Contribution of CD14-159C/T polymorphism to tuberculosis susceptibility: a meta-analysis

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BACKGROUND: CD14 plays an important role in recognising the tuberculosis (TB) antigen and initiating immune response. CD14-159C/T polymorphism has been reported to be associated with susceptibility to TB in some, but not all studies.

OBJECTIVE: To comprehensively evaluate the correlation between CD14-159C/T polymorphism and susceptibility to TB.

METHODS: Relevant studies from six English-language databases were searched up to 15 March 2013. Crude odd ratios (ORs) with 95% confidence interval (CIs) were calculated to assess the strength of associations.

RESULTS: Eight eligible studies including 3583 subjects were retained for the meta-analysis. T-allele and TT homozygosis might increase TB risk in the overall analysis (T vs. C: OR 1.30, 95%CI 1.03–1.64, P = 0.03 and TT vs. CC+CT: OR 1.52, 95%CI 1.12–2.08, P = 0.01). Similar correlations were observed among human immunodeficiency virus negative subjects. Strong associations were also found between CD14-159C/T and TB in Asians. Asian individuals with the T-allele and the TT genotype had a significantly increased risk of TB (T vs. C: OR 1.46, 95%CI 1.27–1.68, P = 0.00; TT vs. CC: OR 1.83, 95%CI 1.38–2.44, P = 0.00 and TT vs. CC+CT: OR 1.84, 95%CI 1.55–2.19, P = 0.00). No associations were detected in the pulmonary TB and extra-pulmonary TB groups.

CONCLUSION: CD14-159C/T contributes to TB susceptibility; the T-allele and TT homozygosis are potential risk factors, particularly in Asians.

KEY WORDS: single nucleotide polymorphism; innate immunity; Hardy-Weinberg equilibrium; genome-wide association study

SUMMARY

TUBERCULOSIS (TB) remains an ongoing serious threat to public health, with high prevalence and mortality worldwide. There were an estimated 8.7 million new TB cases and 1.4 million deaths in 2011. Approximately one third of the world’s population is infected with Mycobacterium tuberculosis, the causative agent of TB. However, only about 10% of infected individuals develop active TB during their lifetimes. Apart from the variability of the bacterial strain and environmental factors, the genetic difference of the host may play a crucial role in an individual’s predisposition to TB. In particular, some single nucleotide polymorphisms (SNPs) in innate immunity genes, such as natural resistance-associated macrophage protein 1 (NRAMP1), toll-like receptors (TLRs), tumour necrosis factor-alpha (TNF-α), interferon-gamma and CD14, are reported to be candidate biomarkers associated with susceptibility to TB.

CD14 is located on chromosome 5q31, which encodes a 55-kDa glycoprotein that is an important receptor for pattern recognition in innate immunity system. When exposed to microbial pathogens, CD14 can anchor antigens by recognising bacterial cell wall components such as lipopolysaccharide. CD14 also serves as the accessory receptor to interact with TLRs to activate cellular signalling and induce the release of pro- and anti-inflammatory cytokines, including TNF-α, interleukin 6 and complement components. The cell activation triggered by CD14-TLRs pathways can initiate an inflammatory response to eliminate the pathogen. Both membrane-bound CD14 (mCD14) and soluble-form CD14 (sCD14) are found in cells. mCD14 is expressed mainly on the surface of monocytes, macrophages and polymorphonuclear leukocytes, whereas sCD14 is abundant in serum.

JZ and GL contributed equally to this work.

