MEDICAL SCIENCE MONITOR

LAB/IN VITRO RESEARCH

e-ISSN 1643-3750
© Med Sci Monit, 2018; 24: 7015-7022
DOI: 10.12659/MSM.908813

Received: 2018.01.04
Accepted: 2018.04.05
Published: 2018.10.03

Association of ERCC2 Gene Polymorphisms with Susceptibility to Diffuse Large B-Cell Lymphoma

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Source of support: Departmental sources

Background: The objective of this study was to detect the association between ERCC excision repair 2, TFIIH core complex helicase subunit (*ERCC2*) gene polymorphisms and diffuse large B-cell lymphoma (DLBCL) susceptibility.

Material/Methods: This study used a case-control design. *ERCC2* gene rs1799793 (Asp312Asn) and rs13181 (Lys751Gln) polymorphisms were genotyped by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) both in DLBCL patients and healthy controls. The association between *ERCC2* gene polymorphisms and DLBCL risk was assessed by χ^2 test. Odds ratios (ORs) with corresponding 95% confidence intervals (95% CIs) were used to address the association strength. Subgroup analyses were also performed to investigate the genetic effects of *ERCC2* polymorphisms on clinical characteristics of DLBCL patients.

Results: A significant association was discovered between the rs1799793 A allele and increased DLBCL risk (P=0.031, OR=1.928, 95% CI=1.052–3.534). The C allele of rs13181 was obviously associated with elevated DLBCL susceptibility (P=0.047, OR=1.820, 95% CI=1.002–3.305). The subgroup analysis demonstrated that rs1799793 and rs13181 polymorphisms had no relationship with serum lactate dehydrogenase level, nidus number, B-symptoms, Ann Arbor stages, or immunological types in DLBCL cases (P>0.05 for all).

Conclusions: Minor allele carriers of *ERCC2* gene rs1799793 (Asp312Asn) and rs13181 (Lys751Gln) polymorphisms had higher susceptibility to DLBCL.

MeSH Keywords: Lymphoma, Large B-Cell, Diffuse • Polymorphism, Genetic 8 Xeroderma Pigmentosum Group D Protein

Full-text PDF: https://www.medscimonit.com/abstract/index/idArt/908813

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Background

Diffuse large B-cell lymphoma (DLBCL) is the most common type of non-Hodgkin lymphoma (NHL) in adults [1]. It is a malignancy of large B cells that is strongly invasive [2]. It can occur in any age, but it is usually diagnosed in older people. In recent years, the morbidity of DLBCL has increased within the aging population [3]. In China, it has a high morbidity and mortality rate [4]. Histological morphology, immune phenotype, and genetic characteristics of DLBCL had significant heterogeneity between patients [5]. DLBCL could originate from lymph nodes and extranodal sites [6,7]. Pathogenesis of DLBCL is still clear, but it is confirmed that immune deficiency, medical system, lifestyle, environmental exposure, and genetic variation may contribute to the occurrence of DLBCL [8–13]. Additionally, genetic factors are the basis for the disease heterogeneity and the therapy response [14,15]. Therefore, gene polymorphisms play a crucial role in disease susceptibility.

DNA is often damaged by endogenous or exogenous mutagenesis in cells, and if the repair for these damages is not timely, it may cause apoptosis or uncontrolled growth of cells [16]. Polymorphisms in the exon regions of DNA repair gene might alter the structure or activity of the protein, then lead to different cancers, including lymphoma [17–19]. ERCC excision repair 2, TFIIH core complex helicase subunit (*ERCC2*), also known as xeroderma pigmentousm complementation group D (XPD), is a protein which participates in the transcription-coupled nucleotide excision repair. Human *ERCC2* gene is located at chromosome 19q13.32. Single nucleotide polymorphisms (SNPs), rs1799793 (Asp312Asn), and rs13181 (Lys751Gln) are respectively located in exon 10 and exon 23 of the *ERCC2* gene. Previous studies indicate that they could alter DNA repair capacity of the protein [20].

