Int J Clin Exp Med 2015;8(7):10248-10255
www.ijcem.com /ISSN:1940-5901/IJCEM0009099

Review Article

DNA repair gene ERCC1 polymorphisms and glioma susceptibility among Chinese population: a meta-analysis

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Received April 14, 2015; Accepted June 10, 2015; Epub July 15, 2015; Published July 30, 2015

Abstract: Background: Excision repair cross complementation group 1 (ERCC1) has been shown to be involved in the progression of glioma susceptibility. However, the results remain conflict. The aim of this study was to systematically review and evaluate the role of ERCC1 C118T and C8092A polymorphisms in glioma risk among Chinese population. Methods: Related case-control studies were searched in online electronic databases. Odds ratio (OR) with its 95% confidence interval (CI) were employed to calculate the extracted data. Results: Total seven articles were retrieved, including 4426 subjects (1926 were glioma patients and 2500 were matched controls). No significant heterogeneity was found between studies (I^2=0%, *P*>0.01). Our results demonstrated that A allele and AA genotype of ERCC1 C8092A polymorphism have a positive association with increasing the risk of glioma in the fixed-effect model (A vs. C: OR=1.13, 95% CI=1.02-1.25, *P*=0.02; AA vs. CC: OR=1.29, 95% CI=1.04-1.61, *P*=0.02; AA vs. CA+CC: OR=1.25, 95% CI=1.01-1.55, *P*=0.04). However, no significant relationship was found between C118T variant and glioma susceptibility. Conclusions: Our results indicated that ERCC1 C8092A, not C118T polymorphism might be a biomarker for patients with glioma among Chinese population. Future studies with more ethnicities are needed to explore the precise association.

Keywords: Glioma, DNA repair gene, ERCC1, polymorphism, meta-analysis

Introduction

Glioma is the most common heterogeneous central nervous system tumor, approximately accounting for 80% of all malignant brain tumors [1]. It consists of different histological subtypes with significant variability in prognosis, leading to high mortality and morbidity [2]. The 5-year survival probability of patients with glioma varies by subtype, but is as low as 4.7% [3]. The aetiology of glioma is largely unknown, and the treatment remains one of the most challenging problems in public health. Therefore, better understanding of the mechanism under the development of glioma and improving therapeutic strategy may contribute to glioma.

Over the past two decades, molecular studies have identified numerous genetic aberrations which are specifically related with individual types of glioma. Known these risk loci may be associated with glioma within a family history of brain tumors [4]. Nucleotide excision repair (NER), a DNA repair pathway, contains more than 30 proteins which have the concerted action in recognizing and excising lesions caused by harmful mutagens [5, 6]. A number of genetic instability resulting from mutations in NER genes underscore the biological importance of this pathway [7, 8]. Among which, the excision repair cross-complementing group 1 (ERCC1), located at 19q13, is a crucial gene in NER pathway and one of the most be studied [9]. Overexpression of ERCC1 mRNA is associated with drug resistance to cisplatin in human glioma [10, 11]. Genetic polymorphisms may influence the expression of ERCC1. Two common polymorphisms in ERCC1 are widely stud-

