The Catalase C-262T Gene Polymorphism and Cancer Risk

A Systematic Review and Meta-analysis

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Abstract: Many studies suggest that catalase C-262T gene polymorphism is associated with cancer risk, but with inconsistent results. This study aimed to summarize the overall association between catalase C-262T polymorphism and cancer risk. Literature search was performed in PubMed, Embase, and other databases, studies regarding the association between catalase C-262T polymorphism and cancer risk were identified, and data were retrieved and analyzed by using Review Manager 5.0.24 and STATA 12.0. A total of 18 publications with 22 case–control studies, including 9777 cancer patients and 12,223 controls, met the inclusion criteria. Meta-analysis results showed significant association between catalase C-262 T polymorphism and cancer risk (TT vs CT + CC: odds ratio [OR] = 1.17, 95% confidence interval [CI] = 1.03–1.31, \( P = 0.01 \)). Subgroup analyses stratified by cancer types suggested the catalase C-262T polymorphism was significantly associated with an increased prostate cancer risk (TT vs CT + CC: OR = 1.61, 95% CI = 1.17–2.22, \( P = 0.004 \)); for subgroup analyses stratified by ethnicity, no associations between this polymorphism and Asians or whites were identified (CT vs CC: odds ratio \( OR = 1.11 \), 95% CI = 0.98–1.26, \( P = 0.09 \) for whites; \( OR = 1.19 \), 95% CI = 0.78–1.80, \( P = 0.42 \) for Asians). In summary, the catalase C-262T polymorphism may be a risk factor for cancer with cancer type-specific effects. Further studies should be performed to confirm these findings.

INTRODUCTION

Cancer is one of the leading causes of death and a severe public health problem worldwide.\(^1\) However, the exact mechanism of carcinogenesis has not been fully elucidated yet, growing studies reported that the reactive oxygen species (ROS) contributes to various aspects of malignant tumors, including carcinogenesis, aberrant growth, metastasis, and angiogenesis.\(^2\) ROS-mediated damage to cellular macromolecules is believed to accumulate as a function of age and to lead to deleterious effects associated with carcinogenesis.\(^3\)–\(^4\) The catalase (CAT) is an important enzyme involved in the production and dissipation of ROS,\(^5\) which can neutralize reactive oxygen species by converting H\(_2\)O\(_2\) into H\(_2\)O and O\(_2\). Some investigators reported a significant reduction of CAT activity in prostate cancer and lung cancer, implicating the possible role of CAT in the carcinogenesis.\(^6\)–\(^8\)

In humans, the CAT gene is encoded by the nuclear chromosome 11p13. The rs1001179 polymorphism (C-262T) of this gene is located on the promoter region and influences transcription factors-binding, altering the basal transcription and consequent expression of this enzyme.\(^9\) Compared with the C allele, the variant T allele of the CAT C-262T gene polymorphism has been associated with lower enzyme activity and hence increased levels of ROS.\(^10\) Thus, it is plausible that the endogenous variability associated with this polymorphism may play a role in the host response to oxidative stress, which accordingly influences the development and progression of cancer. Up till now, a number of case–control studies have been performed to identify the association of CAT C-262T polymorphism with cancer risk; however, the results remain inconsistent and inconclusive.\(^11\)–\(^12\) Since meta-analysis is a powerful tool for analyzing cumulative data from studies in which individual sample sizes are small and the statistical power is low,\(^13\) a meta-analysis based on current available independent studies was performed, which may provide the evidence for the overall association of CAT C-262T polymorphism with cancer susceptibility.

MATERIAL AND METHODS

Identification and Eligibility of Relevant Studies

The electronic databases PubMed, Embase, Web of Science, and Cochrane Library were searched using the Mesh terms: “catalase or CAT,” “polymorphism or variant or
mutation,’’ and ‘‘cancer or tumor or carcinoma or malignancy’’
(Last search update October 15, 2014). Additional eligible
studies on this topic were identified by a hand search of
references of retrieved articles. If studies used partly overlapped
subjects, the study with the largest sample size was selected.
The languages were limited to English. Only the studies with
complete data on comparison of frequency of the CAT C-262T
polymorphism between controls and patients with cancer were
selected, and the distribution of genotypes in the control group
should be consistent with Hardy-Weinberg equilibrium (HWE).
Animal studies, case reports, review articles, abstracts,
editorials, reports with incomplete data, and studies based on
pedigree data were excluded. Institutional review board
approval was not required for this retrospective meta-analysis.

