RESEARCH ARTICLE

Lack of any Association between the Hogg1 Ser326Cys Polymorphism and Breast Cancer Risk: a Systematic Review And Meta-Analysis Of 18 Studies

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Abstract

Background: The human 8-oxoguanine DNA glycosylase (hOGG1) gene may be linked with cancer susceptibility. The aim of this study was to quantitatively summarize any association between the hOGG1 Ser326Cys polymorphism and breast cancer (BC) risk. **Materials and Methods:** A comprehensive search of the PubMed, Embase, and ISI web of knowledge databases for papers published before 1 October 2016 was conducted. Summary odds ratios (ORs) with corresponding 95 % confidence intervals (95 %CIs) were estimated, with fixed-effects or random-effects models when appropriate, to assess any association. **Results:** A total of 9,434 cases and 10,497 controls from 18 studies were included in this meta-analysis. When the eligible studies were pooled, there was no evidence found for a significant association between the hOGG1 Ser326Cys polymorphism and BC in in all genetic contrast models G vs. C (OR=1.19, 95% CI 0.92– 1.53), CG vs. CC (OR = 0.97, 95% CI 0.91-1.04, p = 0.46), GG vs. CC (OR = 1.11, 95% CI 0.91-1.35, p = 0.30), GG + CG vs. CC (OR = 0.98, 95% CI 0.92-1.05, p = 0.67), and GG vs. CG + CC (OR = 1.22, 95% CI 0.98-1.52, p = 0.07). According to subgroup analysis, we also did not find a significant association between the hOGG1 Ser326Cys polymorphism and BC risk in Asians and Caucasians considered separately. **Conclusions:** The current meta-analysis suggests that the hOGG1 Ser326Cys polymorphism is not significantly associated with BC risk.

Keywords: Breast cancer- 8-oxoguanine DNA glycosylase- polymorphism- meta-analysis

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Introduction

Breast cancer (BC) is the most common cancer and the leading cause of cancer deaths in women (Neamatzadeh et al., 2015). Global breast cancer incidence has been increasing by more than one million new cases every year; the incidence is significantly higher in developed countries than in developing countries (Torre et al., 2015). Although substantial progress has been made in BC in the past few decades, the underlying molecular mechanism of BC still remains not fully elucidated (Forat-Yazdi et al., 2015; Yao et al., 2015). The vast majority of risk factors associated to breast cancer susceptibility are related to hormonal exposure, either from endogenous sources such as early age at menarche, late age at menopause, late pregnancy or nullliparity, overweight and obesity, or exogenous sources such as the use of hormone replacement therapy (HRT) (Forman et al., 2013). Other risk factors include alcohol intake, radiation exposure, current age, past history of breast cancer and the history of a breast biopsy (Singletary 2003).

DNA damage generated by different carcinogenic agents can be repaired primarily through base excision repair (BER) pathway, composed of many DNA repair genes (Lange et al., 2011). Common polymorphisms in DNA repair genes may alter protein function and the possibility to repair damaged DNA (Ferguson et al., 2015). Defects in DNA repair pathways may lead to genetic instability and carcinogenesis (Roberts et al., 2011). The Human 8-oxoguanine DNA glycosylase (hOGG1) gene is a key gene in the BER pathway and DNA repair process, and the Ser326Cys polymorphism is reported to be a functional variation in the hOGG1 gene. The 1,245 C/G (Ser326Cys) polymorphism of hOGG1 gene is a well-known polymorphism that results in an amino substitution from Serine to Cystein at codon 326 (Wang et al., 2014; Zhang et al., 2014). Lots of functional studies have showed that the Cys allele was associated with the reduced DNA repair activity, thus increased the cancer risk. The Cys326 has lower ability to prevent mutagenesis by 8-OHdG than Ser326 in human cells in vivo (Niu et al., 2014).