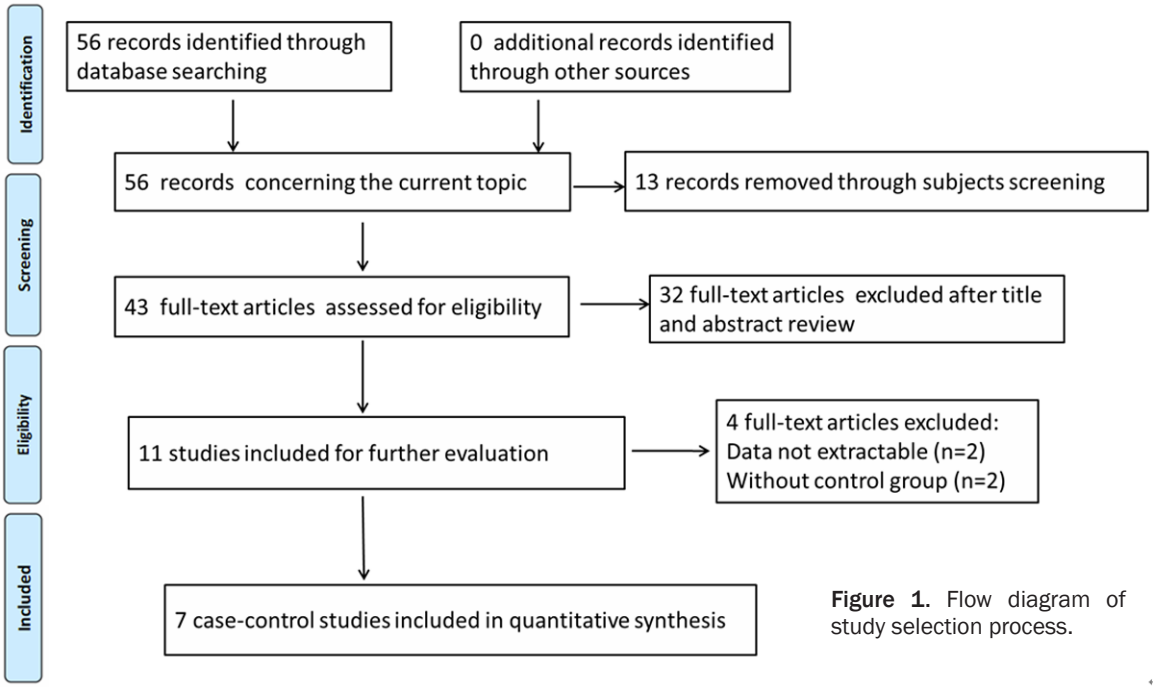

Figure 1. Flow diagram of study selection process.

ied: one was a T to C transversion in codon 118 (C118T, rs11615), leading to asparagine to aspartate substitution; the other was a C to A transversion in codon 8092 of the 3'-untranslated region (C8092A, rs3212986), leading to a substitution of lysine for glutamine. Studies have demonstrated that C118T variant was related to ERCC1 mRNA level and protein expression, and C8092A variant might influence ERCC1 mRNA stability [12].

Recently, several studies have investigated the role of these two ERCC1 polymorphisms in susceptibility to glioma. Polymorphisms in ERCC1 C118T was shown to have a role in the prognosis of glioma [13]. C8092A variant might be low-penetrance glioma-risk genes [14]. However, because of glioma rates vary by demographic factors and geo-political boundaries [15], and genotype distribution of ERCC1 polymorphisms varies among different populations, the results may be inconclusive. Moreover, the annual incidence of glioma was higher in Chinese than that in other populations. Thus, we performed this study to determine the association between ERCC1 polymorphisms and glioma risk among Chinese population.

Materials and methods

Study identification

We conducted a comprehensive literature search in online databases of PubMed, Web of science, Medline and Embase to retrieve relevant articles published between January 2000 and November 2014. The following terms: "glioma or glial cell tumor", "the excision repair cross-complementing group 1 or ERCC1", "DNA repair gene" and "polymorphism or variant or mutation" as well as their combination were employed as the searching items. Meanwhile, references of related studies were searched manually to obtain more studies. The search was only limited on English language.

Selection criteria

The eligible studies should meet the following criteria: 1) case-control study focused on the role of ERCC1 polymorphisms in glioma risk; 2) patients should be diagnosed, histologically confirmed glioma, controls should be age-, sex-matched participants without any history of any type of cancer; 3) the results were presented in odds ratio (OR) with its 95% confidence interval (CI); and 4) the allele and genotype information of patients and controls were available to extract.

Data extraction

According to the PRISMA guidelines, all the data from each included study were extracted and assessed by two investigators independently, any disagreement was discussed with a third expert to achieve a consensus. The follow-

Table 1. Main characteristics of included studies

First author	Year	Mean age		Sample size		Genotype methods
		Cases	Controls	Cases	Controls	
Chen DQ	2012	50.4±7.9	49.6±8.5	393	410	TaqMan
Zhang N	2012	47.6±7.5	46.8±6.2	257	278	TaqMan
Luo KQ	2013	48.7±7.9	50.2±8.1	297	415	Sequenom assay
Pan WR	2013	50.9±9.6	51.2±9.1	443	443	PCR-SBE
Dong YS	2014	44±15	46±18	72	302	Sequenom assay
Gao K	2014	47.5±8.5	48.6±7.4	326	376	PCR-SBE
Hui L	2014	45.2±10.5	44.7±11.2	138	276	TaqMan-RT-PCR

PCR-SBE, polymerase chain reaction amplification and single base extension assays.

