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Original Article

XPD Asp312Asn polymorphism and esophageal cancer risk: an update meta-analysis based on 3928 cases and 6012 controls

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Abstract: Background: Although xeroderma pigmentosum group D (XPD) was reported to be related with esophageal cancer (EC) risk, the results remained inconsistent. The aim of this meta-analysis was to make a more precise estimation of the relationship between XPD Asp312Asn polymorphism and EC risk. Methods: We searched PubMed, Web of Science, Embase, Medline, CNKI and Chinese Biomedical database, covering all publications (up to May, 2014). Statistical analyses were performed with Stata software (version 12.0, USA) and RevMan 5.1 (Copenhagen, 2008). The calculation of odds ratios (ORs) with 95% confidence intervals (CI) was calculated to assess the strength of the association. Results: A total of 15 case-control studies from 13 literatures including 3928 cases and 6012 controls described Asp312Asn genotypes and EC risk. A significant association between XPD Asp312Asn polymorphism and EC risk was found when all the eligible studies were pooled into this meta-analysis. It's also the same result in subgroup analysis of smokers in dominant model (OR=1.63, 95% CI: 1.06-2.50, *P*=0.03). However, in the stratified analysis by ethnicity and source of population controls, no association between them was discovered. Conclusion: The XPD Asp312Asn polymorphism was proved to contribute to the risk of EC in this meta-analysis. Data showed that tobacco consumption may increase the susceptibility of EC.

Keywords: XPD polymorphisms, esophageal cancers, ESCC, EADC, meta-analysis

Introduction

Esophageal cancer (EC) ranked the sixth in leading cause of malignancies in the world, consisting of esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EADC) [1]. The annual morbidity of ESCC which was the predominant histological type in China was 16.7 per 10 million in average with a higher incidence of males than females [2]. However, the etiology of EC has not been fully understood up to now and was supposed to be multifactorial. A lot of researches have focused on the risk factors, such as heredity, Barrett' esophagus, smoking, drinking and environmental factors [3-5].

DNA repair enzymes continuously monitor chromosomes to correct damaged nucleotide residues generation. Polymorphisms of xeroderma pigmentosum complementation group D (XPD), a type of DNA repair enzymes which could continuously monitor chromosomes to correct damaged nucleotide residues generation [6]. Polymorphisms of XPD which was involved in single-strand breaks and nucleotide excision repair (BER) pathway, was considered as a key factor in the development of EC [7]. Evidences have showed that XPD Asp312Asn (Asp→Asn) polymorphisms is a risk factor to digestive system cancers and other cancers [8-12]. However, the underlying mechanism of carcinogenesis was still unknown. Although some studies have reported that there were significant associations between the XPD Asp312Asn polymorphisms and esophageal cancer risk, the results were inconclusive or inconsistent. So we conduct this meta-analysis to evaluate the association between XPD Asp312Asn polymorphisms and the susceptibility of esophageal cancer systematically.

