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Mannose-binding Lectin Two Gene Polymorphisms and Tuberculosis Susceptibility in Chinese Population: A Meta-analysis

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Summary: Numerous studies have been done to explore the association between mannose-binding lectin two (MBL2) gene polymorphisms and the risk of tuberculosis (TB). However, the results are inconsistent. We performed a meta-analysis to investigate whether polymorphisms in the MBL2 gene were associated with TB risk. Databases including PubMed, Medline, Chinese Biomedicine Database, China National Knowledge Infrastructure, Wanfang Database, and Weipu Database were searched to find relevant articles published up to 2 October, 2012. Odds ratio (OR) with 95% confidence interval (CI) was used to evaluate the strength of association. All statistical tests were performed by using Revman 5.1 software and STATA 11.0 software. Six case-control studies including 1106 cases and 1190 controls were accepted in the meta-analysis. The results indicated that individuals carrying the MBL2 codon 54 B allele may have an increased risk of TB as compared with AA homozygotes (BB+AB *vs*. AA: OR=1.52, 95% CI: 1.22—1.88), whereas MBL2 +4 P/Q was possibly not associated with TB susceptibility in Chinese population.
Key words: meta-analysis; tuberculosis; mannose-binding lectin; polymorphism

Tuberculosis (TB) is an ancient disease, but not a disease of the past. It remains a serious global health problem. According to a recent World Health Organization (WHO) report on global TB, there were 8.8 million incident cases of TB in 2010, and approximately 1.4 million people died of TB[1]. While a third of the world's population has ever been infected with TB bacterium, only one in ten of them will develop active TB at some point in their lives[2]. It is supposed that genetic susceptibility and environmental factors may play a crucial role in the development of TB.

Mannose-binding lectin (MBL), also known as mannose-binding protein (MBP), is a liver-derived serum protein of the collectin family and plays an important role in the innate immune system[3]. It can recognize mannose and N-acetylglucosamine rich microorganisms, activate the lectin complement pathway and mediate opsonophagocytosis[4]. Multiple studies have shown that low MBL level appears to predispose individuals to infectious disease and influence the outcome of severe infection[3, 5, 6].

MBL two (MBL2) gene maps to chromosome 10q11.2-q21, and the genetic polymorphisms in exon 1 as well as 5′ untranslated region (5'UTR) have been reported to be associated with serum MBL level[3, 6]. In exon 1, three-point mutations: codon 52 A/D (CGT/TGT or Arg/Cys), codon 54 A/B (GGC/GAC or Gly/Asp), and codon 57 A/C (GGA/GAA or Gly/Glu), have been identified[3]. Wild type allele of these polymorphisms is referred as A, while the B (codon 54), C, (codon 57) and D (codon 52) variants are referred as O, which is associated with the serum level, configuration and function of MBL[6]. One mutation in the 5'UTR (+4 P/Q, or C/T) is also found to be associated with the serum MBL level[7]. In the last decade, the association between MBL2 gene mutation and the risk of TB has been extensively studied. The study conducted by Selvaraj *et al*[8] showed that the genotype frequency of MBL2 functional mutant homozygotes was significantly increased in pulmonary TB patients as compared with control subjects in India. Studies investigated by Bellamy[9] and Mombo *et al*[10] also showed that MBL2 gene polymorphisms were associated with TB in Africa. However, Druszczyńska *et al*[11] found no association between MBL2 gene polymorphisms and TB in a Caucasian Polish population. And El Sahly *et al*[12] discovered that the frequency of B allele was lower among controls than TB patients in African-American individuals, but no difference was found between cases and controls of white and Hispanic ethnicity. These different results may be due to different genetic backgrounds and environmental factors. Therefore, we reviewed the publications of the past and performed a meta-analysis to investigate whether MBL2 gene polymorphism (codon 54 A/B or +4 P/Q) was associated with TB susceptibility in Chinese population.

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1 MATERIALS AND METHODS

1.1 Publication Search

We searched the PubMed, Medline, Chinese Biomedicine Database, China National Knowledge Infrastructure, Wanfang Database, and Weipu Database for all articles about the association between MBL2 gene polymorphisms (coden 54 A/B and +4 P/Q) and the risk of TB (last search updated in 2 October, 2012). The following terms were used in searching: (tuberculosis or TB or mycobacteria) and (MBL or mannose-binding lectin or mannose-binding protein or MBP) and (polymorphism or mutation or variant). The search was not restricted on publication date or language. The reference of the retrieved articles and reviews were hand-searched to find relevant publications.