Data Extraction
Two investigators extracted all data independently accord-
ing to the inclusion and exclusion criteria, and reached a
consensus on all items. In case of disagreement, a third author
assessed these articles and made the final decision. For one
publication with several cancer types, each one was treated as a
single study. From each study, the following information was
extracted: first author’s name, year of publication, country
where the study was conducted, ethnicity of the study popu-
lation, genotyping methods, total number of cancer cases and
controls, and genotype distributions of cases and controls.

Statistical Analysis
Review Manager Software 5.0.24 (Cochrane Collabor-
ation, Oxford, UK) and STATA 12.0 (Stata Corp., College
Station, TX) software were used to perform all statistical
analyses. The following genotype contrasts were evaluated:
allelic contrast (T vs C), additive genetic model (TT vs CC),
dominant genetic model (CT + TT vs CC), and recessive
genetic model (TT vs CT + CC). In addition, we conducted
subgroup analyses by cancer types and ethnicity. The associa-
tion between CAT C-262T polymorphism and cancer risk was
measured by the odds ratio (OR) with 95% confidence interval
(95% CI). The significance of the pooled OR was determined by
the Z test and \( P < 0.05 \) was considered as statistically signifi-
cant. The heterogeneity across studies was calculated using the
chi-squared-based Q-test and the inconsistency index \( I^2 \) with
95% CI. When a significant Q-test (\( P < 0.1 \) or \( I^2 > 50\% \))
indicated heterogeneity among studies, the random-effects
model was used to calculate the pooled OR; otherwise, the
fixed-effects model was used.

Funnel plot asymmetry and Harbord test were used to
determine the potential publication bias.\(^{14}\) Sensitivity analysis
was performed by sequentially excluding individual studies and
recalculating the results.\(^{15}\) HWE was tested by Pearson \( \chi^2 \) test
with significance set at \( P < 0.05 \).

RESULTS
Characteristics of Eligible Studies
A total of 18 publications with 22 case–control studies,
including 9777 cancer patients and 12,223 controls, met our
inclusion criteria and were included in this meta-analysis.\(^{16-33}\)
The study selection process was shown in Figure 1.
### TABLE 1. Clinical Summary of Included Studies and Genotype Distribution