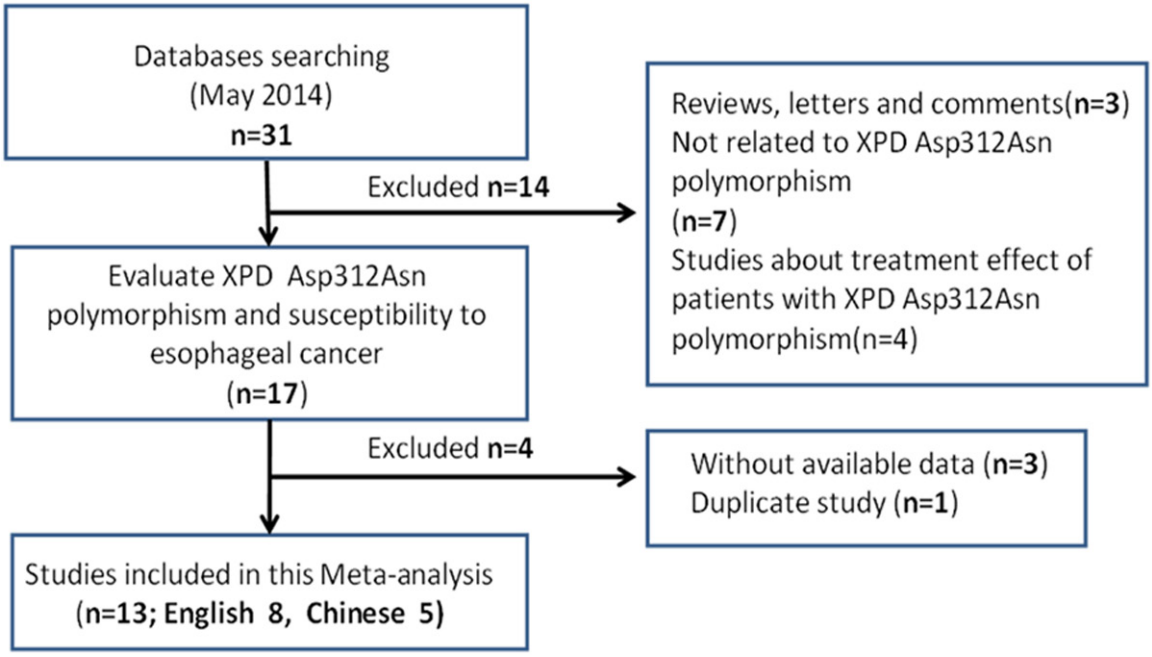

Figure 1. The flow chart of study selection process.

Materials and methods

Search strategy

A computer assisted search was conducted from PubMed, Web of Science, Embase, Medline, CNKI and Chinese Biomedical database (up to May 2014) by using the following key words: 'XPD polymorphisms or XPD Asp312Asn polymorphisms', 'genetic polymorphism or polymorphisms or variant', 'esophageal cancer', 'ERCC1', 'DNA repair gene'. The eligible studies were limited to humans and without language restrictions. If more than one type of cancer were reported in one article, each type of cancer combined with control group was extracted as one independent trial, respectively.

Inclusion and exclusion criteria

Studies were primarily screened by titles and abstracts, and full-texts were obtained to assess the eligibility. The following criteria must be confirmed: (1) the XPD Asp312Asn polymorphisms and esophageal cancer, (2) case-control study design, (3) available data for quantitative synthesis, namely genotype distribution data, (4) for human, (5) studies containing the sample size, the odds ratio (OR) and 95% confidence interval (CI). Major exclusion criteria were: (1) case-only study, (2) no XPD Asp312Asn genotype reported, (3) studies with duplicate data, (4) case reports, and review articles.

Data extraction

All data were extracted from eligible studies by two independent investigators (Guo XF and Wang J) according to the same principle. A consensus results were desired for following analysis. If there were different opinions, discussions should be introduced to reach an agreement. And an expert (Dong WG) would check the information carefully at last. The following characteristics were collected: the first author's name, year of publication, country of origin, ethnicity, cancer type, genotyping methods, number of cases and controls. Ethnicities were categorized as Asian, European and Mixed, while the types of cancer were classified as ESCC and EADC.

Statistical analysis

Cochrane Collaboration RevMan 5.1 (Copenhagen, 2008) was used for this meta-analysis. χ^2-test-based Q statistic test was performed to assess the between- study heterogeneity [13]. And the effect of heterogeneity was quantified by I^2 value. When $P < 0.05$ (Q test) or $I^2 > 50\%$, the heterogeneity across studies was deter-

Table 1. Main characteristics of all studies included in the meta-analysis

Authors	Year	Country	Ethnicity	Control Source	Cancer Type	Genotyping Method	Genotype distribution (case/control)			HWE (*P*)
							Asp/Asp	Asp/Asn	Asn/Asn	
Zhang *et al* [18]	2014	China	Chinese	HB	ESCC	PCR-RFLP	349/354	55/50	1/1	0.579
Huang *et al* [19]	2012	China	Chinese	HB	ESCC	PCR-RFLP	171/298	42/60	0/0	0.084
Wang *et al* [20]	2012	China	Chinese	PB	ESCC	PCR-RFLP	349/354	55/50	1/1	0.580
Li *et al* [21]	2012	China	Chinese	HB	ESCC	PCR-RFLP	342/351	56/47	2/2	0.754
Wu *et al* [22]	2010	China	Chinese	PB	ESCC	PCR-Taq	206/212	28/22	1/1	0.602
Pan *et al* [23]	2009	America	European	HB	ESCC	PCR-Taq	137/201	163/185	43/48	0.581
Zhou *et al* [24]	2007	China	Chinese	PB	ESCC	PCR-RFLP	279/528	46/82	2/2	0.527
Ye *et al* [25]	2006	Sweden	European	PB	ESCC	PCR-RFLP	30/176	41/237	10/57	0.093
Yu *et al* [26]	2004	China	Chinese	HB	ESCC	PCR-RFLP	121/136	14/16	0/0	0.493
Xing *et al* [27]	2003	China	Chinese	HB	ESCC	PCR-RFLP	286/338	38/45	1/0	0.222
Xing *et al* [28]	2002	China	Chinese	HB	ESCC	PCR-RFLP	381/461	49/62	3/1	0.467
Pan *et al* [23]	2009	America	European	HB	EADC	PCR-Taq	16/201	20/185	1/48	0.581
Ye *et al* [25]	2006	Sweden	European	PB	EADC	PCR-RFLP	31/176	51/237	14/57	0.093
Tse *et al* [29]	2008	Canada	Mixed	HB	EADC	PCR-Taq	117/199	150/206	43/49	0.690
Liu *et al* [30]	2007	America	European	HB	EADC	PCR-RFLP	75/144	92/160	16/32	0.190

