

Case Study

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## Association of HLA-DQB1 Gene Polymorphisms to the Progression of Viral Hepatitis B in Burkina Faso: A Case-Control Study

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### ABSTRACT

Clinical outcomes following exposure to hepatitis B virus vary enormously from spontaneous clearance to chronic hepatitis B, often progressing to liver cirrhosis and hepatocellular carcinoma. The presence and expression of polymorphisms in the HLA-DQB1 gene have been shown to correlate with spontaneous clearance and susceptibility to HBV infection. This study aimed to determine the association of HLA-DQB1 gene polymorphisms with chronic hepatitis B infection, cirrhosis, and HBV-related HCC in Burkina Faso. This case-control study was conducted in Ouagadougou, Burkina Faso, from August 2022 to May 2023. It characterized the HLA-DQB1 gene polymorphism (HLA-DQB1\*0303, HLA-DQB1\*0502, HLA-DQB1\*0604) in 111 participants stratified into four groups: HCC (n=35), cirrhosis (n=13), chronic hepatitis B (CHB, n=37), and healthy controls (n=26). HLA-DQB1\*03: 03, \*05: 02, and \*06: 04 alleles were characterized using PCR with sequence-specific primers (PCR-SSP). Data were analyzed using SPSS version 21 and R version 4.3.1. Our data suggest that HLA-DQB1\*0502 (OR=28.4; 95%CI= 5.7-141.3; p-value <0.01; OR=7.1; 95%CI = 1.43-35.02 and p-value <0.01) was a risk factor for progression from hepatitis B to CHB and HCC. However, this allele also appeared to have a protective effect against cirrhosis (OR=0.03; 95%CI=0.004-0.305; p-value=<0.01). Carriers of the HLA-DQB1\*0303 allele iniquitously had a higher risk of developing cirrhosis following chronic HBV infection (OR=29.4; CI95%=3.4-254.67, p-value<0.01). The simultaneous absence of HLA-DQB1\*0502/HLA-DQB1\*0604 alleles showed statistically significant values in healthy controls versus patients with HCC (58.5% versus 41.5%) (OR=0.03; CI95%=0.02-0.38; p-value<0.01). Our results revealed that carrying the HLA-DQB1\*0502 allele could induce susceptibility to the progression of infection to critical stages, and the HLA-DQB1\*0303 allele could be protective against the progression of hepatitis B.

#### Keywords

HBV, HLA-DQB1, polymorphisms, HLA, Cirrhosis, HCC, CHB.

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## **Introduction**

Hepatitis B virus (HBV) infection is an endemic disease, often leading to acute and chronic liver disorders [1]. Worldwide, 260 million people are chronically infected with HBV, and 890,000 die each year from complications arising from the progression of the infection [2]. In addition, around 1.5 million new cases of HBV infection occur every year, a quarter of whom develop liver disease [1]. Deaths from HBV infection are estimated at between 500,000 and over a million a year, representing 1/10th of all causes of mortality worldwide [3]. Sub-Saharan Africa is an area of high prevalence, with a chronic HBV carrier rate of over 8%, compared with less than 1% in Western Europe and North America [4]. Other authors estimate that between 70% and 95% of the adult African population has already been in contact with HBV, of which between 6% and 20% are chronic carriers who may later develop liver cancer or cirrhosis [4].

Burkina Faso is one of the countries with a high prevalence of HBV, with HBs antigen carriage above 8% and an estimated prevalence of 7.8% in women and 10.5% in men [5]. In Africa, after vertical and sexual transmission modes, HBV and HCV infections may be due to cultural practices or medical surgery [6]. Indeed, the potential severity of hepatitis B includes the risk of progression to chronic hepatitis, cirrhosis, and hepatocellular carcinoma [5, 7]. Consequently, significant individual differences in the severity of chronic HBV infection indicate a complex biological process for which the cellular processes and genetic factors involved in pathogenesis are unclear [7]. Thus, factors influencing the risk of chronic HBV infection or spontaneous HBV clearance include age, location, gender, body mass index, ethnicity, viral mutation, HBV genotype, and host genetic and immune responses [6, 8, 9].

