Genetic association of APOA1 rs670 and rs5069 variants with the risk of developing cardiovascular disease: A systematic review and meta-analysis

HARITHA NAGARAJAN and SANTHINI GOPALAKRISHNAN

Department of Biochemistry, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Chettinad Health City, Kelambakkam, Tamil Nadu 603103, India

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Abstract. Apolipoprotein A1 (APOA1) is a major component of high-density lipoprotein and plays a critical role in lipid metabolism and cardiovascular prevention. Genetic variations in APOA1, including rs670 (-75 G>A) and rs5069 (+83 C>T), have been investigated for their association with the risk of developing cardiovascular disease (CVD). The present systematic review and meta-analysis evaluated the associations of APOA1 rs670 and rs5069 polymorphisms with the risk of developing CVD. For this purpose, a systematic literature search was conducted to identify eligible case control studies evaluating APOA1 rs670 and rs5069 polymorphisms and the risk of developing CVD. Odds ratios (ORs) with 95% confidence intervals (CIs) were pooled under allele contrast, dominant, recessive and over dominant genetics models using random effects meta-analyses. Between-study heterogeneity was assessed using Cochran's Q test and the I^2 statistics. Study quality was evaluated using the Newcastle-Ottawa Scale. Sensitivity analysis was performed using the leave-one-out approach, and publication bias was evaluated using funnel plots. A total of ten studies were included in the analysis, with five studies each for rs670 and rs5069. The primary pooled analysis revealed no significant association between either the APOA1 rs670 or rs5069 polymorphisms and the risk of developing CVD under any genetic model. Substantial heterogeneity was detected across studies. Leave-one-out sensitivity analysis confirmed the stability of the pooled estimates. Hardy-Weinberg equilibrium (HWE)-based sensitivity analysis for rs670 revealed a significant association under the allele contrast (OR, 1.81; 95% CI, 1.18-2.75; P=0.005) and dominant models (OR, 1.75; 95% CI, 1.17-2.62; P=0.006) after excluding studies deviating from the HWE, whereas a similar analysis for rs5069 was not feasible as only one HWE compliant study remained. On the whole, the primary pooled analysis found no significant association between the APOA1 rs670 or rs5069 polymorphisms and susceptibility to CVD. However, HWE-based sensitivity analysis suggests that HWE deviation may influence the observed association for rs670. Further large-scale studies involving diverse populations are required to validate these findings.

Correspondence to: Dr Santhini Gopalakrishnan, Department of Biochemistry, Chettinad Hospital and Research Institute, Chettinad Academy of Research and Education, Chettinad Health City, Rajiv Gandhi Salai (OMR), Kelambakkam, Tamil Nadu 603103, India
E-mail: santhini.research@gmail.com

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Introduction

Cardiovascular disease (CVD) is a major cause of mortality and creates a critical public health burden worldwide (1). CVD is a complex condition due to various environmental and genetic factors, and is particularly more complex in those who have a genetic predisposition to CVD. In such cases, genetic and hereditary factors play a major role in the development of CVD (2,3). The risk of developing CVD involves disorders of the heart and blood vessels, which includes coronary artery disease (CAD), myocardial infarction (MI), coronary disease (CD), acute coronary syndrome (ACS), rheumatic heart disease and other conditions (4). Dyslipidemia plays a crucial role in the development of CVD. Particularly, decreased levels of high-density lipoprotein (HDL) cholesterol (5) are highly associated with the development of all forms of CVD (6). Of note, both environmental and genetic factors contribute to inter-individual variations in lipid metabolism and the risk of developing CVD (7).

Apolipoprotein A1 (APOA1) is a key protein responsible for both the structure and function of the HDL particle and plays a crucial role in reverse cholesterol transport by enhancing cholesterol removal from peripheral tissue to the liver (8,9). The APOA1 gene is located on chromosome 11q23. APOA1 is associated with the APOA1 serum concentration and serum levels, which may increase the risk of developing CVD (10,11). Previous studies have observed that rs670 has an association with the risk of developing CVD (12). APOA1 is a component of HDL, which is commonly known as 'good cholesterol', although higher HDL cholesterol levels are epidemiologically associated with a reduced risk of developing CVD. It has been suggested that increasing the HDL concentration alone does not

protect against CVD, and highlights the importance of HDL functions (13). APOA1 also contributes to anti-inflammatory and anti-atherogenic functions; thus, it may provide protection against atherosclerotic plaque formation (14).