Table 2. Distribution of alleles and genotypes for each polymorphism in includedstudies

First author	Cases					Controls				
C118T	CC	CT	TT	C	T	CC	CT	TT	C	T
Chen DQ	171	154	68	496	290	196	152	62	544	276
Zhang N	130	94	33	354	160	149	107	22	405	151
Luo KQ	138	114	45	390	204	188	158	69	534	296
Pan WR	193	171	79	557	329	211	162	70	584	302
Gao K	149	128	49	426	226	179	139	57	497	253
Hui L	61	47	30	169	107	131	89	56	351	201
C8092A	CC	CA	AA	C	A	CC	CA	AA	C	A
Chen DQ	202	141	50	545	241	221	154	35	596	224
Zhang N	123	98	36	344	170	144	105	29	393	163
Pan WR	229	169	45	627	259	241	162	41	644	244
Dong YS	33	32	7	98	46	137	144	21	418	186
Gao K	163	129	33	455	195	197	140	39	534	218
Hui L	67	49	22	183	93	148	92	36	388	164

Table 3. Meta-analysis of the association of ERCC1 C118T and C8092A polymorphisms with the risk of glioma

Comparisons	OR (95% CI)	P	Ph	I^2	Model
C118T					
T vs. C	1.09 (1.00, 1.20)	0.06	0.71	0%	F
TT vs. CC	1.16 (0.97, 1.39)	0.11	0.60	0%	F
CT vs. CC	1.09 (0.95, 1.25)	0.20	0.97	0%	F
TT+CT vs. CC	1.11 (0.98, 1.26)	0.10	0.92	0%	F
TT vs. CT+CC	1.11 (0.94, 1.32)	0.22	0.59	0%	F
C8092A					
A vs. C	1.13 (1.02, 1.25)	0.02	0.96	0%	F
AA vs. CC	1.29 (1.04, 1.61)	0.02	0.86	0%	F
CA vs. CC	1.07 (0.93, 1.24)	0.34	0.98	0%	F
AA+CA vs. CC	1.12 (0.98, 1.28)	0.10	0.99	0%	F
AA vs. CA+CC	1.25 (1.01, 1.55)	0.04	0.79	0%	F

OR, odds ratio; 95% CI, 95% confidence interval; P, *p*-value of pooled ORs; I^2 and Ph, between-study heterogeneity; F, the fixed-effect model.

ing information was extracted: the first author, year of publication, mean age, sample size, genotype, allele frequencies and genotype distributions in glioma cases and controls.

Quality assessment

The strength of association between polymorphisms of ERCC1 gene and glioma risk was measured by the pooled ORs with its 95% CIs under five genetic models: the allele model, dominant model, recessive model, homozygous model, and heterozygous model. The significance of the crude ORs was determined by the Z test, and a *P*-value less than 0.05 was considered as statistical significant. The heterogeneity between these studies was analyzed by the I^2 test and the Q statistic test. The fixed-effect model was used when the effect was homologous (I^2 less than 50% and *P*-value of the Q test more than 0.01); and the random-effect model was employed when they are heterogeneous. All analysis was performed by Review manager 5.2 (The Cochrane Information Management System).

Results

Characteristics of eligible studies

The initial search identified 56 studies concerning the current topic. After applying the inclusion criteria, only seven articles were screened out [16-22], including 4426 subjects (1926 were glioma patients and 2500 were matched controls). The process of selection was presented in **Figure 1**. Among the seven articles, six studies focused on C118T variant, and six

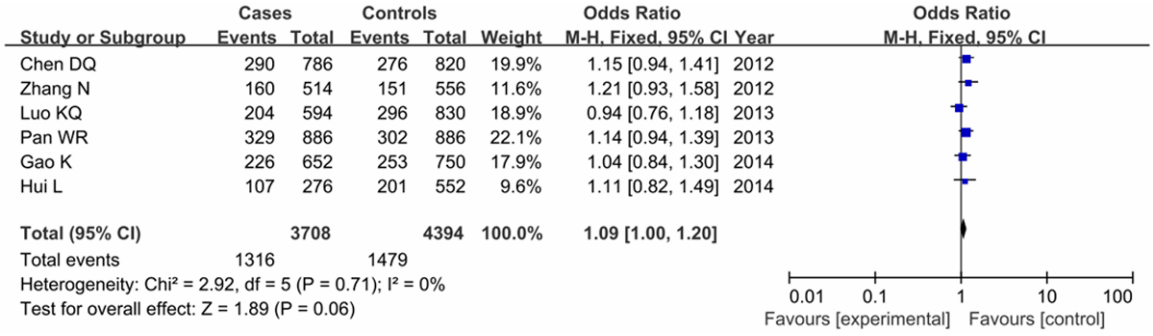

Figure 2. Meta-analysis on the ERCC1 C118T polymorphism and glioma risk under the allele model (T vs. C) in a fixed-effect model.