Human leukocyte antigens (HLA) mediate the immune response [8]. Several studies have reported that different variations of class I and class II genes of the HLA system are an essential part of the immune response and a substantial genetic control gene for immune characteristics involved in the persistence or elimination of HBV [7, 10, 11]. Thus, polymorphisms in HLA loci, particularly HLA-II (rs2856718 and rs7453920), have been associated with spontaneous clearance or susceptibility to HBV infection. Some HLA class II alleles have also been attributed to persistent HBV

infection, such as HLA-DQB1\*0301 (susceptible to chronic HBV infection), HLA-DQA1\*0501, HLA-DQA1\*0302 and HLA - DPA1 \*0202 and HLA-DPB1\*0501 or to HBV resistance to therapies, such as HLA-DQB1\*0604 and HLA-DQB1\*0501 [9, 10, 12, 13]. Accordingly, this study investigated the association of HLA-DQB1\*0303, DQB1\*0502, and DQB1\*064 alleles with HBV infection progression in Burkina Faso.

## **Materials and Methods**

### **Population setting**

This is a case-control study conducted among the population of Burkina Faso at the Piétro Annigoni Biomolecular Research Center (CERBA/LABIOGENE) in Ouagadougou. A total of 111 participants were included in this study and stratified into four groups: 35 patients with HCC, 13 with cirrhosis, 37 with chronic hepatitis B (CHB), and 26 healthy controls. Patients with HCC and cirrhosis were recruited from the hepatogastroenterology departments of CHU-YO and Hôpital Paul VI.

In contrast, CHB cases were recruited from the Centre de Recherche Biomoléculaire Pietro Annigoni, while healthy controls were selected from blood donors at the Centre Régional de Transfusion sanguine de Ouagadougou. HBV-infected patients were identified based on serological markers, including HBsAg, anti-HBs, and anti-HBc. Healthy controls were seronegative for all HBV biomarkers. HCC was diagnosed by serum alpha-fetoprotein (AFP) assays and clinical criteria. All blood samples were confirmed negative for hepatitis C virus and human immunodeficiency virus.

### **Blood sampling and preparation**

Peripheral blood was collected from each participant in a dry and EDTA tubes. Then, samples were centrifuged, aliquoted and stored at -20°C until further analysis. Serum was used for serological screening, while plasma was reserved for molecular analyses.

### **Genomic DNA extraction**

Genomic DNA was extracted from whole blood using the Genomic Pure Link DNA Extraction Mini Kit (INVITROGEN, USA) following the manufacturer's protocol, and stored at -20°C.

## Genotyping of the HLA-DQB1 gene polymorphisms

PCR amplification was performed to characterize the HLA-DQB1\*0303, HLA-DQB1\*0502, and HLA-DQB1\*0604 alleles. Allele-specific primers were used for allele discrimination, while primers targeting the Human Growth Factor (HGF) gene was used as internal control (Table 1). This involved multiplex PCR, simultaneously targeting all three alleles and the HGF gene, using the GeneAmp PCR System 9700 (Applied Biosystem, USA). The total reaction volume was 20  $\mu$ L consisting of 9  $\mu$ L of molecular biology water, 04  $\mu$ L of master mix (FIREPol master mix ready to load), 0.5  $\mu$ L of each primer (0.2  $\mu$ M) and 5  $\mu$ L of DNA.

The following cycling conditions were used: DNA polymerase activation at 94°C for 4 mn, followed by 35 cycles of DNA denaturation at 94°C for 1 mn, hybridization at 65°C for 1 min and extension at 72°C for 1 min. DNA amplicon were separated by gel electrophoresis using 2% agarose. The bands were then visualized under transilluminator UV.

## Statistical analysis

Data were analyzed using SPSS version 21, and R. Fisher and Chi-square tests were used to calculate odds ratios for comparisons. All values were considered statistically significant for  $p \leq 0.05$ .

## Results and Discussion

### Distribution of alleles according to sex and clinical status

Table II shows a higher frequency of the HLA-DQB1\*303 allele in males (54.9%) than females (41.4%), while the HLA-DQB1\*0502 allele was more prevalent in females (20.7%) than males (17.1%). The HLA-DQB1\*0604 allele was found in only one female ( $p$ -value=0,04).