Among the most commonly studied polymorphisms, the rs670 (-75 G>A) variant is present in the promoter region of the APOA1 gene and has been reported to influence transcriptional activity and circulating HDL level (15). The rs5069 (+83 C>T) polymorphism is located downstream of the transcription start site and has been associated with alterations in lipid parameter (16). Several studies have been conducted across different countries and have examined the association of these polymorphisms with the outcomes of patients with CVD, such as CAD, ACS and MI (11,14-16). Although multiple studies have examined the association between APOA1 rs670 and rs5069 polymorphism and CVD risk, the findings remain inconsistent across different populations and study designs. A previous meta-analysis conducted by Dong *et al* (12) evaluated the association between APOA1 rs670 and rs5069 polymorphisms and the risk of developing CAD. In addition, Xu *et al* (13) evaluated the association between the APOA1 rs670 polymorphism and the risk of developing CAD. However, several studies have been published since then, providing new evidence regarding the role of these polymorphisms in cardiovascular disease risk (11,14,17). Therefore, the present systematic review and meta-analysis was conducted to systematically evaluate the association between APOA1 rs670 and rs5069 polymorphism with CVD risk, as reported in eligible case-control studies.

Data and methods

Literature search strategy. A systematic literature search for the present study was conducted using electronic databases, including PubMed, Web of Science, Scopus and Google Scholar to identify potentially relevant studies. The search strategy combined the following key words and their relevant combinations (APOA1 OR apolipoprotein A1), (rs670 or -75 G>A, rs5069 or +83 C>T), (cardiovascular disease or CVD, coronary artery disease or CAD, myocardial infarction or MI, acute coronary syndrome or ACS, coronary disease or CD). No restriction was applied based on the year of publication. The eligible studies identified through the search were published between 1998 to 2026.

A total of ten studies met the inclusion criteria and were included in the meta-analysis. The methodological quality of the included case-control studies was assessed using the Newcastle-Ottawa Scale (NOS) (18). The detailed quality assessment scores are provided in Table SI. No language restrictions were applied during the literature search. However, all the studies that met the inclusion criteria were published in the English language. The final literature search was completed on January 30, 2026. The present systematic review is not prospectively registered in PROSPERO or any other systematic review registry.

Inclusion criteria. Studies were included if they were case-control studies, evaluated APOA1 rs670 and rs5069 polymorphisms, investigated CVD outcomes and included sufficient genotype data. The methodological quality of the included case-control studies was assessed using the NOS, evaluated based on the selection of participants, comparability and exposure.

Exclusion criteria. Studies were excluded in the event that they included undefined data, were letters, lacked genotype data, or were review articles. Studies that did not fulfill the inclusion criteria and studies with duplicate populations were excluded.

Data extraction. Of note, two reviewers had separately carried out the data extraction using a standardized data extraction form. Information extracted from each eligible study included first author detail, year of publication, study region, CVD outcome, genotyping method and genotype allele frequencies. All selected articles were carefully reviewed and relevant data were recorded. Any disagreements between the reviewers were resolved through discussion. In addition, study quality-related information, including total sample size, study design and Hardy-Weinberg equilibrium (HWE) of the control group was also collected. The process of study selection, and screening and data extraction were conducted systematically.

Statistical analysis. All the statistical analyses were performed using the MetaGenyo web tool for genetic association meta-analyses (19). The level of the association between APOA1 rs670 and rs5069 polymorphism and CVD susceptibility was evaluated using odds ratios (ORs) with 95% confidence intervals (CIs). Pooled ORs were calculated under the applied genetic models: Allele contrast, dominant, recessive and over-dominant genotype comparison. The statistical significance of the pooled effect size was assessed using the Z-test and a two-sided P-value <0.05 was considered to indicate a statistically significant difference.

Between-study heterogeneity was evaluated using the Cochran's Q-test and the magnitude of heterogeneity was quantified using I^2 statistics. Due to the presence of substantial heterogeneity across the individual studies, the random-effects model was primarily applied to generate pooled estimates.

Sensitivity analysis was executed by removing one study at a time to assess the robustness of the pooled result. The funnel plot was applied to evaluate probable publishing bias, supported by Egger's regression test, acknowledging the publication bias test has low statistical power when a small number of studies are available. HWE in the control group was assessed using extracted genotype frequency and recorded for each included study (20).

Results

Included studies and study characteristics. The database research identified 155 records; after removing duplicates, review articles, meta-analyses, unrelated articles and animal studies, 42 articles were screened. Additionally, 32 studies with inadequate information were also excluded. Following thorough screening, a total of ten studies were included in the final meta-analysis (3,11,14-17,21). The process of selecting studies is presented as a PRISMA flow diagram according to meta-analysis PRISMA guidelines in Fig. 1.

Among the ten selected studies, five studies described the association of between APOA1 rs670 and the risk of developing CVD (Table IA) (3,11,15,16,21) and five studies described the

Table I. Characteristics of included studies for APOA1 rs670 and rs5069 polymorphisms.

