**Original Article**

**Association of GSTP1 Ile105Val polymorphism with risk of esophageal cancer: a meta-analysis of 21 case-control studies**

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**Abstract:** Background: The association of glutathione s-transferase P1 (GSTP1) Ile105Val polymorphism with risk of esophageal cancer (EC) has been evaluated in many studies; however, the results from these studies are controversial. Thus, further analysis on association between GSTP1 Ile105Val polymorphism and risk of EC is needed among a larger study population. Method: We searched the relevant electronic databases and performed a meta-analysis based on 21 published case-control studies. The Chi-square based I²-statistic test was performed to evaluate possible heterogeneity across the studies. Additionally, random-effects models were used to calculate crude pooled odds ratios (ORs) with 95% confidence intervals (CIs). Results: Overall, this meta-analysis did support a significant association between GSTP1 Ile105Val polymorphism and risk of EC (pooled OR 1.25, 95% CI, 1.05-1.49). Furthermore, the stratified analysis showed that, in comparison to GSTP1 Ile105Val Ile/Ile genotype, the Val/Val genotype was significantly associated with risk of esophageal squamous cell carcinoma (ESCC) (pooled OR 1.45, 95% CI, 1.07-1.96), particularly in the Caucasian population (pooled OR 1.41, 95% CI, 1.01-1.95). Such a significant association was not observed for esophageal adenocarcinoma (EAC) patients or subjects of an Asian ethnicity. Moreover, substantial evidence of heterogeneity among the studies was not observed. Conclusion: The results from this meta-analysis support a significant association between the GSTP1 Ile105Val polymorphism and risk of EC, particularly in a subgroup with ESCC and in the Caucasian population. Further studies with larger sample sizes are needed to validate our findings.

**Keywords:** Esophageal cancer, GSTP1, polymorphism, meta-analysis, cancer risk

**Introduction**

Esophageal cancer (EC) is one of the most common malignancies in the world, with obvious geographical characteristics of its pathogenesis [1]. The development of EC is a multifactorial process. For example, tobacco smoking and alcohol consumption are well-recognized etiological factors for EC [2, 3]. However, not every smoker and/or alcohol consumer develops EC, suggesting that individual susceptibility factors might also be involved in the development of this malignancy.

Evidence indicates that genetic polymorphisms in certain carcinogen-metabolizing genes play an important role in modifying the risk for EC [4]. Sequence variations in these genes can alter the expression, function and activity of the encoded enzymes and may consequently increase or decrease carcinogen activation or detoxification. Among them, the glutathione-s-transferases (GSTs) represent a superfamily of phase II enzymes which catalyze the conjugation reactions between reduced glutathione and reactive intermediates of a variety of endogenous and exogenous electrophilic compounds. Some of these compounds have carcinogenic potential, thereby making them more water-soluble for easy elimination from the body. GSTP1 has a high level of esophageal expression and plays a central role in the inacti-
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The association between GSTP1 polymorphism and risk of EC is unclear, with some studies reporting increased EC risk in subjects carrying GST1/Val105 variant [13-17]; some reporting decreased risk [18]; and others reporting no association [19-28]. A previous meta-analysis investigated the association between EC risk and GSTP1 polymorphism [29]; however, this study also included a limited number of published studies, and new results have since been published. In the current meta-analysis, we gave a more comprehensive overview of risk effect of GSTP1 lle05-Val polymorphism on EC occurrence, including journal articles published up to September 1, 2013.

Materials and methods

Identification of eligible studies

We conducted a literature search through September 1, 2013 using the key words search in the PubMed, Web of Knowledge, MEDLINE, Embase, and Google Scholar electronic databases and search engines. The language of publication was restricted to English. The following search terms were used: glutathione s-transferase p or GST or GSTP1 or glutathione S-transferase P1, and polymorphism or single nucleotide polymorphism or SNP, and esophageal cancer or esophagus or esophageal squamous cell carcinoma or ESCC or esophageal adenocarcinoma or EAC.

