1. Introduction

Hepatocellular carcinoma (HCC) is one of the most common and lethal cancer worldwide. The estimated annual number of patients with HCC has increased by more than 500,000 cases. Although significant advances in the diagnosis and treatment, the prognosis of HCC patients remains poor and the 5-year survival rate in developing countries is only 5%.[12,13] Multiple factors have been demonstrated to be associated with the development of HCC, such as chronic infection with hepatitis B (HBV) or hepatitis C virus, excessive alcohol consumption, high cigarette smoking and many etiological factors.[4] Additionally, HCC has been proven to be induced by inflammation, and virus-associated HCC is the most common type of liver cancer. In China, more than 80% of HCC patients were associated with chronic HBV infection.[5,4]

Today, most diagnoses of virus-related HCC are made after the disease has progressed substantially, and there is no effective therapy for most virus-related HCC patients currently.[7–9] Therefore, effective screening of high-risk populations for chemoprevention is of great significance to the treatment of virus-related HCC.[7,10] Serum alpha-fetoprotein measurement and liver imaging are currently the main methods for screening high-risk groups. However, due to the low sensitivity and specificity, the effectiveness is questionable and limited.[11–13] In order to improve prevention and treatment strategies, the identification of molecular markers associated with the risk of virus-related HCC is necessary.

In recent years, several important signaling pathways have been systematically studied in virus-related HCC. These pathways regulate physiological processes such as the growth and differentiation of tumor cells, the regeneration of blood vessels,
and the migration of tumor cells. Epidermal growth factor (EGF) plays a significant role in cell proliferation, differentiation and tumorigenesis of epithelial tissues. The EGF +61A > G polymorphism (rs4444903) is a functional SNP in the 5\’ untranslated region of the EGF gene. It results in higher EGF levels in individuals with EGF genotype G/G in comparison to the A/A genotype. Studies have shown that EGF signal pathway plays an important role in the occurrence of HCC. It was involved in the proliferation of epithelial and epithelial cells, which has a strong relationship with embryonic growth, tissue repair, regeneration, and tumorigenesis. The transient profile of EGF RNA accumulation suggested that the elevated EGF levels may catalyze a cascade of events preceding the first wave of liver DNA replication in hepatocytes isolated by collagenase perfusion. The EGF could activate the EGF receptor as a ligand with biological effect through signal transduction.

In this study, we performed a meta-analysis of all eligible studies to clarify the relationship between EGF polymorphism and the risk of virus-related HCC.

2. Methods

The Preferred Reporting Items for Systematic Reviews and Meta-analyses criteria were used for this meta-analysis.

2.1. Literature-searching strategy

We performed a computerized literature search by 2 independent researchers in the following 6 electronic databases: Chinese National Knowledge Infrastructure, Wanfang, VIP, Pubmed, Web of Science, and Embase from their start date to September 2022. We used the following keywords and medical subject heading terms: (“Epidermal growth factor” or “EGF”) and (“polymorphism” or “variant” or “SNP” OR “mutation”) and (“hepatocellular carcinoma” or “liver cell carcinoma” or “liver cancer”).

2.2. Inclusion and exclusion criteria

Studies included in the meta-analysis had to meet the following inclusion criteria: evaluating the association between EGF polymorphism and virus-related HCC risk, using unrelated individuals, providing sufficient data for estimating an odds ratio (OR) with its 95% confidence interval (CI), using case-control, cohort or cross-sectional design, published in English or Chinese. The corresponding authors were contacted to obtain missing information, and some studies were excluded if critical missing information was not obtained. Reviews, case reports, family-based studies, case-only studies, and studies without sufficient data were excluded. When a study reported results on different sub-populations based on ethnicity or geographical region, we treated each sub-population as a separate comparison. If more than 1 article was published using the same subjects, only the study with the largest sample size was selected.

2.3. Data extraction

All data were extracted independently by 2 investigators. Disagreement was resolved by discussion. The following data were extracted: authors, name of the journal, year of publication, ethnicity, country of study population, inclusion and exclusion criteria, characteristics of cases and controls, numbers of HCC cases and controls, matching criteria, source of controls, HCC confirmation, study design, genotyping methods, genotype frequencies of cases and controls, and interactions between environmental factors or genes.

