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ORIGINAL PAPER

Meta-analysis of the association between hOGG1 Ser326Cys polymorphism and risk of colorectal cancer based on case–control studies

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Abstract
Purpose Oxidative DNA damage caused by reactive oxygen species plays an important role in cancer development. The association between colorectal cancer and hOGG1 Ser326Cys polymorphisms has been analyzed in several published studies, but mixed findings have been reported. The main purpose of this study was to integrate previous results and explore whether the polymorphism of hOGG1 is associated with susceptibility to colorectal cancer.
Methods PubMed, Embase, Google Scholar, and Cbmdisc were searched for studies on the relationship of hOGG1 SNPs and the incidence of colorectal cancer (CRC). Eligible articles were included for data extraction. The main outcome was the frequency of hOGG1 Ser326Cys polymorphisms between cases and controls. Comparison of the distribution of SNP was mainly performed using Review Manager 5.0.
Results A total of 4,174 cases and 6,196 controls from 12 studies were included for this meta-analysis. Overall, stratified by ethnicity or population source, no significant associations between the hOGG1 Ser326Cys polymorphism and colorectal cancer risk were found for Cys/Cys allele (OR = 1.146; 95 % CI: 0.978–1.342, $P = 0.091$), Cys/Cys + Cys/Ser versus Ser/Ser (OR = 1.045; 95 % CI: 0.975–1.121, $P = 0.213$) Cys/Cys Versus Ser/Ser (OR = 1.243; 95 % CI: 0.979–1.578, $P = 0.074$) and Cys/Cys versus Cys/Ser + Ser/Ser (OR = 1.198; 95 % CI: 0.959–1.496, $P = 0.111$) in a recessive model and (OR = 1.494; 95 % CI: 1.023–2.181, $P = 0.038$) in a homozygote contrast. However, if apart from sensitivity analysis, there was some evidence to indicate that significantly increased risks were found among European plus American subjects, who are mostly Caucasian (OR = 1.444; 95 % CI: 1.017–2.05 Cys/Cys vs. Ser/Cys + Ser/Ser; $P = 0.04$). In the subgroup analyses, we also did not found any association between hOGG1 Ser326Cys polymorphism and certain populations and smokers.
Conclusions This meta-analysis suggests that there is no robust association between hOGG1 Ser326Cys polymorphism and colorectal cancer. Because of the limitation of meta-analysis, this finding demands further investigation.

Keywords hOGG1 · SNP · Colorectal cancer · Meta-analysis

Chang-long Guo and Fei-fei Han contributed equally to this work.

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Introduction

Somatic DNA is continuously exposed to the assaults by various endogenous and exogenous mutagens or carcinogens. Oxidants, as one of the most common threats to genomic stability, are thought to cause oxidative damage to

DNA and mutations, leading to the carcinogenesis (Loft et al. 1998; Marnett 2000). However, multiple DNA repair enzymes protect DNA against such oxidative damage. So DNA repair mechanisms are important for maintaining DNA integrity and preventing carcinogenesis (Kiyohara et al. 2006).

The base excision repair (BER) pathway is the most common route for removal of small lesions from DNA and is an important part of cellular defense against a large variety of structurally unrelated DNA lesions. It is believed to be the predominant pathway used for removal of oxidized and many alkylated bases (Marnett 2000; Barzilai and Yamamoto 2004). BER is initiated by human 8-oxoguanine DNA glycosylase (OGG1). OGG1 maps on chromosome 3p26.2 and encodes the enzyme responsible for the excision of 8-oxoguanine, a mutagenic base byproduct which occurs as a result of exposure to reactive oxygen. The action of this enzyme includes lyase activity for chain cleavage. OGG1 gene has at least 20 validated sequence variants, and one of the most studied functional polymorphism is Ser326Cys (exon 7 of the OGG1 gene, rs1052133) (Dherin et al. 1999; Gerhard et al. 2004; Vodicka et al. 2007). It is believed that 326Cys allele was associated with reduced enzyme activity, DNA repair ability, and increased cancer risk, such as lung cancer (Sugimura et al. 1999), esophageal cancer (Xing et al. 2001), breast cancer (Sangrajrang et al. 2008), colon cancer (Kim et al. 2003), and so on. The exact mechanisms how the hOGG1 Ser326Cys polymorphism affects cancer risk at the molecular level remain to be unraveled, and the published studies on the structure and biological functions of hOGG1 gene as well as their genetic variants did not discover the potential roles of this polymorphism (Obtulowicz et al. 2010; Park et al. 2001).