| Author (Ref)       | Year | Country     | Ethnicity                  | Cancer Type     | Cancer Case | Control | Genotyping Method | Cancer Case | Control | HWE |
|--------------------|------|-------------|----------------------------|-----------------|-------------|---------|-------------------|-------------|---------|-----|
| Ahn et al\(^{15}\) | 2005 | USA         | White                      | Breast cancer   | 1008        | 1056    | HM L/I MS         | 614         | 349     | 45  |
| Castaldo et al\(^{16}\) | 2014 | Portugal    | White                      | Cervical cancer | 119         | 106     | PCR               | 58          | 25      | 36  |
| Cebrian et al\(^{17}\) | 2006 | UK          | White                      | Breast cancer   | 2171        | 2262    | Taqman            | 1351        | 707     | 113 |
| Choi et al\(^{18}\) | 2007 | USA         | White/African American     | Prostate cancer | 508         | 1403    | HM L/I MS         | 317         | 165     | 26  |
| Ezzikouri et al\(^{19}\) | 2010 | France      | Mixed                      | Hepatocellular  | 96          | 222     | PCR               | 76          | 14      | 6   |
| Farawela et al\(^{20}\) | 2012 | Egypt       | White                      | NHL             | 100         | 100     | PCR-RFLP          | 26          | 49      | 25  |
| Funke et al\(^{21}\) | 2009 | Germany     | White                      | Colorectal Cancer | 632       | 605     | Pyrosequencing technology | 374 | 235 | 23 |
| He et al (22)\(^{a}\) | 2010 | USA         | White                      | BCC             | 270         | 796     | Taqman            | 161         | 97      | 12  |
| He et al (22)\(^{b}\) | 2010 | USA         | White                      | Melanoma        | 211         | 796     | Taqman            | 129         | 75      | 7   |
| He et al (22)\(^{c}\) | 2010 | USA         | White                      | SCC             | 266         | 796     | Taqman            | 160         | 96      | 10  |
| Ho et al\(^{23}\)  | 2006 | China       | Asian                      | Lung cancer     | 230         | 240     | PCR-RFLP          | 209         | 19      | 2   |
| Karunasinghe et al\(^{24}\) | 2012 | New Zealand | Mixed                      | Prostate cancer | 258         | 567     | Taqman            | 144         | 99      | 15  |
| Li et al\(^{25}\)  | 2009 | USA         | White                      | Breast cancer   | 497         | 493     | Taqman            | 295         | 176     | 26  |
| Lightfoot et al\(^{26}\) | 2006 | USA/UK      | White/African American     | NHL             | 909         | 1437    | Taqman            | 554         | 298     | 57  |
| Quick et al\(^{27}\) | 2008 | USA         | Mixed                      | Breast cancer   | 616         | 1082    | HM L/I MS         | 379         | 210     | 27  |
| Rajaraman et al (28)\(^{a}\) | 2008 | USA         | Mixed                      | Acoustic neuroma | 63         | 438     | Taqman            | 43          | 17      | 3   |
| Rajaraman et al (28)\(^{b}\) | 2008 | USA         | Mixed                      | Gliona          | 330         | 438     | Taqman            | 195         | 124     | 11  |
| Rajaraman et al (28)\(^{c}\) | 2008 | USA         | Mixed                      | Meningioma      | 120         | 438     | Taqman            | 73          | 39      | 8   |
| Saadat et al\(^{29}\) | 2014 | Iran        | White                      | Breast cancer   | 407         | 395     | PCR               | 261         | 129     | 17  |
| Tang et al\(^{30}\) | 2010 | USA         | Mixed                      | Pancreatic cancer | 551       | 602     | Taqman            | 349         | 174     | 28  |
| Tefik et al\(^{31}\) | 2013 | Turkey      | White                      | Prostate cancer | 155         | 195     | PCR               | 58          | 64      | 33  |
| Tsai et al\(^{32}\) | 2012 | China       | Asian                      | Breast cancer   | 260         | 224     | PCR               | 225         | 35      | 0   |

BCC = basal cell carcinoma, HM L/I MS = high-throughput, matrix-assisted, laser desorption/ionization time-of-flight mass spectrometry, HWE = Hardy-Weinberg equilibrium, NHL = non-Hodgkin lymphoma, PCR = polymerase chain reaction, PCR-RFLP = polymerase chain reaction-restriction fragment length polymorphism, SCC = squamous cell carcinoma.
The included studies' clinical characteristics and genotype distributions were summarized in Table 1.15–32 These studies were published from 2005 to 2014. In all 22 studies, there were 11 studies of whites,16–18,21,23,26,30,32 2 studies of Asians,24,33 2 studies of whites and African-Americans,19,27 and 7 of mixed ethnicity.20,25,28,29,31 The 22 studies included 6 studies on breast cancer,16,18,26,28,30,33 3 studies on prostate cancer,19,25,32 3 studies on brain tumors (including acoustic neuroma, glioma, and meningioma),29 3 studies on skin cancer (including basal cell carcinoma, squamous cell carcinoma, and melanoma),23 2 studies on non-Hodgkin lymphoma (NHL),21,27 1 study on hepatocellular carcinoma,26 1 study on colorectal cancer,22 1 study on lung cancer,24 1 study on cervical cancer,17 and 1 study on pancreatic cancer.23 The distributions of the genotypes in the control groups in all studies were in HWE. Genotyping methods used in the eligible studies included polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP),21,24 general PCR,17,20,30,32,33 Taqman,18,23,25–27,29,31 high-throughput, matrix-assisted, laser desorption/ionization time-of-flight mass spectrometry (HML/I MS),16,19,28 and pyrosequencing technology,23 as listed in Table 1.

### Pooled Analysis

Meta-analysis results showed significant association between CAT C-262T polymorphism and the risk of cancer in additive and recessive genetic models (TT vs CC: OR = 1.19, 95% CI = 1.01–1.40, P = 0.04; TT vs CT + CC: OR = 1.17, 95% CI = 1.03–1.31, P = 0.01, Figure 2), but no evidence of association in other genetic models (T vs C: OR = 1.07, 95% CI = 1.00–1.15, P = 0.06; CT + TT vs CC: OR = 1.05, 95% CI = 0.97–1.13, P = 0.20). These results suggest that individuals who carry the TT homozygote may have an increased risk of cancer compared with the C allele carriers (CC or CT + CC).