2.4. Quality score assessment

The quality of the studies was independently assessed by the same 2 investigators. Any disagreement was resolved by discussion between the 2 investigators. The total scores ranged from 0 (worst) to 24 (best). Studies scoring <16 were classified as “low quality” and those scoring ≥16 as “high quality.”

2.5. Statistical analysis

The unadjusted OR with 95% CI was used to assess the strength of the association between the EGF polymorphism and the risk of virus-related HCC. The pooled ORs were performed under the allelic contrast (G vs A), codominant model (homozygote comparison: GG vs AA, heterozygote comparison: GA vs AA), dominant model (GG + GA vs AA), and recessive model (GG vs GA + AA), respectively. Heterogeneity between studies was measured using a Q statistic test and an I² statistic. P < .10 was considered representative of significant statistical heterogeneity due to the low power of the statistic. I² values of 25%, 50%, and 75% were defined as low, moderate and high estimates. If the significant Q-statistic indicated heterogeneity across studies, the random-effects model was used, otherwise, the fixed-effects model was adopted. The Z test was used to assess the significance of the pooled OR and a P < .05 was considered significant.

Subgroup analyses were stratified by racial descent, study quality, source of controls, type of controls, and the number of cases, respectively. Furthermore, metaregression analysis was performed to investigate 5 potential sources of heterogeneity including ethnicity (Asian populations vs non-Asian populations), study quality (high-quality studies vs low-quality studies), source of controls (hospital-based vs Population-based), type of controls (healthy controls vs controls with chronic liver diseases) and the number of cases (<100 vs ≥100). Statistical significance was defined as a P < .10 because of the relatively weak statistical power.

To evaluate the stability of the results, sensitivity analyses were performed by sequential omission of individual studies under various comparisons in the overall and Asian populations, respectively. Publication bias was investigated by funnel plot. Funnel plot asymmetry was assessed by the method of linear regression test. The Hardy-Weinberg equilibrium was tested using the \( \chi^2 \) test. All P values were 2-sided. Data analyses were performed using the software Stata version 11.0 software.

3. Results

3.1. Eligible studies

As shown in Figure 1, a total of 1124 articles were initially obtained by searching the databases. After duplicate checks by Endnote 20, 905 articles remained. We subsequently excluded 882 articles based on browsing the titles and abstracts. According to the inclusion criteria, 5 of the remaining 23 records were further excluded based on a full-text review. In total, 21 studies (18 articles) with 2692 virus-related HCC cases and 5835 controls were finally included in this meta-analysis.

3.2. Characteristics of study

The characteristics of the 21 included studies are shown in Table 1. Of all eligible studies, 11 were conducted in Asian populations, 2 in European populations, 5 in African populations, and 3 in mixed populations. In all studies, the cases were histologically confirmed (17 studies) or diagnosed by elevated
α-fetoprotein and different iconography changes (abdominal ultrasound and triphasic computed tomography). All controls were free of cancer. Four studies used healthy populations as controls, 5 studies used patients with chronic liver diseases (HBV infection, hepatitis C virus infection, and cirrhosis) as controls, and 12 studies used healthy subjects and patients with chronic liver diseases as controls. The sample size of the total participants ranged from 75 to 1774, with a mean of 406. Quality scores for individual studies ranged from 11.5 to 21, with 10 of the 21 studies classified as high quality. Twenty studies used peripheral blood, and 1 study used FFPE to extract genome DNA. Fourteen studies used the polymerase chain reaction-restriction fragment length polymorphism assay, 6 studies used the Taqman method, and 1 study used Matrix-Assisted Laser Desorption/Ionization Time of flight mass spectrometry to genotype the EGF + 61 A/G polymorphism. The genotype distribution in the controls of all studies was consistent with Hardy-Weinberg equilibrium.

