Research Square

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No Association Between CCL5 rs2280788 Polymorphism and Tuberculosis Susceptibility: A Meta-Analysis

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Abstract

Objective

The association between the CCL5 rs2280788 genetic polymorphism and susceptibility to tuberculosis (TB) has produced inconsistent results across studies. Given the global impact of TB and the potential role of genetic factors in disease risk, clarifying this relationship is important.

Methods

This systematic meta-analysis synthesized data from case-control studies to evaluate the link between the CCL5 rs2280788 polymorphism and TB risk. Comprehensive searches were performed in Scopus, PubMed, Web of Science, Embase, and the China National Knowledge Infrastructure (CNKI). Heterogeneity among studies was assessed, and pooled odds ratios (ORs) along with 95% confidence intervals (CIs) were computed using both fixed-effects and random-effects models.

Results

Nine studies comprising 10 comparisons (2,071 cases and 2,416 controls) were included. The analysis indicated no significant associations between the CCL5 rs2280788 polymorphism and TB risk across multiple genetic models, including allele contrast, heterozygous, homozygous, recessive, dominant, and overdominant models. Substantial heterogeneity was detected under the allele model (I^2 = 97%). However, subgroup analysis by ethnicity revealed an elevated TB risk associated with this polymorphism, specifically within the Arab population, without significant heterogeneity in this subgroup.

Conclusion

These results imply that the CCL5 rs2280788 polymorphism is not a significant risk factor for TB overall, although its potential ethnic-specific effects in Arab populations merit further investigation.

Introduction

Tuberculosis (TB), caused by the bacterium Mycobacterium tuberculosis, remains a critical global health issue and is the most common infectious disease in terms of mortality worldwide, accounting for an estimated 1.60 million deaths in 2023 [1]. Despite the widespread prevalence of Mycobacterium tuberculosis infection, which affects billions of people globally, only approximately 10% of those infected develop active TB during their lifetime, while the majority maintain latent infections that may reactivate under certain conditions [2]. This significant variation in disease manifestation underscores the potential role of host genetic factors, including polymorphisms in immune-related genes, in influencing both susceptibility to TB infection and progression from latent to active disease [3]. Ongoing research aims to

identify specific genetic markers that could inform targeted interventions and improve TB control strategies globally.

The immune response to Mycobacterium tuberculosis, the causative agent of TB involves a highly orchestrated interplay of immune cells, with a primary focus on the recruitment, differentiation, and activation of monocytes and macrophages at infection sites. This cellular mobilization facilitates the formation of granulomas, a key pathological hallmark that encapsulates the bacterium to limit dissemination but paradoxically contributes to the chronic inflammation and tissue damage characteristic of TB [4]. Chemokines are central mediators in this process, directing leukocyte trafficking and activation; notably, C-C motif chemokine ligand 5 (CCL5), also known as RANTES, is integral for its chemotactic properties. Encoded on chromosome 17q11.2-q12, CCL5 promotes T lymphocyte chemotaxis, macrophage polarization, and proliferation, thereby playing a critical role in both acute inflammatory responses and chronic granulomatous inflammation during TB pathogenesis [5, 6]. The CCL5 rs2280788 polymorphism, featuring a cytosine (C) to guanine (G) substitution at the − 28 position within the promoter region, enhances transcriptional efficiency, leading to upregulated CCL5 mRNA synthesis and elevated protein expression in vitro, which amplifies chemokine signaling [7]. As CCL5 directly inhibits the intracellular replication of M. tuberculosis by modulating phagolysosomal fusion and enhancing microbicidal activity in infected macrophages [8], this genetic variant is implicated in altering host susceptibility to TB, potentially conferring resistance through enhanced immune effector functions.

However, case-control studies examining the relationship between the CCL5 rs2280788 polymorphism and TB risk have consistently yielded conflicting outcomes across various research efforts. Specifically, some investigations have demonstrated a protective effect of this polymorphism, potentially reducing susceptibility to TB, as referenced in the literature [9], whereas others have indicated an increased risk, suggesting a detrimental association with increased disease incidence [10]. Additionally, a subset of studies has reported no significant association at all, implying that the polymorphism may not influence TB risk in certain contexts, as noted previously [11]. These discrepancies likely arise from methodological variations, including differences in study design such as case selection criteria and control group matching, the diverse ethnic backgrounds of participant populations that introduce genetic heterogeneity, and variations in sample sizes that affect statistical power and reliability.