Inclusion and exclusion criteria

The following inclusion criteria were used to select studies: (1) case-control study methodology; (2) association of EC with GSTP1 polymorphisms; (3) reported sample size, odds ratios (ORs) and 95% confidence intervals (CIs); and (4) EC cases confirmed using histopathology. The exclusion criteria were as follows: (1) rationale and study design obviously different from our research objectives; (2) not case-control study; (3) malignant tumor cases included in controls; and (4) duplicated studies, reviews, case reports. If duplicate data were presented in more than one study, only the most informative and recent one was included.
Table 1. Characteristics of studies on the association of GSTP1 Ile105Val: Ile/Val vs. Ile/Ile.

<table>
<thead>
<tr>
<th>Study</th>
<th>Study area</th>
<th>Ethnicity</th>
<th>Cases/controls</th>
<th>OR(^a) (95% CI)</th>
<th>OR(^b) (95% CI)</th>
<th>OR(^c) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morita 1998 [18]</td>
<td>Japan</td>
<td>Asian</td>
<td>66/164</td>
<td>0.19 (0.07-0.51)</td>
<td>-</td>
<td>0.18 (0.07-0.48)</td>
</tr>
<tr>
<td>Lin 1998 [19]</td>
<td>China</td>
<td>Asian</td>
<td>42/36</td>
<td>0.83 (0.31-2.22)</td>
<td>0.25 (0.03-2.60)</td>
<td>0.7 (0.3-1.8)</td>
</tr>
<tr>
<td>Van Lieshout 1999 [6, 13]</td>
<td>Caucasian</td>
<td>Caucasian</td>
<td>34/247</td>
<td>3.45 (1.55-7.65)</td>
<td>3.65 (0.88-15.07)</td>
<td>3.47 (1.60-7.57)</td>
</tr>
<tr>
<td>Tan 2000 [20]</td>
<td>China</td>
<td>Asian</td>
<td>150/150</td>
<td>0.89 (0.55-1.44)</td>
<td>1.47 (0.50-4.29)</td>
<td>1.0 (0.81-1.3)</td>
</tr>
<tr>
<td>Lee 2000 [21]</td>
<td>China</td>
<td>Asian</td>
<td>90/270</td>
<td>-</td>
<td>-</td>
<td>0.66 (0.39-1.11)</td>
</tr>
<tr>
<td>Casson 2003 [14]</td>
<td>Canada</td>
<td>Caucasian</td>
<td>45/45</td>
<td>2.5 (1.0-6.3)</td>
<td>0.8 (0.2-3.1)</td>
<td>1.8 (0.84-3.4)</td>
</tr>
<tr>
<td>Wang 2003 [15]</td>
<td>China</td>
<td>Asian</td>
<td>62/38</td>
<td>1.91 (0.82-4.45)</td>
<td>2.48 (0.24-25.44)</td>
<td>5.37 (2.50-11.50)</td>
</tr>
<tr>
<td>Ribeiro Pinto 2003 [16]</td>
<td>Brazilian</td>
<td>Mixed</td>
<td>34/68</td>
<td>-</td>
<td>-</td>
<td>4.09 (1.29-13.00)</td>
</tr>
<tr>