### Subgroup Analysis

We then performed the subgroup analyses stratified by cancer types and ethnicity. The pooled ORs for additive model and recessive model comparison suggested the C-262T polymorphism was significantly associated with an increased prostate cancer risk (TT vs CC: OR = 1.81, 95% CI = 1.07–3.04, P = 0.03; TT vs CT + CC: OR = 1.61, 95% CI = 1.17–2.22, P = 0.004, Figure 3), whereas for breast cancer, NHL, such association was not significant in any genetic model (all P > 0.05). For subgroup analyses stratified by ethnicity, no associations between this polymorphism and Asian or white populations were identified (CT + TT vs CC: OR = 1.11, 95% CI = 0.98–1.26, P = 0.09 for white; CT + TT vs CC: OR = 1.19, 95% CI = 0.78–1.80, P = 0.42 for Asian) (Figure 4). These results suggest that the effects of CAT C-262T polymorphism on cancer susceptibility are ethnic and cancer subtype specific. Meanwhile, as the genotyping method may influence the results, we also performed a subgroup analysis according to genotyping method used in studies. Significant associations were only found in additive and recessive genetic models in studies using PCR (TT vs CC: OR = 1.94, 95% CI = 1.04–3.62, P = 0.04; TT vs CT + CC: OR = 1.83, 95% CI = 1.06–3.16, P = 0.03), whereas for studies using Taqman or HML/I MS, no such associations were observed. The main results of the meta-analysis were summarized in Table 2.

![FIGURE 2](https://example.com/figure2.png)

**FIGURE 2.** Forest plot for meta-analysis of catalase C-262T polymorphism and cancer risk (TT vs CT + CC). The size of the square is proportional to the weight of each study; horizontal lines represent the 95% confidence interval.
Publication Bias and Sensitivity Analysis

The publication bias of the studies was assessed by visual funnel plots and Harbord test. The funnel plots for CT + TT vs CC were shown in Figure 5 and Harbord test did not indicate asymmetry of the plot (P = 0.16), indicating a lack of publication bias. To evaluate the stability of our findings, sensitivity analysis was performed by sequentially excluding each study. Statistically similar results were obtained after sequentially excluding each study, suggesting the stability of the results (Figure 6).

DISCUSSION

CAT is a heme enzyme that plays a predominant role in controlling H2O2 concentration by converting H2O2 into H2O and O2, and protects cells from deleterious effects of oxidative stress; studies suggest that CAT C-262T gene polymorphism influences transcription factors binding thus altering the basal transcription and consequent expression of this enzyme and hence the oxidative status of cells and its microenvironment. Therefore, this polymorphism is believed to play a role in the pathogenesis of cancer. As a number of studies have been published to investigate the potential association between CAT C-262T polymorphism and cancer risk with considerably variable results, we performed this meta-analysis to summarize their overall association.

The present meta-analysis included 18 publications with 22 case–control studies, comparisons of dominant/recessive/additive models and allele frequency were all estimated. In addition, the consistency of genetic effects across different ethnicities and

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**FIGURE 4.** Forest plot for meta-analysis of catalase C-262T polymorphism and prostate cancer risk (CT + TT vs CC). The size of the square is proportional to the weight of each study; horizontal lines represent the 95% confidence interval. (A) White; (B) Asian.
cancer types was investigated. Based on current available evi-
dences, the individuals who carry the TT homozygote have 17% 
increased risk of cancer compared with the C allele carriers, 
indicating that the CAT C-262T gene polymorphism may be a 
risk factor for cancer. Sensitivity analysis was performed to 
evaluate whether a single study influenced the overall results, 
and showed the stability and reliability of our statistical results.15

Although growing studies have suggested population-
specific genetic differences in cancer pathogenesis, no associ-
ation between CAT C-262T polymorphism and cancer risk was 
observed in our subgroup analysis stratified by ethnicity, which 
could be explained by that for certain population, cancer 
susceptibility may be associated with different genes, different 
loci within the same gene, and/or different polymorphisms at 
the same locus.35,36 In addition, 7 studies in our meta-analysis 
included population with mixed ethnicities20,25,28,29,31 and we 
did not find studies performed in Latinos, so it is hard to make 
a definite conclusion about the population-specific genetic

