

Association of the CAT rs1001179 (-262C>T) Variant with Polycystic Ovary Syndrome Risk: An Updated Meta-Analysis

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Polycystic ovary syndrome (PCOS) is a common endocrine–metabolic disorder characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. Growing evidence suggests that oxidative stress plays an important role in the pathogenesis of PCOS, and genetic variations in antioxidant enzymes, particularly catalase (CAT), may influence individual susceptibility to the disorder. This meta-analysis was conducted to examine whether the CAT -262C>T polymorphism (rs1001179) is associated with the risk of developing PCOS. A detailed search of PubMed, Google Scholar, and Semantic Scholar was performed following the PRISMA guidelines. Peer-reviewed case–control studies that provided genotype frequency data were included in the analysis. The quality of the selected studies was assessed using the Newcastle–Ottawa Scale (NOS). Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated under different genetic models. Depending on the level of heterogeneity among the studies, either fixed- or random-effects models were applied. Publication bias was assessed using Begg's and Egger's tests. Four studies comprising 1847 participants (cases: 824 and controls: 1023) met the inclusion criteria. No significant association was observed between the CAT -262C>T polymorphism and PCOS risk under allele, dominant, recessive, or over-dominant genetic models (all $p > 0.05$). Subgroup analyses by ethnicity similarly showed no significant associations. The current evidence does not support a significant role for the CAT -262C>T polymorphism in modifying PCOS risk. Further well-powered studies with diverse populations are necessary to validate these findings and explore potential gene–environment interactions.

Keywords: -262C>T, CAT rs1001179, Catalase, Genetic polymorphism, Meta-analysis, Oxidative stress.

Polycystic ovary syndrome (PCOS) is a multifactorial and complex disorder affecting women during their reproductive years, typified by elevated androgen levels, ovulatory irregularities, and polycystic ovarian morphology.¹ A growing body of evidence implicates Overproduction of

reactive oxygen species (ROS) in comparison to antioxidant capability causing oxidative stress which is a critical factor in the etiology of PCOS.² Excess generation of reactive oxygen species (ROS) may disrupt the body's oxidative balance, leading to damage to cells and surrounding tissues.

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This oxidative imbalance can interfere with normal ovarian follicle maturation and may worsen metabolic disturbances such as insulin resistance and elevated androgen levels. Because these abnormalities can influence each other, they may create a continuous harmful cycle that promotes both the development and progression of PCOS.³

An important gene involved in the oxidative stress pathway is the *CAT* gene, located on chromosome 11 (Figure 1A).⁴ It is a relatively long gene comprising 13 exons (Figure 1B) and contains a key upstream promoter variant (Figure 1C) that can influence gene expression⁵. This variant affects the transcriptional regulation of the gene (Figure 1E) and may alter the activity of catalase, the critical antioxidant enzyme encoded by *CAT*.⁵

The *CAT* protein also interacts with several other oxidative stress-related proteins, including SOD1, SOD2, HAO1, HAO2, and HSD17B4, highlighting its role within a broader antioxidant network (Figure 1F). By converting hydrogen peroxide into water and oxygen, catalase plays a crucial part in cellular defense by shielding cellular components from oxidative damage (Figure 1D).⁶⁻⁸ Different research studies across varied populations have investigated *CAT* gene polymorphisms; however, their findings have been variable,⁹⁻¹² with some reporting significant associations with disease susceptibility while others observed no clear relationship. These inconsistencies may be due to ethnic diversity, sample size differences, environmental exposures, or methodological variations.

Since earlier investigations have reported inconsistent associations, this study was undertaken to provide a more comprehensive understanding of the relationship between *CAT* polymorphisms and the risk of PCOS. To our knowledge, this is the first pooled analysis conducted specifically for this gene. By integrating data from multiple sources and applying a rigorous analytical approach, this study seeks to offer clearer evidence and bridge the gaps left by earlier individual studies.