HWE, Hardy-Weinberg equilibrium; $P<0.05$ was considered statistically significant; PCR-RFLP, polymerase chain reaction-restriction fragment length polymorphism; ESCC, Esophageal Squamous Cell Carcinoma; EADC, Esophageal Adenocarcinoma; HB, Hospital-based; PB, Population-based; *P,* value for HDW.

Table 2. Results of meta-analysis for XPD Asp312Asn polymorphism and esophageal cancer risk

Study group	N	Asn/Asp *vs.* /Asp/Asp		Asn/Asn *vs.* Asp/Asp		(Asn/Asp+Asn/Asn) *vs.* Asp/Asp		Asn/Asn *vs.* (Asn/Asp+Asp/Asp)	
		OR (95% CI	*P*/*P*#	OR (95% CI	*P*/*P*#	OR (95% CI	*P*/*P*#	OR (95% CI	*P*/*P*#
Total	15	1.06 [0.72, 1.57]	0.01/1.00	1.27 [0.99, 1.62]	0.06/0.95	1.14 [1.03, 1.27]	0.01/1.00	1.16 [0.92, 1.46]	0.22/0.94
Cancer type									
ESCC	11	1.13 [0.99, 1.28]	0.07/0.99	1.29 [0.90, 1.86]	0.16/0.99	1.12 [0.99, 1.27]	0.08/0.99	1.18 [0.84, 1.66]	0.34/0.99
EADC	4	1.20 [0.98, 1.48]	0.08/0.95	1.21 [0.88, 1.68]	0.24/0.32	1.20 [0.99, 1.47]	0.06/0.90	1.10 [0.81, 1.49]	0.55/0.30
Ethnicity									
Asian	9	1.10 [0.95, 1.28]	0.20/0.99	1.64 [0.67, 3.98]	0.28/0.97	1.08 [0.94, 1.26]	0.28/1.00	1.62 [0.66, 3.93]	0.29/0.97
European	5	1.20 [0.99, 1.45]	0.06/0.92	1.13 [0.83, 1.52]	0.44/0.56	1.18 [0.99, 1.42]	0.07/0.90	1.02 [0.77, 1.36]	0.87/0.56
Control source									
PB	10	1.12 [0.91, 1.38]	0.27/0.96	1.23 [0.76, 2.00]	0.40/0.97	1.13 [0.93, 1.39]	0.22/0.96	1.84 [0.82, 4.12]	0.14/< 0.05
HB	5	1.16 [1.02, 1.32]	0.02/0.98	1.25 [0.95, 1.66]	0.11/0.69	1.15 [1.01, 1.30]	0.03/0.97	1.13 [0.86, 1.46]	0.38/0.65
Smoking									
Yes	3	1.33 [0.79, 2.24]	0.28/0.52	1.10 [0.17, 7.12]	0.92/0.32	1.63 [1.06, 2.50]	0.03/0.31	1.06 [0.16, 6.86]	0.95/0.33
No	3	1.02 [0.67, 1.57]	0.91/0.73	2.31 [0.40, 13.34]	0.35/0.18	3.29 [0.39, 8.05]	0.28/< 0.05	2.29 [0.40, 13.22]	0.35/0.18

N, number of studies; *P*#, Test for heterogeneity, Random-effect model was used when the *P* value was <0.05, otherwise the fixed-effect model was used; OR, odds ratio; CI, confidence interval; ESCC, Esophageal Squamous Cell Carcinoma; EADC, Esophageal Adenocarcinoma; HB, Hospital-based; PB, Population-based.