Since the original identification of the hOGG1 Ser326Cys polymorphism, a number of studies have investigated the genetic effect of this polymorphism on

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BC susceptibility (Vogel et al., 2003; Rossner et al., 2006 Sangrajrang et al., 2008; Loizidou et al., 2009; Sterpone et al., 2010; Roberts et al., 2011; Kim et al., 2013; Smolarz et al., 2014; Romanowicz et al., 2016). However, the findings are conflicting about the role of the hOGG1 Ser326Cys polymorphism in relation to BC susceptibility. In order to get more accurate results, we performed a meta-analysis. In this study, we intend to explore the possible association between hOGG1 Ser326Cys polymorphism and BC risk. To our knowledge, this is the most comprehensive meta-analysis conducted to date with respect to the association between hOGG1 Ser326Cys polymorphism and BC risk.

Materials and methods

Literature search

We searched all published papers (before 1 October, 2016) in databases of PubMed, Medline, Embase and Google scholar. The keywords were as follows: ''OGG1'', ''hOGG1'', ''polymorphism'' and ''breast cancer''. Articles not written in English were excluded. Additionally, abstracts and unpublished reports were not included. All of the searched studies were retrieved, and the bibliographies were checked for other relevant publications.

Inclusion and exclusion criteria

Studies included in the current meta-analysis had to meet all the following criteria: (a) evaluation of the Ser326Cys polymorphism and BC risk, (b) case–control studies, (c) sufficient published data for estimating an odds ratio (OR) with 95 % confidence interval (95 % CI). The exclusion criteria of studies were as follows: (a) not for BC research, (b) only case population, (c) abstract, comment, case reports, letters, and review, (d) duplicate of previous publication and (e) no sufficient data were provided.

Data extraction

Two investigators extracted the data independently, and the results were reviewed by a third investigator. From each study, the following items were noted: first author, year of publication, country, numbers of cases and controls, frequencies of hOGG1 Ser326Cys polymorphism genotypes, and evidence of the Hardy–Weinberg equilibrium (HWE) in controls. If any disagreement were raised, it would be resolved by discussion and consultation with another researcher. Different ethnic descents were categorized as Caucasians, Asians, and Africans.

Quality assessment

The quality assessment of those included studies mainly conformed to the confirmation of Hardy–Weinberg equilibrium (HWE) for the genotype distribution of Ser326Cys polymorphism in the controls (Zintzaras et al., 2010). If studies departed from HWE in the controls, they were defined as low quality studies. Conversely, studies with the genotype distribution of Ser326Cys polymorphism in the controls in accordance with HWE (P>0.05) were defined as high quality studies.

Statistical analysis

The pooled OR and 95 %CI were used to assess the association between hOGG1 Ser326Cys polymorphism and BC risk for each case–control study. The pooled ORs were performed for allele model (G vs. C), homozygote model (GG vs. CC), heterozygote model (CG vs. CC), dominant model (GG + CG vs. CC), and recessive model (GG vs. CG + CC). Heterogeneity was evaluated with a chi-square-based Q test among the studies (P<0.10 was considered significant) (Huedo-Medina et al., 2006). When the heterogeneity was present, the random effects model was used to calculate the pooled OR, whereas the fixed affects model was used in its absence (DerSimonian et al., 2007). Sensitivity analysis was performed to assess the stability of the results. The I2 value was used as an index for the heterogeneity test, with values less than 25 % indicating low, 25 to 50 % indicating moderate, and greater than 50 % indicating high heterogeneity. The I2 statistic was used to estimate heterogeneity in the pooled studies (Huedo-Medina et al., 2006). Publication bias was assessed by visual inspection of funnel plots, in which the standard error of log (OR) of each study was plotted against its log (OR). Publication bias was qualitatively assessed by performing Begg's funnel plots, and it was quantitatively evaluated by Egger's test. P <0.05 was considered representative of statistically significant publication bias. In addition, an asymmetric plot indicates a possible publication bias (Song 2002). Subgroup analyses were performed according to sample size, ethnicity, source of control, family history status and genotyping method separately. One-way sensitivity analysis was also used to assess the stability of the results by omitting one of the studies each time. All the statistical analyses were performed by comprehensive meta-analysis (CMA) V2.0 software (Biostat, USA). All tests were two-sided, and the P values of < 0.05 were considered statistically significant.

