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PARP-1 Val762Ala (rs1136410) Gene Polymorphism is Associated with Cancer Incidence in Asian Population: A Meta-Analysis

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ABSTRACT

Introduction: Poly(ADP-ribose) polymerase-1 (PARP-1) is an enzyme involved in DNA repair mechanism especially in resolving strand break lesion. Genetic variation in form of polymorphism in PARP-1 Val762Ala (rs1136410) may reduce its enzymatic activity and affect cancer susceptibility. This study aimed to analyze the roles of this SNP in cancer development specifically on Asian population.

Methods: A systematic literature search was conducted up to May 2026 using PubMed, PubMed Central, ScienceDirect, and Nature databases. Eligible studies published from 2005 onward investigating the association between PARP-1 Val762Ala polymorphism and cancer incidence in Asian populations were included. Pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated under different genetic comparison models. Sensitivity analysis and publication bias assessment using Egger's test were also performed.

Result: Our meta-analysis included 21 articles with 22 studies, comprised of a total of 7,866 cancer cases and 12,086 control. Analysis on 4 model analysis resulted as shown (TT vs TC+CC (OR= 1.09 [0.98; 1.22]; 95% CI)), (CC vs TT/TC (OR= 1.14 [1.00; 1.29]; 95% CI)), (CC vs TT (OR = 1.14 [0.98; 1.33]; 95% CI)), and (TC vs TT (OR = 1.05 [0.92; 1.19]; 95% CI)). Sensitivity analysis and Egger's test showed that results of the meta-analysis were fairly stable.

Conclusion: This study indicated that PARP-1 Val762Ala (rs1136410) gene polymorphism is associated with an increased risk of cancer particularly on CC genotype in Asian population.

Keywords: PARP-1 Val762Ala (rs1136410) polymorphism, Cancer Incidence, Asian population.
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INTRODUCTION

Cancer incidence has increased from roughly 18 million to 19 million per year in 2018-2020 period according to Globocan.[1,2] It is also estimated that the number would increase 1.5 fold in 2040 compared to 2020.[3] Many factors may affect cancer incidence, including DNA repair defects. DNA damage may leads to carcinogenesis driven by mutations.[4] Several gene encoded DNA repair protein may account for cancer susceptibility, one of them is PARP-1.[5] Human PARP-1 (Poly ADP-ribose Polymerase) is a gene located on chromosome 1q41-42 with 23 exons and its protein is an enzyme consist of 3 domain.[6–8] DNA binding domain act as sensor for DNA damage lesion such as DNA nicks, DNA overhangs, and DNA blunt end.[9] Auto-modification domain of PARP-1 aid interaction with other proteins associated with PARP-1 activity and become site for ADP-ribosylation activity.[10] Catalytic domain in PARP-1 protein is responsible for catalyzing the poly ADP-ribosylation activity with NAD+ as the substrate.[11] PARP-1 involved in many biological process such as replication, chromatin remodeling, and base excision repair.[12] PARP-1 as DNA repair gene is responsible of protecting genomic stability in normal cells.[12] Other than that, PARP-1 are downstream pathway of caspase-3 and caspase-9 contributing in cell apoptosis. Hyperactivation of PARP-1 is also become one of mechanism in inducing apoptosis and necrosis.[13,14] PARP-1 act mainly as strand break repair for DNA lesion such as Single Strand Break (SSB) and Double Strand Break (DSB).[12]

Single nucleotide polymorphism (SNP), most frequent type of gene polymorphism, is a substitution of single DNA nucleotide and exist in >1% of a population which differentiate polymorphism and mutation. [8] PARP-1 polymorphism Val762Ala (rs11136410) is the most investigated SNP in PARP-1, which cause substitution of single nucleotide from T to C in codon 762 exon 17 resulting in base substitution from valine to alanine in catalytic domain of PARP-1. This SNP lower enzymatic activity in catalytic domain of PARP-1.[15] However, two study found no association of SNP and lower enzymatic activity observed in previous study possibly due to small sample size.[16,17] PARP-1 may contribute in cancer progression as its function is based on DNA repair mechanism. As theoretically lower enzymatic activity of PARP-1 may affect cancer susceptibility, as several studies shows significant association.[18–20] However, several studies found inconclusive result between SNP and

cancer susceptibility.[21,22] Each population has different allele frequency, which makes the effect of certain SNP differ from one region to others. Globally, T allele frequency dominates by more than 80%. However, in Asian population T allele comprises only 57% of the population, which more equally distributed with C allele.[23] Specific study on certain population is necessary to accurately documented the effect of SNP.