Authors	Year of publication	Country of the study	Study design	Outcome	Sample size	Genotyping method	(Refs.)
A, Studies on the rs670 polymorphism							
Chhabra *et al*	2005	India	Case-Control	CAD	370	PCR	(15)
Dawar *et al*	2010	India	Case-Control	MI	100	PCR-RFLP	(16)
Liao *et al*	2015	China	Case-Control	CAD	600	PCR-RFLP	(21)
Reguero *et al*	1998	Spain	Case-Control	CD	176	PCR	(3)
Sohail *et al*	2024	Pakistan	Case-Control	CAD	200	PCR-RFLP	(11)
B, Studies on the rs5069 polymorphism							
Kumari *et al*	2023	India	Case-Control	MI	104	PCR	(17)
Pandith *et al*	2021	India	Case-Control	ACS	240	PCR-RFLP	(14)
Liao *et al*	2015	China	Case-Control	CAD	300	PCR-RFLP	(21)
Sohail *et al*	2024	Pakistan	Case-Control	CAD	200	PCR-RFLP	(11)
Dawar *et al*	2010	India	Case-Control	MI	100	PCR-RFLP	(16)

CAD, coronary artery disease; MI, myocardial infarction; CD, coronary disease; ACS, acute coronary syndrome; PCR, polymerase chain reaction; RFLF, restriction fragment length polymorphism.

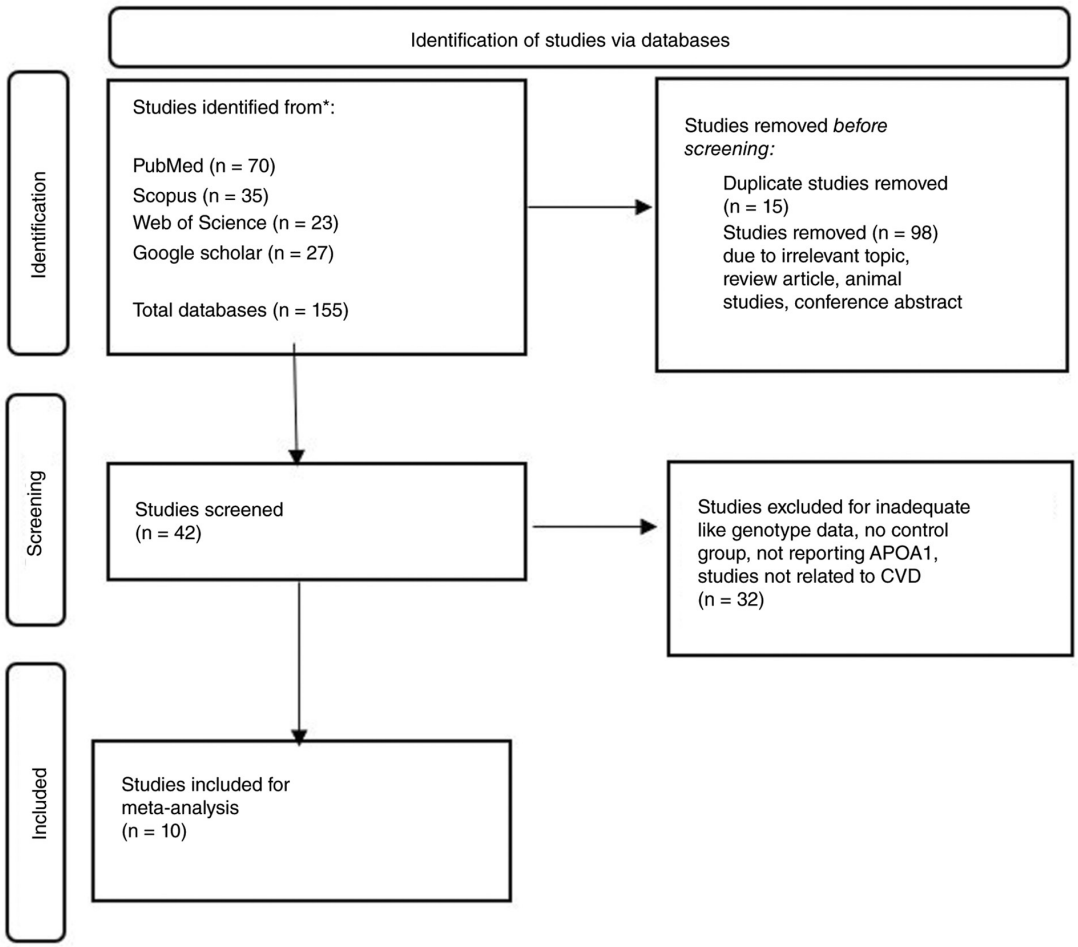

Figure 1. PRISMA flow diagram illustrating the study selection process, including identification, screening, eligibility assessment, and final inclusion of studies in the systematic review and meta-analysis.

Table II. Genotype distribution of rs670 with HWE P-values.