### 3.3. Meta-analysis results

The pooled results of all studies showed that the EGF + 61A/G polymorphism was significantly associated with the susceptibility of virus-related HCC under all genetic models (G vs A: \(OR = 1.56, P < .001, 95\% CI: 1.26–1.94, I^2 = 86.8\%\); GG vs GA + AA: \(OR = 1.67, P < .001, 95\% CI: 1.29–2.15, I^2 = 79\%\); GG + GA vs AA: \(OR = 1.67, P < .001, 95\% CI: 1.26–2.20, I^2 = 70.2\%\); GG vs AA: \(OR = 2.18, P < .001, 95\% CI: 1.50–3.16, I^2 = 76.6\%\); GA vs AA: \(OR = 1.20, P < .001, 95\% CI: 1.03–1.39, I^2 = 23.7\%\)) (Table 2).

In subgroup analyzes based upon ethnicity, significantly associations were observed between EGF + 61A/G polymorphism and the risk of virus-related HCC in Asian populations (G vs A: \(OR = 1.15, P < .001, 95\% CI: 1.02–1.29, I^2 = 40.1\%\)), European populations (G vs A: \(OR = 1.59, P < .001, 95\% CI: 1.05–2.41, I^2 = 0.0\%\)) and African populations (G vs A: \(OR = 4.46, P < .001, 95\% CI: 1.53–13.02, I^2 = 93\%\)) (Fig. 2). When stratifying by study quality, the results showed that EGF + 61A/G polymorphism was associated with the risk of virus-related HCC both in high-quality studies (G vs A: \(OR = 1.20, P < .001, 95\% CI: 1.08–1.34, I^2 = 24.2\%\)) and in low-quality studies (G vs A: \(OR = 1.98, P < .001, 95\% CI: 1.25–3.14, I^2 = 91.7\%\)). In subgroup analyzes by source of controls, the results showed that the EGF + 61A/G polymorphism was significantly associated with the risk of virus-related HCC in hospital-based studies (G vs A: \(OR = 1.72, P < .001, 95\% CI: 1.29–2.29, I^2 = 88.8\%\)), but not in population-based studies (G vs A: \(OR = 1.12, P = .202, 95\% CI: 0.99–1.27, I^2 = 0.0\%\)). Furthermore, according to the type of controls, a significant association was observed between EGF + 61A/G polymorphism and the risk of virus-related HCC when the controls had chronic liver diseases (G vs A: \(OR = 2.02, P < .001, 95\% CI: 1.32–3.09, I^2 = 82.7\%\)), and when the controls were healthy individuals (G vs A: \(OR = 1.40, P < .001, 95\% CI: 1.10–1.79, I^2 = 86.4\%\)) (Table 2).
3.4. Heterogeneity analysis

The Q-statistic indicated statistically significant heterogeneity across all studies under all genetic models except for heterozygote comparison (Table 2). However, in the subgroup analyses by ethnicity, the between-study heterogeneity was not observed in Asian and European populations. Moreover, meta-regression indicated that both ethnicity, type of control, and study quality significantly contributed to the heterogeneity (Table 3).

3.5. Sensitivity analysis and publication bias

Begg’s funnel plots were generated and Egger’s test was performed on the final set of 21 studies to assess publication bias. The results showed that the risk of publication bias may exist in the overall population, but a low risk of publication bias in Asian populations (Fig. 3 and Table 4). Sensitivity analysis was performed by sequential omission of individual studies. Pooled ORs were consistently significant in general populations or Asian populations by omitting 1 study at a time under the allelic contrast, recessive model, and homozygote comparison, suggesting the robustness of our results (Fig. 4).

4. Discussion

HCC is a complex disease in which the environment and the host interact with multiple genes. Currently recognized risk factors for HCC include liver virus infections, aflatoxins, alcoholic liver cirrhosis, etc. However, only a small number of people exposed to the above risk factors eventually develop HCC, which indicates that host genetic factors may play an important role in the pathogenesis of HCC. Accumulating evidence has

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**Table 1**

Main characteristics of eligible studies included in the meta-analysis.