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produced by CD14 secretion and enzymatic cleavage of mCD14. The sCD14 levels are higher in the serum and bronchoalveolar lavage fluid of active TB patients, and decrease to normal levels after antituberculosis treatment, suggesting the critical role of CD14 in the host-mycobacteria interaction.11,12

Some SNPs, including G-2451C, C-1885T, G-1619A, A-1359C, G-1145A, G-809T, A-550G and C-159T, have been identified upstream of the CD14 gene. The C-159T substitution in the 5'-flanking region of CD14 (rs2569190) is one of the most extensively studied SNPs. CD14-159C/T polymorphism was found to influence the interplay between CD14 promoter and transcription factors. Sp family proteins and the T-allele could effectively enhance transcriptional activity and increase CD14 expression levels. CD14-159C/T has thus been taken as a genetic factor for individual variant susceptibility and investigated in many diseases, such as inflammatory bowel disease, Helicobacter pylori infection-related gastric carcinoma, asthma and TB. The associations of CD14-159C/T with risk of TB have been found in some, but not all, studies. This inconsistency may have been the result of relatively small sample sizes and different ethnicities.7,13,19–24

The meta-analysis is a powerful statistical approach, as it involves the collection of data from independent studies and weighting their results according to the precision of the data; this can help overcome sample size limitations and provide more reliable results. To obtain a more comprehensive and precise assessment of the association between CD14-159C/T polymorphism and TB susceptibility, we performed a meta-analysis of eight eligible studies, including 1770 cases and 1813 controls.

MATERIALS AND METHODS

Literature search strategy
To identify all studies on the association between CD14-159C/T polymorphism and TB risk, we searched the literature from six English-language databases, PubMed, Embase, EBSCO, Web of Knowledge, Springer Link and Science Direct, up to 15 March 2013. Search terms were: ‘Mycobacterium tuberculosis’ or ‘tuberculosis’, and ‘CD14’ combined with ‘polymorphism’ or ‘genotype’ or ‘variant’ or ‘allele’. References of the retrieved and review papers were hand-searched.

Inclusion and exclusion criteria
Studies were considered to be eligible based on the following criteria: 1) full-text articles on CD14-159C/T polymorphism and TB susceptibility, 2) case-control studies, and 3) studies with genotype frequency of cases and controls to estimate odds ratios (ORs) and corresponding 95% confidence intervals (CIs). The main exclusion criteria were: 1) not being case-control studies, 2) control populations that included other complex infectious diseases, and 3) overlapping and republished papers, in which case only the most complete and most recent studies were included.

Data extraction
Two authors (JZ and GL) independently extracted data from all eligible papers to reduce bias in data collection. Extracted data included the first author’s family name, year of publication, participants’ country of origin, ethnicity, type of TB, HIV status, sample size, diagnostic methods used in cases, selection criteria for controls and genotyping methods used in detecting CD14-159C/T polymorphism.

Statistical analysis
For each study, the Hardy-Weinberg equilibrium (HWE) was analysed using the χ² test in the control population. The strength of the association between CD14-159C/T polymorphism and TB risk was assessed using ORs and 95%CIs per the Woolf method. Heterogeneity among studies was detected using Cochran’s χ²-based Q-test. As most of the results of the heterogeneity test were P < 0.1, the random effect model with the DerSimonian and Laird method was used to pool the results and yield wider CIs. The significance of the pooled ORs was determined using the Z-test (P < 0.05 was taken as significant). Subgroup analysis was stratified by HIV status (no HIV infection and related information not available), ethnicity (Asian and Caucasian) and type of TB (pulmonary TB [PTB] and extra-pulmonary TB [EPTB]). One-way sensitivity analyses were conducted to assess the stability of the meta-analysis results. Publication bias was tested using both the Beggs funnel plot and the Egger linear regression test. No publication bias was considered to exist if the funnel plot was symmetric and the Egger test was P > 0.05. All statistical tests were performed using Stata software, version 10.0 (Stata Corporation, College Station, TX, USA).

RESULTS

Study characteristics in the meta-analysis
We identified 134 studies investigating the relationship between CD14 and TB. As shown in Figure 1, six reviews and 31 articles not conducted among humans were excluded. After reviewing the titles and abstracts, 13 of the remaining 97 papers were selected; these explored the correlations between polymorphisms and TB susceptibility. When extracting data from the candidate papers, five papers were excluded due to the absence of CD14 polymorphism. A final eight case-control studies examining the association between CD14-159C/T polymorphism and TB risk met our requirements for further meta-analysis. All of these papers were in English and the last update was on 15 March 2013.