1.2 Inclusion and Exclusion Criteria

The following criteria were used to include published studies: (1) evaluate the association between MBL2 gene polymorphisms (codon 54 A/B or +4 P/Q) and TB risk; (2) use a case-control design; (3) genotype data in both cases and controls should be presented for calculating the odds ratio (OR) and 95% confidence interval (CI); (4) genotype distribution of control group must be consistent with Hardy-Weinberg equilibrium (HWE). Studies were excluded if one of the following existed: (1) not relevant to MBL2 gene polymorphisms or TB in Chinese population; (2) the design based on family or sibling pairs; (3) genotype frequencies or number not reported; (4) reviews or abstracts; and (5) genotype distribution of control group not consistent with HWE. For overlapping studies, only the study with larger sample size was included.

1.3 Data Extraction

All data were extracted independently by two independent investigators, and disagreements were resolved by discussion between the two investigators and a third investigator. The following information was collected from each study: first author, year of publication, study location, sample size, selection of control, diagnosis criteria, genotype numbers in cases and controls, and genotyping method.

1.4 Statistical Analysis

We examined the genotype distribution in the control group to see whether it was consistent with HWE by Pearson's χ^2 test. The heterogeneity between studies was evaluated by a Chi square-test based Q-test. I^2 was also measured to test the heterogeneity between studies. When P value was >0.10, it indicates a lack of heterogeneity between studies. The pooled OR was calculated by the fixed-effects model, otherwise, a random-effects model was used. The strength of the association between TB risk and MBL2 gene polymorphisms (codon 54 A/B and +4 P/Q) was estimated by OR and 95% CI. The statistical significance of OR was analyzed by Z test, and P<0.05 was considered as statistically significant. Five genetic models (dominant model, recessive model, heterozygote comparison, homozygote comparison, and variant allele versus wild type allele) for each polymorphism were estimated. Sensitivity analysis was performed by sequentially excluding individual study to assess the stability of the results. Publication bias was examined by using Begg's test[13] and Egger's test[14], and the significant level was set at 0.05.

All statistical tests were performed by using Revman 5.1 software (Nordic Cochrane Center, Denmark) and STATA 11.0 software (Stata Corporation, College Station, USA)

2 RESULTS

2.1 Study Inclusion and Characteristics

The selection process is shown in fig. 1. Briefly, 122 articles were identified after initial search. Forty-three duplications were removed. After reading the abstracts, 60 articles were excluded for not being relevant to genetic variants and TB risk in Chinese population. By reading the full texts of the remaining 19 articles[15-33], one article was excluded for not relevant to MBL2 gene polymorphisms (codon 54 A/B and +4 P/Q)[15]. Four articles were excluded for review articles[16-19]. One article was excluded for not a case-control study[20]. One article was excluded for without a normal control group[21]. Two articles[22, 23] were excluded for overlapping with the other two studies[28, 29]. Three articles were excluded for no genotype frequencies or number[24-26]. One article was excluded for not consistent with HWE[27]. Finally, 5 studies[28-31, 33] for MBL2 codon 54 A/B and three studies[28, 30, 32] for MBL2 +4 P/Q gene polymorphism were included in the meta-analysis. The detailed characteristics of each study are presented in table 1. The genotype and allele distributions of each study are shown in table 2.

2.2 Quantitative Data Synthesis

2.2.1 MBL2 Codon 54 A/B Gene Polymorphism As shown in fig. 2, five case-control studies[28-31, 33] including 880 cases and 959 controls on the association between MBL2 codon 54 A/B gene polymorphism and the risk of TB were included in the meta-analysis. As shown in table 3, there was no significant heterogeneity under the five genetic models (all P>0.10 for heterogeneity). Therefore, the pooled OR values for these comparisons were estimated in the fixed-effects model. Significant association was found in the dominant model (BB+AB *vs*. AA: OR=1.52, 95% CI: 1.22−1.88), homozygote comparison (BB *vs*. AA: OR=2.10, 95% CI: 1.08−4.09), and B *vs*. A (OR=1.45, 95% CI: 1.20−1.75). The results suggested that B allele carriers may have an increased risk of TB as compared with wild type AA homozygotes in Chinese population. No significant association was found in the recessive model (BB *vs*. AB+AA: OR=1.82, 95% CI: 0.94−3.54) and heterozygote comparison (BB *vs*. AB: OR=1.36, 95% CI: 0.69−2.70). The results indicated that BB homozygotes may have no increased risk of TB as compared with AB heterozygotes in Chinese population.