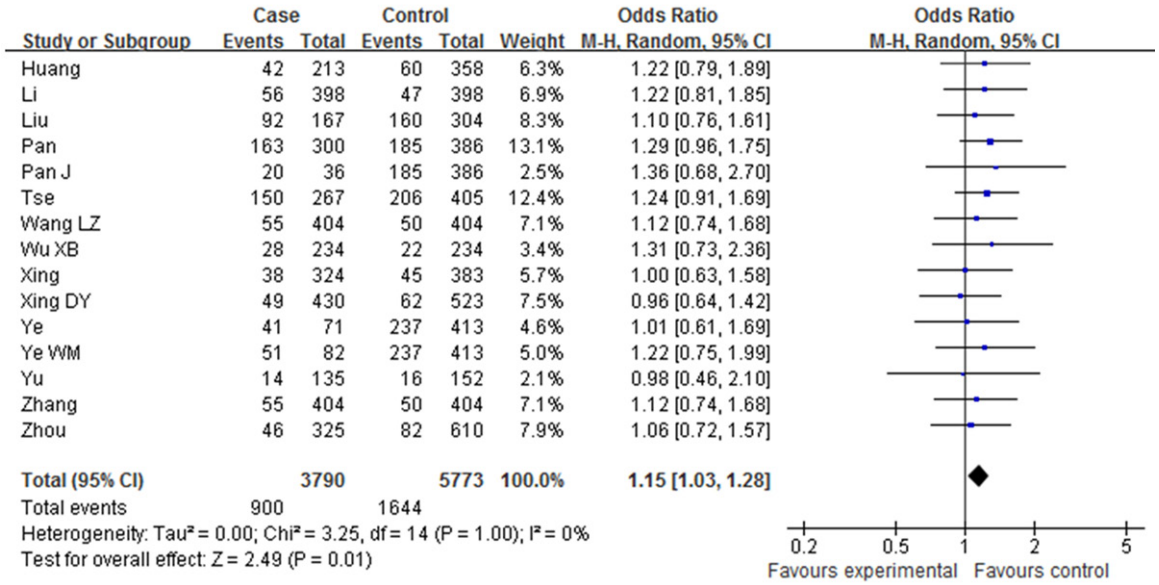

Figure 2. Forest plot of XPD Asp312Asn polymorphisms and EC risk in the overall population. [Asn/Asp *vs.* /Asp/Asp] CI, confidence interval; OR, odds ratio; M-h, Mantel-haenszel.

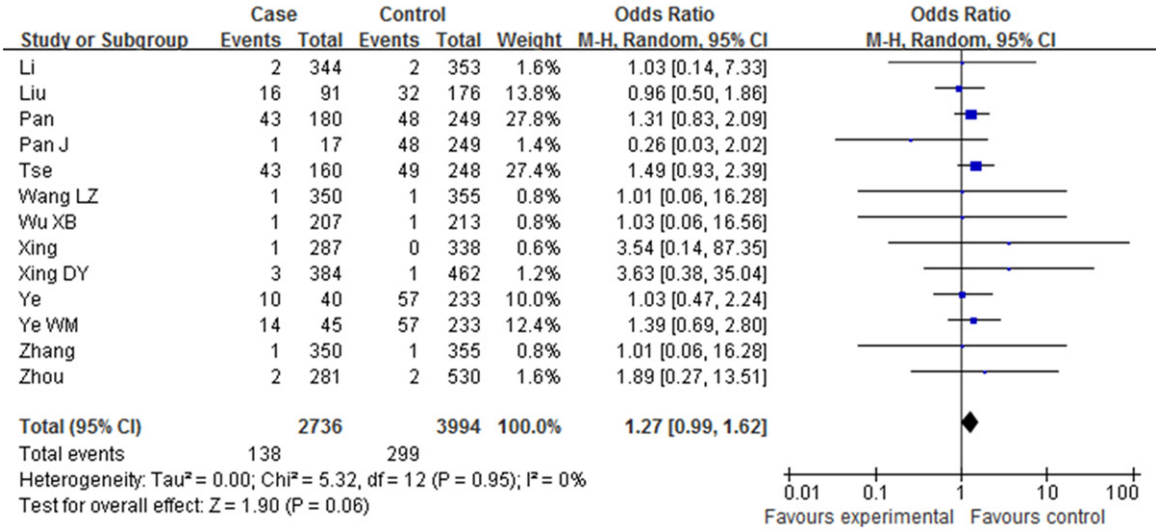

Figure 3. Forest plot of XPD Asp312Asn polymorphisms and EC risk in the overall population. [Asn/Asn *vs.* Asp/Asp] CI, confidence interval; OR, odds ratio; M-h, Mantel-haenszel.

mined, suggesting that the random effects model should be employed [14]. Otherwise, the fixed effects model would be introduced [15]. A professional web-based program (http://ihg2.helmholtz-muen-chen.de/egibin/hw/hwal.pl) was used to assess the Hardy-Weinberg equilibrium of controls. Four genetic comparison models were analyzed in this analysis, including the dominant model (Asn/Asp+Asn/Asn *vs.* Asp/Asp), recessive model (Asn/Asn *vs.* Asn/Asp+Asp/Asp) and the co-dominant model (Asn/Asp *vs.* Asp/Asp and Asn/Asn *vs.* Asp/Asp). The influence of a single study to the whole estimate was tested by removing each study in turn. The publication bias was assessed by Egger's test and Begg' test with STATA Software 12.0 ($P < 0.05$ was considered statistically significant, version 12.0, USA) [16, 17]. Begg's funnel plots were used to evaluate publication bias.