Assessment of allele frequency within the study subgroups showed a statistically significant difference in the distribution of the HLA-DQB1\*0303 allele ( $p$ -value = 0.04) and the HLA-DQB1\*0502 allele ( $p$ -value <0.01). Carrying the HLA-DQB1\*0604 allele showed no statistical significance according to clinical status, as

illustrated in Table 2.

### Association of HLA-DQB1 alleles with hepatitis B infection

As shown in Table 3, the frequency of the HLA-DQB1\*0502 allele was higher in patients with CHB than in healthy controls (OR=28.4; 95%CI= 5.7-141.3;  $p$ -value<0.01). The frequency of the HLA-DQB1\*0502 allele was higher in patients with HCC than in healthy controls (37.1% vs. 7.7%) (OR=7.09; 95% CI =1.43-35.02 and  $p$ -value<0.01) (table 4). Also, individuals carrying HLA-DQB1\*0303/HLA-DQB1\*0502 alleles were more remarkable in patients with HCC than in healthy controls (86.7% vs. 13.3%). (OR=7.09; 95%CI= 51.43-35.02;  $p$ -value<0.01). The simultaneous absence of HLA-DQB1\*0502/HLA-DQB1\*0604 alleles showed statistically significant values in healthy controls versus patients with HCC (58.5% versus 41.5%) (OR=0.03; CI95%=0.02-0.38;  $p$ -value<0.01) as shown in Table 5. No link was observed between allele carriage and the risk of cirrhosis in healthy controls (table 5).

The present study findings revealed that the frequency of HLA-DQB1\*0303 allele was higher in healthy controls than in patients with chronic hepatitis B. But, the HLA-DQB1\*0502 allele was more prevalent in patients with chronic hepatitis B compared to healthy controls.

This suggests that HLA-DQB1\*0502 allele may be a risk allele for the progression of hepatitis B infection to chronicity. Karra *et al.*, (2018) reported similar association in a study of southern and western indian populations [10]. A study conducted in Chinese populations indicated that people carrying the HLA-DQB1\*0303 and DQB1\*0502 alleles were susceptible to CHB [12]. Another study showed that HLA-DQB1\*0303 was susceptibility to CHB [15]. In contrast, a meta-analysis suggests that DQB1\*0502 is a risk factor for CHB, while HLA-DQB1\*0303 is protective factor [7]. In our study, the frequency of HLA-DQB1\*0303/DQB1\*0502 alleles was more significantly high in healthy controls than in patients with cirrhosis. These results suggest that HLA-DQB1\*0303/DQB1\*0502 alleles may protect against progression to cirrhosis. Our findings are contradictory to the study conducted by Fatmawati *et al.*, (2022) in Indonesia, which reported the presence of HLA-DQB1\*0502 allele only in patients with end-stage chronic hepatitis B.

**Table.1** Primer sequences and amplicons size

HLA-DQB1 alleles	Primers sequences	Size (pb)	References
<b>*0303</b>	F: 5' — GACGGAGCGCGTGCGTTA-3' R: 5' — CTGTTCCAGTACTCGGCG-3'	129	[14]
<b>*0502</b>	F: 5'-TGCGGGGTGTGACCAGAC-3' R: 5'-TGTTCCAGTACTCGGCGCT-3'	117	[14]
<b>*0604</b>	F: 5'-CGTGTACCAGITTAAGGGCA-3' R: 5' — GCAGGATCCCCGCGGTACC-3'	254	[14]
<b>HGF</b>	F: 5' — GCC TTC CCA ACC ATT CCC TTA-3 R: 5' -TCA CGG ATTTCT GTT GTG TTTC-3'	432	[14]