ERCC2 rs1799793 and rs13181 SNPs have been widely explored in different cancers [21–23]. In spite of previous studies focused on the association of *ERCC2* SNPs with DLBCL susceptibility, few studies have focused on this topic for the Chinese Han population. Therefore, we carried out this study; subgroup analysis based on different clinical features were also performed.

Material and Methods

Study participants

This case-control study was ratified by the institutional review board of Shenzhen Hospital, Southern Medical University. Written informed consent was signed by every participant prior to enrollment. The study protocol followed the Helsinki Declaration.

DLBCL patients who were hospitalized in Shenzhen Hospital, Southern Medical University were enrolled as participants in the case group. These patients were diagnosed by 2 pathologists using histopathologic examination, x-ray, magnetic resonance imaging (MRI), and routine blood examination [24]. Patients were all older than 18 years of age. Individuals who presented for healthy checkups in the same hospital were recruited as the controls; healthy individuals with histories of leukemia or lymphoma, immune or inflammatory diseases, or other tumors were excluded from the control group. The control group was matched with the case group by age and gender.

Sample collection and genotyping method

We collected 5 mL of peripheral blood samples from the elbow venous of the participants and stored the samples in EDTA tubes. TIANamp Blood DNA Kit (TIANGEN, Beijing) was used to extract the genomic DNA following the manufacturer's instructions. *ERCC2* gene rs1799793 and rs13181 SNPs were genotyped by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) as described in previous studies [25].

Statistical analysis

Continuous variables were compared by Student's *t*-test. Categorical variables were evaluated by chi-square test or Fisher's exact test. Genotype distributions of *ERCC2* gene SNPs were examined by the Hardy-Weinberg equilibrium (HWE) test. The association between *ERCC2* SNPs and DLBCL risk was addressed by odds ratios (ORs) with 95% confidence intervals (CIs). Subgroup analyses performed in this study were based on clinical characteristics: serum lactate dehydrogenase (LDH) level, location, nidus number, B-symptoms, Ann Arbor stages, and immunological type. All of the calculations were performed by PASW 18.0. $P<0.05$ was considered as the existence of statistical significance.

Results

Characteristics of participants

DLBCL patients in the case group included 78 males and 59 females with the mean age of 62.95±15.03 years. The control group included 83 males and 62 females, the mean age was 61.05±13.17 years old. No significant difference in age or gender was discovered between the case group and the control group (Table 1, $P>0.05$), indicating that the age and gender were well matched. In addition, smoking and drinking had no significant difference between the case group and the control group ($P>0.05$).

In DLBCL patients, 80 patients had high serum LDH levels and 57 patients had normal LDH levels; 45 cases occurred

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Table 1. Characteristics of participants.

Characteristics	Case n=137 (%)		Control n=145 (%)		*P*
Basic characteristics					
Age	62.95±15.03		61.05±13.17		0.259
Gender					0.958
Male	78	(56.93)	83	(57.24)	
Female	59	(43.07)	62	(42.76)	
Smoking					0.573
No	96	(70.07)	106	(73.10)	
Yes	41	(29.93)	39	(26.90)	
Drinking					0.475
No	89	(64.96)	100	(68.97)	
Yes	48	(35.04)	45	(31.03)	
Clinical characteristics					
Serum LDH level					
Normal (<226)	57	(41.61)			
High (≥226)	80	(58.39)			
Locations					
Lymph nodes	45	(32.85)			
Extranodal	92	(67.15)			
Nidus number					
<2	87	(63.50)			
≥2	50	(36.50)			
B-symptoms					
Without	85	(62.04)			
With	52	(37.96)			
Ann Arbor stages					
I, II	73	(53.28)			
III, IV	64	(46.72)			
Immunological type					
GCB	40	(29.20)			
ABC	97	(70.80)			

LDH – lactate dehydrogenase; B-symptoms – including fever, weight loss, and night sweats; GCB – germinal center B-cell-like; ABC – activated B-cell-like.

in lymph nodes and 92 cases occurred in extranodal sites; and 50 patients had more than one nidus. In addition, 52 patients had B-symptoms (fever, weight loss, and night sweats); and 73 patients had Ann Arbor I or II stages and 64 patients had Ann Arbor III or IV stages. Based on the immunological types of DLBCL patients, 40 cases were germinal center B-cell-like (GCB) and 97 cases were activated B-cell-like (ABC).