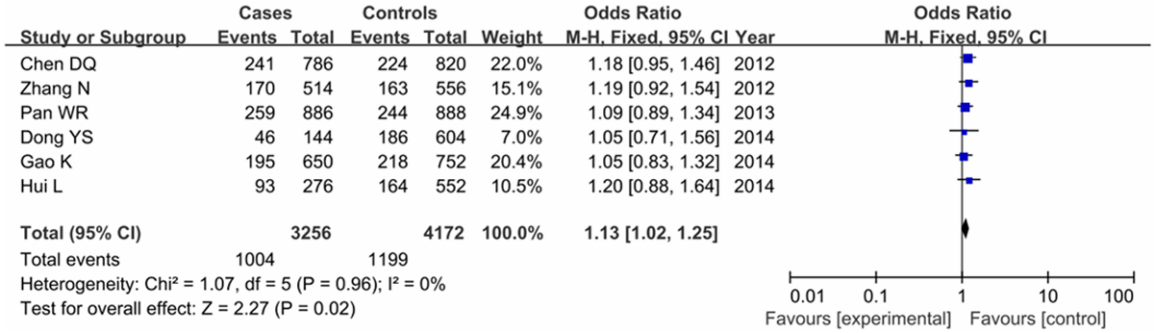

Figure 3. Forest plot of ERCC1 C8092A variant and glioma risk under the allele model (A vs. C) in a fixed-effect model.

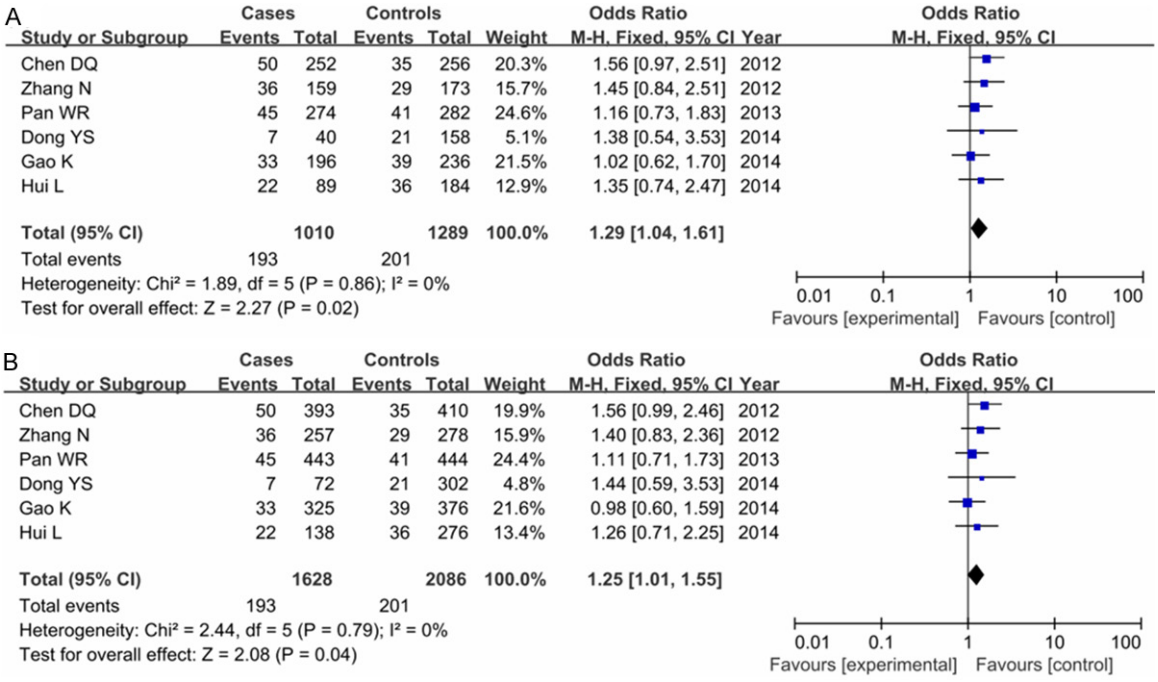

Figure 4. Forest plot of ERCC1 C8092A variant and glioma risk under the homozygous model (A: AA vs. CC) and recessive model (B: AA vs. CA+CC).