| First author     | Year | Country (ethnicity) | Source of controls | Type of controls | Sample origin | Genotyping methods | Sample size (case/control) | Genotype frequency (case/control) | Quality score |
|------------------|------|---------------------|--------------------|------------------|---------------|-------------------|---------------------------|-----------------------------------|---------------|
| Tanabe-FRA       | 2008 | France (European)   | HB                 | Cirrhosis        | Peripheral    | PCR-RFLP         | 44/77                     | GG 15/12, GA 17/37, AA 12/28 | 39.60% Y       | 13.5          |
| Tanabe-USA       | 2008 | USA (mixed)         | HB                 | HBV/HCV/ Cirrhosis/ Healthy/healthyHBV | Peripheral    | PCR-RFLP         | 59/148                    | GG 23/32, GA 27/65, AA 9/51 | 43.60% Y       | 14.5          |
| Qi                | 2009 | China (Asian)       | HB and PB          | Healthy/healthyHBV | Peripheral    | PCR-RFLP         | 215/380                   | GG 102/182, GA 98/160, AA 15/38 | 68.90% Y       | 21            |
| Wang-GX          | 2009 | China (Asian)       | HB                 | Healthy/healthyHBV | Peripheral    | PCR-RFLP         | 376/477                   | GG 190/208, GA 154/221, AA 32/48 | 66.80% Y       | 17.5          |
| Wang-JS          | 2010 | China (Asian)       | HB                 | Healthy/healthyHBV | Peripheral    | PCR-RFLP         | 186/198                   | GG 107/93, GA 65/88, AA 14/17 | 69.20% Y       | 18            |
| Li                | 2010 | China (Asian)       | HB and PB          | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 186/338                   | GG 96/161, GA 82/145, AA 8/32  | 69.10% Y       | 19.5          |
| Abu Dayeh        | 2011 | USA (mixed)         | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 66/750                    | GG 26/178, GA 25/350, AA 15/222 | 47.10% Y       | 16.5          |
| Chen              | 2011 | China (Asian)       | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 120/240                   | GG 62/106, GA 51/110, AA 7/24  | 67.10% Y       | 19            |
| Abbas            | 2012 | Egypt (African)     | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 20/60                     | GG 7/9, GA 9/28, AA 4/23     | 38.30% Y       | 12            |
| Cmet              | 2012 | Italy (European)    | HB and PB          | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 18/361                    | GG 4/66, GA 10/172, AA 4/123  | 42.10% Y       | 16            |
| Shi               | 2012 | China (Asian)       | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 73/117                    | GG 18/13, GA 31/52, AA 24/52  | 33.30% Y       | 13.5          |
| El-Bendary       | 2013 | Egypt (African)     | HB                 | HCV/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 133/105                   | GG 57/9, GA 43/36, AA 33/60  | 25.70% Y       | 12            |
| Suenaga          | 2013 | Japan (Asian)       | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP         | 208/290                   | GG 108/161, GA 89/104, AA 11/25 | 73.40% Y       | 11.5          |
| Wu                | 2013 | China (Asian)       | HB and PB          | Healthy/ Cirrhosis/ HCV | Peripheral    | TaqMan        | 404/1370                  | GG 206/647, GA 153/576, AA 45/147 | 68.20% Y       | 17.5          |
| Yuan-USA         | 2013 | USA (mixed)         | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | TaqMan        | 117/225                   | GG 28/63, GA 61/102, AA 28/60  | 50.70% Y       | 19            |
| Yuan-CHN         | 2013 | China (Asian)       | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | TaqMan        | 250/245                   | GG 25/20, GA 99/107, AA 126/118 | 30.00% Y       | 15            |
| Wei               | 2016 | China (Asian)       | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | MALDI-TOF-MS    | 47/213                    | GG 30/101, GA 15/98, AA 2/14   | 72.1% Y        | 12.5          |
| El Sergany       | 2017 | Egypt (African)     | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | TaqMan        | 50/50                     | GG 42/2, GA 5/6, AA 3/42      | 49.50% Y       | 15.5          |
| Gholizadeh       | 2017 | Iranian (Asian)     | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | PCR-RFLP       | 40/106                    | GG 4/34, GA 25/48, AA 11/24   | 51% Y          | 13            |
| Asar             | 2020 | Egypt (African)     | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | TaqMan        | 30/60                     | GG 11/11, GA 10/34, AA 9/15   | 48.8% Y        | 13            |
| Baghdadi         | 2020 | Egypt (African)     | HB                 | Healthy/ Cirrhosis/ HCV | Peripheral    | TaqMan        | 50/25                     | GG 20/4, GA 23/13, AA 7/8     | 56% Y          | 21            |