**Table.2** Gender-dependent allelic detection

Characteristics		<i>HLA-DQB1*303</i>			<i>HLA-DQB1*502</i>			<i>HLA-DQB1*604</i>		
		Yes (%)	No (%)	p-value	Yes (%)	No (%)	p-value	Yes (%)	No (%)	p-value
Sex	Male	61 (54.9)	2 (1.8)	1	19 (17.1)	44 (39.6)	0.07	0 (0.0)	63 (56.8)	0.43
	Female	46 (41.4)	2 (1.8)		23 (20.7)	25 (22.5)		1 (0.9)	47 (42.3)	
Clinic status	CHB	37 (33.3)	0 (0.0)	0.04	26 (23.4)	11 (9.9)	<0.01	0 (0.0)	37 (33.3)	0.6
	Cirrhosis	13 (11.7)	0 (0.0)		1 (0.9)	12 (10.8)		0 (0.0)	13 (11.7)	
	HCC	31 (27.9)	4 (3.6)		13 (11.7)	22 (19.8)		1 (0.9)	34 (31)	

**Table.3** Allele frequencies between CHB and healthy controls

		CHB n (%)	Controls n (%)	OR	IC (95 %)	P-Value	
<i>HLA-DQB1*0303</i>	Yes	35 (9.2)	25 (96.1)	Reference			
	No	1 (2.8)	1 (3.8)	1.4	0.08-23.46	0.66	
<i>HLA-DQB1*0502</i>	Yes	26 (70.3)	2 (7.69)	Reference			
	No	11 (29.7)	24 (92.3)	28.4	5.69-41.27	<0.01	
<i>HLA-DQB1*0604</i>	Yes	1 (2.7)	1 (3.85)	Reference			
	No	36 (97.3)	25 (96.1)	0.7	0.04-11.63	0.65	
<i>HLA-DQB1*0303/HLA-DQB1*0502/HLA-DQB1*0604</i>		+ / +/-	26 (92.9)	02 (7.1)	28.4	5.7-141.3	<0.01

**Table.4** Allele frequencies between HCC and healthy controls

		HCC n (%)	Controls n (%)	OR	IC (95 %)	p-Value
<b>HLA-DQB1*0303</b>	Yes	31 (88.4)	25 (96.1)	Reference		
	No	04 (11.4)	01 (3.8)	0.31	0.03-2.95	0.28
<b>HLA-DQB1*0502</b>	Yes	13 (3.1)	2 (7.7)	Reference		
	No	22 (8.6)	24 (92.3)	7.09	1.43-35.02	<0.01
<b>HLA-DQB1*0604</b>	Yes	01 (3.8)	01 (3.8)	Reference		
	No	30 (96.8)	25 (96.1)	0.83	0.05-14.01	0.7
<b>HLA-DQB1*0303/HLA-DQB1*0502/HLA-DQB1*0604</b>	+/+/-	13 (86.7)	02 (13.3)	7.1	1.43-35.02	<0.01
	+/-/-	17 (41.5)	24 (58.5)	0.1	0.02-0.38	<0.01

**Table.5** Allele frequencies between cirrhosis and healthy controls

		Cirrhosis n (%)	Controls n (%)	OR	IC (95 %)	p-Value
<b>HLA-DQB1*0303</b>	Yes	12 (92.3)	25 (96.1)	Reference		
	No	01 (7.7)	01 (3.8)	2.1	0.12-36.23	0.56
<b>HLA-DQB1*0502</b>	Yes	01 (7.7)	2 (7.7)	Reference		
	No	12 (92.3)	24 (92.3)	1	0.08-12.16	0.71
<b>HLA-DQB1*0604</b>	Yes	01 (7.7)	01 (3.8)	Reference		
	No	12 (92.3)	25 (96.1)	2.1	0.12-36.23	0.56
<b>HLA-DQB1*0303/HLA-DQB1*0502/HLA-DQB1*0604</b>	+/+/-	01 (33.3)	02 (66.7)	1	0.08-12.16	<0.01
	+/-/-	12 (33.33)	24 (66.7)	1	0.08-12.16	0.72

Therefore, the possibility of a patient having this allele will increase the likelihood of developing cirrhosis [16]. Furthermore, a study by Riazalhosseini *et al.*, on patients with hepatitis B also revealed that 62% of those with liver cirrhosis and HCC carried the HLA-DQB1\*0502 allele [17]. These results further support the potential role of this specific allele in the clinical progression toward advanced liver disease. These discrepancies may be attributed to the small sample size of subjects involved in the study. HLA alleles frequencies are different between inter-ethnic, which can influence the detection of significant association in specific populations. Despite the variation, HLA-DQB1\*0604 allele has been reported as protective allele against progression to CHB in studies conducted in Iranian, Turkmen and Japanese populations [16, 17]. Conjointly, these findings suggest that HLA-DQB1\*06: 04 may play a role as a protective polymorphism in different ethnic groups.