<td>Abbas 2004 [22]</td>
<td>France</td>
<td>Caucasian</td>
<td>70/124</td>
<td>1.12 (0.61-2.07)</td>
<td>1.27 (0.41-3.90)</td>
<td>1.02 (0.55-1.89)</td>
</tr>
<tr>
<td>Jain 2006 [23]</td>
<td>India</td>
<td>Indian</td>
<td>100/137</td>
<td>0.80 (0.47-1.39)</td>
<td>1.29 (0.48-3.45)</td>
<td>0.87 (0.52-1.46)</td>
</tr>
<tr>
<td>Cai 2006 [24]</td>
<td>China</td>
<td>Asian</td>
<td>218/415</td>
<td>0.93 (0.64-1.35)</td>
<td>0.46 (0.13-1.67)</td>
<td>0.88 (0.61-1.27)</td>
</tr>
<tr>
<td>Casson 2006 [25]</td>
<td>Canada</td>
<td>Caucasian</td>
<td>56/95</td>
<td>1.36 (0.65-2.84)</td>
<td>2.22 (0.81-6.06)</td>
<td>1.54 (0.77-3.07)</td>
</tr>
<tr>
<td>Murphy 2007 [26]</td>
<td>Ireland</td>
<td>Caucasian</td>
<td>207/223</td>
<td>0.93 (0.62-1.39)</td>
<td>1.00 (0.53-1.88)</td>
<td>0.94 (0.62-1.41)</td>
</tr>
<tr>
<td>Wideroff 2007 [27]</td>
<td>U.S</td>
<td>Caucasian</td>
<td>67/206</td>
<td>0.71 (0.38-1.32)(^a)</td>
<td>1.73 (0.75-4.02)(^a)</td>
<td>0.87 (0.50-1.50)</td>
</tr>
<tr>
<td>Rossini 2007 [17]</td>
<td>Brazil</td>
<td>Mixed</td>
<td>162/252</td>
<td>1.66 (1.04-2.66)</td>
<td>1.78 (0.89-3.54)</td>
<td>2.12 (1.37-3.29)(^a)</td>
</tr>
<tr>
<td>Zendehdel 2009 [28]</td>
<td>Sweden</td>
<td>Caucasian</td>
<td>175/471</td>
<td>1.21 (0.83-1.75)</td>
<td>1.41 (0.76-2.62)</td>
<td>1.24 (0.87-1.77)</td>
</tr>
<tr>
<td>Liu 2010 [30]</td>
<td>China</td>
<td>Asian</td>
<td>97/97</td>
<td>0.896 (0.478-1.678)(^a)</td>
<td>-</td>
<td>0.825 (0.447-1.52)(^a)</td>
</tr>
<tr>
<td>Moaven 2010 [31]</td>
<td>Iran</td>
<td>Mixed</td>
<td>148/137</td>
<td>0.83 (0.50-1.36)</td>
<td>1.672 (0.678-4.119)(^a)</td>
<td>1.100 (0.688-1.757)(^a)</td>
</tr>
<tr>
<td>Li 2010 [32]</td>
<td>South African</td>
<td>Mixed</td>
<td>245/288</td>
<td>1.01 (0.68-1.48)(^a)</td>
<td>1.21 (0.71-2.07)(^a)</td>
<td>1.00 (0.70-1.43)</td>
</tr>
<tr>
<td>Malik 2010 [4]</td>
<td>Kashmir Valley</td>
<td>Indian</td>
<td>135/195</td>
<td>1.00 (0.62-1.6)(^a)</td>
<td>2.48 (1.03-6.02)(^a)</td>
<td>1.16 (0.74-1.80)</td>
</tr>
<tr>
<td>Matejic 2011 [33]</td>
<td>South African</td>
<td>Mixed</td>
<td>554/902</td>
<td>1.02 (0.79-1.32)</td>
<td>1.03 (0.77-1.38)</td>
<td>1.02 (0.81-1.30)</td>
</tr>
</tbody>
</table>