**TABLE 2. Meta-analysis of Catalase C-262 T Polymorphism and Cancer Association**

| Genetic Contrasts | Group and Subgroups | Studies (n) | Q test P Value | $I^2$ (95% CI) | Model Selected | OR (95% CI) | P |
|-------------------|---------------------|------------|---------------|----------------|----------------|-------------|---|
| **T vs C**         | Overall             | 22         | 0.004         | 50% (18%–69%) | Random        | 1.07 (1.00–1.15) | 0.06 |
|                    | White               | 11         | <0.001        | 69% (43%–84%) | Random        | 1.12 (1.00–1.27) | 0.05 |
|                    | Asian               | 2          | 0.48          | 0% (not applicable) | Fixed    | 1.22 (0.82–1.83) | 0.32 |
|                    | Breast cancer       | 6          | 0.17          | 36% (0%–74%)   | Fixed        | 1.02 (0.95–1.09) | 0.66 |
|                    | Prostate cancer     | 3          | 0.007         | 80% (37%–94%) | Random        | 1.32 (0.97–1.81) | 0.08 |
|                    | NHL                 | 2          | 0.5           | 0% (not applicable) | Fixed    | 1.04 (0.91–1.18) | 0.61 |
|                    | Genotyping by Taqman| 11         | 0.36          | 8% (0%–64%)    | Fixed        | 1.00 (0.95–1.06) | 0.89 |
|                    | Genotyping by PCR   | 7          | 0.002         | 72% (39%–87%)  | Random        | 1.30 (0.98–1.73) | 0.07 |
|                    | Genotyping by HM L/I MS | 3        | 0.86         | 0% (0%–90%)    | Fixed        | 1.10 (1.00–1.20) | 0.06 |
| **TT vs CC**        | Overall             | 22         | 0.08          | 32% (0%–60%)   | Random        | 1.19 (1.01–1.40) | 0.04 |
|                    | White               | 11         | 0.02          | 52% (5%–76%)   | Random        | 1.21 (0.94–1.56) | 0.14 |
|                    | Asian               | 2          | —             | 0% (not applicable) | Fixed    | 5.19 (0.25–108.77) | 0.29 |
|                    | Breast cancer       | 6          | 0.69          | 0% (0%–79%)    | Fixed        | 1.04 (0.86–1.25) | 0.71 |
|                    | Prostate cancer     | 3          | 0.10          | 56% (0%–87%)   | Random        | 1.81 (1.07–3.04) | 0.03 |
|                    | NHL                 | 2          | 0.76          | 0% (not applicable) | Fixed    | 1.21 (0.91–1.77) | 0.16 |
|                    | Genotyping by Taqman| 11         | 0.87          | 0% (0%–60%)    | Fixed        | 1.07 (0.91–1.25) | 0.43 |
|                    | Genotyping by PCR   | 7          | 0.01          | 66% (18%–86%)  | Random        | 1.94 (1.04–3.62) | 0.04 |
|                    | Genotyping by HM L/I MS | 3        | 0.94          | 0% (0%–90%)    | Fixed        | 1.19 (0.91–1.56) | 0.20 |
| **CT + TT vs CC**   | Overall             | 22         | 0.05          | 37% (0%–62%)   | Fixed        | 1.03 (0.98–1.09) | 0.29 |
|                    | White               | 11         | 0.01          | 56% (14%–78%)  | Random        | 1.11 (0.98–1.26) | 0.09 |
|                    | Asian               | 2          | 0.34          | 0% (not applicable) | Fixed    | 1.19 (0.78–1.80) | 0.42 |
|                    | Breast cancer       | 6          | 0.13          | 41% (0%–77%)   | Fixed        | 1.01 (0.94–1.10) | 0.72 |
|                    | Prostate cancer     | 3          | 0.02          | 75% (17%–92%)  | Random        | 1.33 (0.94–1.89) | 0.11 |
|                    | NHL                 | 2          | 0.70          | 0% (not applicable) | Fixed    | 0.98 (0.83–1.16) | 0.84 |
|                    | Genotyping by Taqman| 11         | 0.21          | 25% (0%–63%)   | Fixed        | 0.99 (0.92–1.06) | 0.80 |
|                    | Genotyping by PCR   | 7          | 0.03          | 57% (0%–81%)   | Random        | 1.22 (0.92–1.62) | 0.17 |
|                    | Genotyping by HM L/I MS | 3        | 0.70          | 0% (0%–90%)    | Fixed        | 1.11 (0.99–1.24) | 0.08 |
| **TT vs CT + CC**   | Overall             | 22         | 0.18          | 22% (0%–54%)   | Fixed        | 1.17 (1.03–1.31) | 0.01 |
|                    | White               | 11         | 0.06          | 43% (0%–72%)   | Random        | 1.17 (0.94–1.47) | 0.16 |
|                    | Asian               | 2          | —             | 0% (not applicable) | Fixed    | 5.26 (0.25–110.21) | 0.28 |
|                    | Breast cancer       | 6          | 0.80          | 0% (0%–79%)    | Fixed        | 1.04 (0.86–1.25) | 0.69 |
|                    | Prostate cancer     | 3          | 0.28          | 21% (0%–92%)   | Fixed        | 1.61 (1.17–2.22) | 0.004|
|                    | NHL                 | 2          | 0.77          | 0% (not applicable) | Fixed    | 1.30 (0.95–1.78) | 0.10 |
|                    | Genotyping by Taqman| 11         | 0.90          | 0% (0%–60%)    | Fixed        | 1.08 (0.97–1.36) | 0.35 |
|                    | Genotyping by PCR   | 7          | 0.03          | 61% (3%–94%)   | Random        | 1.83 (1.06–3.16) | 0.03 |
|                    | Genotyping by HM L/I MS | 3        | 0.89          | 0% (0%–90%)    | Fixed        | 1.16 (0.88–1.51) | 0.29 |