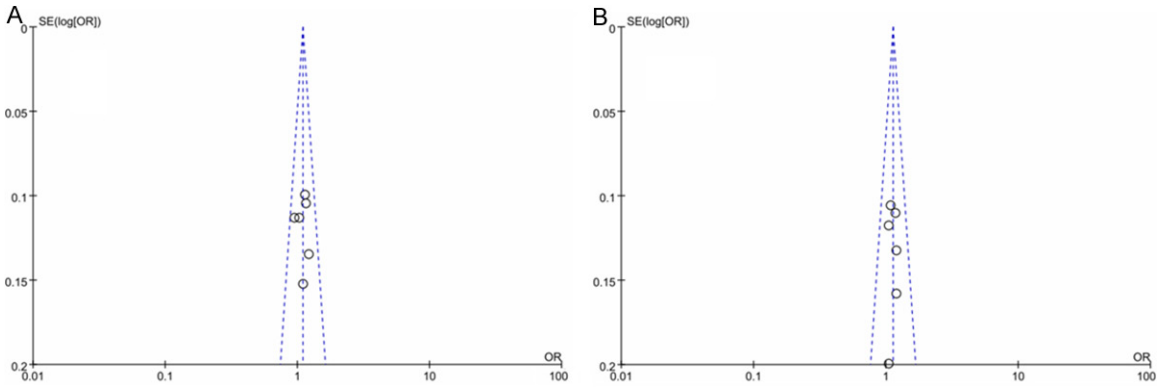

Figure 5. Funnel plot of allele comparison for publication bias (A: T vs. C for C118T polymorphism; B: A vs. C for C8092A variant).

on C8092A variant. All of them were conducted in Chinese population, and written in English. The sample size ranged from 374 to 886. The mean age ranged from 44 to 51.2 years old. **Table 1** showed the main characteristics of included studies. **Table 2** listed the information of alleles and genotypes for each polymorphism.

Association between ERCC1 C118T polymorphism and glioma risk

Table 3 summarized the main results of combined data on ERCC1 polymorphisms based on seven case-control studies. Between-study heterogeneity was examined, and no significant heterogeneity was found (I^2=0%, *P*>0.01). Overall, our analysis did not find a significant association between ERCC1 C118T polymorphism and glioma risk under all five genetic models in a fixed-effect model (T vs. C: OR=1.09, 95% CI=1.00-1.20, *P*=0.06, **Figure 2**; TT vs. CC: OR=1.16, 95% CI=0.97-1.39, *P*=0.11; CT vs. CC: OR=1.09, 95% CI=0.95-1.25, *P*=0.20; TT+CT vs. CC: OR=1.11, 95% CI=0.98-1.26, *P*=0.10; TT vs. CT+CC: OR=1.11, 95% CI=0.94-1.32, *P*=0.22).

Association between ERCC1 C8092A polymorphism and glioma risk

No significant heterogeneity was found between studies. The frequency of A allele was higher in cases than that in controls (30.8% vs. 28.7%), indicating a positive relationship between C8092A polymorphism and glioma risk (A vs. C: OR=1.13, 95% CI=1.02-1.25, *P*=0.02) as shown in **Figure 3**. This significant association was also found in homozygous model (AA vs. CC: OR=1.29, 95% CI=1.04-1.61, *P*=0.02) and recessive model (AA vs. CA+CC: OR=1.25, 95% CI=1.01-1.55, *P*=0.04) as shown in **Figure 4**. While no association was found in heterozygous model (CA vs. CC: OR=1.07, 95% CI=0.93-1.24, *P*=0.34) and dominant model (AA+CA vs. CC: OR=1.12, 95% CI=0.98-1.28, *P*=0.10).

Publication bias

The shapes of the funnel plot for C118T (**Figure 5A**) and C8092A (**Figure 5B**) polymorphisms showed no obvious asymmetry. Egger's test indicated that there is no publication bias (C118T: *P*=0.069; C8092A: *P*=0.071). Both of the results showed no publication bias in included studies.

