SBP
Koh, et al (2011)	RCT	SC	NA	42/NA	Atorvastatins:53 PC:53	20/22	atorvastatin 20 mg/day and amlodipine 10 mg/day	DBP; SBP; HDL-C; LDL-C;TG; Total Cholesterol
Holzhauser,et al (2017)	Retrospective Cojort Study	MC	01/2002-01/2012	762/138	Statin:72 PC:72	488/274	Statin	DBP; SBP;

Table 1. (Continued)

Study	Study type	Single/Multi Center	Study period	Total Patients / Patients in Statins No.	Mean Age, y	Female/ Male of Patients No.	Experimental Intervention	Reported Outcomes
Alves-Cabratosa, et al (2015)	Retrospective Cohort Study	SC	07/2006-12/2015	100276/90462	Statins:68.2 PC:68.1	60518/39758	Statins	Cardiovascular events;
Liu, et al (2014)	RCT	SC	NA	79/39	Rosuvastatins:67.85 PC:65.9	40/39	Rosuvastatins (20mg/d)	DBP; SBP; HDL-C; TG; Total Cholesterol
Nazzaro,et al (1999)	RCT	SC	NA	30/15	Simvastatin:42 PC:44	NA	Simvastatin (10mg/d)	DBP; SBP; HDL-C; Total Cholesterol
Ruszkowski,et al (2019)	RCT	SC	NA	30/20	Fluvastatin:56.5 PC:53.1	12/18	Fluvastatin (80mg/d)	DBP; SBP

Table 1. (Continued)

Study	Study type	Single/Multi Center	Study period	Total Patients / Patients in Statins No.	Mean Age, y	Female/ Male of Patients No.	Experimental Intervention	Reported Outcomes
Sirenko, et al (2023)	RCT	SC	NA	99/59	Atorvastatin:58.02 PC:50.05	41/56	Atorvastatin (20-40mg/d)	LDL-C; Office DBP; Office-SBP; Total Cholesterol
Sirenko, et al (2017)	RCT	SC	NA	587/226	Statins:58.2 PC:55.9	305/282	Statins	DBP; SBP;
Spannella,et al (2020)	Retrospective Cohort Study	SC	NA	738/369	Statin:65.8 PC:65.3	355/383	Statins	Office DBP; Office-SBP;
Danaoğlu,et al (2003)	RCT	SC	NA	39/21	Simvastatin:52 PC:54	29/10	Simvastatin (20mg/d)	DBP; SBP HDL-C; TG;

Abbreviations: RCT, Randomized Controlled Trial; MC, Multi-center; SC, Single-center; PC, Placebo or control; NA, Not acquired; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG,triglycerides; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol;

Primary Outcome

We illustrated the alterations in SBP in hypertensive patients after being subjected to statin treatment in **Figure2**. Notably, outcomes revealed that administering statins did not significantly impact the SBP of hypertensive patients (MD,-1.77; 95% CI, -4.82 to 1.27). Subgroup analyses revealed a decrease in SBP in patients who received rosuvastatin (MD,-1.70; 95% CI,-2.75 to -0.65) and pravastatin (MD,-8.00; 95% CI,-10.79 to -5.21) in **Figure 3**. Outcome indicates changes in DBP following statin therapy in hypertensive subjects in **Figure 4**. The results indicate no significant effect of statin treatment on DBP in hypertensive patients (MD,-2.04; 95% CI,-4.11 to 0.02). However, subgroup analyses suggest that simvastatin (MD,-2.49; 95% CI, -4.91 to -0.07) and pravastatin (MD,-5.00; 95% CI, -6.60 to -3.40) significantly reduced DBP in hypertensive patients in **Figure5**. Statin therapy did not have a significant effect on office SBP (MD,-0.09;95%CI,-2.72 to 2.54; **Figure 6**) and had only a minor effect on office DBP (MD,-0.14; 95%,-5.05 to 4.77; **Figure 7**). No evidence of publication bias was found for SBP (P=0.688) and DBP (P=0.989) based on the results of the funnel plot and Egger test (**Supplementary Figure. 1-4**). **Supplementary Figures 5-6** suggest that the SBP and DBP models were not particularly reliable, as shown by sensitivity analyses.

Figures 8 to 13 demonstrate the evaluation of secondary outcomes. Abbreviations for technical terms were explained upon first use. Common academic sections were included, and author and institution formatting was maintained. The language was formal, objective, and value-neutral. Causal connections were established between statements and the structure had a logical progression. The text was precise, free from grammatical errors, and followed consistent footnote style and formatting features. Biases were avoided, and positions were made clear through hedging. The use of statins resulted in a significant reduction in LDL-C in hypertensive patients (MD, -0.95; 95% CI, -1.32 to -0.65; **Figure 8**), while significantly increasing HDL-C (MD, 0.39; 95% CI, 0.15 to 0.64; **Figure 9**). According to the subgroup analysis, atorvastatin (mean difference (MD) -1.27; 95% confidence interval (CI) -1.61 to -0.94), rosuvastatin (MD -0.62; 95% CI -0.69 to -0.55), and fluvastatin (MD -0.56; 95% CI -1.05 to -0.07) were associated with a reduction in LDL-C in **Supplementary Figure 7**, whilst simvastatin (MD -0.69; 95% CI -1.89 to 0.50) did not demonstrate a significant decrease. In terms of its effect on HDL-C in **Supplementary Figure 8**, pravastatin (MD 0.33, 95% CI 0.15 to 0.51) was found to have no effect when compared with rosuvastatin (MD 0.01, 95% CI -0.11 to 0.14), simvastatin (MD 0.05, 95% CI -0.19 to 0.30), and fluvastatin (MD -0.33, 95% CI -0.80 to 0.14). On the other hand, atorvastatin (MD 1.30, 95% CI -1.66 to 4.27) was found to be more effective. Moreover, statins were shown to significantly reduce TG levels in hypertensive patients (MD -0.14, 95% CI -0.23 to -0.05 in **Figure 10**). In the subgroup analyses in **Supplementary Figure 9**, simvastatin (mean difference [MD], -0.17; 95% confidence interval [CI], -0.24 to -0.11) demonstrated greater efficacy compared to rosuvastatin (MD, -0.01; 95% CI, -0.25 to 0.23), fluvastatin (MD, 0.23; 95% CI, -0.78 to 1.24), and atorvastatin (MD, -0.19; 95% CI, -0.43 to 0.05). Moreover, statins significantly reduced total cholesterol in those hypertensive patients (MD, -1.75; 95% CI, -2.66 to -0.83 in **Figure 11**). Based on the subgroup analysis findings, simvastatin (MD,-1.30; 95% CI,-1.51 to -1.09) and atorvastatin (MD,-2.69; 95% CI,-4.68 to -0.70) were more effective than rosuvastatin (MD,0.00; 95% CI,-0.36 to 0.36) in treating hypertensive patients. Additionally, the results indicate that statins significantly reduced the incidence of cardiovascular events (HR, 0.71; 95% CI, 0.45 to 0.98 in **Figure 12**) and mortality (HR, 0.47; 95% CI, 0.33 to 0.60 in **Figure 13**). Funnel plots and Egger's test indicate no publication bias for LDL-C (P=0.472), HDL-C (P=0.430), TG (P=0.583), and total cholesterol (P=0.643) (**Supplementary Figures 11-18**). **Supplementary Figures 19-22** show that the models for LDL-C, HDL-C, TG, and total cholesterol were less plausible in the sensitivity analyses.