The main characteristics of the studies included are listed in Table 1. Of the eight studies, three were...
performed in Asians,13,21,24 four in Caucasians,19,20,22,23 and one in Caucasian and Indian populations.7 Four papers focused specifically on PTB,13,19,20,22 while another four were on both PTB and EPTB,7,21,23,24 including two that provided detailed information on PTB and EPTB subpopulations.7,23 HIV infection in subjects was a criterion for exclusion in six studies, and the other two did not mention HIV status.19,22 A total of 3583 subjects, including 1770 TB cases and 1813 controls (sample sizes ranging from 204 to 814), were covered by the analysis. Among these studies, most cases were diagnosed according to clinical symptoms, positive sputum smear for acid-fast bacilli, or chest radiographic infiltrates, and confirmed as TB by sputum culture; healthy controls were mainly recruited from unrelated blood donors with no TB history and other immune disorders. The allele and genotype distributions of \(CD14\)-159C/T polymorphism in TB cases and controls are presented in Table 2. Genotype frequencies in all the control groups were in agreement with the HWE.

**Meta-analysis results**

The main results are listed in Table 3. The T-allele was more frequent in TB patients than in controls (T vs. C: OR 1.30, 95%CI 1.03–1.64, \(P = 0.03\)), and individuals with homozygote TT had a 52% increased TB risk compared with C-allele carriers (TT vs. CC+CT: OR 1.52, 95%CI 1.12–2.08, \(P = 0.01\)) in the overall analysis. As people with HIV are more prone to co-infection with *M. tuberculosis* and develop active TB due to a compromised immune system, six of the eight selected studies excluded participants with HIV; correlation trends similar to the above results were also observed in the HIV-negative group.

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**Figure 1** Flow chart of study selection for the meta-analysis. TB = tuberculosis.

**Table 1** Main characteristics of the included studies

<table>
<thead>
<tr>
<th>First author, reference</th>
<th>Year</th>
<th>Country</th>
<th>Ethnicity</th>
<th>TB type</th>
<th>HIV status</th>
<th>Sample size, cases/controls</th>
<th>Selection of controls</th>
<th>Genotype method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xue13</td>
<td>2012</td>
<td>China</td>
<td>Asian</td>
<td>PTB</td>
<td>Neg</td>
<td>311/395</td>
<td>Clinical, radiological and bacteriological investigations</td>
<td>PCR sequence</td>
</tr>
<tr>
<td>Alavi-Naini22</td>
<td>2012</td>
<td>Iran</td>
<td>Caucasian</td>
<td>PTB</td>
<td>NA</td>
<td>120/131</td>
<td>Clinical, sputum smear, radiological and bacteriological tests</td>
<td>ARMS-PCR</td>
</tr>
<tr>
<td>Zhao24</td>
<td>2012</td>
<td>China</td>
<td>Asian</td>
<td>PTB and EPTB</td>
<td>Neg</td>
<td>410/404</td>
<td>Clinical, radiological and bacteriological investigations</td>
<td>PCR-sequence</td>
</tr>
<tr>
<td>Ayaslioglu23</td>
<td>2012</td>
<td>Turkey</td>
<td>Caucasian</td>
<td>PTB and EPTB</td>
<td>Neg</td>
<td>88/16</td>
<td>Clinical, radiological and bacteriological investigations</td>
<td>PCR-RFLP</td>
</tr>
<tr>
<td>Kang21</td>
<td>2009</td>
<td>Korea</td>
<td>Caucasian</td>
<td>PTB</td>
<td>Neg</td>
<td>126/122</td>
<td>Sputum culture-positive</td>
<td>PCR-RFLP</td>
</tr>
<tr>
<td>Rosas-Taraco20</td>
<td>2007</td>
<td>Mexico</td>
<td>Caucasian</td>
<td>PTB</td>
<td>Neg</td>
<td>274/262</td>
<td>Sputum culture-positive</td>
<td>PCR-RFLP</td>
</tr>
<tr>
<td>Druschczyńska15</td>
<td>2005</td>
<td>Poland</td>
<td>Caucasian</td>
<td>PTB</td>
<td>Neg</td>
<td>126/122</td>
<td>Smear of culture-positive</td>
<td>PCR-RFLP</td>
</tr>
<tr>
<td>Pacheco7</td>
<td>2004</td>
<td>USA</td>
<td>Caucasian</td>
<td>PTB</td>
<td>Neg</td>
<td>267/122</td>
<td>Tuberculin-positive healthy controls</td>
<td>PCR-RFLP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>and Indian</td>
<td>EPTB</td>
<td></td>
<td></td>
<td>Tuberculin-positive healthy controls</td>
<td>PCR-RFLP</td>
</tr>
</tbody>
</table>