To address these inconsistencies and synthesize the existing evidence more robustly, we conducted an updated meta-analysis, incorporating a broader range of recent studies and applying advanced analytical techniques to comprehensively assess the connection between the CCL5 rs2280788 polymorphism and susceptibility to TB. By identifying potential sources of heterogeneity, especially via subgroup analyses based on ethnicity and TB type, we hope to elucidate the genetic determinants of TB susceptibility and advance the understanding of TB pathogenesis. This research has been registered in PROSPERO (CRD420251136876).

Materials and methods

Literature Search

In order to ensure a comprehensive and unbiased collection of relevant literature, a systematic search was conducted across several major academic databases, such as Scopus, PubMed, Web of Science, Embase, and the China National Knowledge Infrastructure (CNKI). The search strategy utilized the keywords “CCL5,” “RANTES,” “rs2280788,” “-28 C > G,” “polymorphism,” and “tuberculosis.” There were no restrictions on the start date of publication, and the search extended to January 30, 2026, to incorporate the most recent studies. This method aimed to identify all available case–control studies examining the association between the CCL5 rs2280788 polymorphism and tuberculosis susceptibility.

Inclusion and exclusion criteria

Studies were eligible for inclusion if they (1) were case–control studies exploring the relationship between the CCL5 rs2280788 polymorphism and TB risk; (2) supplied adequate data for the calculation of odds ratios (ORs) and 95% confidence intervals (CIs); and (3) were published in English or Chinese. The following research articles were excluded from analysis: (a) Absence of control, (b) No available data on genotype frequency, and (c) duplicated case-control groups. Studies where the control group deviated from the Hardy–Weinberg equilibrium (HWE) were also excluded to maintain the integrity of the genetic association analysis, as HWE deviations may suggest genotyping inaccuracies or population stratification.The supporting PRISMA checklist is available as supporting information.

Data Extraction

Two reviewers independently extracted the data to reduce errors and ensure uniformity. From each study, the following details were collected: first author’s name, publication year, country of origin, ethnicity of the study population, type of tuberculosis (pulmonary or extrapulmonary), genotyping method, and genotype distributions in cases and controls. Discrepancies between reviewers were resolved through discussion until consensus was reached.

Quality assessment

The quality of the included literature was evaluated by two independent researchers in strict accordance with the Newcastle-Ottawa Scale (NOS). The selectivity, comparability and exposure of each article were assessed, and all documents included were of high quality.

Statistical Analysis

To evaluate the association between the CCL5 rs2280788 polymorphism and TB risk, pooled ORs and 95% CIs were calculated using both fixed effects and random effects models. Model selection was based on the level of heterogeneity among studies, as evaluated by Cochran’s Q test and the I^2 statistic.

A fixed-effects model was employed when heterogeneity was low ($I^2 < 50\%$), and a random-effects model was used when heterogeneity was moderate to high ($I^2 \geq 50\%$). This strategy ensures that interstudy variability is considered, yielding a more reliable overall effect estimate. Heterogeneity was assessed using Cochran's Q test and the I^2 index, which measures the proportion of total variation across studies attributable to heterogeneity rather than chance. Subgroup analyses by ethnicity and TB type were conducted to investigate potential sources of heterogeneity. Sensitivity analysis was performed by iteratively removing each study to test the robustness of the pooled results. This process is vital for determining whether any single study unduly influenced the overall outcomes. Publication bias was evaluated using Egger's linear regression test, which assesses funnel plot asymmetry. A p value < 0.05 was considered to indicate significant publication bias. This analysis helps determine whether the meta-analysis findings might be distorted by the selective publication of studies with significant results. All the statistical analyses were conducted using MetaGenyo, a web-based tool designed for meta-analyses of genetic association studies. MetaGenyo offers a user-friendly interface and robust statistical methodologies, making it well suited for this analysis. The use of this tool improves the reproducibility and transparency of our results.

Results

Study characteristics

The flow chart of study selection is summarized in Fig. 1. Two studies were excluded from this meta-analysis because of deviation from HWE, which could bias the results [12, 13]. In total, nine eligible studies encompassing 10 comparisons for the CCL5 rs2280788 polymorphism (2,071 cases and 2,416 controls) were included [8–11, 14–18]. The detailed characteristics of each study are provided in Table 1.