Discussion

This meta-analysis comprehensively comprised 23 studies (109,694patients), revealing the administration of statin use did not diminish SBP, DBP, office-SBP, office-DBP of patients with hypertension compared with control group. Nevertheless, it was disclosed that statin use contributed to a reduction of LDL-C and elevation of HDL-C, respectively. At the same time, statin use decreased the TG and total cholesterol, related with the lower risk of cardiovascular events and mortality. There are controversial outcomes about effect of statin use on lowering blood pressure. Aligning with our results, a study[30] reported that blood pressure lowering was not affected by statin use plus antihypertensive drugs compared with antihypertensive drug alone. However, previous study[31] reported the efficacy of statin use was associated with reduction of SBP and DBP on patients with blood pressure in normotensive or hypertensive. These results of article included a larger scope of patients. Besides, the addition of statin was associated with a significantly greater reduction of SBP compared with amlodipine monotherapy[32]. A meta-analysis conducted by Ying Wang et al.[33] showed that statin group reduced the risk of cardiovascular events compared with non-statin group, similar with our relevant results.

With the efficacy of elevating nitric oxide bioavailability, statin use is also reported improving arterial compliance so that potentially lower blood pressure.[34, 35]. The effects of statin about potentially reduction in blood pressure may profit from anti-inflammatory properties on hypercholesterolemic patients[36]. Combination of and statin and drugs affecting renin-angiotensin-aldosterone system (RAAS) exerts a positive effect since stains use influences RAAS system. Statin inhibit the synthesis of Ang II and aldosterone from following ways, including downregulating the expression of receptors for angiotensin II (Ang II), inhibiting relevant signalling, oxidative stress and inflammation.[37]However there remains little documented the clinical mechanism of it. There is discrepancy from experimental trials and clinical outcomes of statin effect on blood pressure, lack of further exploration.

The drug pharmacokinetics and effect of statins use lowering lipid may differ among ethnicities [38, 39]. The researchers reported statin treatment resulted in significant reductions in cardiovascular events. Patients in the highest tertile of SBP benefited most from intervention.[40] Rosuvastatin therapy reduces incidence of major cardiovascular events regardless of baseline LDL-C level[41].The positive effect of statins may trace back to potential effect of the central or peripheral homeostasis of blood pressure, inhibiting target organ damage, since statin use mediate endothelial vasoactive, downregulating oxidant stress, or inflammation Furthermore, there is also no evidence that statin therapy reducing cardiovascular event morbidity and mortality differ from patients with hypertension and normal blood pressure[42].

This meta-analysis has certain strengths. This study comprehensively assesses the efficacy of statin on patients with hypertension by a thorough literature search. Besides, the results are reliable shown on outcomes of sensitivity analysis. We also have several limitations. Firstly, the ages and complication of patients differ from included studies. Secondly, there is discrepancy on endpoint of administration of drugs. Lastly, high heterogeneity occurs in our results.

Conclusion

Statin use did not modulate SBP and DBP of patients with hypertension, but SBP was decreased in rosuvastatin or pravastatin subgroup and DBP was decreased in simvastatin or pravastatin subgroup. Statin treatment reduced LDL-C, increased HDL-C, reduced TG and total cholesterol, reduced the risk of cardiovascular events and mortality compared to control groups.

Declarations

Author contributions

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Figures
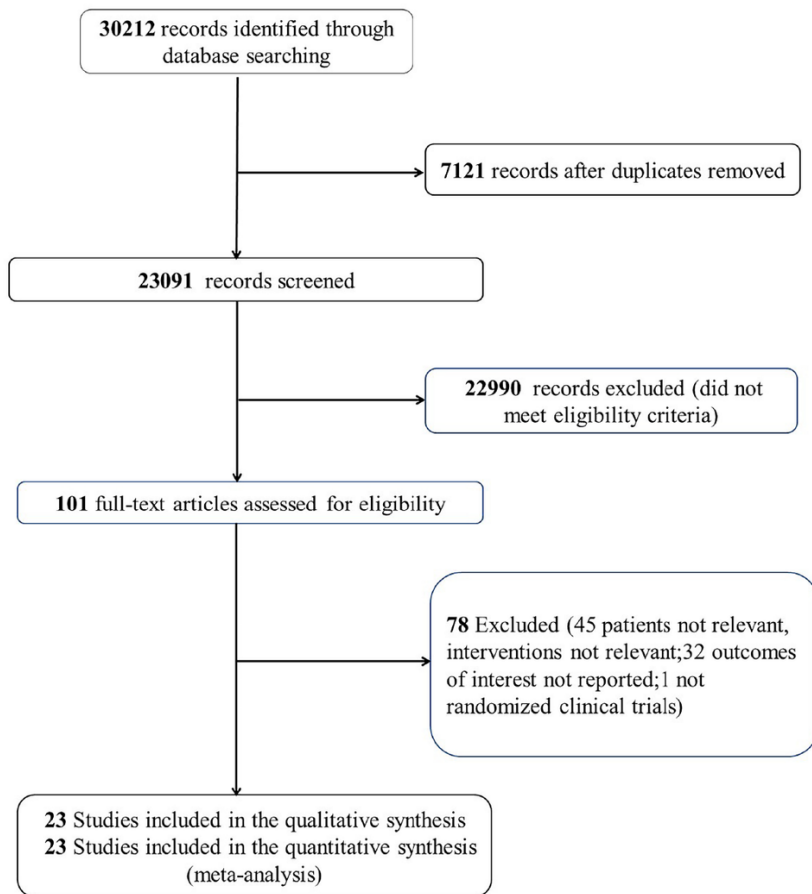
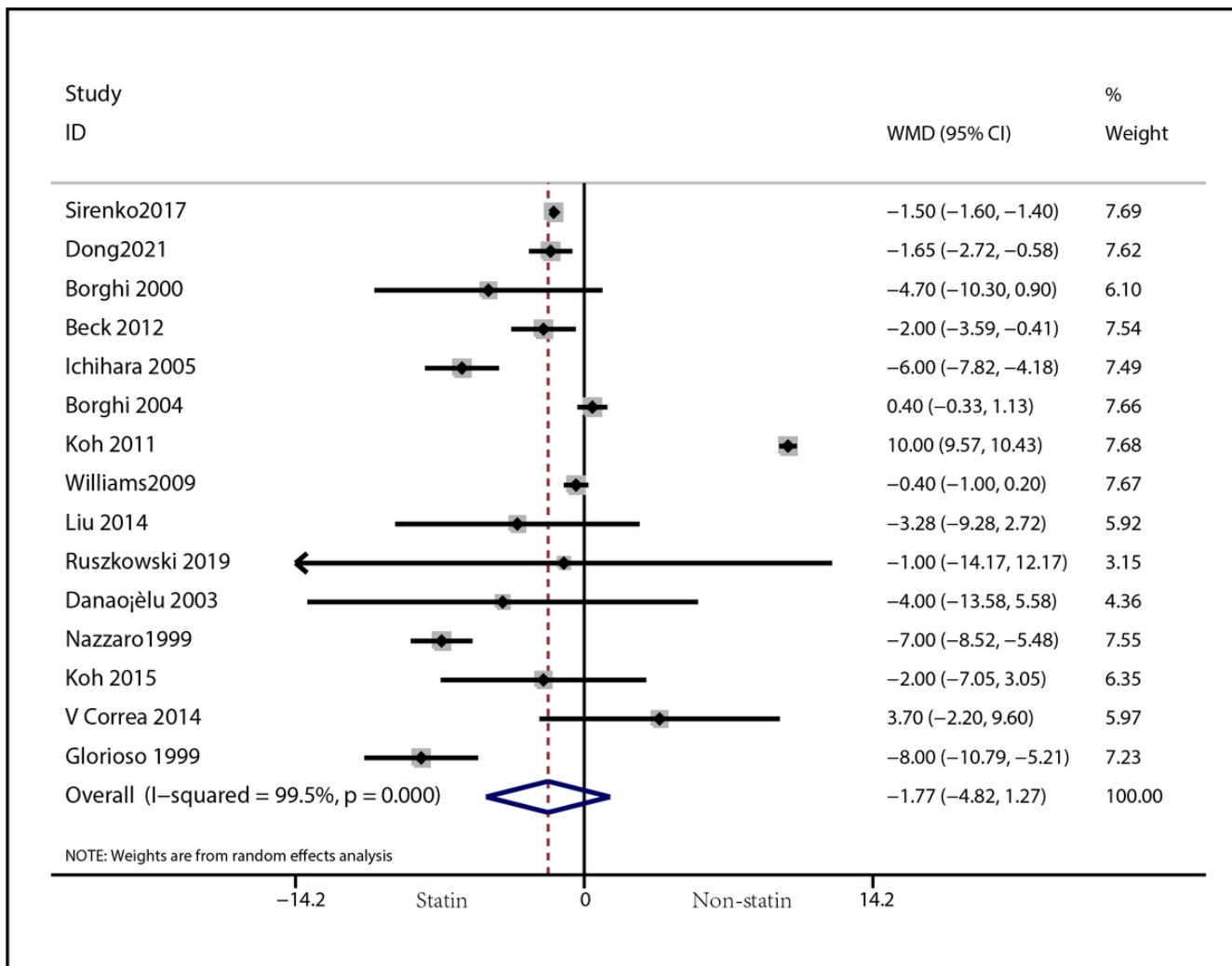**Figure 1**