HB = hospital-based, HBV = control subjects were hepatitis B virus carriers, HCV = control subjects were hepatitis C virus carriers, HWE = Hardy-Weinberg equilibrium in control population, MALDI-TOF-MS = matrix-assisted laser desorption/ ionization time of flight mass spectrometry, N = no, PB = population-based, PCR-RFLP = polymerase chain reaction-restriction fragment length polymorphism, Y = yes.
proved the important role of genetic factors in the occurrence and development of tumors. EGFR activation by GEF pathway is well known to be involved in promoting cell proliferation, differentiation, and inhibition of apoptosis. EGFR promotes cell growth and development by combining with transmembrane EGFR receptors to promote cell proliferation and differentiation, thereby enhancing the carcinogenic rate of various carcinogens. In recent years, research on the relationship between the EGF + 61A/G polymorphism and malignant tumor susceptibility has gradually increased. Including HCC. However, inconsistent findings concerning the association between EGF + 61A/G polymorphism and susceptibility to HCC were observed across the studies.

Herein, 21 cohorts (18 articles) with 2692 virus-related HCC and 5835 controls were included in this meta-analysis to analyze the association between EGF + 61A/G polymorphism and the susceptibility of virus-related HCC. The results suggested that EGF + 61A/G polymorphism was significantly associated with the risk of virus-related HCC. Given the considerable heterogeneity across studies, meta-regression analysis was performed and found the contribution of race, type of controls, and research quality to the heterogeneity. Moreover, the stratified analysis further sheds light on the effect of different variables on the relationship between EGF + 61A/G polymorphism and HCC susceptibility. The sensitivity analysis further strengthened the validity of these positive correlations in the overall population and the Asian population, indicating the credibility of our results.

It is possible that the effects of genetic factors related to cancer are different across various ethnic populations. A large
number of studies have shown that the relationship between EGF + 61A/G polymorphism and HCC susceptibility differs between ethnicity. Tanabe KK et al[41] included 2 independent research populations, that one of the research populations was Caucasian, and the other research population was composed of whites, blacks, Asians, and Hispanics. Abu DB et al[38] compared white people to black people. Jiang G et al[46] studied Asian population, European population, and African population. The same result suggested that the EGF + 61A/G polymorphism was significantly associated with the risk of HCC under all genetic models, and the relationship between EGF + 61A/G polymorphism and HCC susceptibility differs between races. In this study, ethnicity was also identified as a potential source of heterogeneity by meta-regression and subgroup analyses. The results showed that the frequency of the EGF + 61G allele was highest in Asian populations, intermediate in European populations, and lowest in African populations. The higher prevalence of the EGF + 61G allele might lead to a higher HCC prevalence among Asian populations. In subgroup analyses based on ethnicity, a significant association was observed between the EGF + 61G allele and HCC prevalence among Asian populations. In subgroup analyses based on ethnicity, a significant association was observed between the EGF + 61G allele and HCC prevalence among Asian populations. In subgroup analyses based on ethnicity, a significant association was observed between the EGF + 61G allele and HCC prevalence among Asian populations.

### Table 3

| Factor                      | GG vs GA + AA | GG + GA vs AA | GG vs AA | GA vs AA | G vs A |
|-----------------------------|---------------|---------------|----------|----------|--------|
|                             | t             | P             | t        | P        | t      | P      | t      | P      | t      | P      |
| Racial descent              | −2.43         | .025          | −1.76    | .095     | −2.29  | .034   | −1.29  | .212   | −2.17  | .043   |
| Source of controls          | 1.07          | .298          | 0.36     | .722     | 0.73   | .473   | −0.08  | .939   | 0.84   | .41    |
| Type of controls            | 0.91          | .373          | 2.12     | .048     | 1.7    | .106   | 2.77   | .012   | 0.71   | .486   |
| Genotyping methods          | 0.68          | .507          | −0.03    | .975     | 0.25   | .803   | −2.05  | .054   | 0.73   | .477   |
| Sample size                 | −1.22         | .236          | −0.89    | .386     | −1.15  | .265   | −0.50  | .626   | −1.11  | .28    |
| Quality score               | 2.6           | .018          | 1.9      | .973     | 2.34   | .031   | 0.75   | .461   | 2.54   | .02    |

The bold value is <0.05 and considered to be significant.

