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RESEARCH ARTICLE

Association of hOGG1 Ser326Cys polymorphism with colorectal cancer risk: an updated meta-analysis including 5235 cases and 8438 controls

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Abstract It has been suggested that hOGG1 Ser326Cys polymorphism may be a risk factor for colorectal cancer. Published data on its association with colorectal cancer generated contradictory results; thus, we performed an updated meta-analysis of eligible published studies to estimate the effect of hOGG1 Ser326Cys polymorphism on colorectal cancer susceptibility. We reviewed many abstracts and finally included 18 eligible case–control studies comprising 5235 cases and 8438 controls. We pooled data with a fixed or random-effect model. Subgroup analysis by ethnicity was also performed. The overall data indicated a significant association of hOGG1 Ser326Cys polymorphism on colorectal cancer risk (allele model OR=1.14, 95 %CI 1.02–1.27; homozygote model OR=1.32, 95 %CI 0.92–1.92; recessive model OR=1.12, 95 %CI 1.00–1.26; dominant model OR=1.15, 95 %CI 1.00–1.32). Furthermore, in the subgroup analysis by ethnicity, increased cancer risk was observed among Caucasians under the allele, heterogeneity, recessive, and dominant models (allele model OR=1.23, 95 %CI=1.05–1.44; homozygote model OR=1.49, 95%CI 1.05–2.12; recessive model OR=1.40, 95 %CI 1.16–1.69; dominant model OR=1.21, 95 %CI=1.12–1.45). In summary, the present meta-analysis suggested that hOGG1 Ser326Cys polymorphism might modify the susceptibility to colorectal cancer among the total population, especially among Caucasians.

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Introduction

Colorectal cancer is one of the most common malicious tumors [1]. It is one of the major causes of mortality and morbidity, whose 5-year survival rate is low [2]. It is known to all that early diagnosis is very important which improves the chances of patient's survival significantly [3]. The causes of colorectal cancer have not been established [4]. The pathogenesis of colorectal cancer has involved oxidative DNA damage [5]. We know that the base excision repair pathway is the major DNA repair pathway for oxidative DNA damage and genetic variation which is associated with impaired base excision repair and may increase the risk for colorectal cancer [6]. The possible relationship between oxygen-free radicals and cancer development has been reported chiefly in organs that are under a high burden of oxygen-free radicals. 7,8-Dihydro-8-oxoguanine (8oxoG) is one of the most important lesions, which has been produced in DNA by oxygen radical-forming causes. The mispairing of 8oxoG with deoxyadenosine causes mutagenic transversion of G:C to T:A in vitro and in vivo [7, 8]. The *hOGG1* gene encodes a DNA glycosylase/AP-lyase that can catalyze the removal of 8oxoG adducts as part of the base excision repair pathway. The *hOGG1* gene is divided as multiple alternatively spliced isoforms with only one a-form which have a nuclear localization signal [9, 10]. Many studies have found the presence of several polymorphisms at the hOGG1 locus. A C/G polymorphism at position 1245 in the 1a-specific exon 7 of the *hOGG1* gene resulted to an amino acid change from serine to cysteine in codon 326 [11]. Studies on the association of hOGG1 Ser326Cys polymorphism with colorectal cancer generated controversial results, so we performed an updated meta-

analysis of eligible studies to estimate the effect of hOGG1 Ser326Cys polymorphism on colorectal cancer risk [12–17].

Materials and methods

Literature search strategy

We systematically searched PubMed, Embase, Web of Science, and Chinese National Knowledge Infrastructure (CNKI) with no language restrictions, for studies on the association between hOGG1 Ser326Cys polymorphism and colorectal cancer risk. We covered all studies published up to June 2013. We used the following terms: hOGG1, OGG1, Ser326Cys, polymorphism, polymorphisms, colorectal cancer (CRC), colon cancer, rectal cancer, susceptibility, and risk act. All of the searched studies were retrieved, and the bibliographies were checked for other relevant publications. Review articles and bibliographies of other relevant studies identified were searched by hand in order to find additional eligible studies.

Inclusion criteria

The following criteria were used for the literature selection: first, studies should concern the association of hOGG1 Ser326Cys polymorphism with colorectal cancer risk; second, studies should be observational studies (case–control or cohort); third, papers must report the size of the sample, odds ratios (ORs), and their 95 % confidence intervals (95 %CIs), genetic distribution, and ethnicity or must report sufficient data to estimate these correlated data. After rigorous searching, we reviewed all papers according with the criteria defined above for further analysis.