Study search flowchart for searching to Oct 2023.

Figure 2

Forest plot for assessing SBP in hypertensive patients affected by statin use.
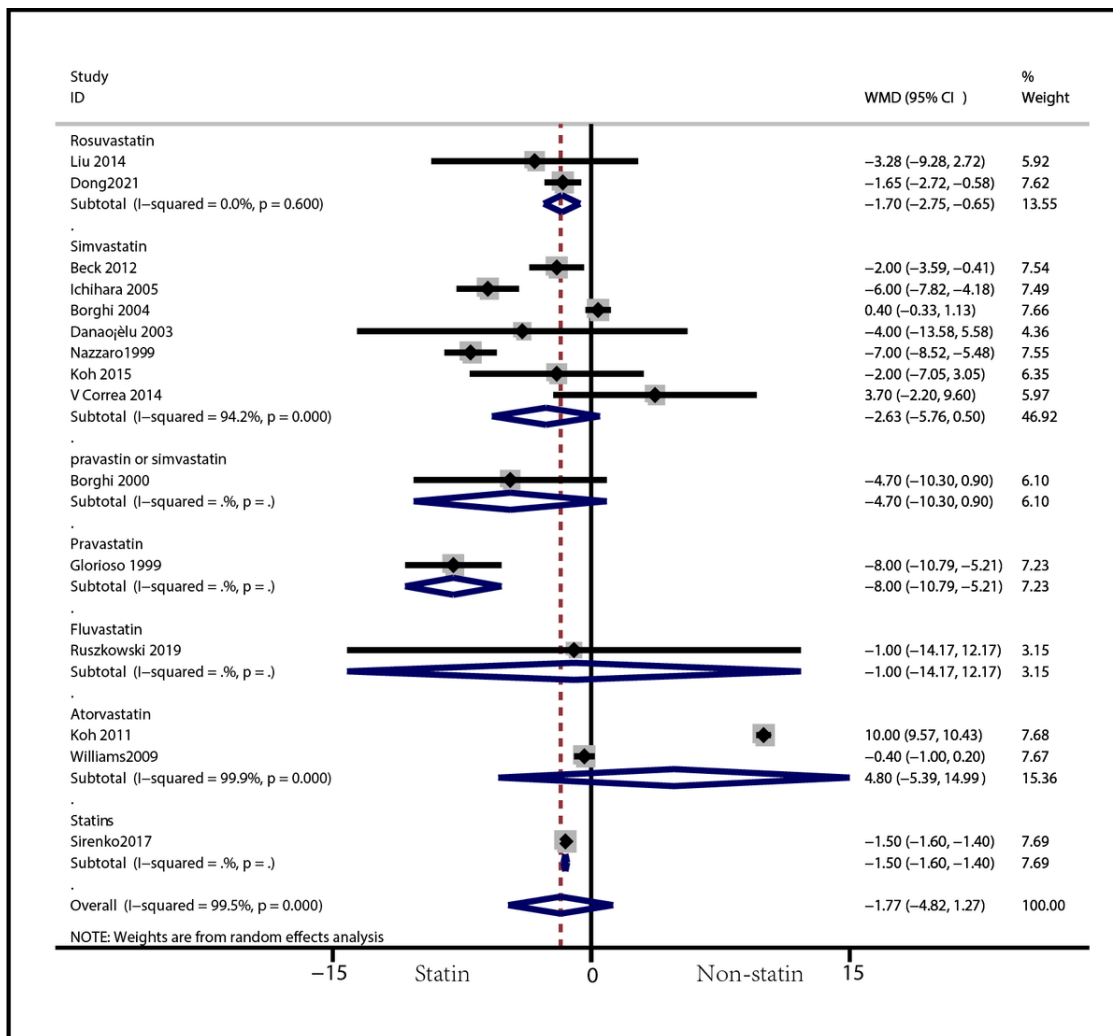
Figure 3