METHOD

Literature survey

This study aimed to investigate whether the *CAT* rs1001179 ("262C>T") polymorphism

is linked to susceptibility to PCOS. To identify relevant studies, we carried out a detailed search of several electronic sources, including PubMed-NCBI, Google Scholar, and Semantic Scholar. The selection process involved searching, screening, assessing eligibility, and including studies according to internationally accepted PRISMA recommendations, which helped maintain a clear, consistent, and reproducible methodology.³⁷

A structured set of targeted keywords and Boolean combinations was used to identify all relevant studies assessing the *CAT* "262C>T" variant and PCOS risk. Search terms included: "*CAT* gene polymorphism and PCOS," "*CAT* rs1001179 and PCOS," "*CAT* "262C>T" variant," "catalase gene polymorphism and polycystic ovary syndrome," and "*CAT* promoter polymorphism and PCOS susceptibility." Non-peer-reviewed sources, including conference abstracts, book chapters, dissertations, and preprints, were eliminated in order to reduce reporting bias. Only full-text, peer-reviewed case-control studies published in English with clearly reported genotype or allele distributions were included, ensuring high-quality evidence and robust pooled estimates.

Inclusion and Exclusion Criteria, Data Extraction, and Quality Assessment

To improve the reliability of this meta-analysis, clear inclusion and exclusion criteria were established (Table 1) so that only relevant and sufficiently detailed studies were included. Each selected study was carefully reviewed for its study design, participant characteristics, and overall methodological quality. Data extraction and quality assessment were performed systematically to ensure the accuracy and consistency of the collected information.

Numerical data were systematically extracted, and missing or unclear information was supplemented using previous meta-analyses, related publications, or by contacting study authors when necessary. Three researchers independently extracted the data, and the corresponding author verified its accuracy. Any differences in assessment were resolved by the corresponding author.¹³

Statistical Analysis

Genotype data from all eligible studies were extracted and analyzed to obtain pooled genotype and allele frequencies using *GeneRiskCalc*.¹⁴ This helped standardize the genetic estimates

and improve comparability among the included studies. The Chi-square test was applied to evaluate Hardy–Weinberg equilibrium (HWE) in the control groups, and $p > 0.05$ was considered consistent with equilibrium. The association between the CAT rs1001179 (“262C>T”) polymorphism and PCOS risk was assessed by calculating odds ratios (ORs) with 95% confidence intervals (CIs) under different genetic models. Cochran’s Q test and the I^2 statistic were used to examine heterogeneity among studies. A random-effects model was used when substantial heterogeneity was observed ($I^2 > 55\%$); otherwise, a fixed-effect model was applied. Potential publication bias was evaluated using Begg’s rank correlation test and Egger’s regression test. Sensitivity analysis was performed by removing one study at a time to determine the stability of the pooled results. All statistical analyses were conducted using the Meta-Genyo online platform for genetic association meta-analysis.¹⁵ All statistical tests were two-sided,

and a P value < 0.05 was considered statistically significant throughout the analyses.

RESULTS

Characteristics of Included Studies

After the stepwise exclusion of the retrieved literature (Figure 2), a total of four eligible case–control studies (Table 2), comprising 1,847 participants (1023 PCOS cases and 824 healthy controls), met the predefined inclusion criteria and were included in the meta-analysis to investigate the association between the CAT “262C>T” polymorphism and susceptibility to PCOS. These studies encompassed populations from diverse ethnic backgrounds, including Asian (Iranian and Chinese), European (Slovenian), and Russian cohorts. All studies employed hospital-based sampling and utilized established genotyping techniques such as PCR-RFLP, RT-PCR, and KASPar assays. PCOS diagnosis was

Table 1. Inclusion and Exclusion Criteria for the Present Study

Feature	Inclusion Criteria	Exclusion Criteria
Study Design	Original peer-reviewed case–control or cohort investigations reporting primary data.	Family-based (pedigree) studies, genome-wide association studies (GWAS), review articles, conference abstracts, or any study design not appropriate for quantitative meta-analysis.
Case Definition	Participants diagnosed with polycystic ovary syndrome (PCOS) using recognized clinical or diagnostic guidelines, such as the Rotterdam criteria.	Studies lacking a confirmed diagnosis of PCOS or including participants with other endocrine disorders or significant co-existing diseases.
Genetic Polymorphism	Investigation of the catalase (CAT) rs1001179 (-262C>T) polymorphism with clearly specified genotype information.	Studies that did not evaluate the rs1001179 variant or failed to clearly identify the genetic polymorphism examined.
Genotype Data	Availability of genotype and/or allele distribution data for both PCOS cases and control subjects.	Publications that did not provide sufficient genotype or allele frequency information for analysis.
Hardy–Weinberg Equilibrium (HWE)	Control population demonstrating genotype distributions consistent with Hardy–Weinberg equilibrium.	Studies in which the control group significantly deviated from Hardy–Weinberg equilibrium.
Effect Size Estimation	Data adequate to derive odds ratios (ORs) and corresponding 95% confidence intervals (CIs).	Studies with insufficient information to estimate genetic association measures.
Methodological Quality	Studies achieving a Newcastle–Ottawa Scale (NOS) score of 5 or higher, indicating acceptable methodological quality.	Studies rated as low quality with an NOS score below 5.

uniformly based on the Rotterdam criteria, with one study additionally applying the ESHRE/ASRM consensus guidelines.