Data extraction

The data were extracted carefully from all eligible publications independently by two of the authors in accordance to the criteria mentioned above. This study used the following information: study design, publication year, matching factors, source of controls, frequencies of hOGG1 Ser326Cys polymorphism genotypes, ethnicity, country, patient characteristics, ORs, and 95 %CIs. The extracted information was entered into a database.

Statistical analysis

The pooled OR and 95 %CI were used to assess the association between hOGG1 Ser326Cys polymorphism and colorectal cancer risk for each case–control study. The pooled ORs were performed for a homozygote model (Cys/Cys vs. Ser/Ser), a dominant model (Cys/Cys+Ser/Cys vs. Ser/Ser), a recessive model (Cys/Cys vs. Ser/Cys+Ser/Ser), and an allele model (Cys vs. Ser). The I^2 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 I^2 statistic was used to estimate heterogeneity in the pooled studies [18]. When $I^2>50$ %, potential sources of heterogeneity were explored through a meta-regression analysis [19]. The data were pooled according to the fixed-effect model (Mantel–Haenszel) if $I^2<50$ %; otherwise, the random-effect model (DerSimonian and Laird) was used [20, 21]. The significance of the pooled ORs was determined by Z test. The Hardy–Weinberg equilibrium (HWE) was assessed by Fisher's exact test. Publication bias was assessed by visual inspection of funnel plots [22], in which the standard error of log (OR) of each study was plotted against its log (OR). An asymmetric plot indicates a possible publication bias. The symmetry of the funnel plot was further evaluated by Begg's linear regression test [23]. Statistical analysis was performed using the program Stata 11.2 software (Stata Corporation, College Station, TX, USA). P values <0.05 were considered statistically significant. There was no funding source for this study. The corresponding author had full access to all the data in the study and had the final responsibility for the decision to submit for publication.

Results

Study characteristics

We retrieved and screened the publications relevant to the keywords originally. Finally, as shown in Table 1, a total of 18 case–control studies were included for further analysis (Table 1). All the eligible studies were written in English. The characteristics of the studies included in the present meta-analysis were shown in the figure principally. According to the figure, we could know the first author, the number, and characteristics of cases and controls for each study as well as other necessary information. As can be seen in Table 1, there were 12 groups of Caucasians [12–16, 24–30] and 6 groups of Asians [17, 31–35]. The distributions of the control groups of all the studies were in line with HWE.

Meta-analysis

The overall data contained a total of 5235 cases and 8438 controls. We analyzed the heterogeneities for the dominant model, recessive model, etc., respectively. As shown in Table 2 and Figs. 1 and 2, the overall data indicate a significant association of hOGG1 Ser326Cys polymorphism with colorectal cancer risk (allele model OR=1.14, 95%CI 1.02–1.27; homozygote model OR=1.28, 95 %CI 1.02–1.62).

Table 1 Characteristics of the included studies in the meta-analysis

Study	Ethnicity	Country	Cases			Controls			HWE
			Ser/Sert	Ser/Cys	Cys/Cys	Ser/Sert	Ser/Cys	Cys/Cys	
[16]	Caucasians	Poland	102	67	3	142	54	4	0.66
[17]	Asians	India	66	41	7	100	89	11	0.12
[15]	Caucasians	Poland	87	41	4	67	33	0	0.05
[13]	Caucasians	Turkey	31	40	8	171	69	7	0.99
[12]	Caucasians	USA	172	117	19	217	127	18	0.92
[14]	Caucasians	Turkey	50	43	17	51	47	18	0.20
[28]	Caucasians	Poland	38	19	17	63	33	1	0.14
[24]	Caucasians	USA	918	570	94	1172	686	93	0.56
[30]	Caucasians	Poland	52	46	2	68	28	4	0.61
[26]	Caucasians	Denmark	220	137	16	467	277	32	0.25
[31]	Asians	China	42	95	60	179	395	261	0.20
[32]	Asians	Japan	17	30	21	39	56	26	0.48
[29]	Caucasians	Czech Republic	336	168	28	331	181	20	0.44
[35]	Asians	Singapore	35	152	116	183	537	439	0.38
[34]	Asians	Korea	91	220	128	120	333	223	0.82
[27]	Caucasians	Spain	225	114	23	210	104	9	0.36
[25]	Caucasians	Denmark	101	55	9	208	164	24	0.26
[33]	Asians	Korea	24	66	35	52	131	64	0.32

Furthermore, as what Table 2 and Fig. 3 are showing, in the subgroup analysis by ethnicity, increased cancer risk was observed among the Caucasians under the allele, homozygote, recessive, and dominant models (allele model OR=1.23, 95 %CI 1.05–1.44; homozygote model OR=1.49, 95 %CI= 1.05–2.12; recessive model OR=1.40, 95 %CI=1.16–1.69; dominant model OR=1.21, 95 %CI=1.12–1.45) (Fig. 3). That means that an increased colorectal cancer risk was shown among Caucasians with 326Cys mutation of hOGG1 polymorphism.

