Review Article

Cytochrome P1B1 4326C/G gene polymorphism correlation to prostate cancer: a meta-analysis

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Abstract: Background: Human cytochrome P1B1 (CYP1B1) gene polymorphism is correlated with the risk of various cancers, but it is unclear whether this polymorphism is correlated with the risk of prostate cancer. Therefore, this study systematically explored the relationship between genetic polymorphism of CYP1B1 4326C/G and the corresponding susceptibility to prostate cancer. Methods: We conducted a comprehensive and detailed search of PubMed, Cochrane Library, CNKI, EMBASE, and WanFang databases to examine the relationship of CYP1B1 4326C/G polymorphism with the susceptibility to prostate cancer, and the strength of this association was assessed using odds ratios (ORs) with 95% confidence intervals (CIs). Results: In total, 16 case-control studies were included in the current study. The results revealed no correlation between CYP1B1 4326C/G polymorphism and the risk of prostate cancer in the overall population. Subgroup analysis of different races determined that Asian people carrying the G allele were associated with an increased risk of prostate cancer, but no statistical correlation was observed. The analysis of subgroups of different control group sources revealed that compared with the population carrying the C allele, the community population carrying the G allele showed a positive correlation with the risk of prostate cancer (homozygous model: OR = 1.34, 95% CI = 1.01-1.77, P = 0.044). Conclusions: This study revealed no significant association between genetic polymorphism of CYP1B1 4326C/G and prostate cancer susceptibility. However, analysis of different control sources showed that the CYP1B1 GG genotype may have increased prostate cancer susceptibility in the general population.

Keywords: Prostate cancer, polymorphism, susceptibility, CYP1B1, meta-analysis

Introduction

Prostate cancer (PC) is the most commonly diagnosed tumor in men worldwide, as well as the most common malignant tumor in the male reproductive system in the Western Hemisphere. Among cancers in male patients in the United States, prostate cancer shows the highest incidence (about 174650 new cases per year), thus posing a serious threat to the health of male patients [1, 2]. The pathogenic factors of prostate cancer have not been fully elucidated. Multitidinous epidemiological studies have demonstrated that the incidence of prostate cancer may be associated with age, race, heredity, eating habits, and lifestyle [3]. Therefore, it is particularly important to clarify the pathogenic mechanism of prostate cancer to provide a reference for early diagnosis and initiate effective treatment for prostate cancer.

Cytochrome P450 (CYP450) is a type of heme-mercaptan protein required for the synthesis and decomposition of diverse molecules and chemicals in cells [4, 5]. CYP450 plays a role in the metabolism of many endogenous substances, including steroids, vitamin D, cholesterol, neurotransmitters, and bile acids [6, 7]. The superfamily of human CYP450 isoymes comprises 57 CYP genes and 58 pseudogenes, which are divided into 18 families and 43 sub-families [8]. CYP1B1, a core member of the CYP450 super-family, is located on chromosome 2 (2p21-22) and contains two introns and three exons [9, 10]. CYP1B1 is usually overexpressed in human malignant tumors and is involved in the hydroxylation of estrogen and activation of other carcinogenic compounds, including aromatic amines, heterocyclic amines, polycyclic aromatic hydrocarbons, and other carcinogens [11, 12]. In most cases,
CYP1B1 can catalyze the production of more toxic intermediate metabolites. Tokizane et al. [13] found that hypomethylation of the promoter/enhancer region can lead to overexpression of the CYP1B1 gene in prostate cancer cells. In animal models, it has been confirmed that CYP1B1-catalyzed metabolites can induce prostate cancer [14]. Based on these findings, it is clear that the CYP1B1 gene plays a vital part in the occurrence, development and prognosis of prostate cancer and is a potential molecular marker for its diagnosis and prognosis.

Numerous studies have reported the association between genetic polymorphism of human CYP1B1 4326C/G and the susceptibility to prostate cancer; however, the results remain controversial [15-28]. Therefore, to elucidate the exact association between genetic polymorphism of CYP1B1 4326C/G and prostate cancer susceptibility, we included all eligible case-control studies to conduct this meta-analysis.

Materials and methods

Data extraction and quality evaluation

According to the previously established inclusion and exclusion criteria, two independent researchers extracted the following data from eligible articles into an Excel table: first author name, country, publication year, race of the study population, frequency of genotypes of cases and controls in each study, source of the control group, gene detection methods, and Hardy-Weinberg equilibrium (HWE) in the control group. In cases where disagreements occurred during data extraction, a consensus was reached through consultation with a third researcher. The included study quality was assessed by two independent authors based on the improved Newcastle-Ottawa scale. The included articles with Newcastle-Ottawa scale scores of more than six stars were deemed to be high-quality studies.