Sample sizes varied, ranging from 26 to 654 cases and 33 to 535 controls. Complete genotype data were reported for all studies, and Hardy–Weinberg equilibrium was confirmed in the control groups ($p > 0.05$). Quality assessment using the NOS yielded scores of 6 for all studies, indicating acceptable methodological quality (Table 2). Detailed study characteristics, including country, ethnicity, genotyping method, diagnostic criteria, sample sizes, genotype distributions, HWE results, and quality scores, are summarized in Table 2.

Genetic Association

No significant relationship was observed between the *CAT* rs1001179 (“262C>T”) polymorphism and PCOS risk across all genetic models. For the allele model, the OR was 0.92 (95% CI: 0.73–1.17; $p = 0.50$) (Figure 3A). The recessive model showed an OR of 0.80 (95% CI: 0.32–2.01; $p = 0.63$), while the dominant model had an OR of 0.92 (95% CI: 0.70–1.20; $p = 0.53$). The over-dominant model yielded an OR of 0.93 (95% CI: 0.71–1.22; $p = 0.61$). Genotype comparisons indicated no significant associations: HR vs HW (OR = 0.73, 95% CI: 0.29–1.88; $p = 0.52$), HR vs HT (OR = 0.92, 95% CI: 0.35–2.40; $p = 0.86$), and HT vs HW (OR = 0.92, 95% CI: 0.70–1.21; $p = 0.56$) (Table 3).