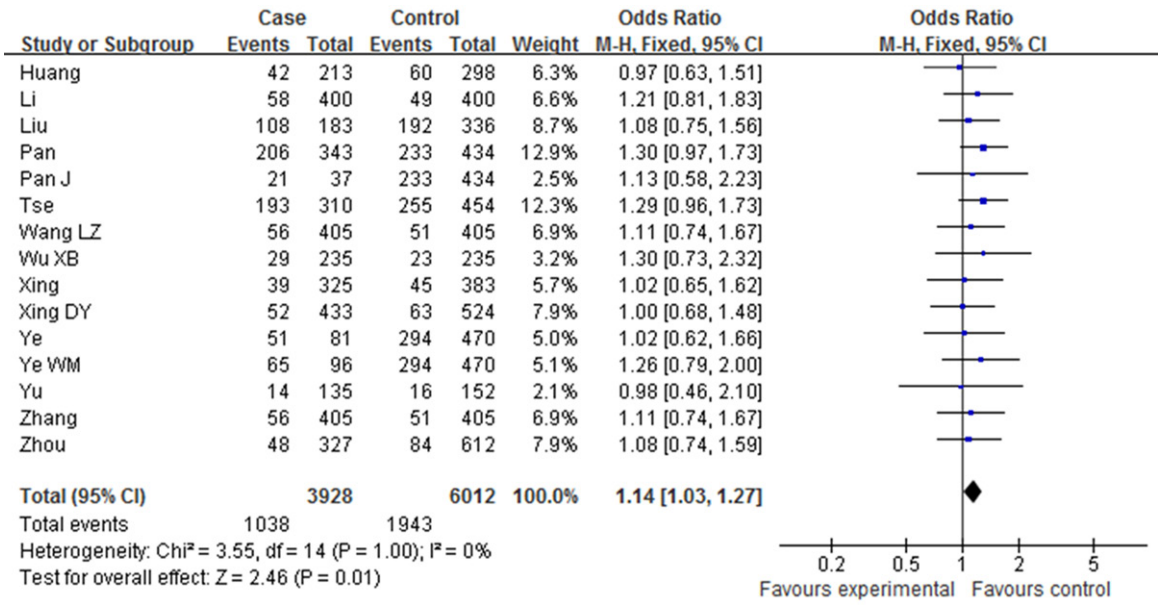

Figure 4. Forest plot of XPD Asp312Asn polymorphisms and EC risk in the overall population. [(Asn/Asp+Asn/Asn) *vs.* Asp/Asp] CI, confidence interval; OR, odds ratio; M-h, Mantel-haenszel.

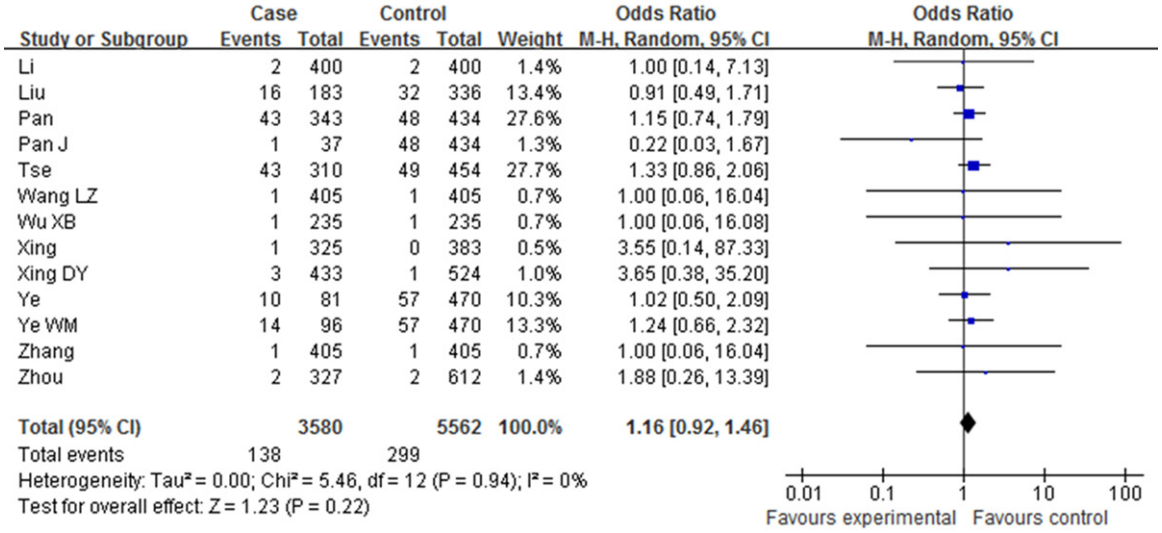

Figure 5. Forest plot of XPD Asp312Asn polymorphisms and EC risk in the overall population. [Asn/Asn *vs.* (Asn/Asp+Asp/Asp)] CI, confidence interval; OR, odds ratio; M-h, Mantel-haenszel.