Results

Study characteristic

In total, 47 studies relevant to the role of hOGG1 Ser326Cys polymorphism on cancer susceptibility were identified through the literature search and selection according to the inclusion criteria. Of these, 13 papers were excluded because of obvious irrelevance by reading the titles and abstracts. Three studies were excluded because of the lack of CC and CG genotype data (Figure. 1). Finally, a total of 18 case-control studies with a total of 9434 cases and 10497 controls were included in the meta-analysis (Vogel et al., 2003; Choi et al., 2003; Huang et al., 2004; Cai et al., 2006; Rossner et al., 2006; Zhang et al., 2006; Romanowicz-Makowska et al., 2008; Sangrajrang et al., 2008; Synowiec et al., 2008; Loizidou et al., 2009; Sterpone et al., 2010; Hsu et al., 2010; Roberts et al., 2011; Xie et al., 2013; Kim et al., 2013; Smolarz et al., 2014; Luo et al., 2014; Romanowicz et al., 2016). The characteristics of included studies were summarized in Table 1. All the eligible studies were written in English. The populations came from different countries, including Denmark, Korea, Japan, Taiwan, China, USA, Poland,

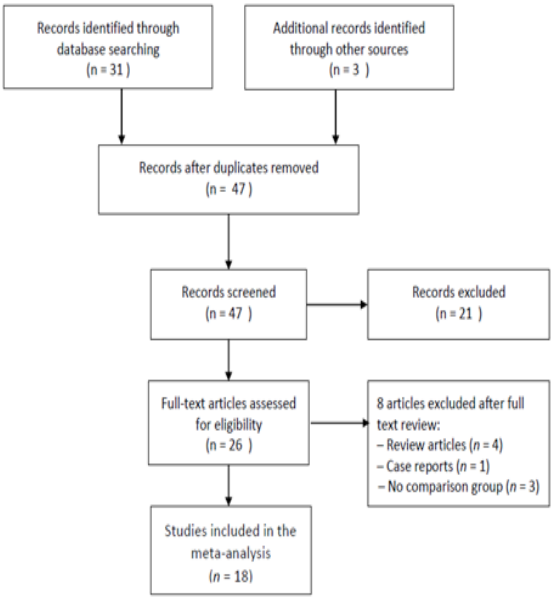

Figure 1. PRISMA Flow Diagram for Inclusion of the Studies Examining the Association of *hOGG1* Polymorphisms with BC Risk

Thailand, Cyprus, and Italy. There were 10 studies of Caucasian descendants (Vogel et al., 2003; Rossner et al., 2006; Zhang et al., 2006; Romanowicz-Makowska et al., 2008; Synowiec et al., 2008; Loizidou et al., 2009; Sterpone et al., 2010; Roberts et al., 2011; Smolarz et al., 2014; Romanowicz et al., 2016) and 8 studies of Asian descendants (Choi et al., 2003; Huang et al., 2004; Cai et al., 2006; Zhang et al., 2006; Sangrajrang et al., 2008; Hsu et al., 2010; Xie et al., 2013; Kim et al., 2013; Luo et al., 2014). In addition, the distribution of genotypes in the controls was consistent with HWE in all studies, except one study (Romanowicz-Makowska et al., 2008).

Quantitative synthesis

As shown in Table 2, no significant association between the hOGG1 Ser326Cys and BC risk was observed in any of the genetic models. Overall, no significant associations were found for G vs. C (OR = 1.07, 95% CI 0.95-1.20, p = 0.24), CG vs. CC (OR = 0.97, 95% CI 0.91-1.04, p = 0.46, Figure. 2A), GG vs. CC (OR = 1.11, 95% CI 0.91-1.35, p = 0.30, Figure. 2B), GG + CG vs. CC (OR = 0.98, 95% CI 0.92-1.05, p = 0.67, Figure. 2C), and GG vs. CG + CC (OR = 1.22, 95% CI 0.98-1.52, p = 0.07, Figure. 2D).