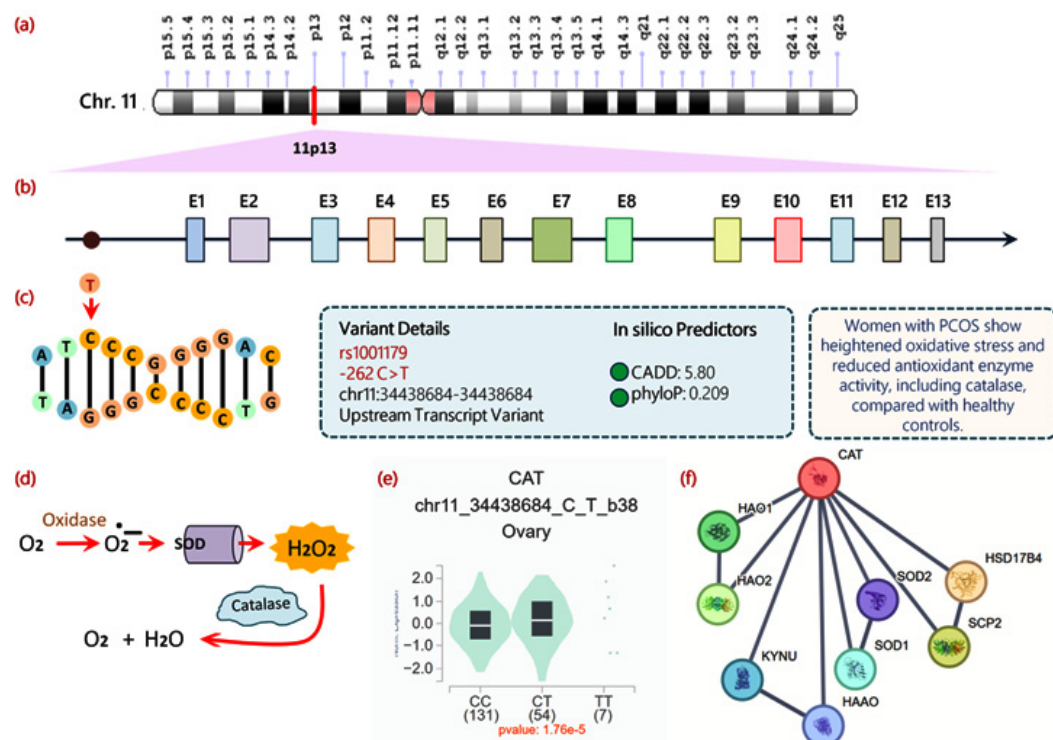


Fig. 1. Overview of the genomic location and functional relevance of the *CAT* rs1001179 (“262C>T”) polymorphism. (a): shows the localization of the *CAT* gene on chromosome 11p13. (b) illustrates the exon organization of the *CAT* gene. (c): presents the position and annotation details of the rs1001179 (“262C>T”) upstream regulatory variant. (d): depicts the role of catalase in the detoxification of H₂O₂ during oxidative stress. (e): shows the GTEx ovary eQTL analysis demonstrating *CAT* expression across different rs1001179 genotypes, and (f): presents the STRING protein–protein interaction network of *CAT* with oxidative stress–related proteins.

Heterogeneity was low across all analyses, with I^2 values of 0% and non-significant Cochran's Q tests, justifying the use of fixed-effect models. Egger's tests for publication bias (Figure 3B) were non-significant in all models (p-values ranged from 0.24 to 0.80), indicating no evidence of small-study effects (Table 3). These findings suggest that the *CAT* "262C>T" polymorphism is not significantly associated with PCOS susceptibility in the studied populations. The pooled effect estimate remained largely unchanged when each study was removed one at a time, suggesting that no individual study had a major influence on the overall results. This indicates that the findings of the meta-analysis were stable and reliable (Figure 4).

The subgroup analysis according to ethnicity did not identify any significant association

between *CAT* "262C>T" and PCOS risk in any genetic model (Table 4). In Asians, allele model OR was 0.99 (95% CI: 0.74–1.34; $p = 0.96$), Europeans 0.78 (95% CI: 0.51–1.19; $p = 0.24$), and Russians 0.86 (95% CI: 0.37–2.02; $p = 0.74$). Other models showed similar non-significant results across ethnicities. Heterogeneity was low ($I^2 = 0\%$) in Asians; heterogeneity and publication bias were not assessed for other groups due to limited studies. These results suggest no significant ethnic-specific effect of *CAT* "262C>T" on PCOS risk.

DISCUSSION

Catalase, a key antioxidant enzyme responsible for the detoxification of hydrogen peroxide and maintenance of cellular redox

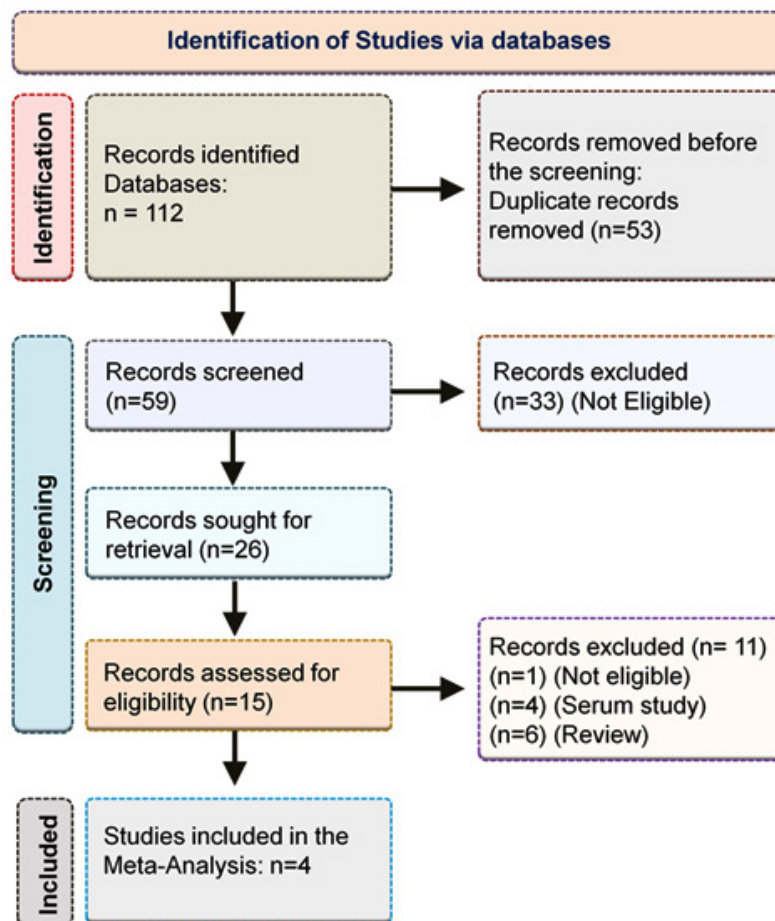


Fig. 2. PRISMA Flow Chart

Table 2. Overview of Eligible Case-Control Studies of CAT rs1001179 ("262C>T") Polymorphism and PCOS Risk