TB = tuberculosis; HIV = human immunodeficiency virus; PTB = pulmonary TB; EPTB = extra-pulmonary TB; PCR = polymerase chain reaction; Neg = negative; NA = not available; ARMS-PCR = amplification refractory mutation system PCR; RFLP = restriction fragment length polymorphism.
population (T vs. C: OR 1.33, 95%CI 1.02–1.73, \( P = 0.04 \)) and TT vs. CC+CT: OR 1.61, 95%CI 1.16–2.24, \( P = 0.00 \)). Ethnicity may affect TB susceptibility due to different genetic backgrounds and living environments. The contributions of CD14-159C/T between Asians and Caucasians were compared; strong associations were detected among Asians, but not in Caucasians. In Asians, the T-allele had higher susceptibility to TB (T vs. C: OR 1.46, 95%CI 1.27–1.68, \( P = 0.00 \)) and risk of TB increased 1.8-fold in homozygote TT carriers compared with those with the CC genotype or the C allele (TT vs. CC: OR 1.83, 95%CI 1.38–2.44, \( P = 0.00 \)) and TT vs. CT: Begg’s test \( P = 0.00 \) and TT vs. CC: Begg’s test \( P = 0.00 \) and TT vs. CC: Begg’s test \( P = 0.00 \). Ethnicity may affect TB susceptibility due to different genetic backgrounds and living environments. The contributions of CD14-159C/T between Asians and Caucasians were compared; strong associations were detected among Asians, but not in Caucasians. In Asians, the T-allele had higher susceptibility to TB (T vs. C: OR 1.46, 95%CI 1.27–1.68, \( P = 0.00 \)) and risk of TB increased 1.8-fold in homozygote TT carriers compared with those with the CC genotype or the C allele (TT vs. CC: OR 1.83, 95%CI 1.38–2.44, \( P = 0.00 \)) and TT vs. CT: Begg’s test \( P = 0.00 \) and TT vs. CC: Begg’s test \( P = 0.00 \) and TT vs. CC: Begg’s test \( P = 0.00 \).

### Table 2: Genotype distribution of CD14-159C/T in cases and controls

<table>
<thead>
<tr>
<th>First author, reference</th>
<th>Allele</th>
<th>Cases</th>
<th>Controls</th>
<th>Genotype</th>
<th>Cases</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Allele</strong></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Xue et al.13</td>
<td>215 (35)</td>
<td>407 (65)</td>
<td>372 (47)</td>
<td>418 (53)</td>
<td>47 (15)</td>
<td>121 (39)</td>
</tr>
<tr>
<td>Alavi-Naini et al.21</td>
<td>102 (43)</td>
<td>136 (57)</td>
<td>147 (56)</td>
<td>115 (44)</td>
<td>18 (15)</td>
<td>66 (55)</td>
</tr>
<tr>
<td>Zhao et al.14</td>
<td>299 (36)</td>
<td>521 (64)</td>
<td>360 (45)</td>
<td>448 (55)</td>
<td>75 (18)</td>
<td>149 (36)</td>
</tr>
<tr>
<td>Ayaslioglu et al.19</td>
<td>75 (45)</td>
<td>131 (53)</td>
<td>93 (30)</td>
<td>145 (50)</td>
<td>19 (19)</td>
<td>118 (43)</td>
</tr>
<tr>
<td>Kang et al.20</td>
<td>196 (36)</td>
<td>352 (64)</td>
<td>359 (43)</td>
<td>485 (57)</td>
<td>39 (19)</td>
<td>118 (43)</td>
</tr>
<tr>
<td>Rosas-Taraco et al.22</td>
<td>87 (39)</td>
<td>135 (61)</td>
<td>210 (60)</td>
<td>138 (40)</td>
<td>17 (15)</td>
<td>53 (48)</td>
</tr>
<tr>
<td>Druszczynska et al.16</td>
<td>107 (58)</td>
<td>78 (42)</td>
<td>97 (54)</td>
<td>84 (46)</td>
<td>48 (38)</td>
<td>59 (47)</td>
</tr>
<tr>
<td>Pacheco et al.23</td>
<td>303 (57)</td>
<td>231 (43)</td>
<td>116 (53)</td>
<td>108 (48)</td>
<td>92 (34)</td>
<td>119 (45)</td>
</tr>
</tbody>
</table>