This study provides latest meta-analysis about PARP-1 Val762Ala (rs1136410) gene polymorphism and its association with cancer susceptibility specifically in Asian population.

MATERIAL AND METHOD

Literature Searching

Database used in this study is PubMed, PubMed Central, Science Direct, Cochrane, and Nature with keyword on ((PARP-1 OR (ADPR) OR (ADPRT) OR (Poly (ADP-ribose) polymerase)) AND ((POLYMORPHISM) OR (SNP) OR (VARIANT)) AND ((VAL762ALA) OR (V762A) OR (rs1136410) OR (T2285C) OR (T2444C) OR (C>T)) AND ((CANCER) OR (NEOPLASM) OR (CARCINOMA)). Last search was updated to 22 May 2026.

Studies retrieved from databases will be further selected based on inclusion and exclusion criteria. Inclusion criteria applied as following: 1) Case Control Study; 2) Study analyzing association of PARP-1 Val762Ala rs1136410 polymorphism with cancer incidence/susceptibility/risk; 3) Study provided sufficient data about genotype analysis and resulted in OR with 95% confidence interval; 4) Research subject is based on Asian population only. Study excluded if met certain criteria: 1) Duplicate study; 2) Family based study; 3) Study on benign tumor; 4) Study with empty cells in one or more genotype group; 5) Study violating HWE.

Data Extraction

For each included study, author reviewed abstract of relevant study characteristics: (i.e. authors name, publication year, cancer type, ethnicity of the study subject, control source, genotype method, score, allelic frequency) and classified the data into a structured form. Author checked all data for completeness and accuracy as well eligibility for inclusion in analysis. Disagreement on study eligibility were resolved through discussion until consensus was made.