S. No	Study Name	Country	Ethnic	Type of Study	Control Sampling	Genotyping Method	Diagnostic Criteria	Sample Size (Case/Control)	HR	HT	HW	HR	HT	HW	HWE	NOS
1	Salahshoor et al. ⁹	Iran	Asian	CC	HB	PCR-RFLP	Rotterdam criteria	175/175	0	58	117	0	54	121	0.0632	6
2	Sun et al. ¹⁰	China	Asian	CC	HB	PCR-RFLP	Rotterdam/ESHRE/ASRM consensus criteria	654/535	2	38	614	1	35	499	0.6408	6
4	Herman et al. ¹¹	Slovenian	European	CC	HB	KASPar assay	Rotterdam criteria	169/83	7	65	96	5	36	40	0.6141	6
3	Ali et al. ¹²	Russia	Russian	CC	HB	RT-PCR	Rotterdam criteria	26/33	2	8	16	3	11	19	0.6141	6

CC: Case-Control, HB: Hospital-Based, PCR-RFLP: Polymerase Chain Reaction-Restriction Fragment Length Polymorphism, KASPar: Competitive Allele Specific PCR, RT-PCR: Reverse Transcription Polymerase Chain Reaction, HWE: Hardy-Weinberg Equilibrium, HR: Homozygous Reference, HT: Homozygous Variant, HW: Homozygous Variant, ESHRE: European Society of Human Reproduction and Embryology, ASRM: American Society for Reproductive Medicine.

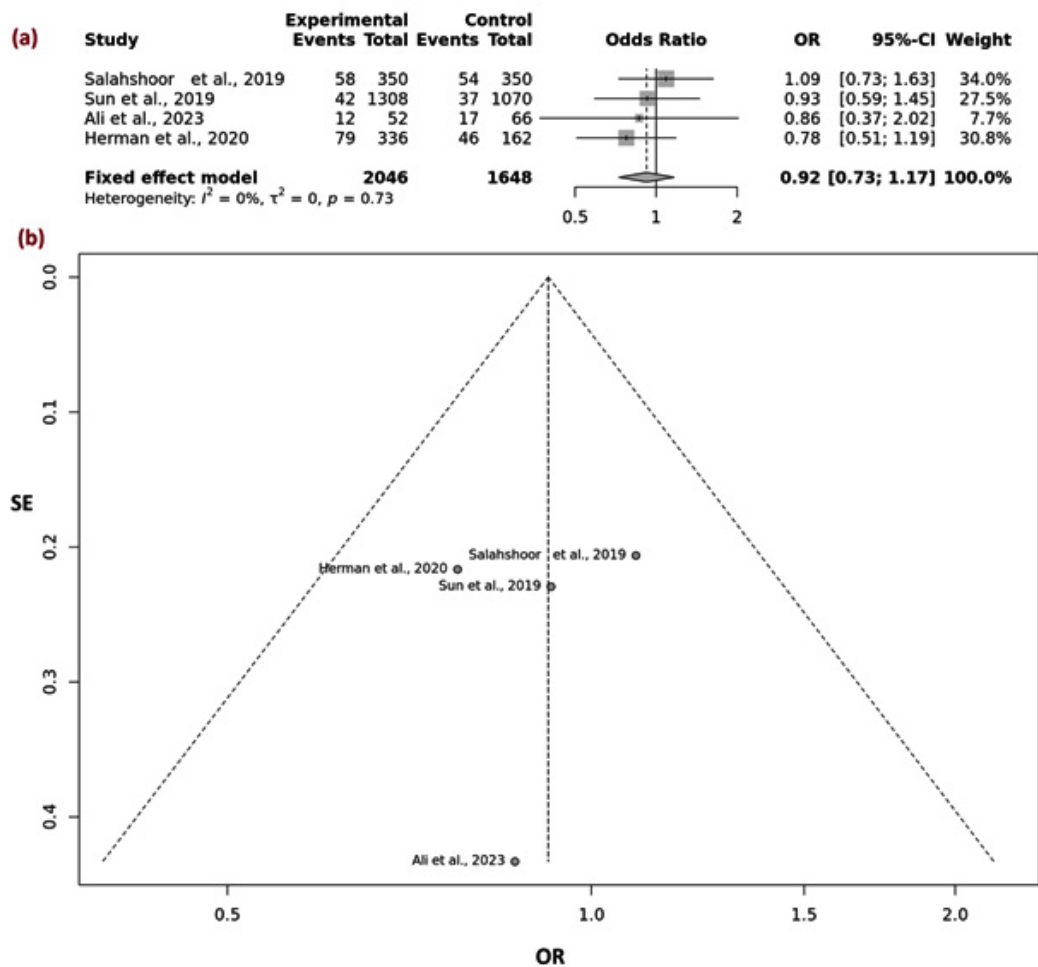
homeostasis, is encoded by the CAT gene.¹⁶⁻¹⁹ By catalyzing the conversion of hydrogen peroxide into water and oxygen, catalase protects cells from oxidative damage and prevents the accumulation of ROS, which are known to impair cellular integrity and metabolic functions. Functional polymorphisms within the CAT gene, particularly those located in promoter or regulatory regions, may alter transcriptional activity and enzyme expression, thereby increasing susceptibility to oxidative stress-related disorders.^{7,20} Among these variants, the CAT -262C>T polymorphism has attracted considerable attention because of its potential influence on catalase activity and oxidative balance.