Statistical analysis

The association between genetic polymorphism of CYP1B1 4326C/G and prostate cancer susceptibility were evaluated via ORs and 95% CI. In the current study, the assessment of the between-study heterogeneity was tested using the Labbe plot, Cochrane’s Q test, and I^2. I^2 > 50% or P < 0.1 indicated remarkable heterogeneity among the studies, in which case the random-effect model (DerSimonian-Laird) was used for pooled analysis [29]. Furthermore, I^2 < 50% or P > 0.1 implied no remarkable heterogeneity; hence, the fixed-effect model (Mante-Hazenszel) was used for pooled analysis [30]. By eliminating each included study one-by-one and then pooling the results, the reliability and robustness of the meta-analysis results were tested. The potential publication bias was detected by Begg’s funnel plot and Egger’s test [31, 32]. All statistical analyses were carried out by Stata 15.1 software.

Results

Literature search

Using the PubMed, Cochrane Library, CNKI, EMBASE, and WanFang databases; 256 possible related articles were preliminarily searched. In total, 79 articles were excluded by reading titles and abstracts: 38 articles were not related to gene polymorphisms, 18 articles
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

The description of included studies

A total of 16 case-control studies, which involved 7859 patients and 7428 controls, were included to assess the relationship between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer. Among them, two studies investigated two different populations, so they could be regarded as two independent studies [16, 17]. The sample size of cases in these articles ranged from 50 to 1419 patients, while that of the control group ranged from 50 to 1235 patients. Of all included studies, four studies (including 1368 patients and 1607 controls) were conducted in Asian populations [22, 25, 26, 28], and 10 studies (including 5885 patients and 5134 controls) were conducted in Caucasian populations [15, 16, 18-21, 23, 24, 27]. Two studies, which involved 606 patients and 687 controls, were conducted in mixed populations [17]. With regard to the source of the control group, eight studies used the hospital patients [15, 16, 19-22, 24], and the other eight studies used the general population [17, 18, 23, 25-28]. Genotypes were detected by MassARRAY nucleic acid mass spectrometry, polymerase chain reaction (PCR), TaqMan determination, and PCR-restriction fragment length polymorphism. The gene frequency distribution of all the included studies was in accordance with HWE, and the quality of the studies was greater than 6 points. The basic characteristics of the included articles are shown in Table 1.

Meta-analysis results

The results of this meta-analysis revealed no statistical correlation between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer in the overall population (allele model: OR = 1.05, 95% CI = 0.94-1.16; homozygous model: OR = 1.08, 95% CI = 0.88-1.33; heterozygous model: OR = 1.03, 95% CI = 0.96-1.11; dominant model: OR = 1.06, 95% CI = 0.93-1.21; recessive model: OR = 1.04, 95% CI = 0.89-1.21), as shown in Figure 2 and Table 2. Subgroup analyses of populations of different races revealed that Asian individuals carrying the G allele were correlated with increased risk of prostate cancer, but there was no statistical correlation (allele model: OR = 1.28, 95% CI = 0.83-1.96; homozygous model: OR = 1.66, 95% CI = 0.60-4.59; heterozygous model: OR = 1.17, 95% CI = 0.81-1.70; dominant model: OR = 1.26, 95% CI = 0.81-1.96; recessive model: OR = 1.53, 95% CI = 0.61-3.84), and similar results were found among the Caucasian population, as shown in Table 2 and Figure 3. Subgroup analysis based on different control group sources indicated that, compared with the population carrying the C allele, the community population carrying the G allele showed a positive correlation with prostate cancer risk (homozygous model: OR = 1.34, P = 0.044), whereas the genetic
polymorphism of CYP1B1 4326C/G was not correlated with prostate cancer susceptibility in the hospital patients, as shown in Table 2 and Figure 4.

Heterogeneity and sensitivity analysis

In the heterogeneity analysis of the results of 16 studies, the summary analysis of most genetic models showed moderate heterogeneity (allele model: $I^2 = 72.9\%, P < 0.001$; dominant model: $I^2 = 63.7\%, P < 0.001$; homozygous model: $I^2 = 65.3\%, P < 0.001$; recessive model: $I^2 = 53.6\%, P = 0.006$) (Figure 5), so the results of this meta-analysis were pooled using a random-effect model. By eliminating each included study one-by-one, the effect sizes of the OR values were combined, and the changes in OR values were observed to detect the reliability and robustness of the combined results. Sensitivity analysis revealed no significant change after excluding any included studies, as shown in Figure 6.