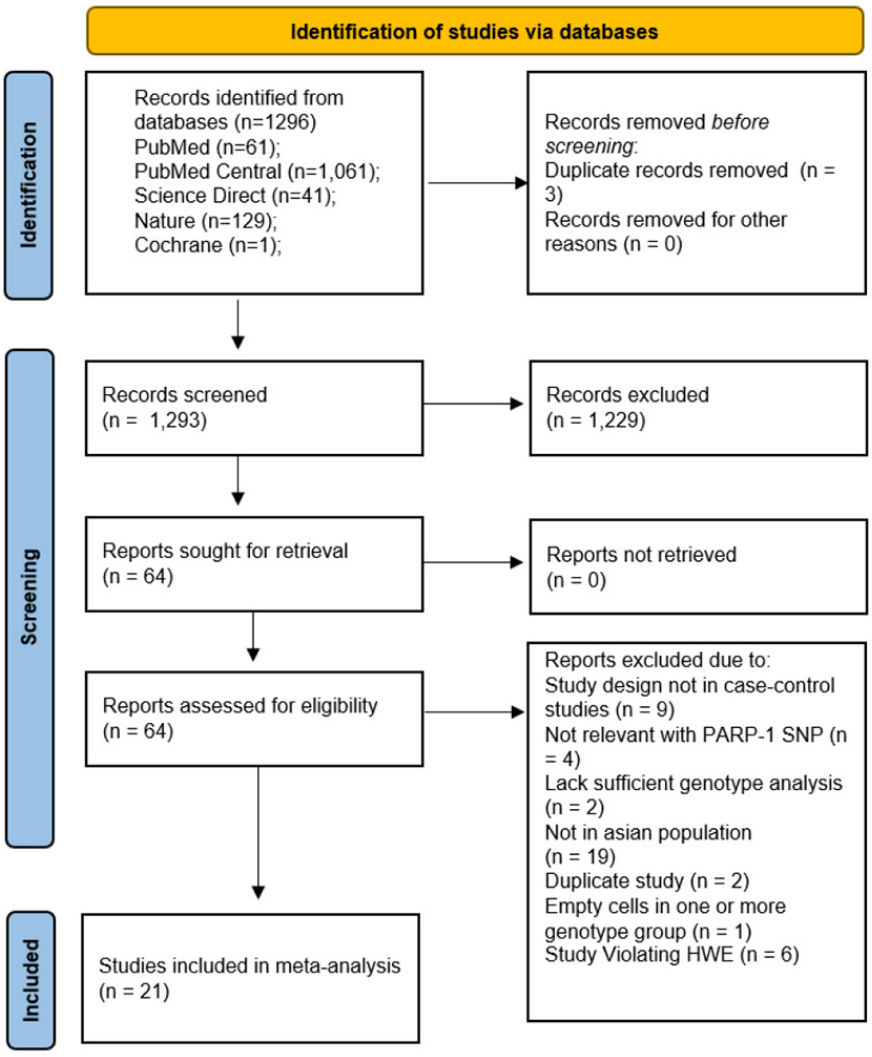


Figure 1. PRISMA Flow Chart of Literature Searching

Statistical methods

We tested study population confirmation to Hardy-Weinberg equilibrium (HWE) using chi-square test and interpreted $p>0.05$ as confirming to the equilibrium. Association between PARP-1 Val762Ala (rs1136410) and cancer risk was assessed by pooling ORs and the corresponding 95% CIs calculated from case-control and exposure-non exposure cross tabulation obtained from each study. Heterogeneity was assessed by I2 value and a value greater than 50% is interpreted as significantly heterogeneous. Heterogenous data was pooled with a random-effect model while homogenous data was pooled with a fixed model. We performed sensitivity analysis by omitting one study at a time and checking the fluctuation of the pooled results. Funnel plot symmetry was assessed to gauge publication bias, and Egger's regression test was employed to quantify the plot's asymmetry. We used R version 4.2.2 with numerous packages to perform statistical analysis.

RESULT

Characteristics of Eligible Studies

A total of 1,296 articles relevant to the keyword were found across 5 databases (PubMed, PubMed Central, Science Direct, Nature, and Cochrane). These records were then screened based on title and abstract, resulting in 61 articles suitable for further selection. Following the inclusion and exclusion criteria outlined above, 21 articles were retrieved

Table 1. Eligible Studies Characteristics

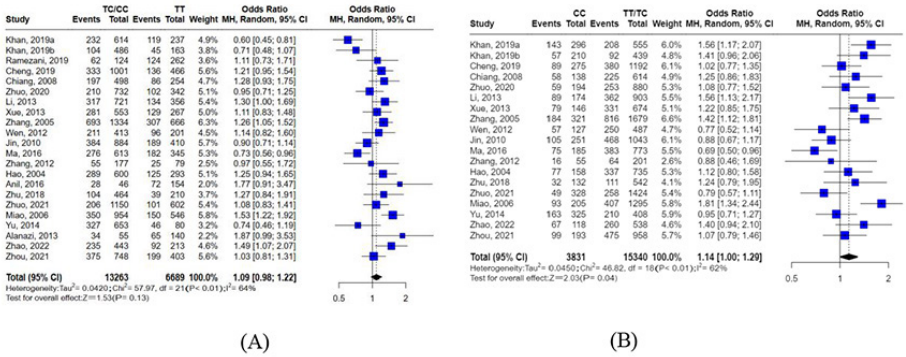
Author	Country	Sample Size (Case/Control)	Cancer Type	SNP Identification Method
Alanazi, 2013	Saudi Arabia	99 / 96	Breast	TaqMan
Anil, 2016	India	100 / 100	OSCC	TaqMan
Cheng, 2019	China	469 / 998	Neuroblastoma	TaqMan
Chiang, 2008	Taiwan	283 / 469	Thyroid	TaqMan
Hao, 2004	China	414 / 479	Esophageal	PCR+PolyPhred
Jin, 2010	Korea	573 / 721	Lymphoma	HRM-PCR
Khan, 2019	Pakistan	500 / 500	Glioma & Meningioma	ARMS-PCR
Li, 2013	China	451 / 626	Colorectal	PCR-RFLP
Ma, 2016	China	458 / 500	Breast	MassARRAY
Miao, 2006	China	500 / 1,000	Gastric	PCR-RFLP
Ramezani, 2019	Iran	186 / 200	Breast	ARMS-RFLP-PCR
Wen, 2012	China	307 / 307	Gastric	MassARRAY
Xue, 2013	China	410 / 410	Lung	PCR-RFLP
Yu, 2014	China	373 / 360	Lung	PCR-RFLP
Zhang, 2005	China	1,000 / 1,000	Lung	PCR-RFLP
Zhang, 2012	China	80 / 176	Cervical	PCR+DNA Chip
Zhao, 2022	China	327 / 329	Endometrial	PCR-LDR
Zhou, 2021	China	574 / 577	Esophageal	PCR-LDR
Zhu, 2018	China	145 / 531	Nephroblastoma	TaqMan
Zhuo, 2020	China	312 / 762	Neuroblastoma	TaqMan
Zhuo, 2021	China	313 / 146	Hepatoblastoma	TaqMan