Colorectal cancer is the third most common cancer and the leading cause of cancer deaths in Western industrialized countries (Hansen et al. 2007). Genetic characteristics and exogenous factors such as smoking, alcohol, polyunsaturated fatty acids, and a diet high in red meat are convincing colorectal cancer (CRC) (Brevik et al. 2010; Kim et al. 2003; Hansen et al. 2007; Kasahara et al. 2008; Stern et al. 2009). Previous researches have revealed the association between hOGG1Ser326Cys polymorphism and colorectal cancer risk (Obtulowicz et al. 2010; Moreno et al. 2006; Pardini et al. 2008; Canbay et al. 2011; Gil et al. 2011). However, the results were conflicting, including an increased risk (Obtulowicz et al. 2010; Moreno et al. 2006), a reduced risk (Hansen et al. 2005), and no association (Park et al. 2007; Kasahara et al. 2008).

The aim of this article is to review and evaluate associations between OGG1Ser326Cys and colorectal cancer risk, focusing on different populations and smoking factors.

Materials and methods

Identification and eligibility of relevant studies

To identify all articles that examined the association between hOGG1 Ser326Cys polymorphism and colorectal cancer risk, we conducted a search in the PubMed, Embase, Google Scholar, and Cbmdisc database (before 2011-5-27) using the terms including human 8-oxoguanine DNA glycosylase or hOGG1 or OGG1 or OGG, polymorphism or genetic variation, and colorectal cancer or colon cancer or rectal cancer. Additional articles were identified through the references cited in the first series of articles selected. Articles included in meta-analysis were in English, with human subjects, published in primary literature, and with no obvious overlap of subjects with other studies. The retrieved literatures were then read in their entirety to assess their appropriateness for the inclusion in this meta-analysis. Conference abstracts, case reports, editorials, review articles, and letters were excluded. Studies included in this meta-analysis had to meet the following criteria: an unrelated case–control design was used, genotype frequency was available, and the genotype distribution in controls was in Hardy–Weinberg equilibrium in all studies (Yuan et al. 2010). Using PubMed database, we identified 12 epidemiological studies that provided information on colorectal cancer occurrence associated with OGG1 Ser326Cys polymorphism.

Data extraction and assessment of study quality

Two authors (Fei-fei Han and Chang-long Guo) extracted data and reached a consensus on all of the eligibility items, including author, journal and year of publication, location of study, selection and characteristics of cancer cases and controls, control source, demographics, ethnicity, smoking status, and genotyping information. For those studies that included subjects of different ethnic groups, data were extracted separately for each of ethnic groups categorized as Asians, Caucasians, and American. We assessed the homogeneity of the study population.

Meta-analysis

The risks (odds ratios, OR) of colorectal cancer associated with hOGG1 Ser326Cys polymorphism were estimated for each study independently. We estimated the risk for the 326Cys allele, Cys/Cys versus Ser/Ser, Cys/Cys plus Ser/Cys versus Ser/Ser, and Cys/Cys versus Ser/Cys plus Ser/Ser.

Statistical analysis

The meta-analysis was performed in a fixed/random effect model. The OR and its 95 % CI were estimated for each study. The chi-squared test-based Q-statistic was used to assess the between-study heterogeneity. Heterogeneity was significant for $P < 0.10$, and then, the result of the random effect model was selected. Otherwise, the result of fixed effect model was selected. Meanwhile, we measured the effect of heterogeneity by another measure, $I^2 = 100\ \% \times (Q - df)/Q$. The I^2 statistic measures the degree of inconsistency in the studies by calculating what percentage of the total variation across studies is as a result of heterogeneity rather than by chance.