Authors	Year of publication	Country	Total no. of cases/ controls	Cases			Controls			HWE	(Refs.)
				GG	GA	AA	GG	GA	AA		
Chhabra *et al*	2005	India	164/36	102	51	11	29	7	0	0.5182	(15)
Dawar *et al*	2010	India	50/50	29	21	0	8	42	0	0.0079	(16)
Liao *et al*	2015	China	300/300	175	104	21	161	100	39	0.0005	(20)
Reguero *et al*	1998	Spain	176/200	117	50	9	152	44	4	0.6983	(3)
Sohail *et al*	2024	Pakistan	135/65	33	88	14	27	36	2	0.0156	(11)

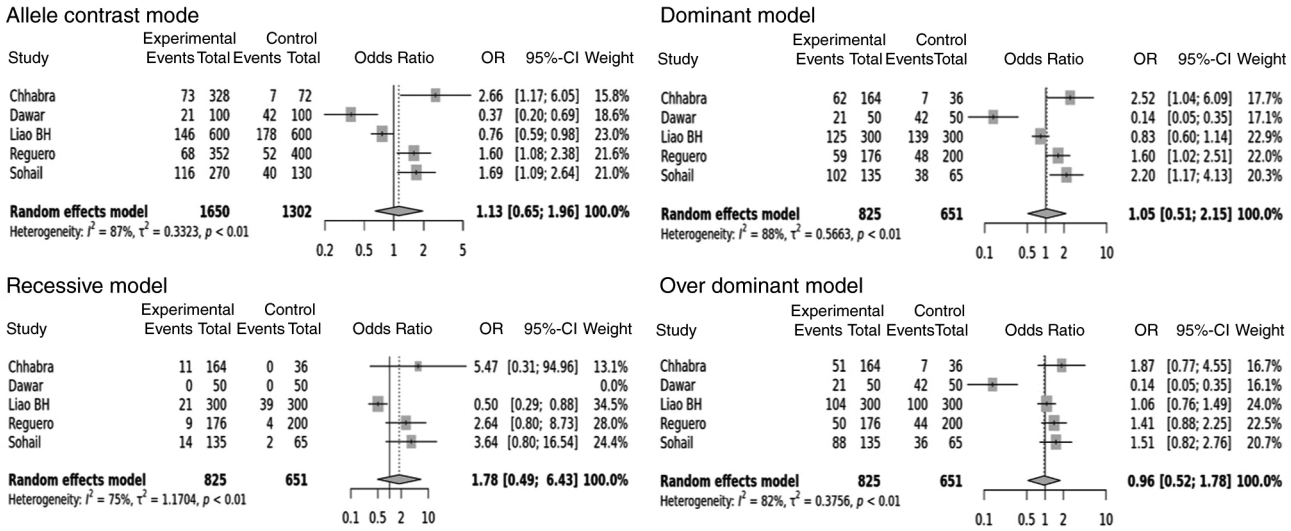

Figure 2. Forest plot illustrating the association between the APOA1 rs670 polymorphism and the risk of developing cardiovascular disease under the allele contrast model, dominant model, recessive model and over-dominant model. Squares represent study-specific odds ratios, horizontal lines indicate 95% confidence intervals and the diamond represent the pooled effect estimate. OR, odds ratio; CI, confidence interval.

association between APOA1 rs5069 and the risk of developing CVD (Table IB) (11,14,16,17,21). The methodological quality of the included studies was evaluated using the NOS. The NOS score ranged from 7 to 8, indicating moderate to high methodological quality (Table SI).

Meta-analysis of studies on APOA1 rs670. A total of five studies were included to evaluate the association between the APOA1 rs670 polymorphism and susceptibility to CVD and their genotype distributions and HWE P-values are presented in (Table II). Under the allele contrast model, no statistically significant association was identified using the random-effects model (OR, 1.13; 95% CI, 0.65-1.96; P=0.675). Similarly, no significant association was observed under the recessive model (OR, 1.78; 95% CI, 0.49-6.43; P=0.382), dominant model (OR, 1.05; 95% CI, 0.51-2.15; P=0.904) and the over-dominant model (OR, 0.96; 95% CI, 0.52-1.78; P=0.905) (Fig. 2). To evaluate the influence of studies deviating from the HWE, an additional sensitivity analysis was performed with only two studies [Chhabra *et al* (15) and Reguero *et al* (3)], excluding the studies by Dawar *et al* (16), Liao *et al* (21) and Sohail *et al* (11). Following that, a statistically significant association was observed under the allele contrast model (OR, 1.81; 95% CI, 1.18-2.75; P=0.005) and dominant model (OR, 1.75; 95% CI, 1.17-2.62; P=0.006). No significant association was identified under the recessive model (OR, 2.94; 95% CI, 0.97-8.86; P=0.055) or over-dominant model (OR, 1.49; 95% CI, 0.98-2.26; P=0.056) (Table SII). These findings suggest that studies deviating from the HWE may have influenced the pooled estimates observed in the primary analysis.

High heterogeneity was found to be present in the selected studies (I^2=75 to 88%, P<0.01), suggesting inconsistency across studies; this may be due to differences in ethnicity, study design, sample size and outcomes across the studies (Table III and Fig. 2) (3,11,15,16,21).