Association between *ERCC2* polymorphisms and DLBCL risk

Genotype distributions of *ERCC2* gene rs1799793 and rs13181 SNPs were accorded with the HWE test in control group (Table 2, $P>0.05$), suggesting good representativeness of participants.

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Table 2. Association between *ERCC2* polymorphisms and DLBCL susceptibility.

Genotype/allele	Case n=137 (%)	Control n=145 (%)	P	OR (95% CI)
rs1799793				
GG	109 (79.56)	128 (88.28)	–	–
GA	25 (18.25)	16 (11.03)	0.076	1.835 (0.932–3.613)
AA	3 (2.19)	1 (0.69)	0.340	3.523 (0.361–34.358)
G	243 (88.69)	272 (93.79)	–	–
A	31 (11.31)	18 (6.21)	0.031	1.928 (1.052–3.534)
P_{HWE}	0.289	0.529		
rs13181				
AA	110 (80.29)	128 (88.28)	–	–
AC	23 (16.79)	15 (10.34)	0.101	1.784 (0.887–3.588)
CC	4 (2.92)	2 (1.38)	0.422	2.327 (0.418–12.950)
A	243 (88.69)	271 (93.45)	–	–
C	31 (11.31)	19 (6.55)	0.047	1.820 (1.002–3.305)
P_{HWE}	0.056	0.062		

Table 3. Effects of *ERCC2* polymorphisms on serum LDH level in DLBCL patients.

Genotype/allele	Normal LDH, n=57, %	High LDH, n=80, %	P	OR (95% CI)
rs1799793				
GG	47 (82.46)	62 (77.50)	–	–
GA	8 (14.04)	17 (21.25)	0.308	1.611 (0.641–4.050)
AA	2 (3.51)	1 (1.25)	0.417	0.379 (0.033–4.306)
G	102 (59.48)	141 (88.12)	–	–
A	12 (10.52)	19 (11.88)	0.728	1.145 (0.532–2.465)
rs13181				
AA	44 (77.19)	66 (82.50)	–	–
AC	11 (19.30)	12 (15.00)	0.488	0.727 (0.295–1.794)
CC	2 (3.51)	2 (2.50)	0.689	0.667 (0.091–4.910)
A	99 (86.84)	144 (90.00)	–	–
C	15 (13.16)	16 (10.00)	0.332	0.688 (0.322–1.470)

GA and AA genotypes of rs1799793 SNP had higher frequencies in the case group compared to the control group, but the difference was not statistical significant (Table 2, $P>0.05$). The allele of rs1799793 had significantly higher frequency in DLBCL patients, which indicated that this allele was positively correlated with DLBCL risk ($P=0.031$, OR=1.928, 95% CI=1.052–3.534).

Frequencies of rs13181 AA, AC, and CC genotypes respectively were 80.29%, 16.79%, 2.92% in DLBCL patients, and 88.28%, 10.34%, 1.38% in healthy controls. No significant difference in rs13181 genotypes were discovered between the case group and the control group ($P>0.05$). The significantly higher frequency of rs13181 C allele in DLBCL patients compared to the controls demonstrated that the C allele was distinctly correlated with an elevated DLBCL risk ($P=0.047$, OR=1.820, 95% CI=1.002–3.305).

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Table 4. Genetic association of *ERCC2* polymorphisms with locations in DLBCL patients.

Genotype/allele	Lymph nodes, n=45, %		Extranodal sites n=92, %		P	OR (95% CI)	
rs1799793							
GG	37	(82.22)	72	(78.26)	–	–	
GA	7	(15.56)	18	(19.57)	0.568	1.321	(0.507–3.447)
AA	1	(2.22)	2	(2.17)	0.982	1.028	(0.090–11.709)
G	81	(90.00)	162	(88.04)	–	–	
A	9	(10.00)	22	(11.96)	0.673	1.193	(0.525–2.708)
rs13181							
AA	35	(77.78)	75	(81.52)	–	–	
AC	9	(20)	14	(15.22)	0.498	0.726	(0.287–1.837)
CC	1	(2.22)	3	(3.26)	0.773	1.400	(0.141–13.942)
A	79	(87.78)	164	(89.13)	–	–	
C	11	(12.22)	20	(10.87)	0.740	0.876	(0.400–1.917)

Table 5. Genetic effects of *ERCC2* polymorphisms on nidus number in DLBCL cases.