\(^a\)OR of esophageal cancer associated with GSTP1 Ile105Val: Ile/Val vs. Ile/Ile. \(^b\)OR of esophageal cancer associated with GSTP1 Ile105Val: Val/Val vs. Ile/Ile. \(^c\)OR of esophageal cancer associated with GSTP1 Ile105Val: Ile/Val + Val/Val vs. Ile/Ile. *adjusted for potential confounding variables. OR, odds ratio and CI, confidence interval.

**Data extraction**

Three investigators (Song, Zhou, and Du) reviewed and extracted information independently from selected publications in accordance with the above-mentioned inclusion and exclusion criteria. Any conflicts over study/data inclusion were settled by discussion between the investigators. Following the criteria above, 21 articles [4, 13-28, 30-33] were included in the present analyses. The steps taken towards article selection are shown below (Figure 1). The following data were extracted from included studies: authors of study, study area and period, the number of cases and controls, OR and 95% CIs. If crude and adjusted ORs and 95% CIs were both offered, we extracted the results that were adjusted for the most potential confounding variables. When the ORs were not presented, we calculated unadjusted ORs from the exposure data given in the articles. The details of each study are shown in Table 1.

**Statistical analysis**

Deviations from Hardy--Weinberg equilibrium (HWE) were tested using Fisher’s exact test to evaluate the genetic equilibrium of each study [34]. For all studies, we evaluated the risk of the hetero- and homozygous carriers of the variant Val allele, both together and separately, compared with the wild type Ile allele. Then, we calculated the overall ORs of the polymorphisms.

Tests for heterogeneity were made among studies using the Cochran’s Q and I2 test statistic [35]. For the Cochran’s Q test statistic, a P value < 0.10 was accepted as statistically significant heterogeneity. Random-effects models were used to estimate summary ORs and 95% CIs [36]. To examine potential sources of heterogeneity, we also conducted subgroup analyses by histological types (EAC and ESCC) and by ethnicity (Asian and Caucasian population). Meta regression analysis was also performed to identify sources of heterogeneity according to several variables, such as number of cases, source of controls, covariates adjusted, and publication time.

Sensitivity analyses were conducted to assess the strength of our findings by excluding one study at a time. Begg’s funnel plot and Egger’s regression test [37] were used to evaluate publication bias. In Egger’s test, when P value <
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Results

Literature search and studies' characteristics

Our keyword search identified 71 papers, from which 44 papers (7 reviews and 37 did not meet criteria) were excluded after review of the abstracts. After reading the full texts of the remaining 27 papers, we eliminated an additional 6 papers, including 1 duplicated report, 4 failure to meet inclusion criterion (1) and 1 failure to meet inclusion criterion (3) (Figure 1). In summary, a total of 21 case-control studies evaluating the association between GSTP1 Ile105Val polymorphism and risk of EC were identified, with 2,757 cases and 4,560 controls included [4, 13-28, 30-33]. Among these 21 studies, 9 were performed in Asian populations, 7 in Caucasians, and 5 in mixed ethnicity populations. Controls in 7 studies were population-based and controls in the other 14 studies were hospital-based. The study characteristics are shown in Table 1.

Meta-analysis results

The summary OR for Ile/Val + Val/Val vs. Ile/Ile, Ile/Val vs. Ile/Ile and Val/Val vs. Ile/Ile was 1.14 (95% CI 0.94-1.38), 1.05 (95% CI 0.88-1.24), 1.25 (95% CI 1.05-1.49), respectively. (Figures 2-4).
Subgroup analyses

Subgroup analyses according to histological types are shown in Table 2. Compared with wild-type (Ile/Ile), variant homozygote (Val/Val) of GSTP1 Ile105Val was associated with a significantly increased risk of ESCC (Val/Val vs. Ile/Ile: OR = 1.45, 95% CI = 1.07-1.96; P = 0.134 for heterogeneity test). However, we failed to find any significant association for GSTP1 Ile105Val with risk of EAC in different genetic models. We also performed stratified analysis by ethnicity (Asian and Caucasian group). As shown in Table 2, a significant association between GSTP1 Ile105Val polymorphism and risk of EC among the Caucasian population was found (OR = 1.41, 95% CI = 1.01-1.95; P = 0.602 for heterogeneity test). Such a significant association was not observed in the Asian study population.

Meta-regression analyses

We performed meta-regression analyses regarding the number of cases, ethnicity, adjusted covariates, and the publication time. We found that all of these variables did not appear to be main causes of heterogeneity, with P values equal to 0.542, 0.453, 0.992, and 0.873, respectively.