Publication bias

The Begg’s funnel plot and Egger’s test were employed for the estimation of the potential publication bias. As shown in Figure 7, there was no significant asymmetry in the shape of the funnel plot. Moreover, the Egger linear regression test showed no publication bias in the genetic comparison models (allele model: $P = 0.628$; homozygous model: $P = 0.873$; heterozygous model: $P = 0.591$; recessive model: $P = 0.934$; dominant model: $P = 0.402$), as shown in Table 2.

Discussion

The occurrence of tumors is an extremely complex multi-stage process. Genetic studies have shown that the occurrence and development of prostate cancer may be the result of a combination of environmental and genetic factors, which determine the susceptibility of a patient, and single-nucleotide polymorphisms (SNPs) show a particularly significant impact [37]. From a theoretical point of view, genetic polymorphisms among a biological population usually contain two or more discontinuous variants, genotype, and allele phenomenon. SNPs are genotypes, or alleles. As the most common type of genetic variation in the human genome, SNPs are caused by a single-nucleotide differences in the DNA sequence [38, 39]. SNPs occur randomly throughout the genome and are more likely to occur in the non-coding regions of DNA [39].

CYP1B1 is a member of the family of CYP450 genes and is a primary enzyme involved in estrogen hydroxylation, which is a key res-
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

Figure 2. Meta-analysis for the association between CYP1B1 4326C/G polymorphism and prostate cancer in the overall populations. A. G vs. C; B. CG vs. CC; C. CG + GG vs. CC; D. GG vs. CC + CG.
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

Table 2. Association between CYP1B1 4326C/G polymorphism and prostate susceptibility

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Subgroup</th>
<th>Studies</th>
<th>Heterogeneity test</th>
<th>Association test</th>
<th>Publication bias</th>
<th>( p )-value</th>
<th>I(^2 )%</th>
<th>OR (95% CI)</th>
<th>( p )-value</th>
<th>Model</th>
<th>Begg</th>
<th>Egger</th>
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<td>1.17 (0.98-1.40)</td>
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<td>58.1</td>
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<td>74.1</td>
<td>1.02 (0.91-1.15)</td>
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<td>84.60%</td>
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<td>Overall</td>
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<td>0.051</td>
<td>40.7%</td>
<td>1.03 (0.96-1.11)</td>
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<td>9</td>
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<td>1.03 (0.93-1.14)</td>
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<td>Asian</td>
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<td>Overall</td>
<td>16</td>
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<td>65.30%</td>
<td>1.08 (0.88-1.33)</td>
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<td>1.34 (1.01-1.77)</td>
<td>0.044</td>
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<td>HP</td>
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<td>0.006</td>
<td>64.60%</td>
<td>0.91 (0.69-1.20)</td>
<td>0.51</td>
<td>R</td>
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<td>0.001</td>
<td>68.80%</td>
<td>1.02 (0.82-1.27)</td>
<td>0.829</td>
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<tr>
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<td>4</td>
<td>0.004</td>
<td>77.10%</td>
<td>1.66 (0.60-4.59)</td>
<td>0.327</td>
<td>R</td>
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<td>0.564</td>
<td>0.00%</td>
<td>1.40 (0.84-2.31)</td>
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<td>GG + GC vs. CC</td>
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<td>&lt; 0.001</td>
<td>63.70%</td>
<td>1.06 (0.93-1.21)</td>
<td>0.354</td>
<td>R</td>
<td>0.137</td>
<td>0.402</td>
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<td>PB</td>
<td>8</td>
<td>&lt; 0.001</td>
<td>74.00%</td>
<td>1.23 (0.98-1.54)</td>
<td>0.077</td>
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<td>0.117</td>
<td>39.30%</td>
<td>0.97 (0.84-1.11)</td>
<td>0.617</td>
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<td>61.40%</td>
<td>1.03 (0.89-1.18)</td>
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<td>1.26 (0.81-1.96)</td>
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<td>1.13 (0.66-1.93)</td>
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<td>GG vs. CC + CG</td>
<td>Overall</td>
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<td>0.006</td>
<td>53.60%</td>
<td>1.04 (0.89-1.21)</td>
<td>0.602</td>
<td>R</td>
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<td>1.16 (0.94-1.45)</td>
<td>0.166</td>
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<td>0.033</td>
<td>54.20%</td>
<td>0.94 (0.76-1.17)</td>
<td>0.599</td>
<td>R</td>
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<td>0.013</td>
<td>57.20%</td>
<td>1.03 (0.87-1.21)</td>
<td>0.724</td>
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<td>0.012</td>
<td>72.80%</td>
<td>1.53 (0.61-3.84)</td>
<td>0.366</td>
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<td>0.913</td>
<td>0.00%</td>
<td>1.01 (0.79-1.29)</td>
<td>0.928</td>
<td>R</td>
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Abbreviations: OR, odds ratio; CI, confidence interval; R, random-effect model; F, fixed-effect model.