The sensitivity analysis for APOA1 rs670, was executed by removing one study at a time; this demonstrated that the pooled effect remained mostly unaltered following the sequential exclusion of each study, indicating that the observed association was stable and not driven by any single study. A summary of the meta-analysis of studies on rs670 is presented in Table III.

Meta-analysis of APOA1 rs5069. A total of five studies were included to evaluate rs5069 and their genotype distributions and HWE p-values are presented in (Table IV).

Table III. Pooled OR and 95% CI values of the association between APOA1 rs670 and risk of developing cardiovascular disease.

rs670 genetic model	Comparison	No. of studies	Pooled OR	95% CI	Z-test P-value	Model	I^2	P-value heterogeneity
Allele	A vs. G	5	1.12	0.64-1.96	0.675	Random effects model	86	<0.001
Dominant	AA/GA vs. GG	5	1.04	0.50-2.15	0.904	Random effects model	87	<0.001
Recessive	AA vs. GA/GG	5	1.77	0.48-6.43	0.382	Random effects model	75	0.0073
Over-dominant	GA vs. GG/AA	5	0.96	0.52-1.77	0.905	Random effects model	81	<0.001

Table IV. Genotype distribution with HWE P-value for rs5069.

Authors	Year of publication	Country	Total no. of cases/ controls	Cases			Controls			HWE	(Refs.)
				CC	CT	TT	CC	CT	TT		
Kumari *et al*	2023	India	52/52	31	4	17	42	6	4	0.0003	(21)
Pandith *et al*	2021	India	90/150	75	13	2	82	50	18	0.0235	(14)
Liao *et al*	2015	China	300/300	221	54	25	230	39	31	0	(20)
Sohail *et al*	2024	Pakistan	65/135	23	32	10	35	73	27	0.3217	(11)
Dawar *et al*	2010	India	50/50	12	20	18	18	32	0	0.0009	(16)

Table V. Pooled OR and 95% CI values of the association between APOA1 rs5069 and the risk of developing cardiovascular disease.

Rs5069 Genetic Model	Comparison	No. of studies	Pooled OR	95% CI	Z-test P-value	Model	I^2 %	P-value heterogeneity
Allele	T vs. C	5	1.13	0.53-2.39	0.752	Random effects model	92	<0.001
Dominant	TT/CT vs. CC	5	0.95	0.44-02.02	0.892	Random effects model	85.5	<0.001
Recessive	TT vs. CT/CC	5	1.38	0.43-4.39	0.590	Random effects model	83	<0.001
Over-dominant	CT vs. CC/TT	5	0.65	0.34-1.23	0.192	Random effects model	76	<0.002

No statistically significant association was observed between rs5069 and disease susceptibility under the allele contrast model (OR, 1.13; CI, 0.53-2.39; P=0.752) using the random-effects model. Similarly, no significant association was found under the dominant (OR, 0.95; 95% CI, 0.45-2.02; P=0.892), recessive (OR, 1.38; 95% CI, 0.43-4.39; P=0.590) and over-dominant (OR, 0.65; 95% CI, 0.35-1.24; P=0.192) models (Fig. 3). HWE sensitivity analysis was considered for APOA1 rs5069. However, the exclusion of all studies whose control groups deviated from HWE left only one eligible study for analysis. Therefore, a pooled HWE-based sensitivity analysis could not be performed due to insufficient data.

Substantial heterogeneity was present (I^2=92%; P<0.01) reflecting the effect of sizes across the included studies (Table V and Fig. 3) (11,14,16,17,21).

The sensitivity analysis for APOA1 rs5069 was executed by removing one study at a time, demonstrating that the pooled estimates did not materially change upon removal of individual studies. A summary of the meta-analysis is presented in Table V.

Publication bias. Publication bias was evaluated for both rs670 and rs5069 by applying funnel plot and Egger's regressing test. The funnel plots revealed mild asymmetry. However, only five studies were available for each polymorphism; therefore, the statistical power of these methods to detect publication bias was limited, which should be interpreted cautiously. The funnel plots are presented in Figs. 4 and 5.

Discussion

The present meta-analysis systematically evaluated the association between the APOA1 rs670 and rs5069 polymorphisms with susceptibility to CVD by pooling evidence from eligible case-control studies published between 1998 to 2026. The methodological quality of the studies was assessed using the NOS, and all studies were of moderate to high quality. The pooled analysis demonstrated that there was no statistically significant association between the APOA1 rs670 polymorphism and susceptibility CVD under the allele contrast, dominant, recessive and over-dominant genetic models. Similarly, the APOA1 rs5069 polymorphism