Sensitivity analysis was performed through sequentially excluding individual studies and statistically similar results were obtained, suggesting stability of the meta-analysis. As shown in table 3 there was no evident publication bias in the recessive model, heterozygote comparison and homozygote comparison (P>0.05), but in dominant model (P<0.05).

2.2.2 MBL2 +4 P/Q Gene Polymorphism As shown in fig. 3, three case-control studies[28, 30, 32] including 598 cases and 669 controls on the association

between MBL2 +4 P/Q gene polymorphism and the risk of TB were included in the meta-analysis. As shown in table 3, there was no significant heterogeneity under the five genetic models (all P>0.10 for heterogeneity). Therefore, the pooled OR values for these comparisons were estimated in the fixed-effects model. No significant association was found in these five genetic models.

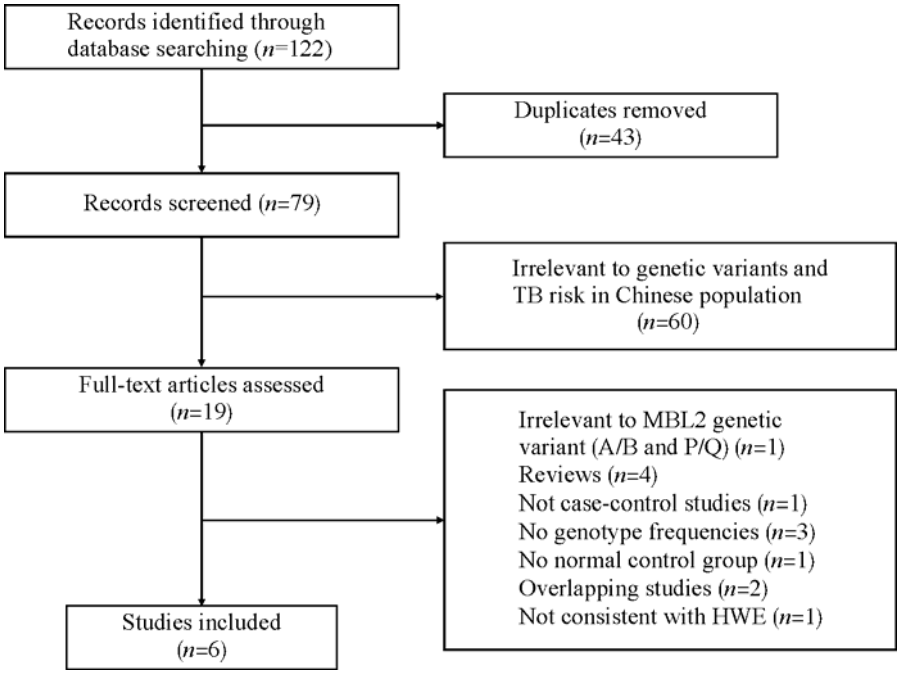

Fig. 1 Flow diagram of included/excluded studies

Table 1 Characteristics of the six studies included in the meta-analysis

First author	Areas	Case/Control	Diagnosis criteria	HIV	HWE
Liu W, 2006[28]	Beijing	141/212	China's national diagnostic criteria	Neg	Yes
Feng FM, 2010[29]	Hebei	182/190	China's TB classification criteria	Neg	Yes
Li Y, 2011[30]	Xinjiang Han	231/226	China's national diagnostic criteria	Neg	Yes
Fang GX, 2011[31]	Guangxi Zhuang	100/100	Bacteriology, radiographic examination and pathologic diagnosis	Neg	Yes
Zhou J, 2011[32]	Xinjiang Uighurs	226/231	Pulmonary tuberculosis diagnosis and treatment guidelines	Neg	Yes
Zhou J, 2012[33]	Xinjiang Uighurs	226/231	Pulmonary tuberculosis diagnosis and treatment guidelines	Neg	Yes

HIV: human immunodeficiency virus; HWE: Hardy-Weinberg equilibrium; Neg: negative

Table 2 Genotype and allele distributions of MBL2 codon 54 A/B and +4 P/Q polymorphisms in TB patients and controls

Studies	A/B (Case/Control)			P/Q (Case/Control)			Genotyping method
	AA	AB	BB	PP	PQ	QQ	
Liu W[28]	103/166	34/42	4/4	118/171	22/39	1/2	PCR-SSP, PCR-SSOP
Feng FM[29]	158/168	23/20	1/2	NA	NA	NA	PCR-SSP
Li Y[30]	171/186	57/37	3/3	189/181	39/41	3/4	PCR-SSP
Fang GX[31]	74/75	25/25	1/0	NA	NA	NA	PCR-RFLP
Zhou J[32]	NA	NA	NA	112/117	90/89	24/25	PCR-SSP
Zhou J[33]	106/146	106/80	14/5	NA	NA	NA	PCR-SSP

PCR: polymerase chain reaction; PCR-SSP: PCR-sequence specific priming; PCR-SSOP: PCR-sequence specific oligonucleotide probe; PCR-RFLP: PCR-restriction fragment length polymorphisms; NA: not available

Sensitivity analysis was performed through sequentially excluding individual studies and statistically similar results were obtained, suggesting stability of the meta-analysis. As shown in table 3, there was no evident publication bias in the five genetic models (P>0.05).