HWE = Hardy-Weinberg equilibrium.

These analyses indicate that our results are statistically robust.

### DISCUSSION

With the advances in sequencing technologies, a number of case-control studies, such as genome-wide association studies (GWAS), have been performed to explore genetic determinants of TB. In 2010, Thye et al. launched the first GWAS of PTB (2237 cases and 3122 controls) in Africa, where they identified the locus (rs4331426) on chromosome 18q11.2 in a gene-desert region to be associated with TB risk.39 Using imputing data from the 1000 Genomes Project onto the Ghanaiian genome-wide data set, the rs2057178 on chromosome 11p13 downstream of the WTI gene was subsequenntly demonstrated to be another novel TB susceptibility locus.30 However, in a recent investigation among the Chinese population, neither rs4331426 nor rs2057178 were associated with TB.31Although the GWAS on TB was performed on a large sample, the SNPs identified as influencing TB susceptibility were within the regions of unknown functions; it is therefore difficult to interpret the underlying mechanisms of pathogenesis for TB risk. In the context of a complicated infectious disease such as TB, genetic factors affecting the immune defence against M. tuberculosis infection are more likely to be promising candidates for TB susceptibility.

CD14 plays an important role in recognising TB antigens and activating the innate immune system. The SNP in the -159 C→T promoter of CD14 could influence CD14 expression, which is thought to be associated with TB development. Many studies have therefore examined the correlations, but the results are still inconclusive. A study in Koreans found that the -159T allele had stronger promoter activity and was a risk factor for TB.21 Similar results were observed in Chinese, Iranian and Mexican populations, and where the genotype distributions of CD14-159TT were more frequent in TB patients than in healthy controls.13,20,22,24 However, investigations in Caucasian and Indian, Polish and Turkish populations did not detect obvious correlations between CD14-159CT.
We conducted this meta-analysis including eight eligible studies with 3583 subjects to better evaluate the association between CD14-159C/T polymorphisms and TB susceptibility. Our results indicated that the T-allele had a greater likelihood than the C-allele of increasing the TB risk ($P = 0.025$), and that TT carriers had prominent TB susceptibility compared with individuals with CC and C alleles ($P = 0.008$) in the overall analysis. Among Asians, the T-allele was a more significant risk factor ($P = 0.000$) and TB risk was 1.8-fold higher in TT carriers than in those with the CC genotype or C alleles ($P = 0.000$). Previous studies have provided some clues to understanding these results. The CD14-159T allele could reduce the affinity between the CD14 promoter and transcriptional inhibitor Sp3, enhance transcriptional activity and increase CD14 expression, particularly circulating sCD14 levels. sCD14 might compete with mCD14 to interact with M. tuberculosis and inhibit mCD14-mediated uptake of M. tuberculosis into human microglia. In addition, the T-allele of CD14 could also reduce immunoglobulin E (IgE) levels in the serum, which may regulate adaptive IgE responses. Furthermore, patients with -159TT had lower IFN-γ release in peripheral blood mononuclear cells than -159CC cases. IFN-γ is an important cytokine that can activate macrophages to eliminate M. tuberculosis or suppress its growth. The downregulation of IFN-γ may attenuate host defences against M. tuberculosis. These potential effects may account for higher TB susceptibility among CD14-159T carriers.