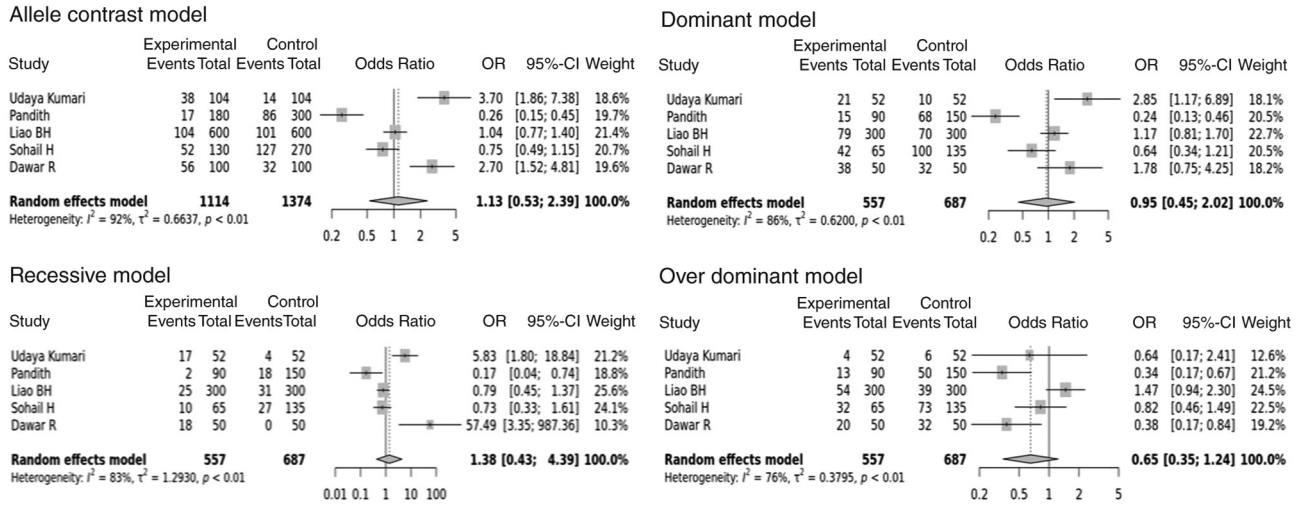

Figure 3. Forest plot illustrating the association between the APOA1 rs5069 polymorphism and the risk of developing cardiovascular disease under the allele contrast model, dominant model, recessive model and over-dominant model. Squares represent study-specific odds ratios, horizontal lines indicate 95% confidence intervals and the diamond represent the pooled effect estimate. OR, odds ratio; CI, confidence interval.