Subgroup analysis by ethnicity

Subgroup analyses by ethnicity were primarily performed in the Asian and Caucasian populations. Nine case–control studies involving 3,781 cases and 4,207 controls on the relationship between hOGG1 Ser326Cys and BC risk were carried out among Asians and ten ones with 5,653 cases and 6,290 controls were among Caucasians, respectively. Similarly, no statistically significant association was observed in Asians and Caucasians under all genetic models (Table 2).

Heterogeneity analysis and publication bias

The results for heterogeneity analysis among the included studies were summarized in Table 2. The heterogeneity was assessed between each of the studies using the Q test. The between-study heterogeneity among total studies was significant in dominant and homozygote genetic models (I2 = 85%, Ph < 0.001; I2 = 70%, Ph < 0.001, respectively) (Table 2). No significant

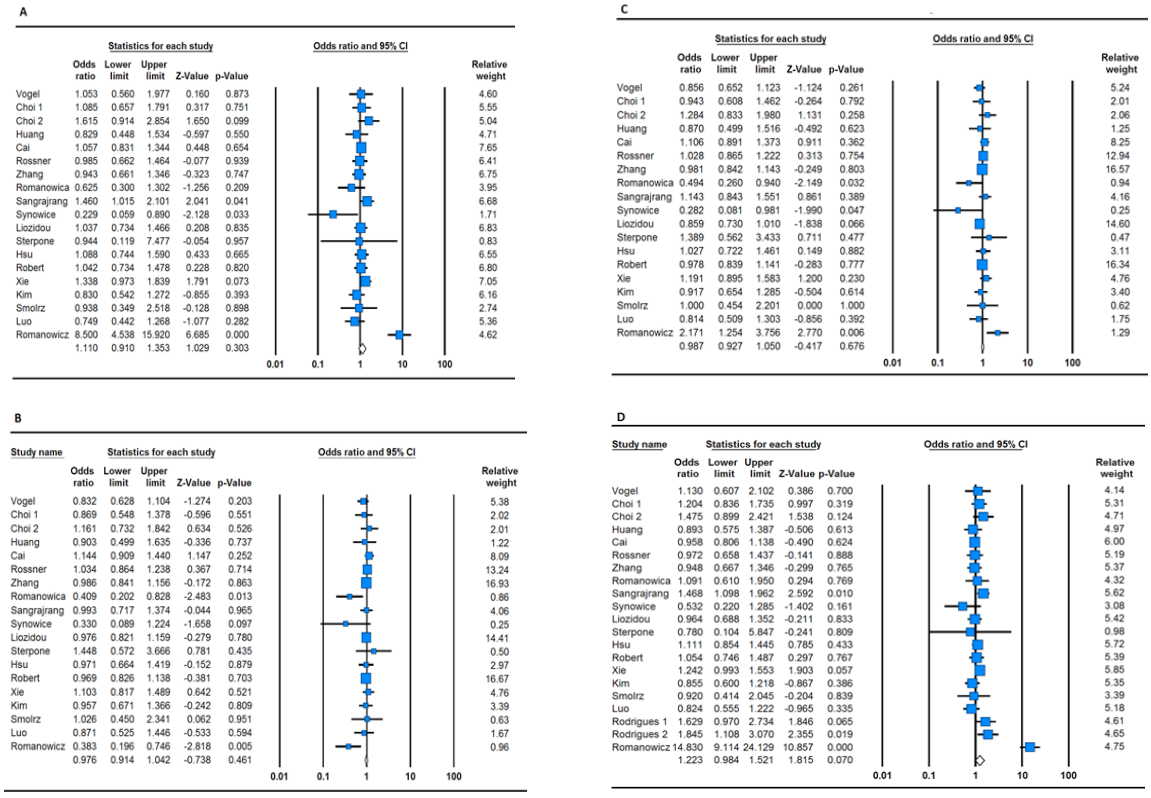

Figure 2. OR with 95 %CI for the Association of *hOGG1* Ser326Cys Polymorphism with BC. A: GG vs. CC, B: CG vs. CC, C: GG + CG vs. CC, D: GG vs. CG + CC.