Oxidative stress has been widely recognized as a central contributor to the pathogenesis of PCOS.²¹⁻²⁵ Elevated ROS levels and impaired antioxidant defense systems have been consistently reported in women with PCOS, suggesting that oxidative imbalance may contribute to both the initiation and progression of the disorder. Excessive ROS production can disrupt ovarian steroidogenesis, impair follicular maturation, induce chronic low-grade inflammation, and aggravate insulin resistance, all of which are key pathological features of PCOS.²⁶ Moreover, oxidative stress may influence reproductive outcomes by negatively affecting oocyte quality and endocrine signaling pathways, further emphasizing the importance of antioxidant-related genes in PCOS susceptibility. Given the role of catalase in antioxidant defense, this systematic review examined the association between the CAT "262C>T" polymorphism and susceptibility to PCOS. To our knowledge, this is the first comprehensive meta-analysis focused on this association.

The pooled analysis demonstrated no statistically significant association between the CAT -262C>T variant and PCOS susceptibility under any of the evaluated genetic models (Table 3). Similar findings were observed across allelic, dominant, recessive, homozygous, and heterozygous comparisons, indicating that the variant does not appear to exert a major independent effect on disease risk. Importantly, the meta-analysis revealed low inter-study heterogeneity across most genetic comparisons, suggesting strong consistency among the included studies.

Table 3. Pooled Effect Estimates for CAT rs1001179 (-262C>T) Polymorphism Under Different Genetic Models for overall population

Model	Test of association			Test of heterogeneityPublication bias p-val (Egger's test)			
	OR	95% CI	p-val	Model	p-val	I ²	
Allele	0.92	0.7279-1.1663	0.495897	Fixed	0.726	0	0.7983
Recessive	0.80	0.3180-2.0073	0.633003	Fixed	0.8008	0	0.2791
Dominant	0.92	0.7025-1.1987	0.52853	Fixed	0.7018	0	0.6645
OD	0.93	0.7114-1.2215	0.610664	Fixed	0.7972	0	0.7141
HR vs HW	0.73	0.2873-1.8764	0.518655	Fixed	0.7536	0	0.2394
HR vs HT	0.92	0.3530-2.3977	0.864545	Fixed	0.8246	0	0.4128
HT vs HW	0.92	0.7024-1.2106	0.559305	Fixed	0.7452	0	0.669

**Fig. 3.** (A) Forest plot presenting the pooled OR with 95% CI and the heterogeneity analysis for the included studies under the allelic model. (B) Funnel plot used to evaluate potential publication bias among the studies included in the meta-analysis.