The bold values mean that their association is significant. CI = confidence interval, HM L/I MS = high-throughput, matrix-assisted, laser desorption/ionization time-of-flight mass spectrometry, NHL = non-Hodgkin lymphoma, OR = odds ratio, PCR = polymerase chain reaction.

**FIGURE 5.** Funnel plot to detect publication bias.
differences between the CAT polymorphism and cancer risk; further studies should pay attention to the ethnic-specific effects on cancer risk. Moreover, our results also showed significant association between C-262T gene polymorphism and increased prostate cancer risk, but not risks of other cancer types, revealing that although the etiology of cancers may overlap, the different cancers appear to have different genetic risk profiles and environmental factors may also contribute to at least part of the cancer subtype bias observed here in the association between the CAT C-262T polymorphism and cancer risk.37

It is worth mentioning a recent study by Tefik et al,32 which found that compared with the CC genotype, the TT genotype in CAT C-262T gene had a 1.94- and 3.83-fold increased risk for high-stage disease and metastasis, respectively, implying that this polymorphism may also be a risk factor in tumor progression and metastasis. In addition, numerous studies have paid attention to the potential of CAT in the treatment of cancer.38 It has been reported that inhibition of CAT with shRNA results in high H2O2 production with increased cell migration and invasion in CL1–0 cells,39 whereas CAT overexpression in mammary cancer cells leads to a less aggressive phenotype and an altered response to chemotherapy,40 suggesting that CAT-mediated oxidative stress might be an important therapeutic target in cancer. Therefore, to make a better understanding of CAT-related genetic, epigenetic, environmental, and clinical factors may also lead to more effective prevention and treatment of cancer.

There are several points that should be addressed in our meta-analysis. First, a relatively small number of studies and subjects were included in this meta-analysis, which may reduce the statistical power for identifying possible associations between the CAT C-262T polymorphism and cancer risk. Secondly, only published studies were included in this meta-analysis; unpublished data and ongoing studies were not sought. As studies reporting positive findings are more likely to be accepted for publication, this may lead to outcome reporting or publication bias, which brings inflation of the associations. Thirdly, lack of the original data of the reviewed studies limited our further investigation of potential interactions between genes because one gene may enhance or hinder the expression of another gene. Fourthly, in this study, we observed that genotyping method may also influence the assay results; further studies should pay attention to these aspects. Last but not least, the included publications were majorly limited to Asian and white populations, so future work should examine other populations, such as Latinos.

CONCLUSION

In summary, our results suggest that the CAT C-262T gene polymorphism may be a risk factor for cancer with cancer type-specific effects. Large well-designed, multicenter epidemiological studies should be carried out in these and other ethnic populations to confirm our findings.

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