Table 1
The main characteristics of the included studies

Study	Country	Ethnicity	Cases	Controls	Type	Genotyping	p_{HWE}
Ben-Selma et al, 2011(pTB)	Tunisia	Arab	168	150	pTB	PCR-RFLP	0.4027
Ben-Selma et al, 2011(epTB)	Tunisia	Arab	168	150	epTB	PCR-RFLP	0.4027
Chu et al,2007	China	East Asian	412	465	TB	PCR-RFLP	0.5772
Mhmoud et al, 2013	Sudan	Arab	191	206	TB	PCR-RFLP	0.8881
Sánchez-Castañón et al,2009	Spain	Caucasian	76	157	pTB	PCR-RFLP	0.5011
Selvaraj et al,2011	India	South Asian	212	213	pTB	PCR-RFLP	0.8897
Sui et al, 2011	China	East Asian	338	390	pTB	PCR-RFLP	0.9663
Xu et al,2015	China	East Asian	300	300	epTB	PCR-RFLP	0.6705
Zhang et al,2011	China	East Asian	239	270	pTB	PCR-RFLP	0.153
Zhang et al, 2014	China	East Asian	78	97	pTB	PCR-RFLP	0.2945
pulmonary tuberculosis (pTB); extra pulmonary tuberculosis (epTB)							

Genotype and Allele Frequency Distribution

Table 2 displays the genotype and allele frequency distributions for the CCL5 rs2280788 polymorphism in both the case and control groups. In the case group, the genotype frequencies were as follows: CC (74.08%), CG (19.02%), and GG (6.90%). In the control group, the frequencies were as follows: CC (73.26%), CG (23.10%), and GG (3.64%). The C allele frequency was 86.58% in cases and 86.73% in controls, while the G allele frequency was 13.42% in cases and 13.27% in controls. These findings indicate a comparable distribution of the CCL5 rs2280788 polymorphism between cases and controls, with only slight variations in genotype and allele frequencies.

Table 2
Genotype distribution of studies included in this meta-analysis

Study	Case			Control		
	CC	CG	GG	CC	CG	GG
Ben-Selma et al, 2011(pTB)	21(12.5%)	67(40.0%)	80(47.5%)	90(60.0%)	50(33.3%)	10(6.7%)
Ben-Selma et al, 2011(epTB)	8(14.5%)	22(40.0%)	25(45.5%)	90(60.0%)	50(33.3%)	10(6.7%)
Chu et al,2007	328(79.6%)	79(19.2%)	5(1.2%)	214(46.0%)	199(42.8%)	52(11.2%)
Mhmoud et al, 2013	183(95.8%)	1(0.5%)	7(3.7%)	202(98.1%)	4(1.9%)	0(0.0%)
Sánchez-Castañón et al,2009	56(73.7%)	14(18.4%)	6(7.9%)	141(90.0%)	16(10.0%)	0(0.0%)
Selvaraj et al,2011	211(99.5%)	1(0.5%)	0(0.0%)	208(98.1%)	4(1.9%)	0(0.0%)
Sui et al, 2011	274(81.0%)	60(17.8%)	4(1.2%)	287(73.5%)	95(24.4%)	8(2.1%)
Xu et al,2015	227(75.7%)	62(20.7%)	11(3.6%)	241(80.4%)	55(18.3%)	4(1.3%)
Zhang et al,2011	164(68.0%)	73(30.3%)	4(1.7%)	200(74.4%)	67(24.9%)	2(0.7%)
Zhang et al, 2014	62(79.5%)	15(19.2%)	1(1.3%)	97(82.9%)	18(15.4%)	2(1.7%)

pulmonary tuberculosis (pTB); extra pulmonary tuberculosis (epTB)

Meta-Analysis Results

The results from all of the comparisons are shown in Fig. 2. Our analysis revealed no significant correlations between the CCL5 rs2280788 polymorphism and TB risk across all the genetic models. However, considerable heterogeneity was noted among studies under the allele model (I^2 = 97%).

Subgroup analyses by ethnicity and TB type were performed to explore potential sources of heterogeneity. In pulmonary TB patients, no associations were found in any comparisons, and significant heterogeneity persisted. Within the Arab population, the meta-analysis indicated an association in all the models except the overdominant model, with little to no heterogeneity. This implies that ethnicity may be a key factor contributing to heterogeneity. Conversely, no significant association was detected in East Asian populations.