The effect of association was indicated as OR with the corresponding 95 % confidence interval (CI). The combined OR was estimated using fixed effects (FE) models (Mantel–Haenszel) and random effects (RE) models (DerSimonian and Laird) (Lau et al. 1997). We did the Q test to assess the heterogeneity between these studies, and it was considered statistically significant with $P < 0.10$ (Yuan et al. 2010). The heterogeneity was quantified by I^2 metric ($I^2 = 100\ \% \times (Q - df)/Q$), which is independent of the number of studies in the meta-analysis ($I^2 < 25\ \%$ no heterogeneity; $I^2 = 25$–$50\ \%$ moderate heterogeneity; $I^2 > 50\ \%$ extreme heterogeneity) and P value ($P > 0.1$ no heterogeneity). Publication bias was investigated by funnel plot and Egger's linear regression test (Egger et al. 1997). The significance of asymmetry was determined by t test, and $P < 0.05$ was considered a significant publication bias. Hardy–Weinberg equilibrium (HWE) was tested by the chi-square test. Meta-analysis was performed using stata/MP 11.0. Sensitivity analysis was performed by sequential removal (statistics of study remove) of individual studies (Review Manager 5.0 software).

Results

Eligible studies for meta-analysis

The studies focusing on hOGG1 Ser326Cys polymorphism and colorectal cancer were chosen. After a careful evaluation of the published literature, only 12 studies met our inclusion criteria for the meta-analysis. The retrieved papers were then read in their entirety to assess their appropriateness for the inclusion in this meta-analysis. The basic information including cancer type, ethnicity of the study populations, and the number of cases and controls of each study are listed in Table 1. In all studies, the cases were histologically confirmed, and the controls were free of colorectal cancer.

In the total 12 studies, 2 articles provided the data of smoking status (Kim et al. 2003; Curtin et al. 2009) and three of them have colon cancer data (Kim et al. 2003; Pardini et al. 2008; Curtin et al. 2009). These researches were conducted in different populations of ethnicities: seven studies were involved in the European populations (Engin et al. 2010; Hansen et al. 2005; Moreno et al. 2006; Pardini et al. 2008; Sliwinski et al. 2009), two were American populations (Brevik et al. 2010; Curtin et al. 2009), and three were Asian populations (Kim et al. 2003; Park et al. 2007; Stern et al. 2007).

Summary statistic

We included 4,026 cancer patients and 5,862 control subjects in the final analysis. Details were listed in Table 1. Frequency of genotype and allele was shown in Table 2. All studies of control were in HWE ($P > 0.05$).

Table 1 Characteristics of included studies in meta-analysis

Year	First author	National	Cases				Controls				HW (P)
			Ser/Ser	Ser/Cys	Cys/Cys	Total	Ser/Ser	Ser/Cys	Cys/Cys	Total	
2010	Asgeir Brevik	USA	172	117	19	308	217	127	18	362	0.916317
2010	Ayse Basak Engin	Turkish	50	43	17	110	51	47	18	116	0.202622
2007	B. Pardini	Czech	336	168	28	532	331	181	20	532	0.436796
2011	Emel Canbay	Turkish	31	40	8	79	171	69	7	247	0.990075
2007	Hye-Won Park	Korea	91	220	128	439	120	333	223	676	0.822604
2003	Jae-IL Kim	Korea	24	66	35	125	52	131	64	247	0.320287
2009	Karen Curtin	USA	918	570	94	1,582	1,172	686	93	1,951	0.562843
2007	Mariana C. Stern	Singapore	35	152	116	303	183	537	439	1,159	0.379570
2005	Rikke Hansen	Norway	101	55	9	165	208	164	24	396	0.262194
2010	Tomasz Obtulowicz	Poland	38	19	17	74	63	33	1	97	0.138524
2009	Tomasz Sliwinski	Poland	52	46	2	100	68	28	4	100	0.606619
2006	Victor Moreno	Spanish	225	114	23	362	210	104	9	323	0.360000