Genotype/allele	Nidus number <2, n=87,%		Nidus number ≥2, n=50,%		P	OR (95% CI)	
rs1799793							
GG	73	(83.91)	36	(72.00)	–	–	
GA	12	(13.79)	13	(26.00)	0.076	2.197	(0.911–5.298)
AA	2	(2.30)	1	(2.00)	0.991	1.014	(0.089–11.556)
G	158	(90.85)	85	(85.00)	–	–	
A	16	(9.15)	15	(15.00)	0.144	1.743	(0.821–3.697)
rs13181							
AA	68	(78.16)	42	(84.00)	–	–	
AC	17	(19.54)	6	(12.00)	0.272	0.571	(0.209–1.564)
CC	2	(2.30)	2	(4.00)	0.633	1.619	(0.220–11.932)
A	153	(87.93)	90	(90.00)	–	–	
C	21	(12.07)	10	(10.00)	0.603	0.810	(0.365–1.796)

Effects of *ERCC2* polymorphisms on clinical characteristics of DLBCL

To certify the effects of *ERCC2* polymorphisms for DLBCL risk, we performed subgroup analysis based on the serum LDH level, location, nidus number, B-symptoms, Ann Arbor stages, and immunological type. We found that *ERCC2* rs1799793 and rs13181 polymorphisms had no association with serum LDH level, location, nidus number, B-symptoms, Ann Arbor stages, or immunological type in DLBCL patients (Tables 3–8, $P>0.05$ for all).

Discussion

We failed to find any significant association between the rs1799793 genotypes and DLBCL susceptibility. The significantly higher frequency of rs1799793 A allele in the case group compared to the control group suggested that the A allele was positively correlated with 1.928 times increased DLBCL susceptibility. Worrillow et al. suggested that although rs1799793 AA genotype had a higher trend for DLBCL, this SNP had no significant relationship with DLBCL risk in an English population [26]. Meanwhile, El-Din et al. found no significant association between rs1799793 SNP and DLBCL susceptibility in an

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Table 6. Effects of *ERCC2* polymorphisms on B-symptoms in case group.

Genotype/allele	Without B-symptoms, n=85, %		With B-symptoms, n=52, %		P	OR (95% CI)
rs1799793						
GG	67	(78.82)	42	(80.77)	–	–
GA	16	(18.82)	9	(17.31)	0.814	0.897 (0.364–2.214)
AA	2	(2.35)	1	(1.92)	0.855	0.798 (0.070–9.071)
G	150	(88.23)	93	(89.42)	–	–
A	20	(11.27)	11	(10.58)	0.763	0.887 (0.407–1.935)
rs13181						
AA	73	(85.88)	37	(71.15)	–	–
AC	11	(12.94)	12	(23.08)	0.094	2.152 (0.867–5.340)
CC	1	(1.18)	3	(5.77)	0.089	5.919 (0.595–58.887)
A	157	(92.35)	86	(82.69)	–	–
C	13	(7.65)	18	(17.31)	0.014	2.528 (1.182–5.407)

Table 7. Genetic correlation of *ERCC2* polymorphisms with DLBCL Ann Arbor stages.