Subgroup analysis of SBP in hypertensive patients affected by statin use according to different types of statins.
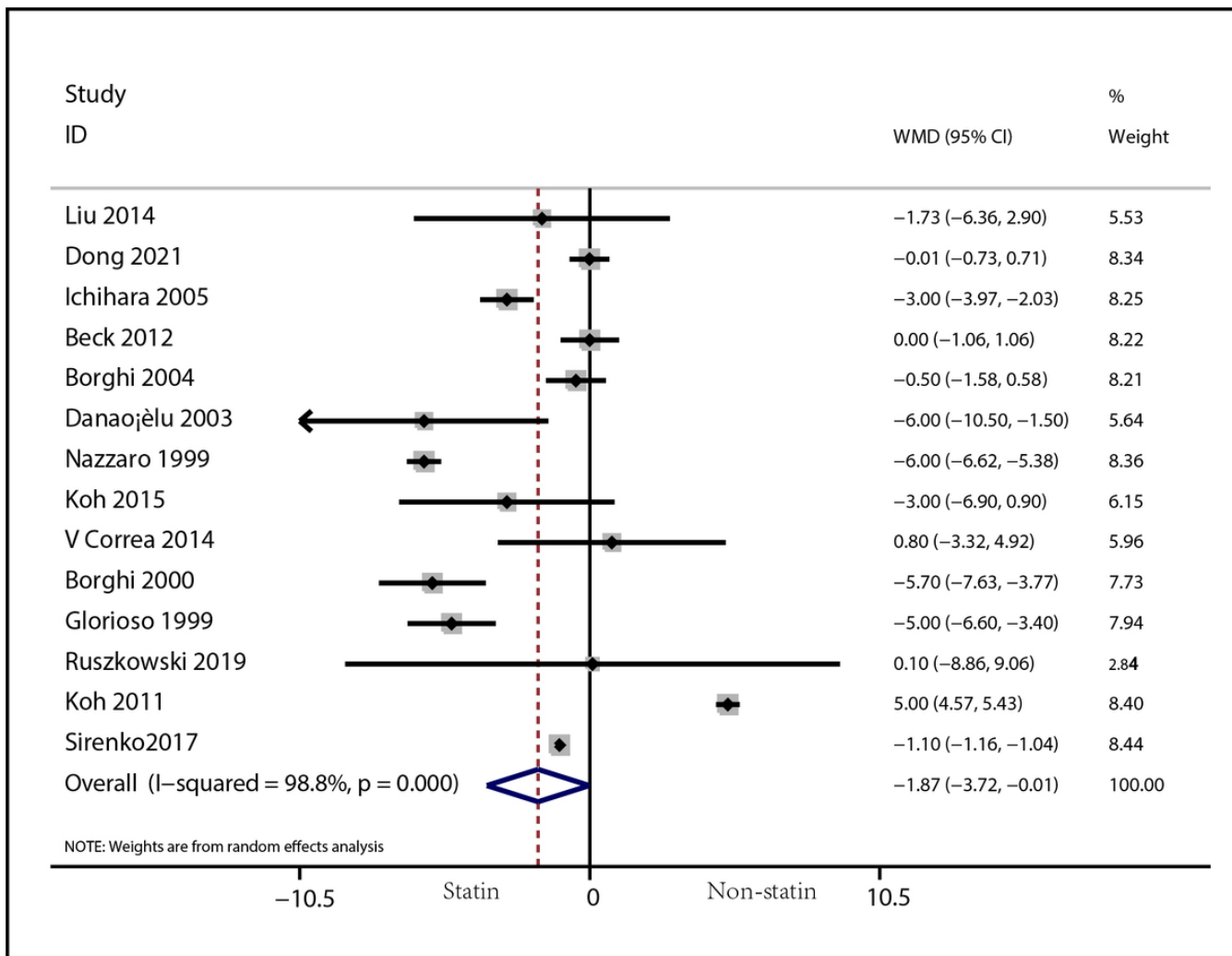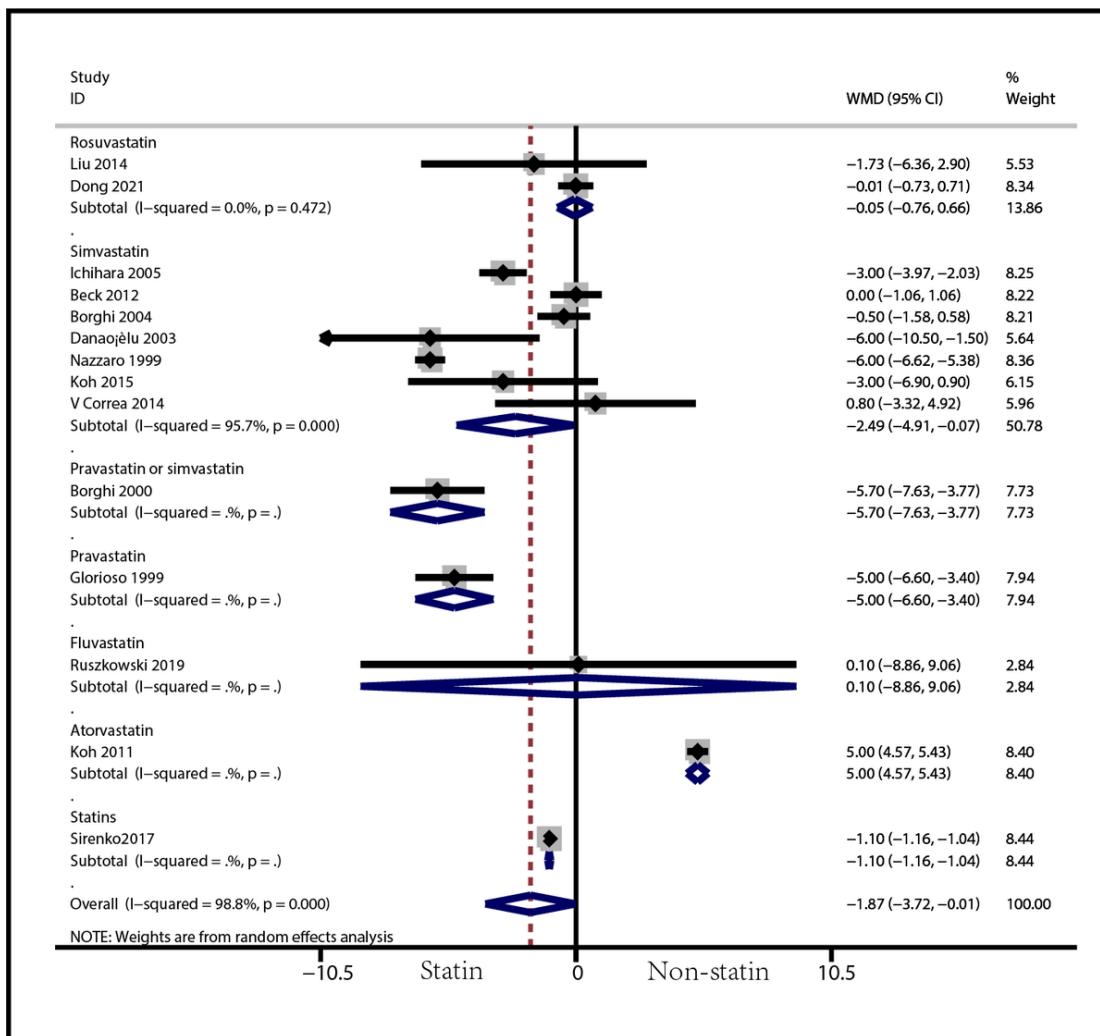
Study ID		WMD (95% CI)	% Weight
Liu 2014		−1.73 (−6.36, 2.90)	5.53
Dong 2021		−0.01 (−0.73, 0.71)	8.34
Ichihara 2005		−3.00 (−3.97, −2.03)	8.25
Beck 2012		0.00 (−1.06, 1.06)	8.22
Borghi 2004		−0.50 (−1.58, 0.58)	8.21
Danaojèlu 2003		−6.00 (−10.50, −1.50)	5.64
Nazzaro 1999		−6.00 (−6.62, −5.38)	8.36
Koh 2015		−3.00 (−6.90, 0.90)	6.15
V Correa 2014		0.80 (−3.32, 4.92)	5.96
Borghi 2000		−5.70 (−7.63, −3.77)	7.73
Glorioso 1999		−5.00 (−6.60, −3.40)	7.94
Ruszkowski 2019		0.10 (−8.86, 9.06)	2.84
Koh 2011		5.00 (4.57, 5.43)	8.40
Sirenko2017		−1.10 (−1.16, −1.04)	8.44
Overall (I−squared = 98.8%, p = 0.000)		−1.87 (−3.72, −0.01)	100.00

NOTE: Weights are from random effects analysis

−10.5   Statin   0   Non-statin   10.5

Figure 4

Forest plot for assessing DBP in hypertensive patients affected by statin use.