*abbreviations and explanations : OSCC: Oral Squamous Cell Carcinoma; RFLP: Restriction Fragment Length Polymorphism; LDR: Ligase detection reaction; ARMS: Amplification-refractory Mutation System ; TaqMan: an RT-PCR procedure with an extra TaqMan probe

for analysis, as depicted in Figure 1. This study encompassed **7,866** cancer cases and **12,086** control subjects, with the specific characteristics of each study listed in **Table 1.**

Quantitative Analysis

The main results of the meta-analysis can be observed in Figure 2-5. The pooled results indicated that PARP-1 Val762Ala (rs1136410) was associated with increased cancer incidence in the recessive model CC vs TT/TC (OR= 1.14 [1.01; 1.29]; 95% CI) but no significant association were found in other models (**Figure 2-5**). The lack of sufficient studies in each cancer type precluded the performance of subgroup analysis.



(A) (B)

Figure 2. Forest plot analysis on Dominant model (TT vs CC/TC). Blue Squares and horizontal lines represent risk estimates of each study based on Odd Ratio on 95% CI. Diamond Represents overall risk estimation on 95% CI (A).

Forest plot analysis on Recessive model (CC vs TT/TC). Blue Squares and horizontal lines represent risk estimates of each study based on Odd Ratio on 95% CI. Diamond Represents overall risk estimation on 95% CI (B).

*TT = Val/Val (AA) ; TC = Val/Ala (AG) ; CC = Ala/Ala (GG)

*TT = Val/Val (AA) ; TC = Val/Ala (AG) ; CC = Ala/Ala (GG)

Forest plot analysis on Heterozygote model (TC vs TT). Blue Squares and horizontal lines represent risk estimates of each study based on Odd Ratio on 95% CI. Diamond Represents overall risk estimation on 95% CI (B).

*TT = Val/Val (AA) ; TC = Val/Ala (AG) ; CC = Ala/Ala (GG)

*TT = Val/Val (AA) ; TC = Val/Ala (AG) ; CC = Ala/Ala (GG)

Sensitivity Analysis, Heterogeneity Analysis, and Conformation to Hardy-Weinberg Equilibrium

A significant heterogeneity was found in dominant (I2 = 64%), recessive (I2 = 62%), homozygote (I2 = 67%), and heterozygote (I2 = 67%). Therefore, a random-effect model was applied to pool ORs and their respective 95% Cis from the included studies. In addition,

the conducted sequential sensitivity analysis was conducted showed great reliability as no significant fluctuation was found on the pooled results following omittance of any included study in all models (Table x). Out of the 27 studies included in the analysis, six did not conform to the Hardy-Weinberg Equilibrium and are therefore excluded from the pooling process.

Publication Bias

The results of the publication bias assessment, employing Begg's funnel plot and Egger's test, suggest that there is no clear evidence of publication bias in the current meta-analysis. The symmetrical outlines of Begg's funnel plots indicate a lack of visual asymmetry, traditionally associated with publication bias. Additionally, the quantitative Egger's test, which assesses funnel plot asymmetry, supports the conclusion of the absence of significant publication bias across all analyzed models.

DISCUSSION

The current study encompassing 27 studies indicates a significant relationship between the PARP-1 Val762Ala (rs1136410) gene polymorphism and cancer susceptibility in Asian population. PARP-1 is an enzyme located on chromosome 1q41-42 involved in several cellular process with its main role is DNA repair activity particularly on strand break lesion.[24,25] PARP-1 Val762Ala (rs1136410) gene polymorphism causes a base change from T to C altering the amino acid from valine to alanine in codon 762 which reduced PARP-1 catalytic activity, potentially increasing the risk of developing cancer.[15,18] Decreased catalytic activity demonstrated by Wang et al. by analyzing PAR protein, which is the result of PARP-1 enzymatic activity.[15] This particular SNP garners frequent investigation due to its coding region alteration, specifically in the domain encoding the catalytic region of PARP-1 within the C-terminal.[26]. This study further substantiates the effect of this SNP on the Asian population, particularly highlighting its impact on the CC genotype.