Table 2 The ORs of hOGG1 Ser326Cys polymorphism, ethnicity, and smoking status with colorectal cancer

Allele and genotype	Populations	OR	I^2 (%)	$P_{heterogeneity}$	P	Model
326Cys allele (an additive model)	All	1.082 (0.956, 1.223)	70.8	0	0.212	D+L pooled OR
	Asian	0.994 (0.905, 1.092)	0	0.598	0.902	M−H pooled OR
	European	1.234 (0.973, 1.565)	73.1	0.001	0.084	D+L pooled OR
	American	0.952 (0.731, 1.242)	75.2	0.45	0.719	D+L pooled OR
	European + American	1.148 (0.95, 1.338)	78.2	0.00	0.152	D+L pooled OR
	Colon carcinoma	1.056 (0.966, 1.155)	0	0.884	0.23	M−H pooled OR
Cys/Cys versus Ser/Ser (homozygote contrast)	All	1.243 (0.979, 1.578)	53	0.016	0.074	D+L pooled OR
	Asian	1.000 (0.835, 1.199)	0	0.599	0.996	M−H pooled OR
	European	1.717 (0.936, 3.149)	65.2	0.008	0.081	D+L pooled OR
	American	1.269 (0.966, 1.667)	0	0.942	0.087	M−H pooled OR
	European + American	1.494 (1.023, 2.181)	54.9	0.023	0.038	D+L pooled OR
	Colon carcinoma	1.235 (0.975, 1.564)	0	0.825	0.081	M−H pooled OR
Cys/Cys + Cys/Ser versus Ser/Ser (dominant genetic model)	All	1.045 (0.975, 1.121)	24.3	0.205	0.213	M−H pooled OR
	Asian	1.007 (0.894, 1.136)	0	0.804	0.904	M−H pooled OR
	European	1.135 (0.918, 1.403)	55.2	0.037	0.243	D+L pooled OR
	American	1.059 (0.947, 1.185)	0	0.76	0.315	M−H pooled OR
	European + American	1.093 (0.957, 1.248)	40.8	0.095	0.188	D+L pooled OR
	Colon carcinoma	1.03 (0.92, 1.17)	43.6	0.17	0.58	M−H pooled OR
Cys/Cys versus Cys/Ser + Ser/Ser (recessive genetic model)	All	1.198 (0.959, 1.496)	51.6	0.019	0.111	D+L pooled OR
	Asian	0.962 (0.818, 1.131)	0	0.654	0.64	M−H pooled OR
	European	1.64 (0.936, 2.873)	60.8	0.018	0.084	D+L pooled OR
	American	1.246 (0.952, 1.63)	0	0.99	0.11	M−H pooled OR
	European + American	1.444 (1.017, 2.05)	49.5	0.045	**0.04**	D+L pooled OR
	Colon carcinoma	1.226 (0.976, 1.541)	0	0.782	0.08	M−H pooled OR
	Smokers	1.354 (0.967, 1.896)	0	0.908	0.077	M−H pooled OR
	Non-smokers	1.132 (0.832, 1.58)	0	0.886	0.431	M−H pooled OR

Bold value indicates that this result was not robust enough

Allele and subgroup analysis

Individuals carrying the hOGG1 Cys/Cys genotype did not have significantly increased colorectal cancer risk compared with those carrying the Ser/Ser genotype (OR = 1.234; 95 % CI: 0.979–1.578; $P = 0.074$) and Cys/Cys allele (OR = 1.146; 95 % CI: 0.978–1.342, $P = 0.091$). Similarly, no significant association with colorectal cancer risk was found in either a recessive model (OR = 1.198; 95 % CI: 0.959–1.496, $P = 0.111$ for Cys/Cys vs. Ser/Cys + Ser/Ser) or a dominant model (OR = 1.045; 95 % CI: 0.975–1.121, $P = 0.213$ for Cys/Cys + Ser/Cys vs. Ser/Ser).