Figure 5

Subgroup analysis of SBP in hypertensive patients affected by statin use according to different types of statins.
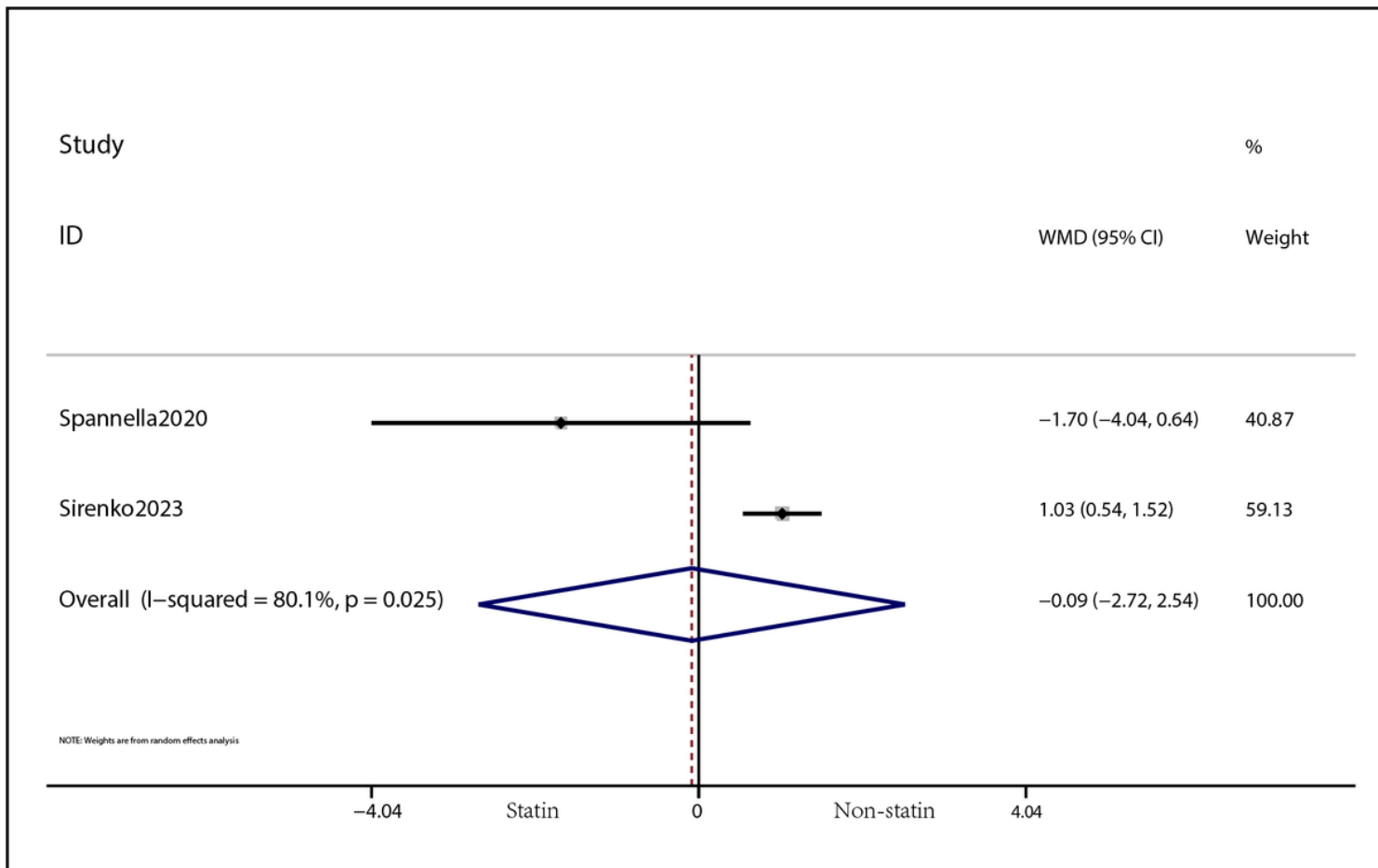
Figure 6

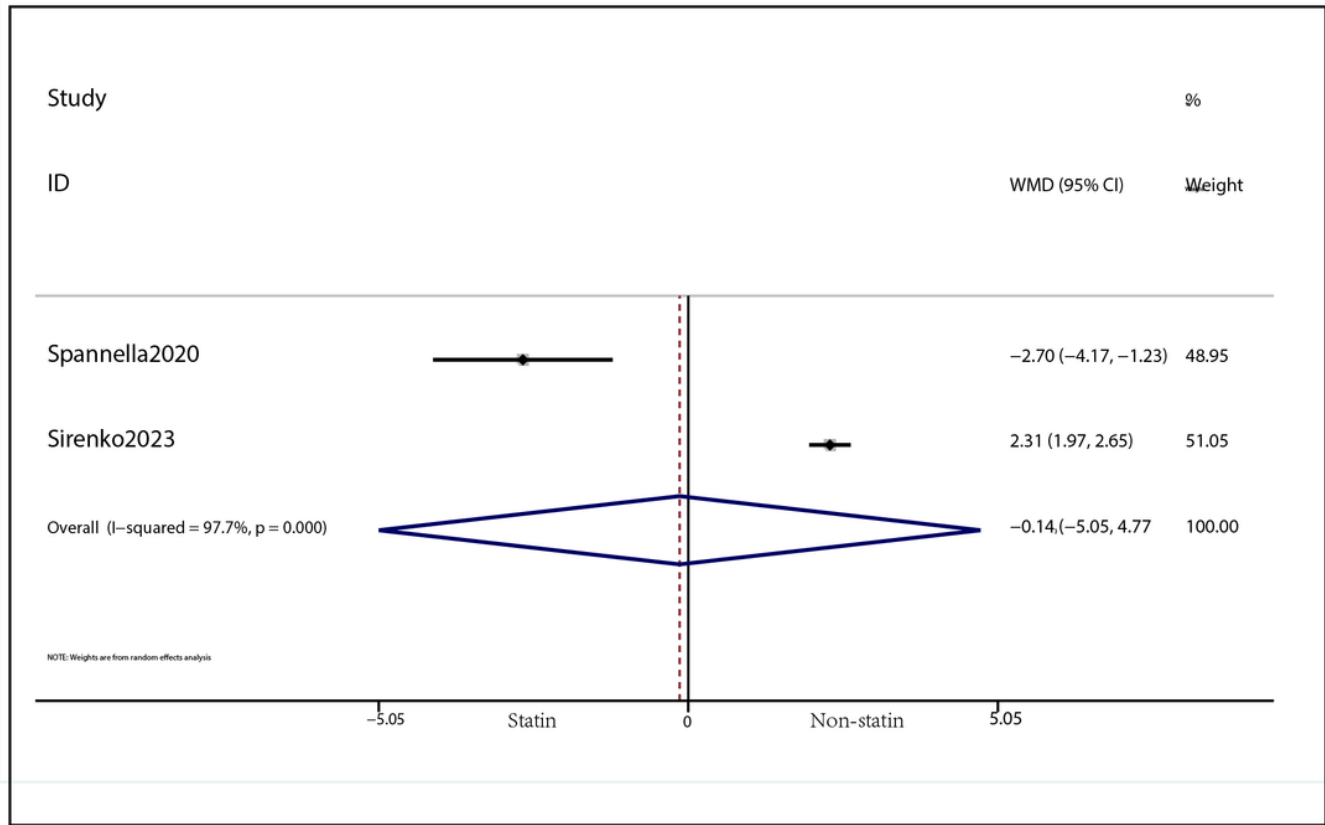Forest plot for assessing office-SBP in hypertensive patients affected by statin use.