Funnel plots were created to evaluate possible publication bias. Then, Egger's linear regression tests were used to assess the symmetries of the plots. The data suggest that the funnel plots were symmetrical for the overall data under the allele model (t=1.82; P=0.087>0.05), suggesting that there was no publication bias.

Table 2 Meta-analysis of the association of hOGG1 Ser326Cys polymorphism with colorectal cancer

Model	Ethnicity	OR		Analysis model	Heterogeneity analysis	
		OR (95 %CI)	P_{OR}		I^2 (%)	P_H
Allele model	Overall	1.14 (1.02–1.27)	0.020	Random	67.7	0.000
	Caucasians	1.23 (1.05–1.44)	0.009	Random	72.8	0.000
	Asians	0.99 (0.90–1.09)	0.890	Fixed	30.9	0.203
Homozygote model	Overall	1.28 (1.02–1.62)	0.033	Random	50.5	0.008
	Caucasians	1.49 (1.05–2.12)	0.025	Random	53.3	0.015
	Asians	1.04 (0.85–1.27)	0.717	Fixed	30.5	0.206
Recessive model	Overall	1.12 (1.00–1.26)	0.059	Fixed	43.6	0.025
	Caucasians	1.40 (1.16–1.69)	0.001	Fixed	45.6	0.042
	Asians	0.98 (0.84–1.13)	0.752	Fixed	0.00	0.541
Dominant model	Overall	1.15 (1.00–1.32)	0.051	Random	62.2	0.000
	Caucasians	1.21 (1.12–1.45)	0.032	Random	69.1	0.000
	Asians	1.01 (0.85–1.19)	0.905	Fixed	37.4	0.157

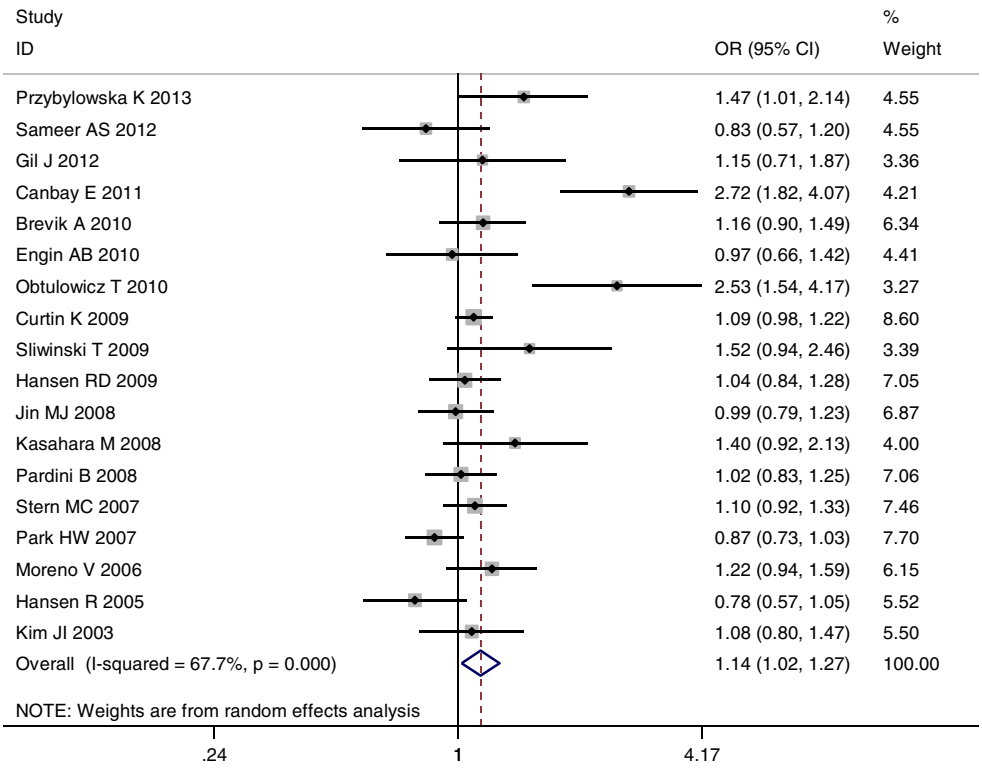

Fig. 1 OR with 95 %CI for the association of hOGG1 Ser326Cys polymorphism with colorectal cancer under the allele model (Cys vs. Ser)