In the stratified analysis by ethnicity, significantly increased risks were found among European plus American subjects, who are mostly Caucasian (OR = 1.444; 95 % CI: 1.017–2.05 Cys/Cys vs. Ser/Cys + Ser/Ser; $P = 0.04$) in a recessive model and (OR = 1.494; 95 % CI: 1.023–2.181; $P = 0.038$) in a homozygote contrast. But in sensitivity analysis, we found that several studies influence the corresponding pooled ORs. So we divided this group into two subjects, one for European population and another for American population. Then the result suggested that there were no association between hOGG1 Ser326Cys polymorphism and colorectal cancer among both of these two populations (Table 2).

However, among Asian subjects, no significant association with colorectal cancer risk was found in either a recessive model (OR = 0.962; 95 % CI: 0.818–1.131 for Cys/Cys vs. Ser/Cys + Ser/Ser; $P = 0.64$) or a dominant genetic model (OR = 1.007; 95 % CI: 0.894–1.136; $P = 0.804$).

There was no significant association with hOGG1 Ser326Cys allele in a homozygote contrast and colorectal cancer (OR = 1.354, 95 % CI: 0.967–1.896 for Cys/Cys vs. Cys/Ser + Ser/Ser; $P = 0.077$) in smokers.

Publication bias

Funnel plots and Egger's test were performed to assess the publication bias. Publication bias was significant in the meta-analysis for Cys allele ($t = 3.4$, $P = 0.007$), while its meta-analysis was not significant. The Egger's test data suggested that there was no evidence of publication bias in hOGG1 Cys/Cys versus Ser/Cys + Ser/Ser ($t = 2.33$, $P = 0.05$), Ser326Cys Cys/Cys + Ser/Cys versus Ser/Ser ($t = 1.41$, $P = 0.189$), and Cys/Cys versus Ser/Ser ($t = 2.01$, $P = 0.072$) analysis.

Discussion

The results of epidemiological studies of common polymorphisms in DNA repair genes, if large and unbiased, can provide insight into the relationships between DNA repair genes, polymorphisms, pathways, and colorectal cancer risk, and also lead to an increased understanding of the public health dimension of DNA repair variation. Meta-analysis is a powerful method for resolving inconsistent finding with a relatively large number of subjects. Mounts of researches about the risks between hOGG1 Ser326Cys polymorphism and different kinds of cancers are very big, such as lung cancer, esophageal cancer, breast cancer, and colon cancer. Most of those cancer types have been summarized by meta-analysis. In the past several years, many studies have indicated that hOGG1 Ser326Cys polymorphism was associated with colorectal cancer risk, maybe because of the relatively small sample size and different genetic background, and the results remain inconsistent. So far, there is no report of meta-analysis about colon cancer and hOGG1 Ser326Cys polymorphism. In this study, meta-analysis was used to summarize the association from the current literatures and to explore sources of heterogeneity.

A total of 12 publications containing 4,026 cases and 5,826 controls were selected in the study. However, no significant effects were observed in 326Cys allele compared to 326Ser allele and other genetic contrasts (homologous contrast, dominant genetic model, and recessive genetic model) on colorectal cancer risk in all subjects. As all know, the pathogenesis of colorectal cancer is complex: several factors including different ethnicity, environmental factors, and the interactions between gene–gene and gene–environment are all involved in this process and make contribution to the genesis of colorectal cancer. Subsequently, we evaluated the role of Ser326Cys polymorphism in different subgroups (European population, Americans, and Asians). Firstly, European and American people were considered together as one group (Caucasians), and results revealed that the people carrying hOGG1 Cys326Cys might have more risk than other types. However, we found that several studies influenced the OR in the sensitivity analysis, so it cannot get the robust conclusion that the Caucasians have more risks than other people. But when analyzed separately, it got negative result. In short, the analysis suggested that there is no association between hOGG1 Ser326Cys polymorphism and colorectal cancer both in European and in American populations.