Sensitivity analysis was conducted to evaluate the influence of each individual study on the pooled results. The sequential removal of each study from the overall analysis did not significantly alter the

results across any genetic models. Moreover, no substantial evidence of publication bias was found for the G allele versus C allele comparison (Fig. 3, p = 0.3201).

Discussion

Genome-wide association studies (GWASs) have revealed that host genetic factors make a substantial contribution to the development of TB [19, 20]. Susceptibility to TB seems to be complex and varied, and a multitude of host genetic factors are likely involved in the progression to active disease. In the present meta-analysis, we did not find an association between the genetic polymorphism of CCL5 rs2280788 and TB. The rs2280788 variant has a positive transcriptional effect on the RANTES/CCL5 gene [7]. Mycobacterium tuberculosis are taken up by macrophages and then induce a pro-inflammatory response in macrophages by increasing the production of TNF-α, RANTES, and iNOS [21]. MM-CCL23 cells derived from monocytes after stimulation with Mycobacterium tuberculosis are characterized by high production of the chemokines CCL23 and CXCL5, and may serve as a marker for Mycobacterium tuberculosis infection [22].

The frequency of the − 28G allele has been reported to be very low in Caucasians (≤ 2%) and more common in Asians (> 20%) [23, 24]. Research conducted by Kouhpayeh et al. indicated that both PTB patients and healthy controls displayed the GG genotype, suggesting that this variant is not polymorphic within the Iranian population [25]. Among individuals born in the U.S. who were reported to have TB, 36% were classified as Black and 28% as White. Among individuals not born in the U.S. who were diagnosed with TB, 48% were identified as Asian and 32% as Hispanic [26]. The influence of the CCL5 gene on susceptibility to pathogenic infections differs across diverse populations. CCL5 is associated with an increased risk of HIV-1 infection in Caucasian populations, whereas in Asian populations, it seems to have a protective effect [27].

The role of the CCL5 rs2280788 polymorphism in TB risk appears to be inconsistent across various meta-analyses. Lu et al. reported that the rs2280788 variant serves as a protective factor against the development of TB [28]. Conversely, Hu et al. reported a significant association between the rs2280788 polymorphism and an increased risk of TB [29]. However, Alqumber et al. did not find any association between the rs2280788 polymorphism and TB risk [30]. We recognize that the quality of the studies included in a meta-analysis is crucial for the credibility of its findings. It is essential to perform a meta-analysis that excludes results that deviate from the HWE to improve the reliability of conclusions. After recalculating the HWE for the control group in the study, we found that the HWE of Chu et al.'s study was 0.02 rather than 0.2. It is plausible that a mistake took place in their computational procedure.

The significant heterogeneity observed across previous studies warrants deeper investigation regarding its influence on the overarching conclusions drawn from the meta-analysis. Methodological differences, variations in sample characteristics, and diverse outcome measures contributing to this heterogeneity substantially and potentially confound the outcomes of the statistical synthesis. Consequently, the degree to which this observed variability affects the robustness, validity, and generalizability of the

pooled effect estimates merits considerable further discussion to accurately interpret the meta-analytic findings and assess their reliability for guiding practice or informing policy.

It is also important to recognize several limitations of our study. First, our analysis concentrated exclusively on the association between a single genetic polymorphism and TB, without considering potential combinations of polymorphisms. Haplotype-based approaches could furnish a more powerful method for mapping genes related to disease. Second, the population included in this meta-analysis was composed mainly of individuals with PTB. The lack of data from cases of extrapulmonary TB might yield conflicting results, since the genetic associations could vary between the two forms of the disease. Third, the results we provide are based on unadjusted estimates. If individual-level data were available, a more refined analysis stratified by factors such as age and sex could be performed. The absence of such detailed data could influence the accuracy of our findings. Consequently, these limitations might bias our results and should be taken into account when interpreting the conclusions of our study.

Overall, this investigation revealed no evidence for an association between the CCL5 rs2280788 polymorphism and the risk for TB. The role of the CCL5 rs2280788 polymorphism in TB susceptibility remains unresolved, and our current outcomes should be regarded as a preliminary stage in this intricate field of investigation. Future research should consider larger, more varied populations, including those with extrapulmonary TB, and investigate the potential interactions between multiple genetic polymorphisms. In addition, future studies should strive to gather and analyze individual-level data to enable more detailed and stratified analyses. This information will aid in clarifying the role of genetic factors in TB susceptibility and possibly identifying novel targets for intervention and treatment.