Additionally, publication bias analyses showed no significant asymmetry, indicating that the pooled findings were unlikely to be influenced by selective reporting or small-study effects (Table 3). These observations strengthen the reliability and robustness of the current results. The findings are also in agreement with previously published individual case-control studies conducted in different populations which similarly failed

to identify a significant relationship between the CAT -262C>T polymorphism and PCOS susceptibility.^{9,10,12} Furthermore, ethnicity-based subgroup analyses did not reveal any statistically significant association in either Asian or non-Asian populations (Table 4), suggesting that the lack of association is unlikely to be population-specific. This indicates that the CAT -262C>T polymorphism may not play a substantial role in

Table 4. Ethnicity-Stratified Meta-Analysis of CAT rs1001179 ("262C>T) Polymorphism Across Genetic Models

Model	Ethnicity	#Studies	Test of association			Test of heterogeneity			Publication bias p-val (Egger's test)
			OR	95% CI	p-val	Model	p-val	I ²	
Allele	Asian	2	0.99	0.73-1.33	0.9641	F	0.6835	0	NA
	Europeans	1	0.78	0.50-1.18	0.2396	F	NA	NA	NA
	Russian	1	0.86	0.37-2.02	0.7371	F	NA	NA	NA
Recessive	Asian	2	0.92	0.13-6.32	0.9359	F	0.4362	0	NA
	Europeans	1	0.66	0.20-2.14	0.4913	F	NA	NA	NA
	Russian	1	0.83	0.12-5.39	0.8483	F	NA	NA	NA
Dominant	Asian	2	1.00	0.72-1.37	0.9805	F	0.5669	0	NA
	Europeans	1	0.73	0.42-1.24	0.2499	F	NA	NA	NA
	Russian	1	0.85	0.29-2.42	0.7584	F	NA	NA	NA
Over-dominant	Asian	2	1.00	0.72-1.38	0.9997	F	0.4719	0	NA
	Europeans	1	0.79	0.46-1.34	0.3867	F	NA	NA	NA
	Russian	1	0.89	0.29-2.67	0.8343	F	NA	NA	NA
HR vs HW	Asian	2	0.93	0.13-6.37	0.9422	F	0.4485	0	NA
	Europeans	1	0.58	0.17-1.94	0.3808	F	NA	NA	NA
	Russian	1	0.79	0.11-5.33	0.8104	F	NA	NA	NA
HR vs HT	Asian	2	0.96	0.13-6.73	0.9684	F	0.3882	0	NA
	Europeans	1	0.78	0.22-2.62	0.6822	F	NA	NA	NA
	Russian	1	0.92	0.12-6.82	0.9323	F	NA	NA	NA
HT vs HW	Asian	2	1.00	0.71-1.38	0.9814	F	0.4897	0	NA
	Europeans	1	0.75	0.43-1.30	0.3100	F	NA	NA	NA
	Russian	1	0.86	0.27-2.66	0.7989	F	NA	NA	NA

HW: Homozygous Recessive, HT: Heterozygous, HR: Homozygous Recessive, OR: Odds Ratio, NA: Not Applicable, F: Fixed

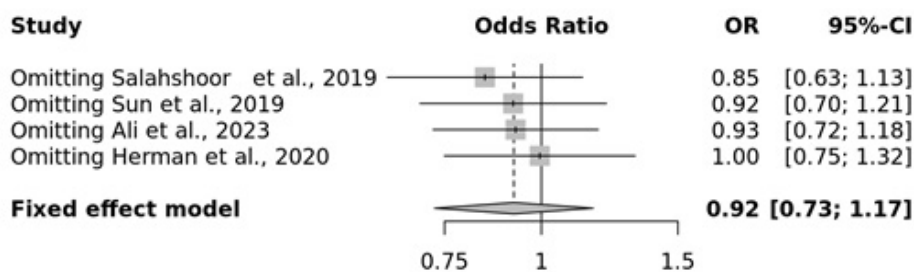


Fig. 4. Sensitivity analysis based on one-study-removed evaluation of the pooled OR.

determining genetic susceptibility to PCOS across diverse ethnic backgrounds.