Table 1. General Characteristics of Studies Included in the Meta-Analysis

First author	Country	Ethnicity	Case/Control	AR					non-AR					HWE
				Genotype			Allele		Genotype			Allele		
				CC	CG	GG	C	G	CC	CG	GG	C	G	
Vogel 2003	Denmark	Caucasian	425/434	256	147	22	659	191	245	169	20	659	209	0.17
Choi 2003	Korea	Asian	265/284	48	132	85	228	302	49	155	80	253	315	0.07
Choi 2003	Japan	Asian	201/184	57	95	49	209	193	62	89	33	213	155	0.91
Huang 2004	Taiwan	Asian	136/232	25	63	48	113	159	38	106	88	182	282	0.52
Cai 2006	China	Asian	1102/1167	186	534	382	906	1298	214	537	416	965	1369	0.08
Rossner 2006	USA	Caucasian	1041/1093	615	375	51	1605	477	653	385	55	1691	495	0.85
Zhang 2006	USA	Caucasian	1571/1244	967	532	72	2466	676	760	424	60	1944	544	0.93
Romanowica 2008	Poland	Caucasian	100/106	32	34	34	98	102	20	52	34	92	120	0.98
Sangrajrang 2008	Thailand	Asian	506/424	112	232	162	456	556	104	217	103	425	423	0.62
Synowiec 2008	Poland	Caucasian	41/48	10	19	12	39	43	4	23	21	31	65	0.5
Loizidou 2009	Cyprus	Caucasian	1108/1174	615	422	71	1652	564	647	455	72	1749	599	0.49
Sterpone 2009	Italy	Caucasian	43/34	18	23	2	59	27	17	15	2	49	19	0.57
Hsu 2009	China	Asian	401/533	64	165	172	293	509	87	231	215	405	661	0.06
Roberts 2011	USA	Caucasian	1054/1887	634	366	54	1634	474	1125	670	92	2920	854	0.54
Xie 2012	China	Asian	630/777	96	310	224	502	758	137	401	239	675	879	0.16
Kim 2013	Korea	Asian	346/361	92	181	73	365	327	90	185	86	365	357	0.63
Smolarz 2013	Poland	Caucasian	70/70	16	39	15	71	69	16	38	16	70	70	0.47
Luo 2014	China	Asian	194/245	42	87	65	171	217	45	107	93	197	293	0.15
Romanowicz 2016	Poland	Caucasian	200/200	23	24	153	70	330	44	120	36	208	192	0.004

Table 2. Meta-Analysis of the Association of *hOGG1* Ser326Cys Polymorphism with BC

	Genetic model	Type of model	Heterogeneity		Odds ratio		
			I^2 (%)	P_H	OR	95% CI	P_{OR}
Overall							
	G vs. C	Random	84	<0.001	1.07	0.95-1.20	0.24
	CG vs. CC	Fixed	17	0.23	0.97	0.91-1.04	0.46
	GG vs. CC	Random	70	<0.001	1.11	0.91-1.35	0.3
	GG + CG vs. CC	Fixed	33	0.07	0.98	0.92-1.05	0.67
	GG vs. CG + CC	Random	85	<0.001	1.22	0.98-1.52	0.07
Ethnicity							
Caucasian							
	G vs. C	Random	91	<0.001	1.1	0.87-1.38	0.41
	CG vs. CC	Random	54	0.02	0.87	0.73-1.03	0.12
	GG vs. CC	Random	83	<0.001	1.11	0.68-1.83	0.65
	GG + CG vs. CC	Random	55	0.01	0.95	0.83-1.09	0.51
	GG vs. CG + CC	Random	92	<0.001	1.29	0.67-2.49	0.43
Asian							
	G vs. C	Fixed	32	0.15	1.04	0.98-1.11	0.13
	CG vs. CC	Fixed	0	0.96	1.01	0.92-1.11	0.75
	GG vs. CC	Fixed	17	0.28	1.09	0.96-1.23	0.15
	GG + CG vs. CC	Fixed	0	0.81	1.03	0.94-1.13	0.47
	GG vs. CG + CC	Fixed	37	0.11	1.07	0.97-1.17	0.13