Results

Study identification and study characteristics

After being examined carefully according to the inclusion criteria, 15 case-control studies from 13 literatures were included in the meta-analysis, containing 3928 cases and 6012 controls, because there were two cancer types in studies of Pan and Ye [22, 24]. The process of selection was shown in **Figure 1**. As shown in **Table 1**, 9 studies were conducted among Chinese population [18-22, 24, 26-28] and 5 studies were among European population [23, 25, 30], while the last one performed by Tse focused on mixed population [29]. In stratified analysis of cancer type, 11 studies were about ESCC [18-28] and the other 4 studies [23, 25, 29, 30] were about EADC. When stratified by population source, controls of 10 studies were from hospital and

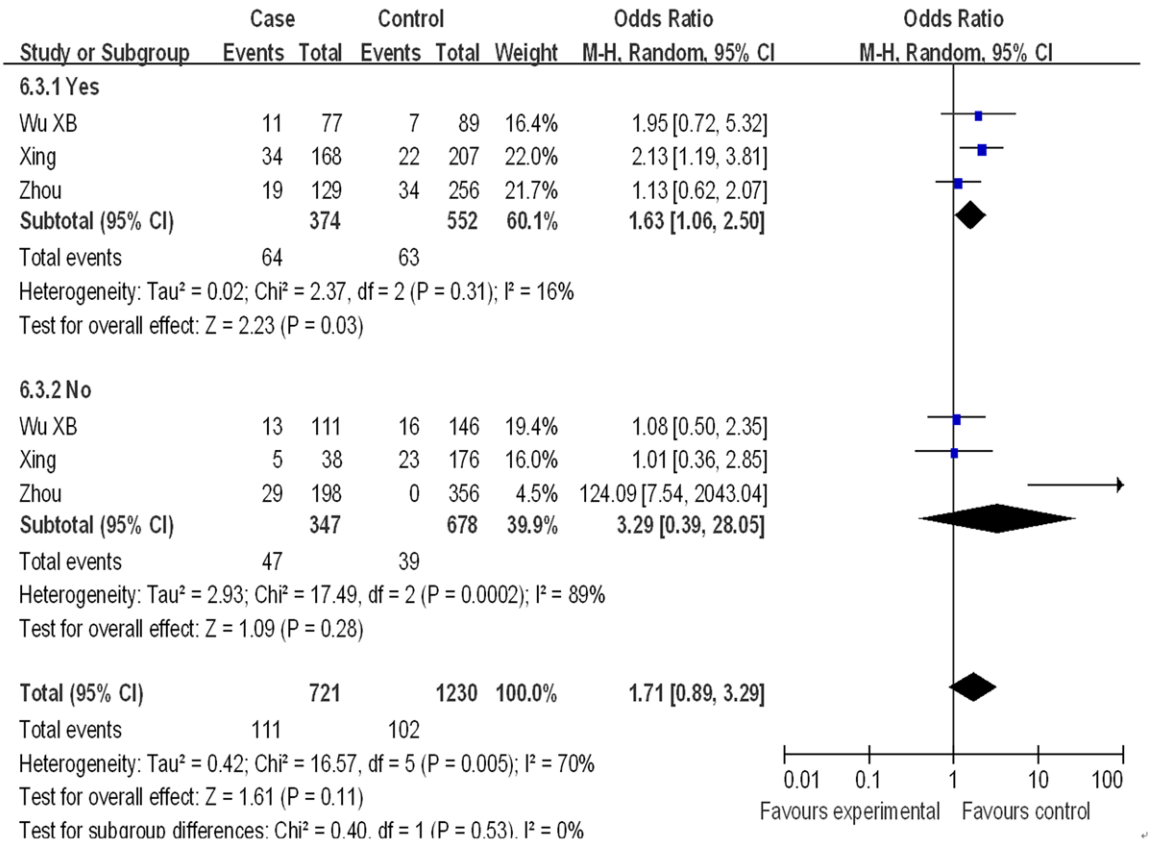

Figure 6. Subgroup analysis by smokers of ORs with a random-effects model for association between XPDAsp312Asn polymorphism and EC risk. [(Asn/Asp+Asn/Asn) *vs.* Asp/Asp] CI, confidence interval; OR, odds ratio; M-h, Mantel-haenszel.

the rest 5 studies were population-based. Controls of all the studies were in accordance with HWE ($P > 0.05$).