Figure 7

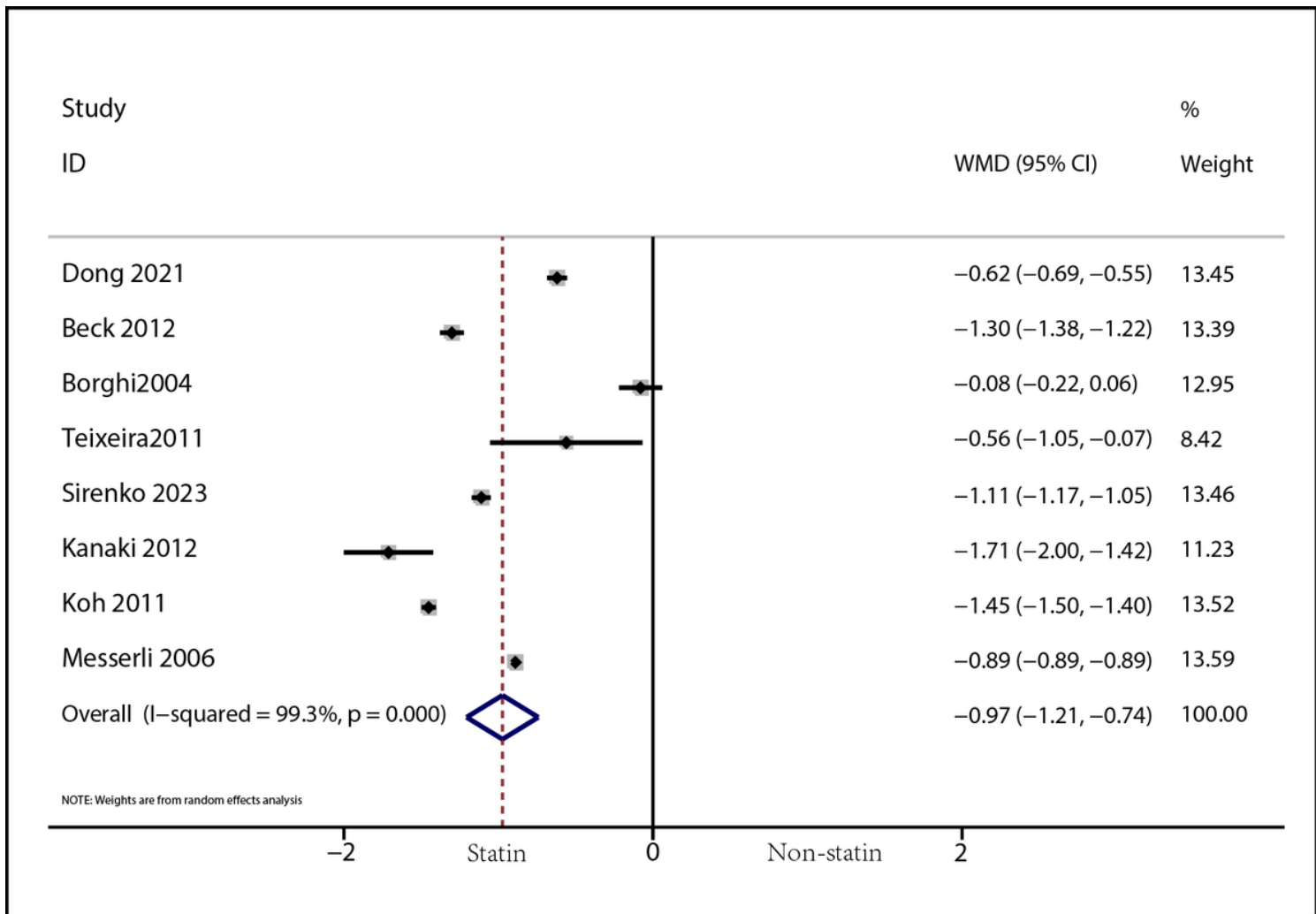Forest plot for assessing office-DBP in hypertensive patients affected by statin use.
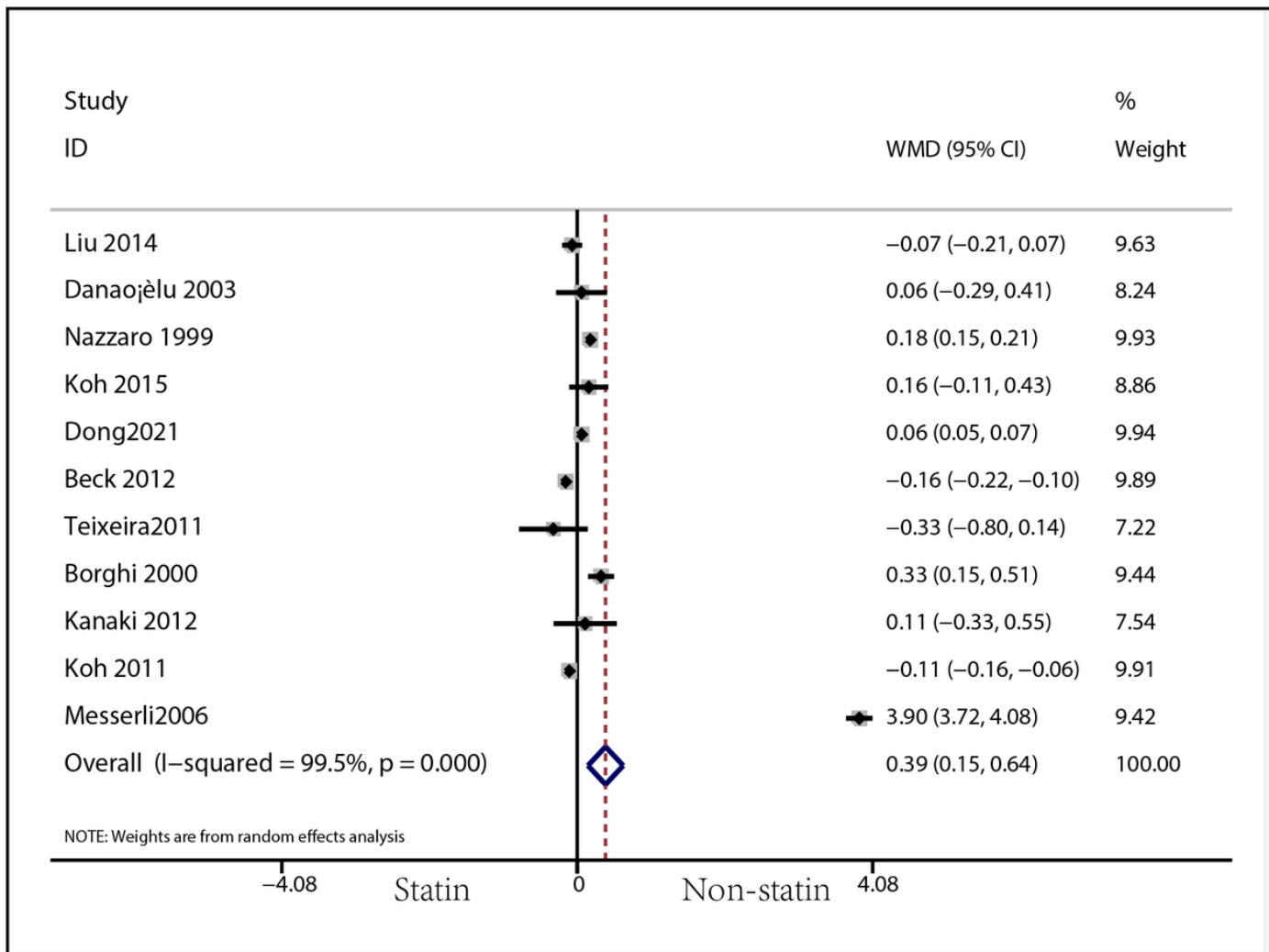
Study ID	WMD (95% CI)	% Weight
Dong 2021	−0.62 (−0.69, −0.55)	13.45
Beck 2012	−1.30 (−1.38, −1.22)	13.39
Borghi2004	−0.08 (−0.22, 0.06)	12.95
Teixeira2011	−0.56 (−1.05, −0.07)	8.42
Sirenko 2023	−1.11 (−1.17, −1.05)	13.46
Kanaki 2012	−1.71 (−2.00, −1.42)	11.23
Koh 2011	−1.45 (−1.50, −1.40)	13.52
Messerli 2006	−0.89 (−0.89, −0.89)	13.59
Overall (I−squared = 99.3%, p = 0.000)	−0.97 (−1.21, −0.74)	100.00

NOTE: Weights are from random effects analysis

Statin   Non-statin

Figure 8

Forest plot for assessing LDL-C in hypertensive patients affected by statin use.