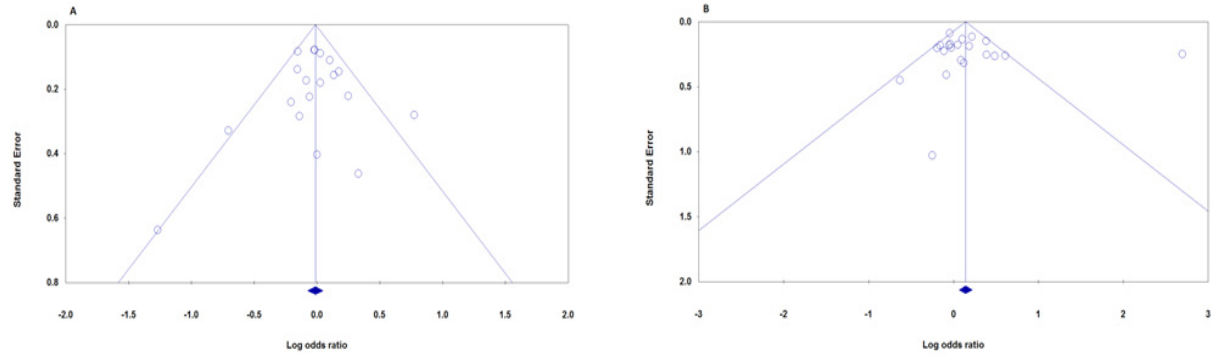

Figure 3. Begg's Funnel Plot with Pseudo 95% Confidence Limits of Publication Bias Test for hOGG1 Ser326Cys Polymorphism. C: GG + CG vs. CC, D: GG vs. CG + CC

heterogeneity was found in studies among Asians but not Caucasians, indicating that the publications in Caucasians were probably the main source of heterogeneity in the current meta-analysis.

Sensitivity analyses and publication bias

Sensitivity analysis was performed to assess the influence of each individual study on the pooled OR by sequential removal of individual studies. The results suggested that no individual study significantly affected the overall OR dominantly.

Publication bias

In this meta-analysis, we performed funnel plot and Egger's test to access the publication bias. Funnel plot's shape of all contrasts failed to indicate obvious evidence of asymmetry, and all the P values of egger's tests were more than 0.1 providing statistical evidence of funnel plot's symmetry (Figure. 3). Therefore, the results revealed that publication bias was not significant in this meta-analysis.

Discussion

The presence of 8-oxodG residues, one of the most abundant oxidative products of cellular DNA, leads to GC/TA transversions since it preferentially pairs with adenine instead of cytosine during DNA replication. An increase in 8-oxodG in DNA can contribute to the incidence of different cancer risk (Agnez-Lima et al., 2012).

In this study, we analyzed the data from 18 available case – control studies. The results are conflicting about the role of the hOGG1 Ser326Cys polymorphism in relation to BC susceptibility. Thus far, the association remains not fully understood because of inconsistent results across independent studies. Eight studies found an increased risk for BC associated with the 326Cys allele (Huang et al., 2004; Rossner et al., 2006; Sangrajrang et al., 2008;