Also smoking is an oxidative stress risk factor; to evaluate the association between smoking and susceptibilities of colorectal cancer, data of smokers and non-smokers were extracted and analyzed from two researches, and the results showed that there was no evidence for smokers carrying Cys/Cys had more risk for colorectal cancer than non-smokers. More detailed and complete researches are needed for this issue to get a more precise conclusion.

Limitation of this meta-analysis should be acknowledged. Some studies evaluated associations between hOGG1 Ser326Cys and colorectal cancer risk in several subgroups of populations, such as associations among smokers with colorectal cancer. There are only two previous studies that consistent with our standard provide the association between smoking and hOGG1 Ser326Cys polymorphism. It is difficult for a meta-analysis to derive such specific associations, because the results from previous studies were not presented in a uniform standard.

Epidemiological studies revealed that many factors were included in the pathology of colorectal cancer. However, none of the single element had been reported to play the pivotal role in the genesis of colorectal cancer. Further synthetic and intimate researches about BER (hOGG1, XRCC1, and APEX1) and other related causes such as age at diagnosis, eating habit, medical history, and stage at diagnosis were needed for the etiological factor of colorectal cancer.

In summary, this meta-analysis of the 12 studies strongly suggests that hOGG1 Ser326Cys polymorphism is not associated with colorectal cancer. However, due to the small subjects included in analysis and the selection bias existed in some studies, the results for the smoking population should be interpreted with caution.

Conflict of interest All authors have no conflicts of interest.

References

Barzilai A, Yamamoto K (2004) DNA damage responses to oxidative stress. DNA Repair (Amst) 3:1109–1115

Brevik A, Joshi AD, Corral R, Onland-Moret NC, Siegmund KD, Le Marchand L, Baron JA, Martinez ME, Haile RW, Ahnen DJ, Sandler RS, Lance P, Stern MC (2010) Polymorphisms in base excision repair genes as colorectal cancer risk factors and modifiers of the effect of diets high in red meat. Cancer Epidemiol Biomarker Prev 19:3167–3173

Canbay E, Cakmakoglu B, Zeybek U, Sozen S, Cacina C, Gulluoglu M, Balik E, Bulut T, Yamaner S, Bugra D (2011) Association of APE1 and hOGG1 polymorphisms with colorectal cancer risk in a Turkish population. Curr Med Res Opin 27:1295–1302

Curtin K, Samowitz WS, Wolff RK, Ulrich CM, Caan BJ, Potter JD, Slattery ML (2009) Assessing tumor mutations to gain insight into base excision repair sequence polymorphisms and smoking in colon cancer. Cancer Epidemiol Biomarkers Prev 18:3384–3388

Dherin C, Radicella JP, Dizdaroglu M, Boiteux S (1999) Excision of oxidatively damaged DNA bases by the human alpha-hOgg1 protein and the polymorphic alpha-hOgg1(Ser326Cys) protein which is frequently found in human populations. Nucleic Acids Res 27:4001–4007

Egger M, Davey Smith G, Schneider M, Minder C (1997) Bias in meta-analysis detected by a simple, graphical test. BMJ 315:629–634

Engin AB, Karahalil B, Engin A, Karakaya AE (2010) Oxidative stress, *Helicobacter pylori*, and OGG1 Ser326Cys, XPC Lys939Gln, and XPD Lys751Gln polymorphisms in a Turkish population with colorectal carcinoma. Genet Test Mol Biomarkers 14:559–564