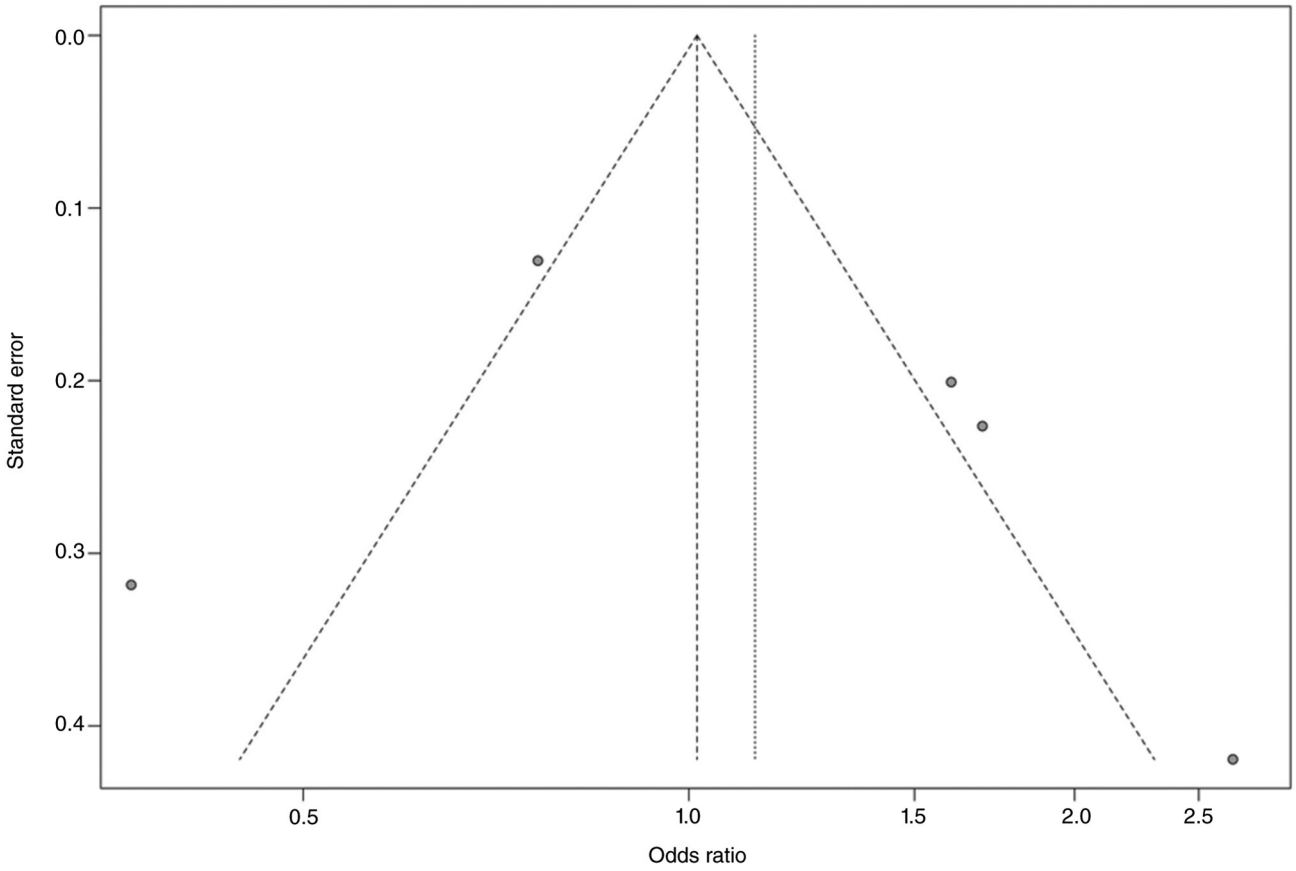

Figure 4. Funnel plot assessing potential publication bias among studies evaluating the association between the APOA1 rs670 polymorphism and the risk of developing cardiovascular disease.

did not exhibit any significant association with the risk of developing CVD under any of the evaluated genetic models.

However, the HWE-based sensitivity analysis revealed a significant association between the APOA1 rs670 polymorphism and the risk of developing CVD under the allele contrast model and dominant model following the exclusion of studies deviating from the HWE. This finding suggests that HWE violations may have contributed to the substantial heterogeneity observed in the primary analysis. By contrast, for APOA1 rs5069, an HWE-based sensitivity analysis was not feasible as the exclusion of studies deviating from the HWE had left only one study for analysis.

Ethnicity-based subgroup analysis was also limited as only one study included in the APOA1 rs670 analysis originated