Overall analyses

Table 2 showed the main results and the heterogeneity test of meta-analysis. Since there were no heterogeneities among the selected studies in the overall analysis of all the four genetic models ($P > 0.05$, $I^2=0\%$), fixed-effect model was employed in each genotype. The pooled ORs showed significant association between XPD Asp312Asn polymorphism and Esophageal cancer risk in two genetic models [Dominant model (OR=1.14, 95% CI: 1.03-1.27, P=0.01); Asp/Asn vs. Asn/Asn (OR=1.06, 95% CI: 0.72-1.57, P=0.01)]. However, such associations were not found in other two comparisons [Asn/Asn vs. Asp/Asp (OR=1.27, 95% CI: 0.99-1.62, P=0.06); Recessive model (OR=1.16, 95% CI: 0.92-1.46, *P*=0.22) (**Figures 2**-**5**).

Subgroup analyses

In the stratified analysis based on control source, a moderate association between XPD Asp312Asn polymorphism and EC risk were found in hospital-based population [Asp/Asn *vs.* Asn/Asn (OR=1.16, 95% CI: 1.02-1.32, *P*=0.02); Dominant model (OR=1.15, 95% CI: 1.01-1.30, *P*=0.03)], but not in population-based group. When stratified by smoking, there was a significant association only in the dominant model of smokers (OR=1.63, 95% CI: 1.06-2.50, *P*=0.03) (**Figure 6**), indicating that smoking could increase susceptibility of EC. No association was found when stratified by cancer type and ethnicity. Detailed results were shown in **Table 2**.

Sensitive analysis

The influence of each individual study on the pooled results was detected by omitting each-one-out method. There was no heterogeneity in