Discussion

Colorectal cancer, the third most common cause of cancer death in the world, has 150,000 new cases and 50,000 deaths in the USA annually. In North America and Europe, the incidence rate is approximately 30–50/100,000. For colorectal cancer, one of the most common cancers, early diagnosis is critical. The susceptibility of colorectal cancer contains many factors. Studies have pointed that individual risks for colorectal cancer depend on genetic factors. Data has indicated an

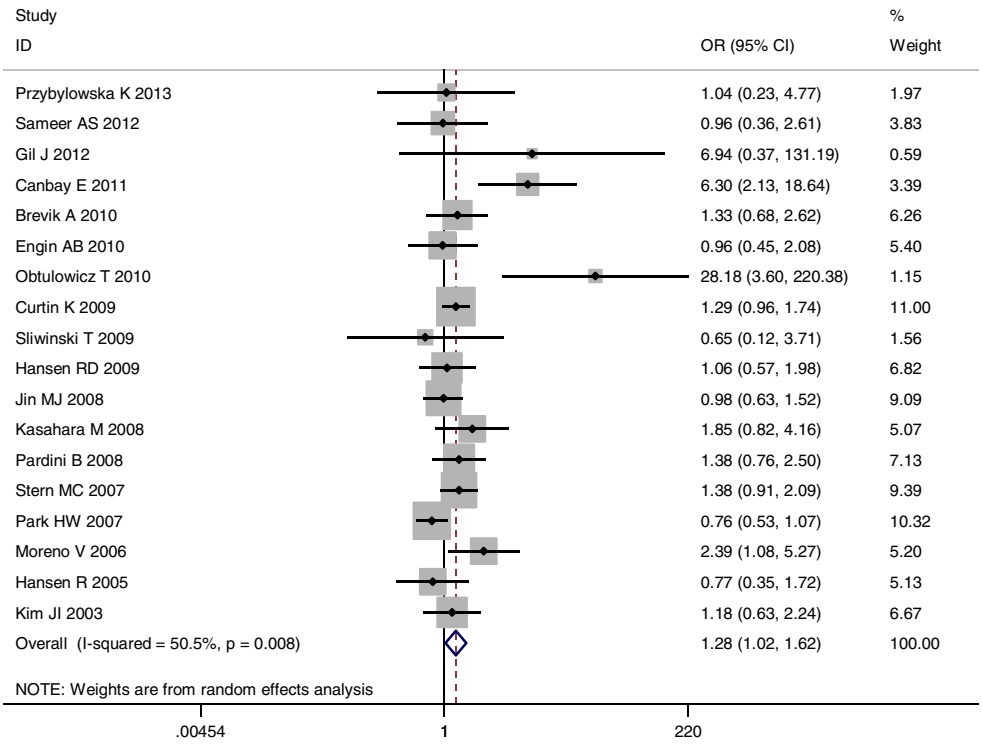

Fig. 2 OR with 95 %CI for the association of hOGG1 Ser326Cys polymorphism with colorectal cancer under the homozygote model (Cys/Cys vs. Ser/Ser)