When interpreting the results of this meta-analysis, several limitations should be kept in mind. First, we carried out an unadjusted assessment of the association between CD14 and TB alone, as some detailed information, such as body mass index, sex and age, was not available; these potential confounders could therefore not be further evaluated. Second, most genetic models were heterogeneous due to the different populations, environmental exposures, heterogeneous cases and controls included in each study. For example, some studies included only PTB cases, while some had both PTB and EPTB cases. Moreover, the controls were not uniform. Some studies used unrelated blood donors, while some included household contacts who may have been latently infected and progressed to active TB as controls. Third, Africa has a high incidence of TB, but no studies on CD14 and TB in African populations have been reported and this requires further study.

Despite these limitations, the current meta-analysis also has several advantages. All of the studies included met all of the inclusion criteria, and all the control groups accord with the HWE, which ensured population homogeneity. To obtain a more convincing association and overcome heterogeneity, all results were calculated using the random-effect model despite wider 95% CIs. To date, this is the first meta-analysis to study the association between CD14-159C/T and TB risk in a large number of pooled cases and con-
Figure 2  Forest plots of the association between CD14 C-159T polymorphism and risk of tuberculosis using the random-effects model stratified by ethnicity: A) T vs. C; B) TT vs. CC; C) TT vs. CC+CT. The grey squares and horizontal lines show the study-specific ORs and corresponding 95% CIs. The area of each square represents its proportion to the sample size and the weight for this analysis. The subtotal ORs are represented by hollow diamonds. OR = odds ratio; CI = confidence interval.
CONCLUSIONS

Our results suggest that the T-allele and the CD14-159C/T TT genotype are associated with increased TB susceptibility, especially in Asians. These findings may help to identify high-risk populations and create more effective prevention and treatment strategies. Further studies are required to explore the effects of gene-gene and gene-environmental interactions on TB development.

Acknowledgements

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Conflict of interest: none declared.

References

**CONTEXTE :** CD14 joue un rôle important dans la reconnaissance des antigènes de la tuberculose (TB) et dans la mise en route de la réponse immunitaire. On a signalé un polymorphisme CD14-159C/T en association avec une sensibilité à la TB dans certaines études, mais pas dans toutes.

**OBJECTIF :** Évaluer de manière complète la corrélation entre ces deux facteurs.

**MÉTHODES :** On a recherché les études pertinentes dans six bases de données en anglais jusqu’au 15 mars 2013. On a calculé les odds ratios (ORs) bruts et les intervalles de confiance (IC) à 95% pour évaluer la solidité de ces associations.

**RÉSULTATS :** On a pu rassembler pour la méta-analyse huit études éligibles comportant 3583 sujets. L’allèle T et l’homozygose TT pourraient augmenter le risque de TB dans l’analyse globale (T vs. C : OR 1,30 ; IC95% 1,03–1,64 ; P = 0,03 et TT vs. CC+CT : 1,52 ; IC95% 1,12–2,08 ; P = 0,01). On a observé des corrélations similaires chez les patients négatifs pour le virus de l’immunodéficience humaine. On a trouvé également des associations solides entre CD14-159C/T et la TB chez les sujets asiatiques. Chez les individus asiatiques avec un allèle T et le génotype TT le risque de TB augmente de façon significative (T vs. C : OR 1,46 ; IC95% 1,27–1,68 ; P = 0,00 ; TT vs. CC : OR 1,83 ; IC95% 1,38–2,44 ; P = 0,00 et TT vs. CC+CT : OR 1,84 ; IC95% 1,55–2,19 ; P = 0,00). On n’a détecté aucune association dans les deux groupes de TB, pulmonaire et extrapulmonaire.

**CONCLUSION :** CD14-159C/T contribue à la sensibilité à l’égard de la TB ; l’allèle T et l’homozygose TT sont des facteurs potentiels de risque, en particulier chez les sujets asiatiques.