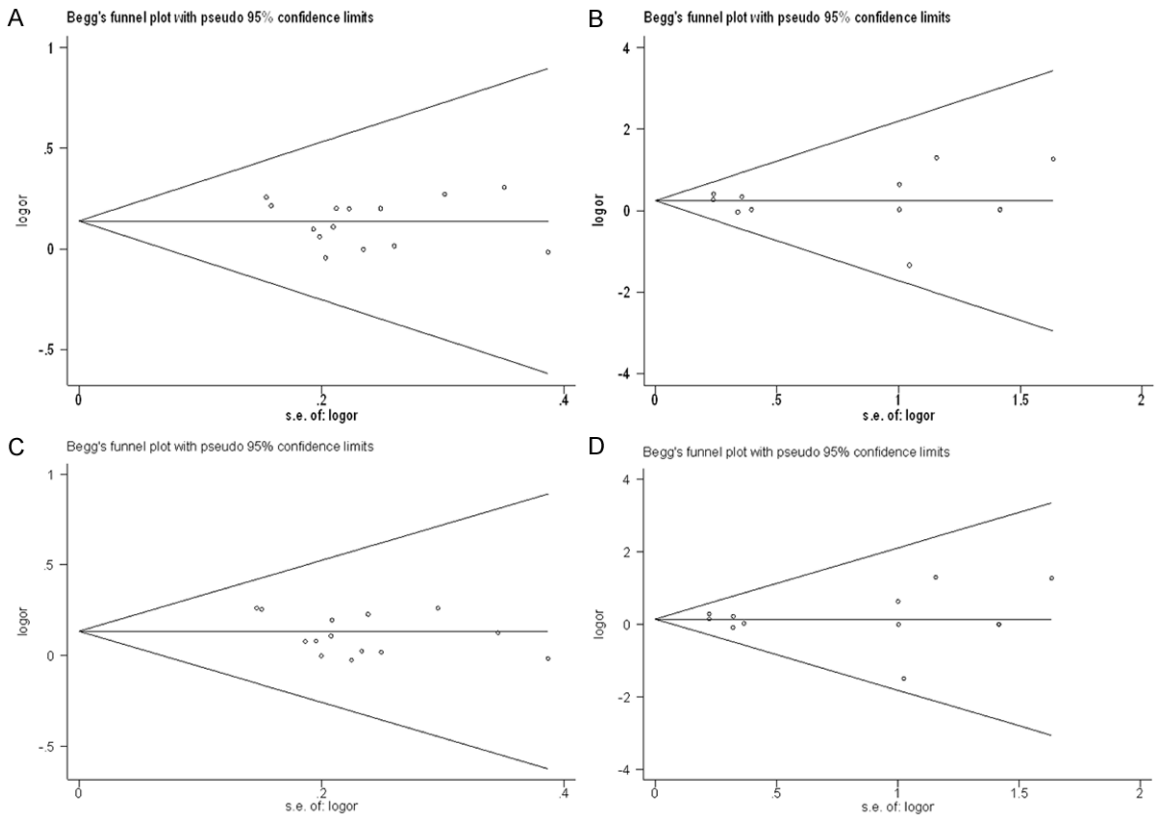

Figure 7. Begg's funnel plot for publication bias of all selected studies. Symmetrical distributions of dots which represent different studies indicated that there was no significant publication bias among selected studies. [A: Asn/Asp *vs.* /Asp/Asp, *P*=0.478; B: Asn/Asn *vs.* /Asp/Asp, *P*=0.732; C: Asn/Asp+Asn/Asn *vs.* Asp/Asp, *P*=0.096; D: Asn/Asn *vs.* Asn/Asp+Asp/Asp, *P*=0.923].

overall and stratified studies, showing that most evidences from our meta-analysis were stable and convincing.

Publication bias

Publication bias of the included trials was assessed by Begg's funnel plot and Egger's test. Overall population analysis of all the four models were tested (**Figure 7**). Results of Egger's test also suggested there was no publication bias in overall populations of this meta-analysis (Asn/Asp *vs.* /Asp/Asp, *P*=0.478; Asn/Asn *vs.* /Asp/Asp, *P*=0.732; Asn/Asp+Asn/Asn *vs.* Asp/Asp, *P*=0.096; Asn/Asn *vs.* Asn/Asp+Asp/Asp, *P*=0.923).

Discussion

XPD gene, also named as excision repair cross-complementing rodent repair deficiency complementation group 2 gene (ERCC2) [18], was reported to encode an ATP-dependent DNA helicase involved in NER (nucleotide excision repair) [29, 30]. Common polymorphisms in DNA repair genes were supposed to alter the functions of corresponding proteins [7]. XPD Asp312Asn (rs1799793) was one of four NER polymorphisms, which was determined to play an important role in the development of EC [3]. However, there were still some disagreements among the investigations. Therefore, in order to clarify the associations between XPD Asp-312Asn polymorphisms and esophageal cancer risk systematically, we performed this meta-analysis on the basis of the selected studies which included 3928 cases and 6012 controls.

In the present meta-analysis, we found that the people with XPD Asn allele had a higher risk of becoming EC than those with normal XPD gene in overall analysis (Dominant model: OR=1.14, 95% CI: 1.03-1.27, P=0.01; Asp/Asn vs. Asn/Asn: OR=1.06, 95% CI: 0.72-1.57, *P*=0.01).

Some outside factors, such as smoking, alcohol consumption or environmental factors, may increase the risk of EC. When stratified by tobacco consumption, borderline significantly increased risk were found in dominant model [OR=1.63, 95% CI: 1.11-2.38, *P*=0.01], which implied that cigarette smokers carrying Asp/Asn or Asn/Asn genotypes were increased risk of EC, but the same results were not found in non-smokers. Moreover, a significant association was presented in the stratified analysis on hospital-based controls rather than on population-based controls. These results showed that Asn, as a risk allele gene, could increase the susceptibility of EC. However, as for the subgroup analysis on different cancer types and ethnicities, no positive results were found, which were inconsistent with a previous meta-analysis by Duan XL [31]. The most possible reason may be that the study by Tse was considered as mixed population [28]. And the increased number of cases and controls in this meta-analysis was also an important factor.

Some limitations of this meta-analysis should be acknowledged. Firstly, the numbers of cases and controls in each study were not enough. Secondly, the control resources were not from the uniformed population, which may cause misclassification bias to some extent. Thirdly, although the gene-smoking and drinking factors were included in this meta-analysis, the included studies were too less to perform further investigations. Finally, only studies in English and Chinese were included in our analysis, which may result in some bias publication bias. In spite of this, our meta-analysis had some key advantages. Firstly, the number of cases and controls were increased, which significantly strengthened statistical power of the analysis. Secondly, the quality of case-control studies included in current meta-analysis was satisfactory and met our inclusion criterion. Thirdly, no heterogeneity was found between study and the forest map showed our results were statistically robust.

Conclusion

Our meta-analysis provided moderate evidence that the XPD Asp312Asn polymorphism contributes to the development of EC in overall population. Tobacco consumption, as an external factor, may stimulate the susceptibility of EC. Large and well designed epidemiological studies are necessary to validate the exact association between XPD Asp312Asn polymorphism and EC in the future.

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Disclosure of conflict of interest

None.

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