Response to hormone carcinogenesis [40, 41]. Therefore, CYP1B1 is usually associated with hormone-mediated tumors, such as breast cancer, ovarian cancer, prostate cancer, and endometrial cancer. CYP1B1 is highly expressed in these cancers and is responsible for hormone metabolism and the formation of toxic metabolites from endogenous and exogenous molecules [40, 41]. Multitudinous studies have examined the relationship between CYP proteins and their SNPs as well as their role in the occurrence and development of prostate cancer. A single study may be insufficient to fully demonstrate this complex genetic correlation due to a relatively small sample size and low statistical efficiency. Therefore, to determine the exact association between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer, we included all eligible case-control studies in the current study.

This study included 16 case-control studies consisting of 15,287 participants to assess the relationship between genetic polymorphism of human CYP1B1 4326C/G and the susceptibility to prostate cancer. The results revealed no correlation between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer. Subgroup analy-
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

![Figure 3](image-url)

**Figure 3.** Meta-analysis for the association between CYP1B1 4326C/G polymorphism and prostate cancer in the different ethnicity populations. A. G vs. C; B. CG vs. CC; C. CG + GG vs. CC; D. GG vs. CC + CG.
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

Figure 4. Meta-analysis for the association between CYP1B1 4326C/G polymorphism and prostate cancer in the different control sources. A. G vs. C; B. CG vs. CC; C. CG + GG vs. CC; D. GG vs. CC + CG.

Association between CYP1B1 4326C/G and susceptibility to prostate cancer

Figure 5. Labbe plot analysis to examine between-study heterogeneity. A. G vs. C; B. CG vs. CC; C. CG + GG vs. CC; D. GG vs. CC + CG.

ses based on different races detected no relationship between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer in Asian and Caucasian men. Upon conducting subgroup analysis of different control group sources, it was found that compared with the population with the C allele, the community population with the G allele showed a positive correlation with the susceptibility to prostate cancer. The effect of the polymorphism on prostate cancer susceptibility is affected by race. It is reported that African-Americans have the highest incidence of prostate cancer, which not only emphasizes the ethnic background of the disease, but also confirms the interaction of genetic and environmental factors in prostate cancer [42]. The interaction of genetic and lifestyle factors, including dietary fat intake, obesity, and sexual factors, can explain these differences to some extent. Undoubtedly, a larger study will provide further insight into the relationship between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer among different ethnic backgrounds, especially among African-Americans.

The present study has certain limitations: (i) most included studies examined Caucasian people, which may limit the generalizability of our research conclusions to some extent; (ii) due to limited data in the literature, this study was unable to conduct subgroup analyses of patients’ age, sex, family history, lifestyle, and other factors; therefore, it was not possible to determine whether these factors are risk...
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

**Figure 6.** Sensitivity analysis on the association between CYP1B1 4326C/G polymorphism and prostate cancer. A. G vs. C; B. CG vs. CC; C. CG + GG vs. CC; D. GG vs. CC + CG.
Figure 7. Begg’s funnel plot analysis to examine publication bias. A. G vs. C; B. CG vs. CC; C. CG + GG vs. CC; D. GG vs. CC + CG.
Association between CYP1B1 4326C/G and susceptibility to prostate cancer

factors for the incidence and prognosis of prostate cancer; and (iii) the included studies did not provide the relevant original data, and the meta-analysis did not elaborate on interactions among genes or between genes and environments.

In conclusion, the results demonstrated that there is no significant correlation between genetic polymorphism of CYP1B1 4326C/G and the susceptibility to prostate cancer; however, based on the analyses of different races and control group sources, CYP1B1 GG genotypes may increase the risk of prostate cancer in Asians as well as the general population.

Disclosure of conflict of interest

None.

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References


Association between CYP1B1 4326C/G and susceptibility to prostate cancer


[40] Singh PB, Ragavan N, Ashton KM, Basu P, Nadeem SM, Nicholson CM, Krishna RK, Matanhelia SS and Martin FL. Quantified gene expression levels for phase I/II metabolizing enzyme and estrogen receptor levels in benign prostate from cohorts designated as high-risk (UK) versus low-risk (India) for adenocarcino-
Association between CYP1B1 4326C/G and susceptibility to prostate cancer