Sensitivity analyses

We performed sensitivity analyses by removing one study at a time and then estimating summary OR of the remaining studies. We found the results to be stable (data not shown).
Assessment for publication bias

A Begg's funnel plot was generated, showing nearly symmetrical pattern (Figure 5), indicating low possibility of publication bias. Egger's test was also used to quantitatively evaluate publication bias, which confirmed no evidence of bias (p = 0.570).

Discussion

GSTP1 Ile105Val polymorphism has been evaluated as a potential susceptibility factor contributing to the risk of developing various cancers including cancer of the breast, prostate and lung, with the variant Val105 genotype more likely to be associated with increased risk of cancer [38-40]. Using a meta-analytic approach, we consistently found a statistically significant association between variant homozygote (Val/Val) of GSTP1 Ile105Val and increased risk of EC. A previous meta-analysis of 13 published case-control studies found no significant general main effects for GSTP1 Ile105Val polymorphism on EC risk [29]. In the present study, we analyzed data from 2,757 cases and 4,560 controls in 21 studies to have the statistical power to detect differences and provide more precise risk estimates than the previous meta-analysis and individual studies, the majority of which suffered from limited sample size [4, 13-28, 30-33].

Results from our meta-analysis stratified by histological types of EC indicate that individuals carrying variant homozygous Val/Val genotype had significantly higher risk of ESCC than individuals carrying wild-type Ile/Ile genotype. However, such a significant association was not
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Table 2. Subgroup analyses for the association of GSTP1 Ile105Val polymorphism and esophageal cancer

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>No of studies</th>
<th>OR (95% CI)</th>
<th>I² statistics (%)</th>
<th>Test for heterogeneity* (p value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histological subtype</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESCC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ile/Val + Val/Val vs. Ile/Ile</td>
<td>13</td>
<td>1.19 (0.93-1.51)</td>
<td>68.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Ile/Val vs. Ile/Ile</td>
<td>12</td>
<td>1.06 (0.91-1.24)</td>
<td>14.4</td>
<td>0.303</td>
</tr>
<tr>
<td>Val/Val vs. Ile/Ile</td>
<td>11</td>
<td>1.45 (1.07-1.96)</td>
<td>33.1</td>
<td>0.134</td>
</tr>
<tr>
<td>EAC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ile/Val + Val/Val vs. Ile/Ile</td>
<td>9</td>
<td>1.18 (0.90-1.55)</td>
<td>29.9</td>
<td>0.179</td>
</tr>
<tr>
<td>Ile/Val vs. Ile/Ile</td>
<td>9</td>
<td>1.21 (0.88-1.67)</td>
<td>43.9</td>
<td>0.075</td>
</tr>
<tr>
<td>Val/Val vs. Ile/Ile</td>
<td>9</td>
<td>1.29 (0.90-1.84)</td>
<td>0</td>
<td>0.84</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ile/Val + Val/Val vs. Ile/Ile</td>
<td>7</td>
<td>0.88 (0.55-1.42)</td>
<td>82.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Ile/Val vs. Ile/Ile</td>
<td>6</td>
<td>0.83 (0.55-1.27)</td>
<td>59.9</td>
<td>0.029</td>
</tr>
<tr>
<td>Val/Val vs. Ile/Ile</td>
<td>4</td>
<td>0.75 (0.33-1.68)</td>
<td>75.6</td>
<td>0.006</td>
</tr>
<tr>
<td>Caucasian</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ile/Val + Val/Val vs. Ile/Ile</td>
<td>8</td>
<td>1.36 (0.99-1.88)</td>
<td>53.8</td>
<td>0.034</td>
</tr>
<tr>
<td>Ile/Val vs. Ile/Ile</td>
<td>7</td>
<td>1.28 (0.91-1.79)</td>
<td>55.8</td>
<td>0.035</td>
</tr>
<tr>
<td>Val/Val vs. Ile/Ile</td>
<td>7</td>
<td>1.41 (1.01-1.95)</td>
<td>0</td>
<td>0.602</td>
</tr>
</tbody>
</table>

*Test for heterogeneity: random effect modeling was used.