In alignment with recent research conducted by Anjali et al., there is a noticeable association between the PARP-1 Val762Ala polymorphism, specifically the CC genotype, and the incidence of gallbladder cancer.[19] Similarly, a study by Zhao et al. echoes these findings, demonstrating significant correlation between the SNP and increased risk of endometrial carcinoma among individuals with CC genotype.20 In contrast, Roszak et al.'s study involving Caucasian subjects indicates an elevated risk of cervical cancer, particularly in individuals with TC genotype.[27] However, some studies within the Asian population showcase a potentially protective effect of the PARP-1 Val762Ala gene polymorphism or find no substantial association with breast cancer susceptibility. Ma et al.'s study hints at a protective effect of the SNP, as evidenced by odds ratio values below 1.[28] Conversely, research by Ramezani et al. in Iran concludes no significant association with breast cancer risk.[29]

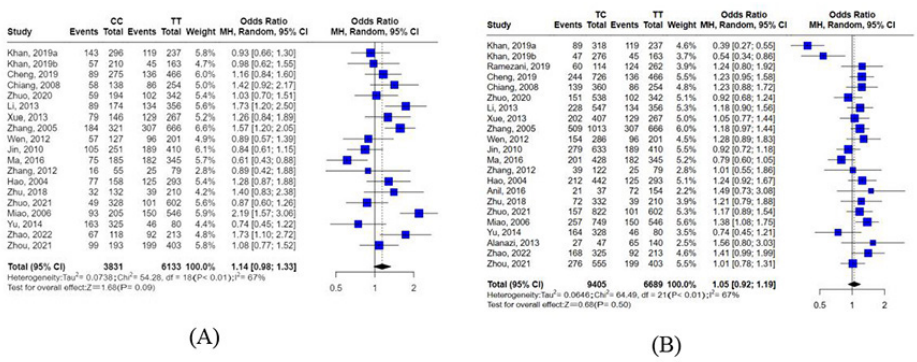


Figure 3. Forest plot analysis on Homozygote model (CC vs TT). Blue Squares and horizontal lines represent risk estimates of each study based on Odd Ratio on 95% CI. Diamond Represents overall risk estimation on 95% CI (A).

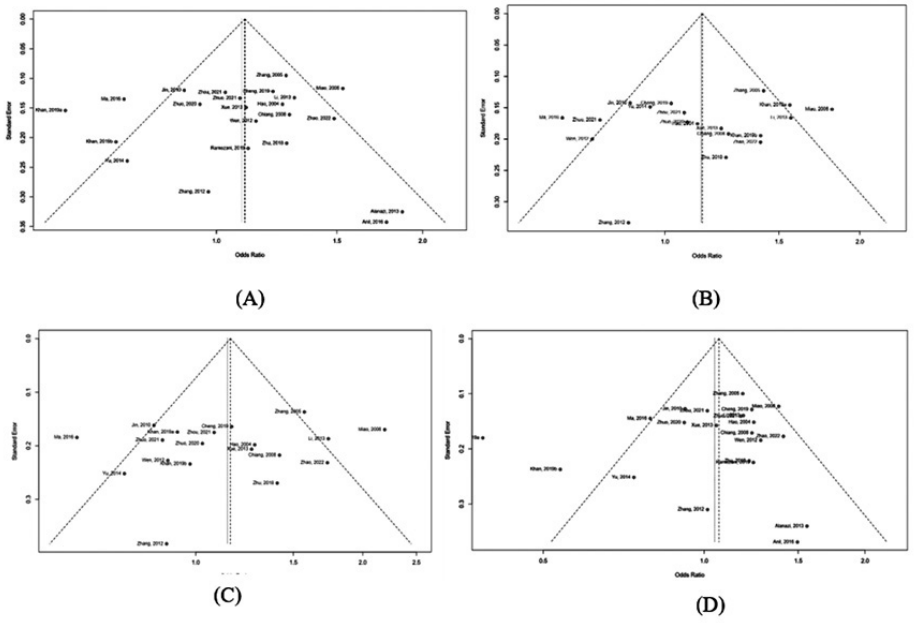


Figure 4. Publication bias detection by Funnel Plot analysis in Dominant Model (TC/CC vs TT) (A); Publication bias detection by Funnel Plot analysis in Recessive Model (CC vs TT/TC) (B); Publication bias detection by funnel plot analysis in Homozygote Model (CC vs TT) (C); Publication bias detection by funnel plot analysis in heterozygote model (TC vs TT) (D)