EGF = epidermal growth factor, HCC = hepatocellular carcinoma.
There are several limitations in the present study. This study revealed that the EGF + 61A/G polymorphism was significantly associated with the risk of HCC in a hospital-based study, but not in a population-based study. Therefore, the results should be treated with caution, as the controls from hospital-based studies may not be representative of the general population. Larger population-based studies are needed to further confirm the association between EGF + 61A/G polymorphism and HCC susceptibility. Herein, 11 of the 21 eligible studies were classified as low-quality studies, which might not rule out the true influence of factors that could bias estimates and lead to erroneous conclusions. In addition, aside from genetic factors, there are other factors related to the development of HCC, such as exposure to aflatoxin B1, smoking, and habitual alcoholism, which should be considered. Finally, the number of studies included in the meta-analysis for European populations and African populations was relatively small, which may lead to low statistical power and generate fluctuation in estimation.

This study combined currently published research on the relationship between EGF + 61A/G polymorphism and HCC susceptibility and generated credible pooled results. However, due to the limitations mentioned above, more studies with a more rigorous design, larger sample size, and wider perspectives are required to obtain more reliable gene effects and more precision. The inherent relationship between EGF gene polymorphism and HCC susceptibility provides better preventive measures and treatment options for HCC.

5. Conclusion
In summary, this meta-analysis demonstrated that EGF + 61A/G polymorphism was significantly associated with the risk of HCC. Further studies with more rigorous designs, larger sample sizes, and wider perspectives are needed to validate our findings.

Acknowledgments
We are grateful to all researchers in the enrolled studies.
Author contributions

Min Zhang and Qinjing Wang had full access to all the data in the study and takes responsibility for the integrity of the data and the precision of the data analysis; the concept and design were carried out by Lingling Xu, Min Zhang, and Qinjing Wang; acquisition of data was carried out by Lingling Xu and Qianbo Wu; analysis and interpretation of the data was performed by Lingling Xu and Qianbo Wu; drafting of the manuscript was carried out by Lingling Xu, Qinjing Wang, and Jing Zhang; critical review of the manuscript for important intellectual content was carried out by Min Zhang and Jing Zhang; statistical analysis was carried out by Lingling Xu, Qinjing Wang, and Qianbo Wu; administrative, technical or material support: none; supervision, None;

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Figure 4. Visualization of the sensitivity analysis based on overall populations (A) and Asian populations (B).

|                  | Lower CI Limit | Estimate | Upper CI Limit |
|------------------|----------------|----------|----------------|
| Tanabe−FRA (2008)|                |          |                |
| Tanabe−USA (2008)|                |          |                |
| Qi (2009)        |                |          |                |
| Wang−GX (2009)   |                |          |                |
| Wang−JS (2009)   |                |          |                |
| Li (2010)        |                |          |                |
| Abu Dayyeh (2011)|                |          |                |
| Chen (2011)      |                |          |                |
| Abbas (2012)     |                |          |                |
| Cmet (2012)      |                |          |                |
| Shi (2012)       |                |          |                |
| El−Bendary (2013)|                |          |                |
| Suenaga (2013)   |                |          |                |
| Wu (2013)        |                |          |                |
| Yuan−USA (2013)  |                |          |                |
| Yuan−CHN (2013)  |                |          |                |
| Wei (2016)       |                |          |                |
| El Sergany (2017)|                |          |                |
| Gholizadeh (2017)|                |          |                |
| Asar (2020)      |                |          |                |
| Baghdadi (2020)  |                |          |                |

|                  | Lower CI Limit | Estimate | Upper CI Limit |
|------------------|----------------|----------|----------------|
| Qi (2009)        |                |          |                |
| Wang−GX (2009)   |                |          |                |
| Wang−JS (2009)   |                |          |                |
| Li (2010)        |                |          |                |
| Chen (2011)      |                |          |                |
| Shi (2012)       |                |          |                |
| Suenaga (2013)   |                |          |                |
| Wu (2013)        |                |          |                |
| Yuan−CHN (2013)  |                |          |                |
| Wei (2016)       |                |          |                |
| Gholizadeh (2017)|                |          |                |
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