Figure 5. Begg’s funnel plot for publication bias assessment.

observed with EAC. GSTP1 is known to metabolize tobacco-related carcinogens and eliminate the oxidative products of thymidine or uracil propenal [41]. GSTP1 has an effect upon benzo(a)pyrene and its major metabolites [9], which are the major components of cigarette smoke [42]. Moreover, epidemiological studies have shown that GSTP1 polymorphisms seem to be more associated with tobacco-related cancers [19]. ESCC and EAC have significant divergence in etiology while sharing a few etiological factors. Smoking is generally accepted as a risk factor for both ESCC and EAC, but the effect is much stronger in ESCC than in EAC [43]. Thus, the difference in main effects of GSTP1 variant genotype in these two histological types of EC is biologically plausible.

Our stratified analyses by ethnicity revealed a significant association between GSTP1 Ile105Val polymorphism and EC in Caucasians, while the association was not significant in the Asian subgroup. This appears to contradict the above stratification analysis by histological type because ESCC is the major type of EC in the Asian population. However, the result in the Asian subgroup was based on 4 studies in Chinese populations showing a high level of between-study heterogeneity, suggesting that...

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the studies do not estimate the same effect due to different degree of bias. In addition, there are significant differences regarding etiological profiles between high and low incidence areas within China [44]. This difference may also be responsible for variation in EC risk in these studies and the overall non-significance. More large studies are warrant to determine the effect of GSTP1 Ile105Val polymorphism on EC risk in Asians.

Although no significant association between GSTP1 Ile105Val polymorphism and EC risk was observed in dominant model (Ile/Val + Val/Val vs Ile/Ile) or in pair comparison between Ile/Val and Ile/Ile genotype, we cannot exclude that we may fail to detect the effect because of between-study heterogeneity (p < 0.001 for heterogeneity test for both meta-analyses). Although we conducted subgroup analyses by ethnicity and histological type to explore potential sources of heterogeneity, other possible sources such as publication year, case-control matching, and sample size were unable to be examined in our analyses. Uncontrolled confounding factors could be a major source of between-study heterogeneity because most studies used crude ORs and 95% CI. Additionally, the design of some studies was not optimal, some having small sample sizes [14-16, 19], and some including populations with highly heterogeneous ethnic backgrounds or of unclear ethnicity [16, 17, 31-33]. Case-control study design is prone to selection bias, which could be a source of heterogeneity. Furthermore, because GSTP1 polymorphisms might contribute to susceptibility to non-cancer disease, using hospital-based controls may introduce heterogeneity to the meta-analysis. Therefore, more optimal and well-designed studies are required to evaluate the genetic association between GSTP1 Ile105Val polymorphism and EC risk in the future.

Considerable effort was made to test for possible associations between GSTP1 Ile105Val polymorphism and risk of EC, and a significant effect of GSTP1 variant homozygous (Val/Val) genotype on EC risk was found with no significant between-study heterogeneity. However, there are still some limitations to the meta-analysis. First, we cannot control for confounding factors that were not adjusted for in individual studies, such as age, sex, family history of cancer, smoking, alcohol consumption, and other potential risk factors. These factors might modify or even change the direction of observed effect. Second, GSTP1 may interact with environmental factors or interact with other genes in creating susceptibility for EC. However, due to lack of these data in individual data, we were unable to explore potential gene-gene or gene-environment interactions. Thirdly, although we did not find evidence of publication bias, it is possible as we do not have information on unpublished studies.

In conclusion, this meta-analysis demonstrates that GSTP1 Ile105Val significantly modified the risk of EC, especially ESCC, and that the effect of modification was particularly pronounced in the Caucasian population. To confirm our findings, more, well-designed large-scale studies in diverse ethnic populations are warranted.

Abbreviations
GSTP1, glutathione s-transferase P1; EC, esophageal cancer; ORs, odds ratios; CIs, confidence intervals; SNP, single-nucleotide polymorphism; ESCC, esophageal squamous cell carcinoma; EAC, esophageal adenocarcinoma.

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