Declarations

Ethics approval and consent to participate

Ethical approval was waived because this article does not contain any studies with human or animal subjects performed by any of the authors.

Authors' contributions

L.A. and S.C. wrote the draft. S.C. conceived the study and revised the manuscript. L.A. and S.C. collected the data and performed the statistical analysis. All authors read and approved the final manuscript.

Declaration of competing interest

The authors declare no conflicts of interest.

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Figures

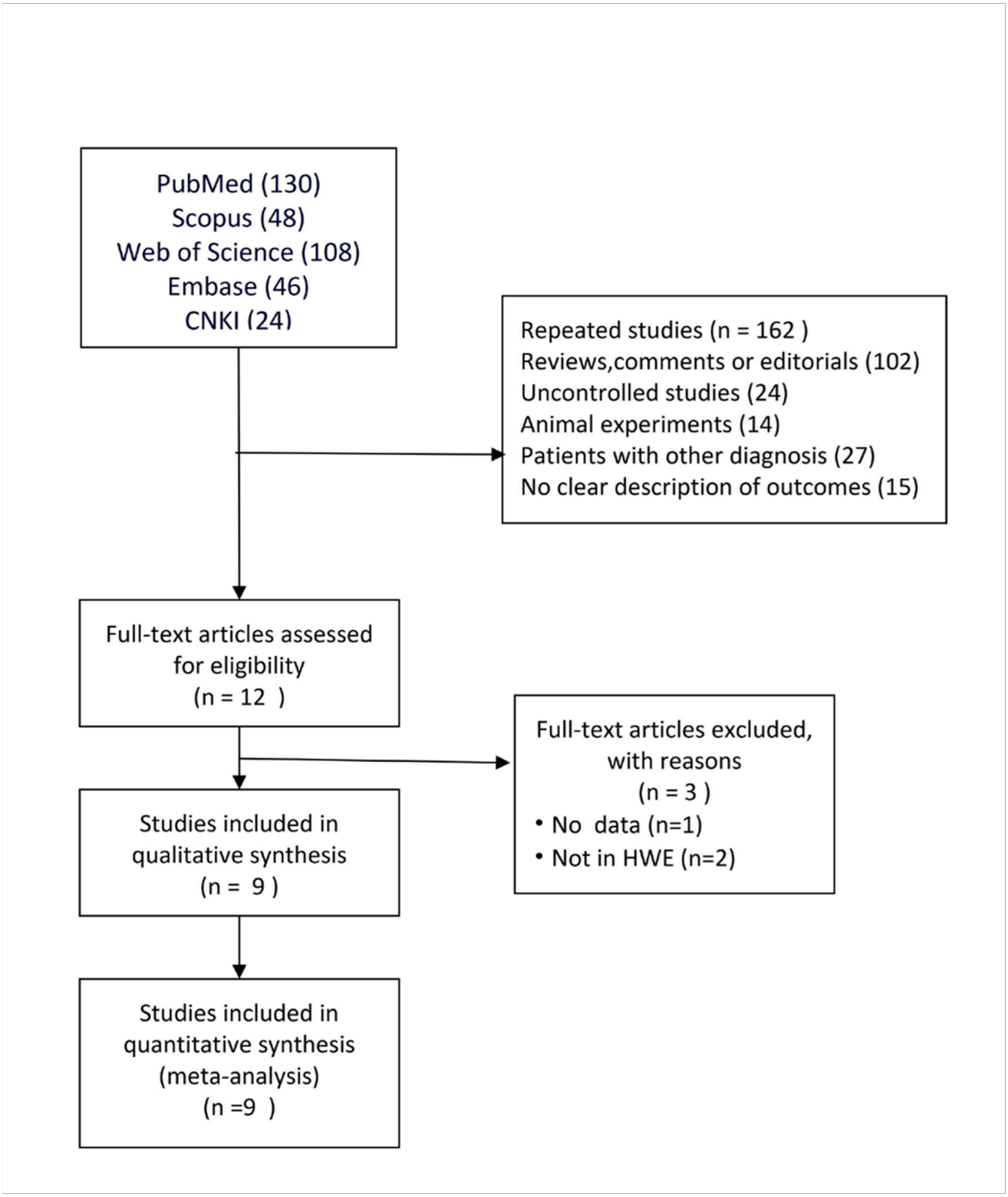


Figure 1

The process for the screening of potentially relevant studies included in the meta-analysis.