Table 3 Pooled odds ratios for the association of the MBL2 codon 54 A/B and +4 P/Q polymorphisms with risk of TB

	n	Genetic model	Test of association			M	Heterogeneity			Publication bias	
			OR (95% CI)	*Z*	*P*		χ^2	*P*	I^2%	Begg	Egger
A/B	5	BB+AB *vs.* AA	1.52 (1.22−1.88)	3.78	0.0002	F	4.04	0.40	1	0.027	0.003
	5	BB *vs.* AB+AA	1.82 (0.94−3.54)	1.77	0.08	F	2.65	0.62	0	0.462	0.366
	5	BB *vs.* AB	1.36 (0.69−2.70)	0.88	0.38	F	2.49	0.65	0	0.806	0.491
	5	BB *vs.* AA	2.10 (1.08−4.09)	2.18	0.03	F	3.36	0.50	0	0.462	0.315
	5	B *vs.* A	1.45 (1.20−1.75)	3.84	0.0001	F	3.43	0.49	0	0.086	0.002
P/Q	3	QQ+PQ *vs.* PP	0.95 (0.73−1.22)	0.43	0.67	F	0.62	0.73	0	0.296	0.074
	3	QQ *vs.* PQ+PP	0.93 (0.54−1.59)	0.26	0.79	F	0.16	0.92	0	1.000	0.262
	3	QQ *vs.* PQ	0.92 (0.52−1.63)	0.28	0.78	F	0.05	0.98	0	1.000	0.466
	3	QQ *vs.* PP	0.94 (0.54−1.64)	0.21	0.83	F	0.21	0.90	0	1.000	0.246
	3	Q *vs.* P	0.95 (0.77−1.18)	0.47	0.64	F	0.63	0.73	0	0.296	0.034

M: model; F: fixed-effects model

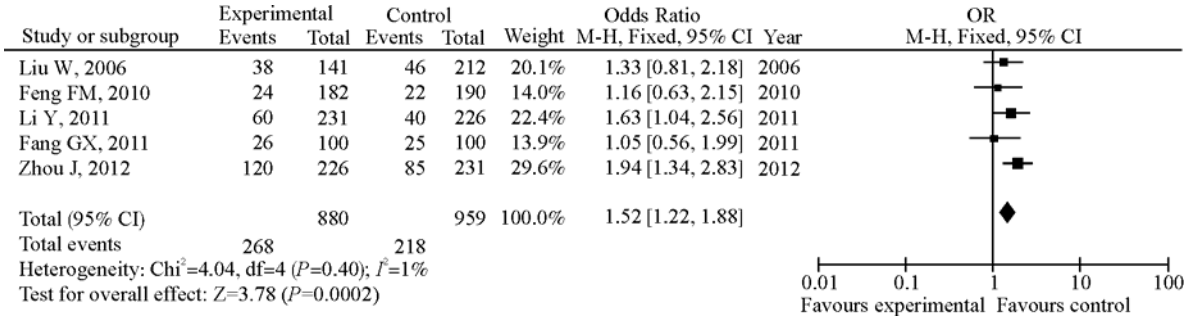

Fig. 2 Meta-analysis with the fixed-effects model for the association between TB risk and MBL2 codon 54 A/B polymorphism (BB+AB *vs.* AA)

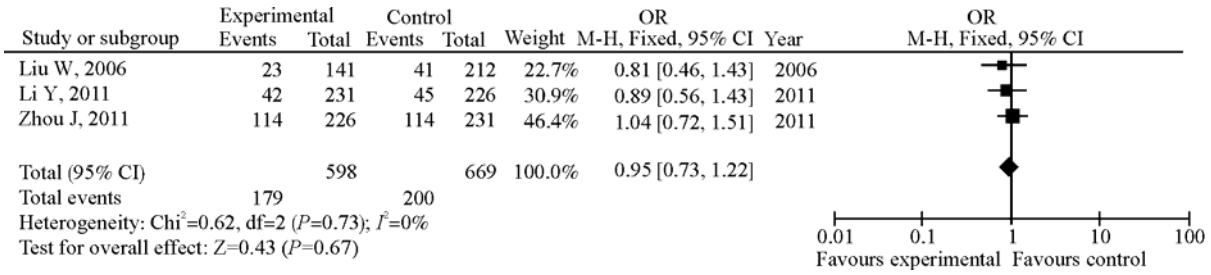

Fig. 3 Meta-analysis with the fixed-effects model for the association between TB risk and MBL2 +4 P/Q polymorphism (QQ+PQ *vs.* PP)