Gerhard DS, Wagner L, Feingold EA, Shenmen CM, Grouse LH, Schuler G, Klein SL, Old S, Rasooly R, Good P, Guyer M, Peck AM, Derge JG, Lipman D, Collins FS, Jang W, Sherry S, Feolo M, Misquitta L, Lee E, Rotmistrovsky K, Greenhut SF, Schaefer CF, Buetow K, Bonner TI, Haussler D, Kent J, Kiekhaus M, Furey T, Brent M, Prange C, Schreiber K, Shapiro N, Bhat NK, Hopkins RF, Hsie F, Driscoll T, Soares MB, Casavant TL, Scheetz TE, Brownstein MJ, Usdin TB, Toshiyuki S, Carninci P, Piao Y, Dudekula DB, Ko MS, Kawakami K, Suzuki Y, Sugano S, Gruber CE, Smith MR, Simmons B, Moore T, Waterman R, Johnson SL, Ruan Y, Wei CL, Mathavan S, Gunaratne PH, Wu J, Garcia AM, Hulyk SW, Fuh E, Yuan Y, Sneed A, Kowis C, Hodgson A, Muzny DM, McPherson J, Gibbs RA, Fahey J, Helton E, Ketteman M, Madan A, Rodrigues S, Sanchez A, Whiting M, Madari A, Young AC, Wetherby KD, Granite SJ, Kwong PN, Brinkley CP, Pearson RL, Bouffard GG, Blakesly RW, Green ED, Dickson MC, Rodriguez AC, Grimwood J, Schmutz J, Myers RM, Butterfield YS, Griffith M, Griffith OL, Krzywinski MI, Liao N, Morin R, Palmquist D, Petrescu AS, Skalska U, Smailus DE, Stott JM, Schnerch A, Schein JE, Jones SJ, Holt RA, Baross A, Marra MA, Clifton S, Makowski KA, Bosak S, Malek J (2004) The status, quality, and expansion of the NIH full-length cDNA project: the Mammalian Gene Collection (MGC). Genome Res 14:2121–2127

Gil J, Ramsey D, Stembalska A, Karpinski P, Pesz KA, Laczmanska I, Leszczynski P, Grzebieniak Z, Sasiadek MM (2011) The C/A polymorphism in intron 11 of the XPC gene plays a crucial role in the modulation of an individual's susceptibility to sporadic colorectal cancer. Curr Med Res Opin 27(7):1295–1302

Hansen R, Saebo M, Skjelbred CF, Nexo BA, Hagen PC, Bock G, Bowitz Lothe IM, Johnson E, Aase S, Hansteen IL, Vogel U, Kure EH (2005) GPX Pro198Leu and OGG1 Ser326Cys polymorphisms and risk of development of colorectal adenomas and colorectal cancer. Cancer Lett 229:85–91

Hansen RD, Sorensen M, Tjonneland A, Overvad K, Wallin H, Raaschou-Nielsen O, Vogel U (2007) XPA A23G, XPC Lys939Gln, XPD Lys751Gln and XPD Asp312Asn polymorphisms, interactions with smoking, alcohol and dietary factors, and risk of colorectal cancer. Mutat Res 619:68–80

Kasahara M, Osawa K, Yoshida K, Miyaishi A, Osawa Y, Inoue N, Tsutou A, Tabuchi Y, Tanaka K, Yamamoto M, Shimada E, Takahashi J (2008) Association of MUTYH Gln324His and APEX1 Asp148Glu with colorectal cancer and smoking in a Japanese population. J Exp Clin Cancer Res 27:49

Kim JI, Park YJ, Kim KH, Kim JI, Song BJ, Lee MS, Kim CN, Chang SH (2003) hOGG1 Ser326Cys polymorphism modifies the significance of the environmental risk factor for colon cancer. World J Gastroenterol 9:956–960

Kiyohara C, Takayama K, Nakanishi Y (2006) Association of genetic polymorphisms in the base excision repair pathway with lung cancer risk: a meta-analysis. Lung Cancer 54:267–283

Lau J, Ioannidis JP, Schmid CH (1997) Quantitative synthesis in systematic reviews. Ann Intern Med 127:820–826

Loft S, Deng XS, Tuo J, Wellejus A, Sorensen M, Poulsen HE (1998) Experimental study of oxidative DNA damage. Free Radic Res 29:525–539

Marnett LJ (2000) Oxyradicals and DNA damage. Carcinogenesis 21:361–370

Moreno V, Gemignani F, Landi S, Gioia-Patricola L, Chabrier A, Blanco I, Gonzalez S, Guino E, Capella G, Canzian F (2006) Polymorphisms in genes of nucleotide and base excision repair: risk and prognosis of colorectal cancer. Clin Cancer Res 12:2101–2108