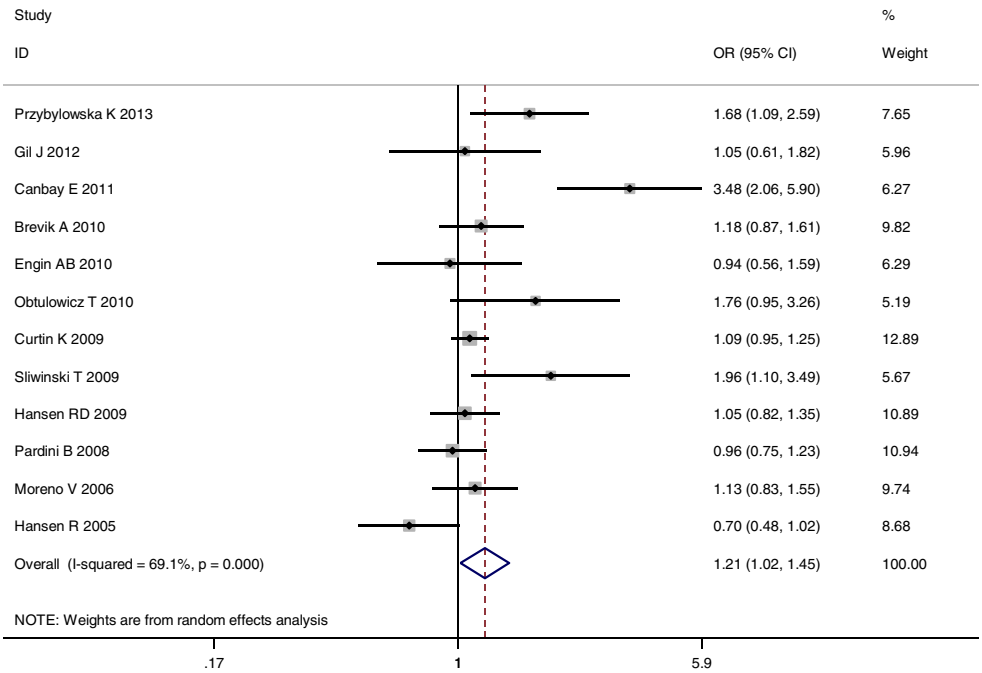

Fig. 3 OR with 95 %CI in the subgroup analysis by ethnicity under the dominant model

association of the descent productivity of DNA repair and the increased susceptibility of colorectal cancer [36]. It is widely recognized that mismatch repair pathway is an etiological factor of individual risk to colorectal cancer [37]. Many study data indicated an association of hOGG1 polymorphisms which were involved in oxidative DNA lesions repair with the risk occurrence of colorectal cancer patients [38]. Researchers found that hOGG1 polymorphisms may affect DNA repair capacity which suggested its role in colorectal cancer pathogenesis [33].

Relations of hOGG1 polymorphisms with cancer susceptibility have been certified by several analyses. As shown above, at present, studies suggest that hOGG1 polymorphisms might modify the susceptibility to colorectal cancer among the total population (allele model and homozygote model). Meanwhile, in the subgroup analysis of ethnicity, increased cancer risk was observed among Caucasians under the heterogeneity, recessive, and dominant models. The difference may be induced by ethnic differences in genetic backgrounds and the environment where they lived. Different socioeconomic status could also affect colorectal cancer risk. The most important etiological factors of sporadic colorectal tumors are inflammation, fat metabolism, tobacco smoking, and consumption of meat and alcohol [12, 39, 40]. This might explain the racial disparities. The difference may be caused by the limited number of included studies. Insufficient statistical power for assessment might be caused by small sample sizes, too. Thus, we should interpret the results carefully.

We used the fixed-effect model in the recessive model for the overall data. Because we observed between-study heterogeneity in the allele, dominant, and homozygote models for the overall data, we used the random-effect model in these models. In every model, we divided the data into subgroups with regard to ethnicity. Then, heterogeneity was removed in Asians, but not in the overall and in the Caucasian populations, which suggested that the heterogeneities may be multifactorial. Other factors, such as living environment, age, gender, and lifestyle, might induce the heterogeneities. At the same time, publication bias, as an important factor, should be considered in the meta-analysis. Funnel plot evaluation and Begg's linear regression tests were used to assess the possible publication bias. We found no evident bias, so the potential publication bias might cause little influence on the results.

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 living environment, age, gender, lifestyle, and other factors have not been expressed. So, selection bias might exist. Meanwhile, in further studies, gene–gene and gene–environment interactions should also be noted. Further well-designed studies with more rigorous matching criteria and large sample sizes are required to assess the relationship between hOGG1 polymorphisms and colorectal cancer risk. Despite the limitations, the overall data indicated a significant association of hOGG1 polymorphisms on colorectal cancer risk (allele model OR=1.14, 95%CI 1.02–1.27; homozygotemodel OR=1.32, 95 %CI 0.92–1.92; recessive model OR=1.12, 95 %CI 1.00–1.26; dominant model OR=1.15, 95 %CI 1.00–1.32). Furthermore, in the subgroup

analysis by ethnicity, increased cancer risk was observed among Caucasians under the allele, heterogeneity, recessive, and dominant models (allele model OR=1.23, 95 %CI=1.05–1.44; homozygote model OR=1.49, 95 %CI 1.05–2.12; recessive model OR=1.40, 95 %CI 1.16–1.69; dominant model OR=1.21, 95 %CI=1.12–1.45). In summary, the present meta-analysis suggested that hOGG1 Ser326Cys polymorphism might modify the susceptibility to colorectal cancer among the total population, especially among Caucasians. In summary, the data of the present studies suggest that hOGG1 polymorphisms might modify the susceptibility to colorectal cancer among the total population, especially among Caucasians.

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Conflicts of interest None.

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