Figure 9

Forest plot for assessing HDL-C in hypertensive patients affected by statin use.
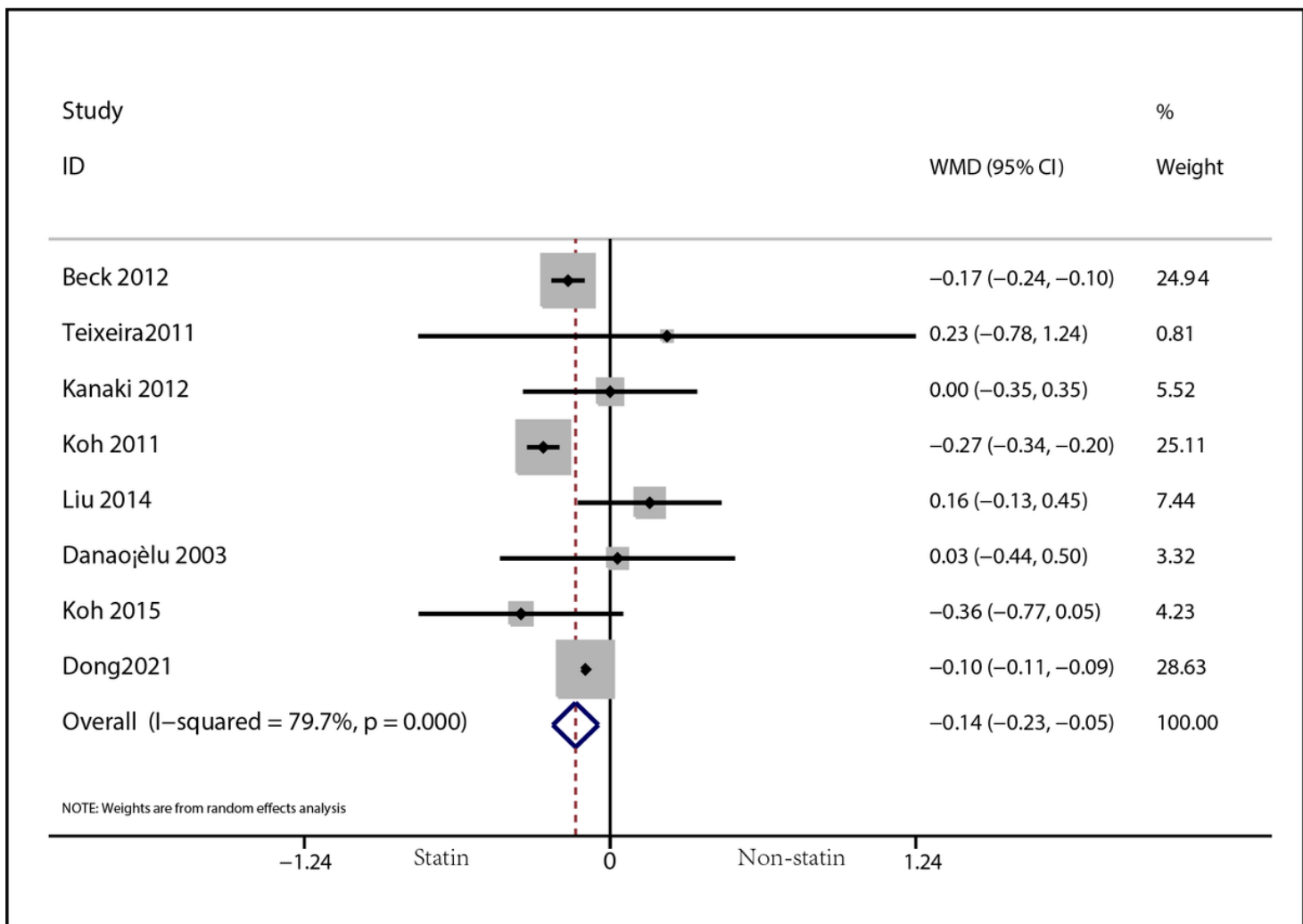
Figure 10

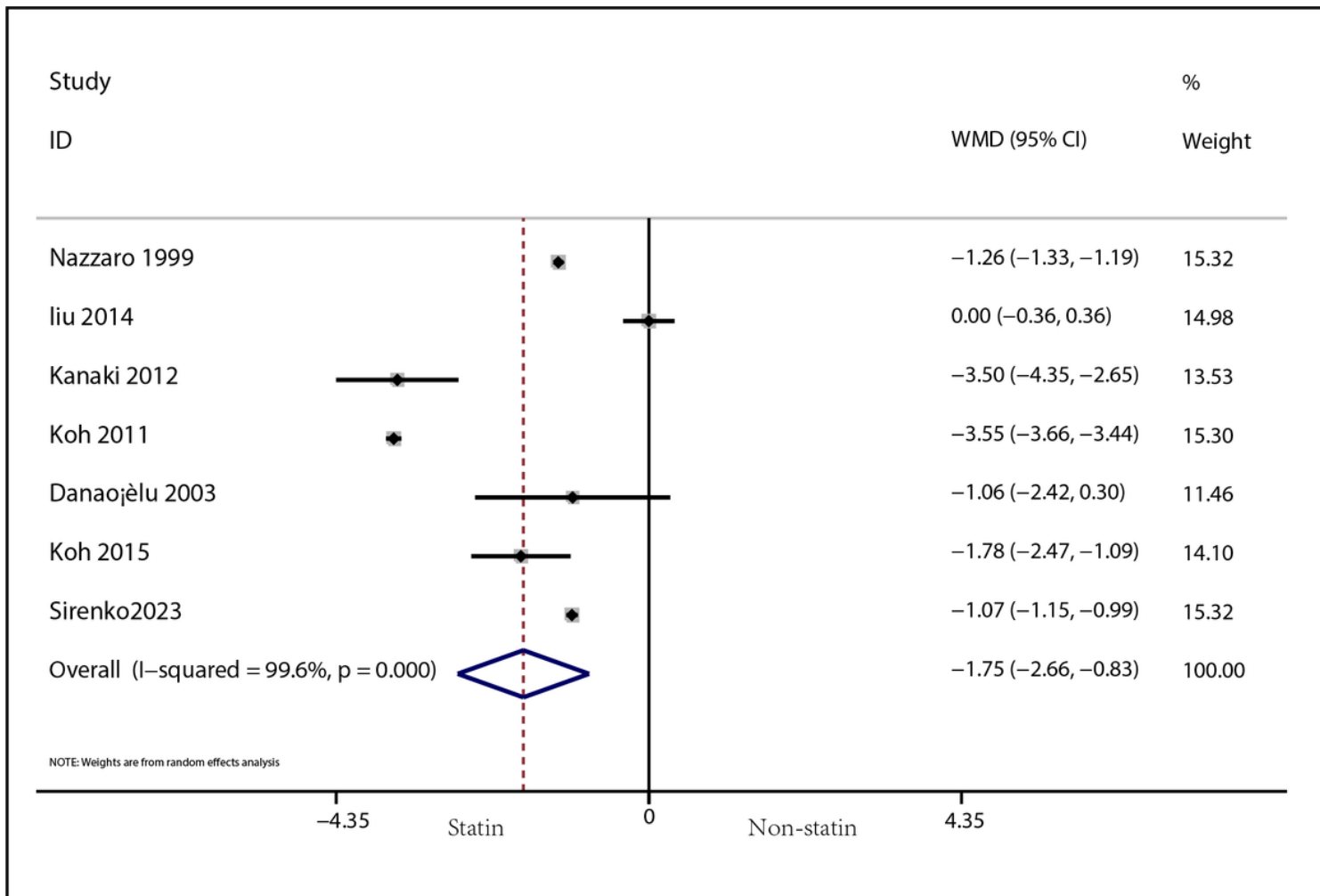Forest plot for assessing TG in hypertensive patients affected by statin use.