Nuerous meta-analyses have explored PARP-1 SNP's association with cancer risk. Li et al.'s meta-analysis in 2020, involving 23,996 cases and 33,015 controls, suggests that the PARP-1 rs1136410 gene polymorphism might contribute to an increased risk of gastric, thyroid, and cervical cancers within the Asian population, but not African and Caucasian populations. Their study highlights a significant association in the recessive model, elevating cancer risk, yet no substantial associations were found in the homozygous, heterozygous, and dominant models.[30] Palaban et al.'s study in 2012 shows a significant association of the Alanine variant, indicating a protective effect

against the risk of cancer in the Chinese population.[31] Meanwhile, Yu et al. in 2012 found no significant results across all models but identified an increased cancer risk in the Asian population with the PARP-1 Val762Ala polymorphism upon stratified analysis.[32]

This study presents the results of analyzing the effect of one type of PARP-1 polymorphism, rs1136410. It does not look at the effect of other PARP-1 polymorphisms because the study of other PARP-1 polymorphisms such as rs907187, rs4653734 and rs3219145 is still limited.[28,29] It should also be noted that there are also proteins upstream and downstream of the pathway related to PARP-1 function. Several proteins with a PARP-1 binding motif may interact with PARP-1 in a reciprocal manner to achieve proper DNA repair activity which is not considered in this study.[33] This study limitation persist in nature of one gene polymorphism specific analysis, which is not considering another possible gene which may affect PARP-1. Therefore, to get a better picture of the effect of increased cancer risk, a combined analysis with other gene polymorphisms is needed.

CONCLUSION

This study underscores that the PARP-1 Val762Ala (rs1136410) gene polymorphism, especially within the CC genotype, significantly elevates cancer incidence among Asian populations. Given that most studies on this topic are centered around Caucasian and Asian populations, further investigations involving other ethnicities are warranted.

CONFLICT OF INTEREST

The authors disclose that research was carried out without any financial or commercial ties that might be seen as having a conflict of interest.

FUNDINGS

No specific funding was provided for this research.

AUTHOR CONTRIBUTION

AAGCW, MDWA, GBB, designed the study framework. AAGCW, MAKA, MDWA, GBB, and IGBBAR conducted the comprehensive literature search, screened the databases, and synthesized the initial manuscript draft. IGPS and NNAD supervised the project, critically reviewed and revised the manuscript for intellectual content, and finalized the formatting. All authors read and approved the final version of the manuscript.

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Supplementary materials 1. Quality Assessment Table

Study	Design	Selection				Comparability	Exposure			Total Score	Interpretation
		1	2	3	4	1	1	2	3		
Zhou, et al (2021)	Case-Control Study	*	*	*	*	**	*	*		8	Good
Alanazi, et al (2013)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Ramezani, et al (2019)	Case-Control Study	*	*	*	*	*	*	*		7	Good
Ma, et al (2016)	Case-Control Study	*	*	*		**	*	*	*	8	Good
Hao, et al (2004)	Case-Control Study	*	*	*	*	* *	*	*	*	9	Good
Anil, et al (2016)	Case-Control Study	*			*	*	*	*	*	6	Fair
Zhu, et al (2018)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Wang, et al (2015)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Zhuo, et al (2021)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Miao, et al (2006)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Khan, et al (2019)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Zhang, et al (2009)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Bashir, et al (2018)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Cheng, et al (2019)	Case-Control Study	*	*		*	*	*	*	*	7	Good
Chiang, et al (2008)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Nakao, et al (2012)	Case-Control Study	*			*	**	*	*	*	7	Good
Yu, et al (2014)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Ye, et al (2012)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Anjali, et al (2021)	Case-Control Study	*	*	*	*	*	*	*	*	8	Good
Zhao, et al (2022)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Zhuo, et al (2020)	Case-Control Study	*	*		*		*	*		5	Fair
Li, et al (2013)	Case-Control Study	*	*			**	*		*	6	Fair
Xue, et al (2013)	Case-Control Study	*	*		*	**	*	*	*	8	Good
Zhang, et al (2005)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Wen, et al (2012)	Case-Control Study	*	*	*	*	**	*	*	*	9	Good
Jin, et al (2010)	Case-Control Study	*	*		*	*	*	*	*	7	Good