The present study findings showed that the HLA-DQB1\*0502 allele was more significantly prevalent in patients with HCC than in healthy controls. Accordingly, the presence of this allele may confer a genetic predisposition to hepatic malignancy, substantially increasing the risk of HCC in these patients.

The present study findings are concordant with those reported by Watmawati *et al.*, (2022), who established in an Indonesians population that the HLA-DQB1\*05: 02 allele was exclusively present in patients with advanced-stage CHB [16]. This further highlight the potential role of this allele as a genetic biomarker for disease severity. These findings were also reported by Riazalhosseini *et al.*, (2018), who showed similar association in their study [18]. A recent meta-analysis investigating the association between HLA-DQB1 alleles and HCC, revealed that the HLA-DQB1\* 0502 allele was a significant risk factor for the development of HCC [19].

The HLA-DQB1\*0604 allele frequency was insignificant in healthy controls or the patients with HCC. Still, studies in Japanese and Chinese populations have suggested that this allele might be a protective allele against the progression from CHB to HCC [19]. HLA class II molecules are present on the surface of antigen-presenting cells (APCs) [20-22]. They are responsible for presenting exogenous antigens to helper CD4+ T cells. The efficiency of antigen presentation can vary due to genetic loci [12]. In this study, the findings demonstrate that HLA-DQB1 polymorphisms substantively modulate host immune response and therefore, influence the evolution of HBV infection. HLA genetic polymorphisms cause differences in the HLA molecular structure, focusing on antigenic peptide binding pockets and determining the selectivity of individual HLA molecules' binding to antigenic peptides [12,23]. Consequently, genome-wide association studies have shown that single nucleotide polymorphisms (SNPs) near HLA-DQ loci significantly correlate with outcomes of HBV infection. Diversities in the HLA class II gene are significantly associated not only with persistent HBV infection but also with clearance, spontaneous HBV seroconversion, disease progression, and the development of HBV-related liver cirrhosis and hepatocellular carcinoma [24, 25]. In conclusion, our study indicates that in the population of Burkina Faso, the HLA-DQB1\*03: 03 allele appears to exert a protective effect on the natural history of hepatitis B by potentially stabilizing the infection in a chronic state and limiting progression to severe liver disease. Conversely, the HLA-DQB1\*05: 02 allele was associated with an increased risk of disease progression. Interestingly, the presence of both HLA-DQB1\*03: 03 and DQB1\*05: 02 appeared to offer protection against cirrhosis. While HLA-DQB1\*06: 04 seemed to be an independent factor in viral evolution, it may increase the risk of cirrhosis during chronic infection. Given that HBV pathogenesis is multifactorial, larger cohorts are necessary to definitively establish the link between these alleles and clinical outcomes. Future research should integrate HBV genotyping with broader HLA profiling and epigenetic factors, such as cfDNA methylation, to provide a more comprehensive diagnostic framework

### **List of abbreviations**

AFP - Alpha Fetoprotein  
CHB - Chronic Hepatitis B  
DNA - Deoxyribonucleic acid  
HBV - Hepatitis virus

HCC - Hepatocellular carcinoma  
HGF - Human Growth Factor  
HLA - Human leukocyt antigen  
LC - Liver cirrhosis  
OR - Odds ratio  
PCR - Polymerase Chain Reaction

### **Authors' Contributions**

STS and LT developed the study protocol. LO and VZ performed laboratory analysis and data interpretation, APS participated to samples collection. LO, ATY, STS drafted the manuscript, FWD and JS approved the final version of the manuscript.

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### **Data Availability**

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

### **Declarations**

### **Ethics approval and informed consent**

This study was approved by the Burkina Faso Health Research Ethics Committee (deliberation number 2022-02-027). Participants in this study were asked to consent before participating in the survey. Each participant was assigned a unique identification number to guarantee anonymity and confidentiality. An interview and information phase was carried out with each participant; at the end, free, informed, and signed consent will be given before inclusion and biological sampling.

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