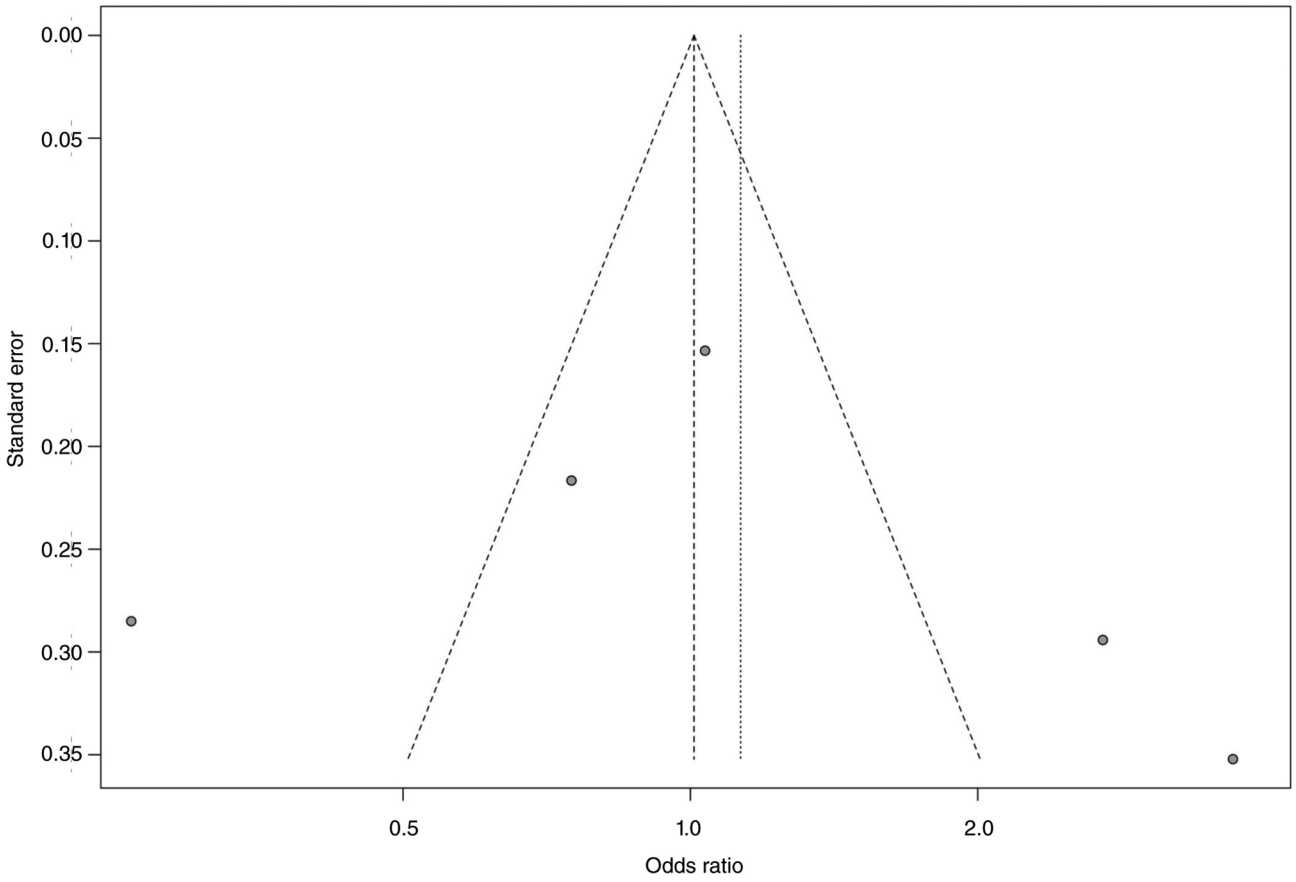

Figure 5. Funnel plot assessing potential publication bias among studies evaluating the association between the APOA1 rs5069 polymorphism and the risk of developing cardiovascular disease.

from a European population (3), while the remaining studies were conducted in Asian populations (11,15,16,21). Similarly, all studies included in the APOA1 rs5069 analysis were derived from Asian populations (11,14,16,17,21). Therefore, meaningful ethnicity-based subgroup analyses could not be performed.

Overall, the findings of the present meta-analysis did not reveal a statistically significant association between APOA1 rs670 and APOA1 rs5069 with susceptibility to CVD in the pooled analysis. However, previous studies have reported that the APOA1 rs670 and APOA1 rs5069 polymorphisms may influence lipid parameters in certain populations (17,21). However, the sensitivity analysis based on HWE indicated significant association between the rs670 polymorphism and the risk of developing CVD under specific genetic models, suggesting that this variant may contribute to disease susceptibility under certain conditions. However, no conclusion could be drawn for rs5069 due to limited data.

Xu *et al* (13) and Dong *et al* (12) evaluated the association between the APOA1 polymorphism and CAD. However, the present meta-analysis implemented an overall CVD approach by including studies investigating multiple cardiovascular outcomes including CAD, MI and ACS. Therefore, the present meta-analysis provides a comprehensive assessment of the APOA1 rs670 and rs5069 polymorphisms associated with susceptibility to CVD overall, rather than a single phenotypic characteristic. Furthermore, the present study incorporated recent evidence published up to 2026 providing updated synthesis of available literature.

Substantial heterogeneity was observed across the included studies. This variable may be explained by difference in ethnicity, sample size, cardiovascular disease phenotype, genotyping methods, environmental exposure and study design. The sensitivity analysis of studies on APOA1 rs670 demonstrated that the pooled estimates remained relatively stable by excluding individual studies, suggesting that no single study substantially influenced the overall findings.

The present meta-analysis has several strengths. It provides an updated analysis of available evidence by including recently published studied and systematically evaluated two commonly investigated APOA1 polymorphisms using multiple genetic models. The performance of sensitivity analysis, HWE assessment has enhanced the robustness od the findings.

However, the present meta-analysis has several limitations which should be considered when interpreting the findings. First, the number of included studies was relatively small, which may have limited the statistical power of pooled analysis. Second, substantial heterogeneity was observed across studies, potentially reflecting difference in ethnicity, study design, sample size, cardiovascular outcomes, and genotyping methods. Third, potential gene-gene and gene-environment interactions could not be evaluated due to insufficient data and inadequate reporting of environmental exposures in the included studies. Finally, publication bias assessment should be interpreted cautiously as fewer than ten studies where available for each polymorphism, limiting the reliability of funnel plot and Egger's test results.

In conclusion, the present meta-analysis found no significant association between the APOA1 rs670 and rs5069 polymorphisms and the overall risk of developing CVD in the primary pooled analyses under any genetic model. However, substantial heterogeneity was observed across studies. Notably, HWE-based sensitivity analysis for rs670 revealed a significant association under the allele contrast and dominant models. However, such an analysis for rs5069 was not feasible due to limited data. Overall, these findings should be interpreted cautiously and further studies are warranted to confirm these findings and provide further insight.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

HN and SG conceptualized the study. HN was involved in the study methodology, MetaGenyo software for genetic association meta-analysis provision, resources of literature databases , data curation, in the writing of the original draft of the manuscript, and in the writing, reviewing and editing of the manuscript. SG was involved in the formal analysis and investigative aspects of the study. SG were involved in data validation, visualization, supervision, project administration. HN and SG confirm the authenticity of all the raw data. Both authors have read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Use of artificial intelligence tools

During the preparation of this work, AI tools (ChatGPT) were used to improve the readability and language of the manuscript or to generate images, and subsequently, the authors revised and edited the content produced by the AI tools as necessary, taking full responsibility for the ultimate content of the present manuscript

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