3 DISCUSSION

TB is an infectious disease caused by Mycobacterium tuberculosis. However, a relatively small proportion of people infected with *Mycobacterium tuberculosis* will develop TB. Recently, MBL2 gene polymorphisms have been reported to be associated with the risk of TB. However, the results were inconsistent and inconclusive. Therefore, a meta-analysis was performed to assess the associations between them. Denholm *et al*[34] had done a meta analysis on the association between MBL2 gene polymorphism and the risk of TB. The result suggested that there was no significant association between MBL2 gene polymorphism and pulmonary TB infection. However, the MBL2 gene variants, B (codon 54 A/B), C (codon 57 A/C) and D (codon 52 A/D) are referred to collectively as O while A is the wild-type in their meta-analysis. Studies showed that codon 54 A/B and codon 57 A/C, especially codon 54 A/B, affect the serum level as well as configuration and function of MBL more significantly than codon 52A/D[6]. The difference between the three gene polymorphisms may affect the result of their meta-analysis. In addition, different genetic backgrounds and environmental factors may also influence the results. In China, the MBL2 codon 54 A/B gene polymorphism is more common than the other two (codon 57 A/C and codon 52A/D) variants[28]. And multiple studies have been done on MBL2 +4 P/Q gene polymorphism in China, too. We performed a meta-analysis to explore the association between them and the risk of TB in Chinese population. For the MBL2 codon 54 A/B gene polymorphism, significant association was found in the dominant model (OR=1.52, 95% CI: 1.22—1.88), homozygote comparison (OR=2.10, 95% CI: 1.08—4.09), and B *vs*. A (OR=1.45, 95% CI: 1.20—1.75). The results suggested that individuals with B allel may have an increased risk of TB as compared with wild type AA homozygotes in Chinese population. No significant association was found between MBL2 +4 P/Q gene polymorphism and the risk of TB.

The exon 1 variant B, leading to the replacement of the glycine by aspartic acid in the glycine-rich motif, is

associated with a decreased stability of the collagenous region of MBL. They are rapidly degraded and exist as lower order oligomers, which have a lower binding capacity to mannose and do not activate complement[35]. MBL has been shown to bind with lipoarabinomannan, one component of *Mycobacterium tuberculosis,* and promote opsonophagocytosis[36, 37]. Low serum level of the protein may contribute to the susceptibility to TB. A large study in India showed that a significantly increased genotype frequency of MBL mutant homozygotes was seen in pulmonary TB patients as compared with that in control subjects[8]. And this result was consistent with our meta-analysis.

MBL2 +4 P/Q gene polymorphisms in the 5'UTR have been reported to be in strong linkage disequilibrium with two promoter polymorphisms (-550 H/L, -221 X/Y), the H variant is always linked to the P variant, X only occurs as LXPA, and LY is associated with either P or Q[6, 38]. Four common haplotypes (LXP, LYP, LYQ and HYP) were identified, HYP is associated with medium to high levels of MBL whereas LXP is associated with low levels of MBL[39].

In our meta-analysis, no significant association was found between MBL2 +4 P/Q and the risk of TB. However, since the complexity of the three polymorphisms (-550 H/L, -221 X/Y, +4 P/Q) linkage condition, more studies on haplotypes are needed to assess the association between them and the risk of TB in the future.

The present meta-analysis may have some limitations that require consideration. The studies included in this meta-analysis were limited and the sample size was small. Haplotype analysis was not performed due to inadequacy of data. And TB is a complex disease affected by multiple factors, such as the bacterial virulence, the host genetic background and the environmental factors, which are not considered in the meta-analysis.

In conclusion, this meta-analysis suggests that individuals carrying the MBL2 codon 54 B allele may have an increased risk of TB as compared with AA homozygotes, whereas MBL2 +4 P/Q gene polymorphism is possibly not associated with TB in Chinese population. For better understanding of the association between these polymorphisms and the risk of TB, larger sample size and more studies, especially those investigating haplotypes as well as gene-gene and gene-environment interactions, are required in the future.

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