Crucially, these findings do not indicate that catalase activity or the *CAT* gene lacks relevance in the pathogenesis of PCOS. The absence of an association for the -262C>T variant cannot be generalized to the entire gene or its biological function, as the present analysis evaluated only a single promoter polymorphism. Substantial functional evidence supports the involvement of *CAT* in ovarian physiology and PCOS-related processes. Several studies have reported reduced catalase activity in women with PCOS, reflecting impaired antioxidant defense and increased oxidative stress.²⁷⁻²⁹ Similarly, another study observed decreased *CAT* activity along with elevated malondialdehyde (MDA) levels, indicating enhanced lipid peroxidation and oxidative imbalance in PCOS patients.³⁰

In contrast, some studies have reported increased *CAT* activity in PCOS, which has been interpreted as a compensatory antioxidant response to elevated reactive oxygen species. Multiple study groups demonstrated enhanced *CAT* activity in PCOS models associated with oxidative stress and inflammation.^{31,32} Additionally, another study reported altered oxidative stress markers, including *CAT* and MDA, in a letrozole-induced PCOS model, while L-carnitine treatment improved antioxidant status and ovarian function.³³ Collectively, these findings suggest that oxidative stress dysregulation and altered catalase activity play important roles in PCOS pathophysiology, although the direction of *CAT* activity changes may vary depending on disease stage, metabolic condition, and compensatory antioxidant responses. In addition, oxidative stress imbalance and altered catalase activity have been observed in the follicular fluid of PCOS patients, further supporting a role for *CAT* in the ovarian microenvironment.¹² Notably, catalase activity appears to be influenced by metabolic status and clinical heterogeneity within PCOS. Bausenwein *et al.*,³⁴ reported significantly higher catalase activity in the follicular fluid of obese women as compared with non-obese women with and without PCOS, suggesting a compensatory antioxidant response to obesity-related oxidative stress. Further, it has been observed that women with PCOS and insulin

resistance have been shown to exhibit reduced catalase activity, highlighting the interaction between oxidative stress, metabolic dysfunction, and antioxidant defense mechanisms.³⁵ Notably, antioxidant supplementation has been shown to restore and enhance catalase activity in PCOS patients who initially exhibit reduced antioxidant capacity.³⁶

Collectively, these findings suggest that while the *CAT*-262C>T polymorphism alone may not influence PCOS susceptibility, catalase activity is dynamically regulated by genetic, metabolic, and environmental factors and likely contributes to PCOS pathophysiology through oxidative stress-related pathways. Future investigations incorporating multiple *CAT* variants, functional analyses, and metabolic stratification are required to fully elucidate the gene's role in PCOS.

Strengths, Limitations and Future Perspective

This meta-analysis rigorously synthesized data from multiple peer-reviewed case-control studies, increasing statistical power to detect associations between the *CAT*-262C>T polymorphism and PCOS risk. The use of standardized quality assessment tools and adherence to PRISMA guidelines ensured methodological rigor and transparency. Subgroup analyses by ethnicity further enhanced the interpretability of results across diverse populations. Despite these strengths, limitations exist. The relatively small number of available studies and modest sample sizes limited the ability to detect subtle genetic effects. Heterogeneity in study designs, diagnostic criteria, and genotyping methods may have introduced variability. Additionally, the absence of comprehensive individual-level data prevented the investigation of any gene-gene and gene-environment interactions.

Future research should focus on large-scale, well-designed multicenter studies incorporating standardized diagnostic criteria and genotyping protocols. Investigations into combined effects of multiple oxidative stress-related gene polymorphisms and environmental factors are warranted to better understand the complex pathophysiology of PCOS. Functional studies elucidating the biological impact of *CAT* variants will also provide mechanistic insights.

CONCLUSION

This meta-analysis reported no significant association between the *CAT*rs1001179 ("262C>T") polymorphism and susceptibility to PCOS across multiple genetic models and ethnic groups. While oxidative stress remains a critical factor in PCOS pathogenesis, this particular genetic variant does not appear to substantially influence disease risk. Continued research incorporating larger cohorts and integrative approaches is necessary to clarify the genetic architecture underlying PCOS and its oxidative stress components.

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Ethics Statement

This research did not involve human participants, animal subjects, or any material that requires ethical approval.

Informed Consent Statement

This study did not involve human participants, and therefore, informed consent was not required.

Clinical Trial Registration

This research does not involve any clinical trials.

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Not Applicable.

Author Contributions

Amrit Sudershan: Data Curation, Formal Analysis, Investigation, Writing – Original Draft, Visualization; Ankush Bala: Data Curation, Formal Analysis, Investigation, Writing – Original

Draft; Bashir Ahmad Sheikh: Formal Analysis, Investigation; Srishty Sudershan: Data Curation, Formal Analysis, Investigation, Writing – Original Draft; Mohd Younis: Conceptualization, Supervision, Visualization, Writing – Review & Editing; Parvinder Kumar: Conceptualization, Supervision, Writing – Review & Editing, Project Administration.

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