Obtulowicz T, Swoboda M, Speina E, Gackowski D, Rozalski R, Siomek A, Janik J, Janowska B, Ciesla JM, Jawien A, Banaszkiewicz Z, Guz J, Dziaman T, Szpila A, Olinski R, Tudek B (2010) Oxidative stress and 8-oxoguanine repair are enhanced in colon adenoma and carcinoma patients. Mutagenesis 25:463–471

Pardini B, Naccarati A, Novotny J, Smerhovsky Z, Vodickova L, Polakova V, Hanova M, Slyskova J, Tulupova E, Kumar R, Bortlik M, Barale R, Hemminki K, Vodicka P (2008) DNA repair genetic polymorphisms and risk of colorectal cancer in the Czech Republic. Mutat Res 638:146–153

Park YJ, Choi EY, Choi JY, Park JG, You HJ, Chung MH (2001) Genetic changes of hOGG1 and the activity of oh8Gua glycosylase in colon cancer. Eur J Cancer 37:340–346

Park HW, Kim IJ, Kang HC, Jang SG, Ahn SA, Lee JS, Shin HR, Park JG (2007) The hOGG1 Ser326Cys polymorphism is not associated with colorectal cancer risk. J Epidemiol 17:156–160

Sangrajrang S, Schmezer P, Burkholder I, Waas P, Boffetta P, Brennan P, Bartsch H, Wiangnon S, Popanda O (2008) Polymorphisms in three base excision repair genes and breast cancer risk in Thai women. Breast Cancer Res Treat 111:279–288

Sliwinski T, Krupa R, Wisniewska-Jarosinska M, Pawlowska E, Lech J, Chojnacki J, Blasiak J (2009) Common polymorphisms in the XPD and hOGG1 genes are not associated with the risk of colorectal cancer in a Polish population. Tohoku J Exp Med 218:185–191

Stern MC, Conti DV, Siegmund KD, Corral R, Yuan JM, Koh WP, Yu MC (2007) DNA repair single-nucleotide polymorphisms in colorectal cancer and their role as modifiers of the effect of cigarette smoking and alcohol in the Singapore Chinese Health Study. Cancer Epidemiol Biomarkers Prev 16:2363–2372

Stern MC, Butler LM, Corral R, Joshi AD, Yuan JM, Koh WP, Yu MC (2009) Polyunsaturated fatty acids, DNA repair single nucleotide polymorphisms and colorectal cancer in the Singapore Chinese Health Study. J Nutrigenet Nutrigenomics 2:273–279

Sugimura H, Kohno T, Wakai K, Nagura K, Genka K, Igarashi H, Morris BJ, Baba S, Ohno Y, Gao C, Li Z, Wang J, Takezaki T, Tajima K, Varga T, Sawaguchi T, Lum JK, Martinson JJ, Tsugane S, Iwamasa T, Shinmura K, Yokota J (1999) hOGG1 Ser326Cys polymorphism and lung cancer susceptibility. Cancer Epidemiol Biomarkers Prev 8:669–674

Vodicka P, Stetina R, Polakova V, Tulupova E, Naccarati A, Vodickova L, Kumar R, Hanova M, Pardini B, Slyskova J, Musak L, De Palma G, Soucek P, Hemminki K (2007) Association of DNA repair polymorphisms with DNA repair functional outcomes in healthy human subjects. Carcinogenesis 28:657–664

Xing DY, Tan W, Song N, Lin DX (2001) Ser326Cys polymorphism in hOGG1 gene and risk of esophageal cancer in a Chinese population. Int J Cancer 95:140–143

Yuan W, Xu L, Feng Y, Yang Y, Chen W, Wang J, Pang D, Li D (2010) The hOGG1 Ser326Cys polymorphism and breast cancer risk: a meta-analysis. Breast Cancer Res Treat 122:835–842