Figure 11

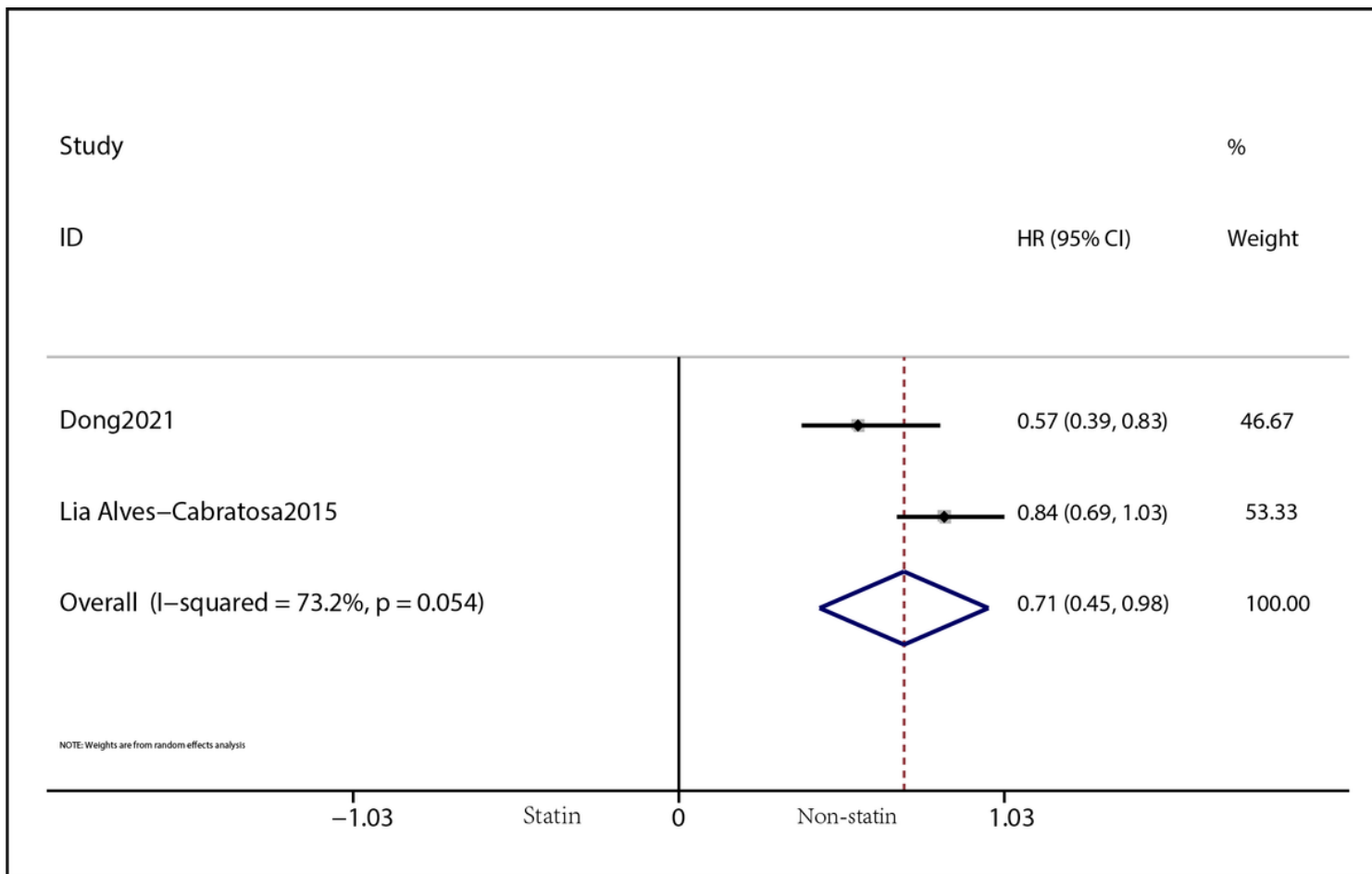Forest plot for assessing total cholesterol in hypertensive patients affected by statin use.

Figure 12

Forest plot for assessing risk of cardiovascular event in hypertensive patients affected by statin use.
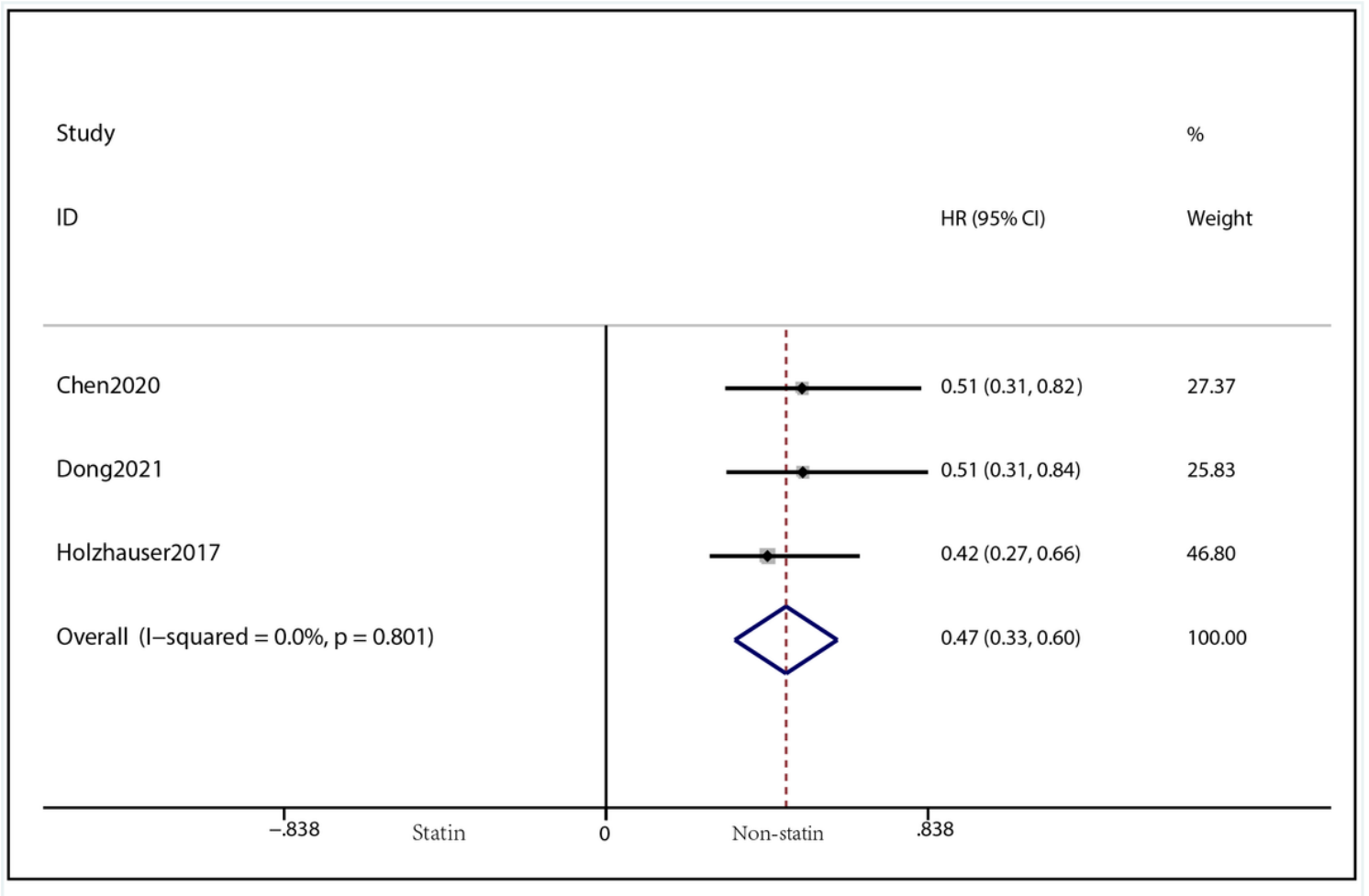
Figure 13

Forest plot for assessing risk of mortality in hypertensive patients affected by statin use.

Supplementary Files

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