Genotype/allele	Ann Arbor stage I, II, n=73, %		Ann Arbor stage III, IV, n=64, %		P	OR (95% CI)
rs1799793						
GG	55	(75.34)	54	(84.38)	–	–
GA	16	(21.92)	9	(14.06)	0.221	0.573 (0.233–1.408)
AA	2	(2.74)	1	(1.56)	0.558	2.037 (0.179–23.130)
G	126	(86.30)	117	(91.45)	–	–
A	20	(13.7)	11	(8.55)	0.183	0.592 (0.272–1.289)
rs13181						
AA	60	(82.19)	50	(78.13)	–	–
AC	11	(15.07)	12	(18.75)	0.557	1.309 (0.532–3.220)
CC	2	(2.74)	2	(3.13)	0.858	1.200 (0.163–8.828)
A	131	(89.73)	112	(87.5)	–	–
C	15	(10.27)	16	(12.5)	0.562	1.248 (0.590–2.636)

Egyptian population, despite A allele carriers having a higher trend for DLBCL [27]. Inversely, a recent meta-analysis study showed that rs1799793 was negatively correlated with DLBCL risk under Asn (A) *vs.* Asp (G) genetic model [28]. These differences might be caused by differences in ethnicity or regions.

In this study, no significant association has been discovered between rs13181 genotypes and DLBCL risk; while an approximately 1.820 times elevated DLBCL risk was associated with rs13181 C allele. El-Din et al. indicated that rs13181 CC genotype carriers had higher susceptibility for DLBCL, but the association was not significant [28]. However, Worrillow et al. demonstrated that rs13181 CC genotype was significantly associated with reduced risk for DLBCL in an English population [26]. In addition, meta-analysis studies also failed to find a significant association between this SNP and DLBCL risk [23,28].

We performed the subgroup analyses to certify the influence of *ERCC2* polymorphisms for DLBCL risk. Genotype distributions of *ERCC2* gene rs1799793 and rs13181 polymorphisms showed no significant difference between different subgroups of patients. Thus, the polymorphisms of rs1799793 and rs13181

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Table 8. Effects of *ERCC2* polymorphisms on DLBCL immunological types.

Genotype/allele	GCB n=40, %		ABC, n=97, %		P	OR (95% CI)
rs1799793						
GG	31	(77.50)	78	(80.41)	–	–
GA	8	(20.00)	17	(17.53)	0.724	0.845 (0.331–2.157)
AA	1	(2.50)	2	(2.06)	0.853	0.795 (0.070–9.086)
G	70	(87.50)	173	(89.17)	–	–
A	10	(12.50)	21	(10.83)	0.691	0.850 (0.381–1.896)
rs13181						
AA	31	(77.50)	79	(81.44)	–	–
AC	8	(20.00)	15	(15.46)	0.527	0.736 (0.284–1.909)
CC	1	(2.50)	3	(3.09)	0.889	1.177 (0.118–11.753)
A	70	(87.50)	173	(89.17)	–	–
C	10	(12.50)	21	(10.83)	0.691	0.850 (0.381–1.896)

Table 9. Subgroup analysis of *ERCC2* polymorphisms with DLBCL susceptibility based on Immunological type.

Genotype/allele	GCB		ABC	
	P	OR (95% CI)	P	OR (95% CI)
rs1799793				
GG	–	–	–	–
GA	0.123	2.065 (0.811–5.259)	0.137	1.744 (0.833–3.649)
AA	0.359	4.129 (0.251–67.862)	0.560	3.282 (0.293–36.796)
G	–	–	–	–
A	0.060	2.159 (0.954–4.884)	0.067	1.834 (0.950–3.541)
rs13181				
AA	–	–	–	–
AC	0.095	2.202 (0.857–5.657)	0.216	1.620 (0.751–3.495)
CC	0.486	2.065 (0.181–23.505)	0.377	2.430 (0.397–14.866)
A				
C	0.080	2.038 (0.907–4.578)	0.094	1.731 (0.905–3.314)

did not have an obvious association with the clinical characteristics of DLBCL patients; however, due to the limited sample size, the results needed further verification.

There were several limitations in this study. First, the sample size was relatively small; thus, the statistical power might be reduced. Second, the ethnicity might affect present results. Third, various potential confounding environmental and genetic factors involved in the occurrence of DLBCL were not considered in this current study. Therefore, the conclusions of this study should be verified in further studies with larger sample size.

Conclusions

ERCC2 gene rs1799793 and rs13181 polymorphisms might be risk factors for DLBCL. However, rs1799793 and rs13181 polymorphisms have no relationship with serum LDH level, tumor location, nidus numbers, B-symptoms, Ann Arbor I and II stages, or immunological type in DLBCL patients.

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