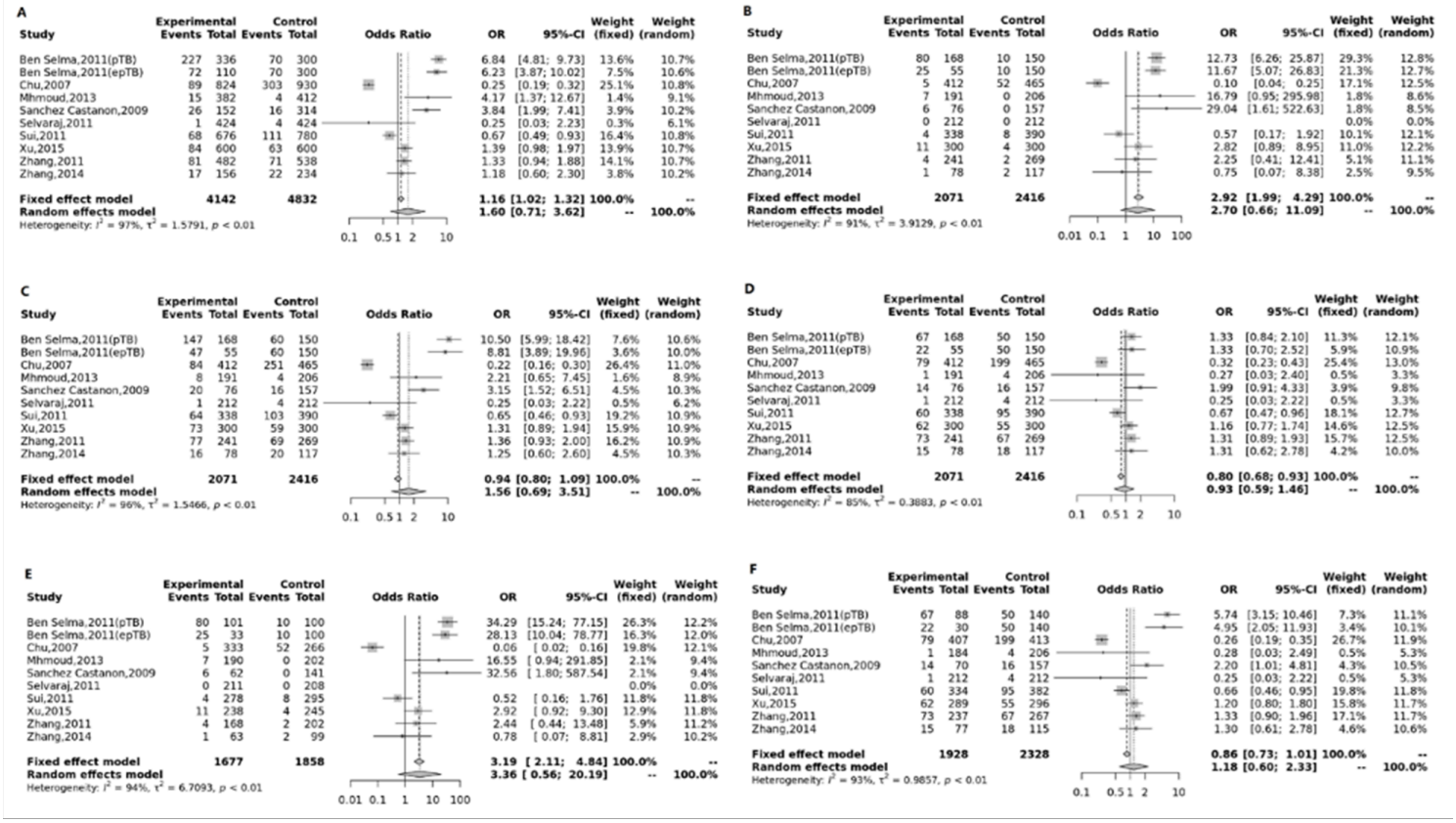


Figure 2

Forest plot analysis for assessing the overall TB risk associated with the CCL5 rs2280788 polymorphism. (A) Allele contrast (G vs. C); (B) recessive model (GG vs. GC+CC); (C) dominant model (GG+GC vs. CC); (D) overdominant model (GC vs. GG+CC); (E) homozygous (GG vs. CC); (F) heterozygous (CG vs. CC).