Discussion

In this meta-analysis, we found that A allele and AA genotype of ERCC1 C8092A polymorphism were associated with increasing the risk of glioma in the Chinese population, while no association was found between ERCC1 C118T polymorphism and glioma risk. Our results were in accord with four previous meta-analysis which suggested that the AA genotype of C8092A polymorphism might increase the susceptibility of glioma among Asians [23-26], while not consistent with analysis conducted by Yuan et al. which found C118T variant increased glioma susceptibility among Asian [24].

NER was shown to have an important role in preventing the deleterious effects of oxidative DNA damage [27]. It eliminates various struc-

turally unrelated DNA lesions by a multiwise 'cut and patch'-type reaction [28]. Mutations in genes of the NER pathway are associated with diseases. ERCC1 is a key element in transcription-coupled NER pathway and be critical for efficient DNA repair capacity, cleaving DNA structures near the site of the platinum-DNA adduct, thereby allowing elimination of the lesion [29]. It functions as a biomarker for patient survival or treatment efficacy at the genomic level, transcriptional level and protein level. It is also essential for the maintenance of genetic stability. Studies have shown that ERCC1 status is highly predictive for patients who treated with adding oxaliplatin to 5-FU chemotherapy for stage III colon cancer, while mismatch repair status had no predictive value [30]. ERCC1 was also shown as a promising predictive and prognostic biomarker in advanced non-small cell lung cancer [31], and a meta-analysis demonstrated that ERCC1 expression might be a favorable prognostic and a drug resistance predictive factor for non-small cell lung cancer [32]. High ERCC1 expression predicts poor locoregional control in nasopharyngeal carcinoma [33]. ERCC1 plays an important role in target therapy as well. ERCC1, combined with xeroderma pigmentosum group F (XPF) endonuclease, forms a heterodimer which catalyzes the 5' incision in the process of excising the DNA lesion. This heterodimer acts as not only as an attractive therapeutic target, but also as a biomarker to predict treatment outcome [34].

ERCC1 variants may influence its expression, thus be associating with human diseases and affecting drug reaction. Researchers have identified that ERCC1 C118T, not C8092A polymorphism, might serve as a biomarker of lung cancer risk and significant association with overall survival of patients with advanced non-small cell lung cancer undergoing platinum-based treatment [35]. ERCC1 C118T may be a predictive marker of treatment response to 5-FU/platinum chemotherapy for colorectal cancer in Han Chinese [36]. It also might be a useful prognostic factor in platinum-treated gastroenterology cancer patients [37]. Studies have indicated that ERCC1 C8092A was a predictive marker of the cisplatin/5-FU-based neoadjuvant setting, and also suggest that it's used as a marker for selecting the appropriate therapeutic approach in esophageal cancer [38]. For glioma, Chen et al. firstly reported a significant association of ERCC1 C8092A variant with the risk of brain tumors [39]. Subsequently, there were several studies conducted to explore the role of ERCC1 polymorphisms in glioma risk. ERCC1 C8092A variant, individually or together with other DNA repair genes, was found to contribute to adult glioma risk [40]. This variant may be also associated with risk of glioblastoma multiforme, the most aggressive type of glioma [41]. Chen et al. indicated that ERCC1 118 T/T genotypes conferred a significantly better prognosis for patients with glioma [16]. While Wrensch et al. found no association between C8092A polymorphism and adult glioma in total study population [42]. Zhang et al. showed that both C8092A and T118C polymorphisms were not associated with the risk of glioma in a Chinese population [21].

Several limitations were presented in our study. First, glioma epidemiology and frequency distribution of ERCC1 polymorphisms were different in different populations, our study may have some population bias, and be too limited to identify precise gene associations. Second, ERCC1 may interact with other DNA repair genes such as XPF, XPD which should also be considered. Third, factors such as age, gender and stage of glioma may influence the role of ERCC1 polymorphisms in glioma risk. Last one, participants in controls were complicated, for some are healthy population, some are subjects without a history of cancer.

In conclusion, our results suggested that A allele and AA genotype of ERCC1 C8092A polymorphism were significantly associated with increasing the risk of glioma among Chinese population. Further studies with large sample size and more ethnicities are needed to do to identify precise gene associations.

Acknowledgements

The study was supported by the Zhejiang Science and Technology Project (grants 20-10KYB085), the Medical and Health Technology Projects of Hangzhou (grants 2010A002) and Hangzhou Key Disciplines Fund.

Disclosure of conflict of interest

None.

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