Synowiec et al., 2008; Hsu et al., 2010; Xie et al., 2013; Romanowicz et al., 2016) and the other ten did not detect the association between Ser326Cys polymorphism and BC (Vogel et al., 2003; Choi et al., 2003; Cai et al., 2006; Zhang et al., 2006; Romanowicz-Makowska et al., 2008; Loizidou et al., 2009; Sterpone et al., 2010; Roberts et al., 2011; Kim et al., 2013; Smolarz et al., 2014; Luo et al., 2014). The conflicting findings among different case–control studies might be attributed to different sample size, source of controls, genotyping method and matching criteria of subjects, and so on. In addition, the potential gene – gene and gene – environment interactions may also play vital roles in the pathogenesis of BC. Single study especially the one with relatively sample size may have not enough statistical power to identify a genetic association. Meta-analysis has the capability of combining quantitatively and evaluating synthetically in terms of the studies with the same objective and multiple independent results so as to improve the inspection efficiency. By means of the meta-analysis, this paper reviewed the case–control studies from home and abroad related to the association of the hOGG1 gene Ser326Cys polymorphism and BC, which provided evidence for BC risk assessment comprehensively upon hOGG1 Ser326Cys polymorphism and BC risk.

When all the eligible studies were pooled into analysis, it failed to uncover any evidence that there was an association between the Ser326Cys polymorphism and BC susceptibility overall. No statistical evidence was found in a dominant model, either in a recessive model, an additive model or a homozygote model. Moreover, the association of the Ser326Cys polymorphism and BC could not be found in Asians or Caucasians. Some meta-analyses were performed to solve the association between Ser326Cys polymorphism and BC risk. Yuan et al., in a meta-analysis reported that the hOGG1 Ser326Cys polymorphism is not associated with BC risk and Gu et al., concluded that the OGG1 Ser326Cys polymorphism might not be a potential candidate risk factor for the development of BC (Gu et al., 2010; Yuan et al., 2010). In the stratified analysis by ethnicity, source of controls, and menopausal status, Gu et al., not observed significant association still in all genetic models (Gu et al., 2010). However, Yuan et al. suggested that the hOGG1 Ser326Cys allele plays a significant protective effect to breast cancer in European women (Yuan et al., 2010).

Our results are consistent with the study performed by Wang et al., (2011) Ni et al., (2012) Guo et al., (2012) and Zhong et al., (2012) which got a negative result between the polymorphisms in hOGG1 Ser326Cys and gastric cancer, colorectal cancer and bladder cancer risk, respectively. However, Wang et al., (2014) Zhang et al., (2013) and Zhu et al., (2012) reported that the hOGG1 Ser326Cys genotype under the recessive model confers protection for digestive system cancer, esophageal squamous cell carcinoma and prostate cancer, respectively. These findings indicate that the hOGG1 Ser326Cys polymorphism exerts different effect on various types of cancers. So, it is necessary to get a better understanding of hOGG1 Ser326Cys polymorphism on BC susceptibility, especially when inclusive and controversial findings still exist.

There were several limitations in our meta-analysis. First, in this meta-analysis, the included 18 studies regarded only Caucasians and Asians, but not other races. Data about other ethnicities, for example, African, should be noticed in the future. Second, because we could not obtain sufficient data from the present publications, in this study, subgroup analyses regarding age, lifestyle, and other factors have not been expressed. Finally, gene – environment interactions were not addressed in our meta-analysis. In addition, it was reported that the combination of hOGG1 Ser326Cys polymorphism with other BER genes such as XRCC1 and APEX1 was significantly related to an elevated risk of BC (Sangrajrang et al., 2008; Peng et al., 2014). In addition, several genes including BRCA1, BRAC2, and P53 were identified to significantly mutate in BC patients (Vaclová et al., 2012). Thus, the possible gene – gene and gene – environment interactions may play central roles in the BC pathogenesis and need further confirmation in future studies.

In conclusion, this meta-analysis found that the hOGG1 Ser326Cys polymorphism was not associated with significantly increased risk of BC. However, further studies are warranted to validate the association between the hOGG1 Ser326Cys polymorphism and BC risk with larger sample size and more detailed data.

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