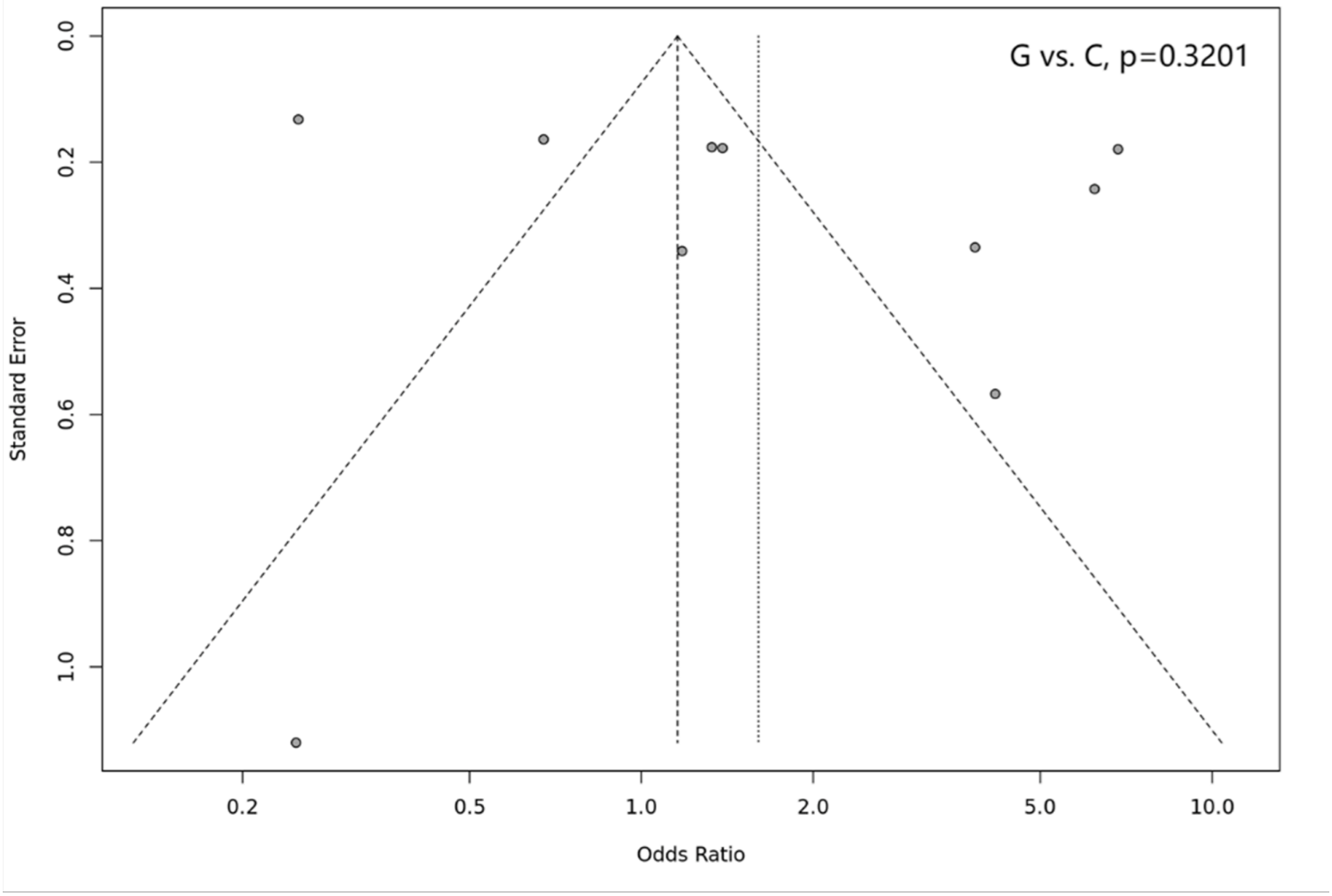


Figure 3

Funnel plot for the publication bias test (G vs. C). Each point represents an individual study for the indicated association.