Scientific Research Publishing

Advances in Bioscience and Biotechnology, 2025, 16(4), 138-150
https://www.scirp.org/journal/abb
ISSN Online: 2156-8502
ISSN Print: 2156-8456

Involvement of IL10 rs1800872 and rs1800896 in the Acquisition and Progression of Dengue Virus Infection to Severe Forms of the Disease in Burkina Faso

Aziz Sidi Aristide Tapsoba[1,2*], Fadilatou Tassembedo[1], Lassina Traore[1], Shoukrat Ohuwa Toyin Bello[3], Mousso Sawadogo[1], Teega-Wende Clarisse Ouedraogo[1], Rogonoma Alice Ouedraogo[4], Patoinéwendé Denise Ilboudo[1,5*], Potiandi Serge Diagbouga[6], Florencia Wendkuuni Djigma[1*], Jacques Simpore[1,3]

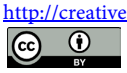
[1]Laboratoire de Biologie Moléculaire et de Génétique (LABIOGENE), Université Joseph KI-ZERBO, Ouagadougou, Burkina Faso
[2]Centre Hospitalier Régional de Koudougou (CHR/KDG), Koudougou, Burkina Faso
[3]Centre de Recherche Biomoléculaire Pietro Annigoni (CERBA), Ouagadougou, Burkina Faso
[4]Centre Universitaire de Gaoua, Université NAZI Boni, Bobo-Dioulasso, Burkina Faso
[5]Université Yembila Abdoulaye Toguyeni, Fada N'Gourma, Burkina Faso
[6]Institut de Recherche en Sciences de la Santé, Centre National de la Recherche Scientifique et Technologique (IRSS/CNRST), Ouagadougou, Burkina Faso
Email: *aristap87@gmail.com, *denise.ilboudo@gmail.com, *florencia.djigma@gmail.com

How to cite this paper: Tapsoba, A.S.A., Tassembedo, F., Traore, L., Bello, S.O.T., Sawadogo, M., Ouedraogo, T.-W.C., Ouedraogo, R.A., Ilboudo, P.D., Diagbouga, P.S., Djigma, F.W. and Simpore, J. (2025) Involvement of IL10 rs1800872 and rs1800896 in the Acquisition and Progression of Dengue Virus Infection to Severe Forms of the Disease in Burkina Faso. *Advances in Bioscience and Biotechnology*, **16**, 138-150.
https://doi.org/10.4236/abb.2025.164008

Received: March 30, 2025
Accepted: April 26, 2025
Published: April 29, 2025

Abstract

Background: Dengue fever is a major public health problem in Burkina Faso. Some people infected by dengue virus develop a severe dengue (SD) and potentially fatal form of the disease which are dengue hemorrhagic syndrome or dengue shock syndrome. Host genetic factors may be relevant, predisposing some individuals to severe disease. Polymorphisms of pro-inflammatory cytokines have been associated with the pathogenesis of dengue fever (DF). The aim of this study was to assess the involvement of IL10 cytokine gene polymorphisms to restriction sequence (rs), rs1800872 and rs1800896 in the pathogenesis of dengue fever and even in its progression to severe forms of the disease in Burkina Faso. We conducted a descriptive and analytical case-control study. A total of 246 subjects were recruited including, with 117 people who had never been in contact with the dengue virus considered as controls and 129 dengue disease patients. Polymorphisms in the IL10-592 and IL10-1082 genes were obtained by restriction fragment length polymorphism (PCR-RFLP). We found that the AA genotype (46.81%/DF, 53.57%/DS and 23.08%/control) of the IL10-592 polymorphism was a risk factor associated with the DS form and the CA heterozygote (21.31%) tended not to favor DF



infection. The GG genotype of IL10-1082 was associated with dengue fever and possible progression from DF to DS, while the AG genotype was associated with a higher risk of DF infection. The A allele of IL10-1082 (OR = 1.521 CI [1.046 - 2.215], p-value = 0.027) was associated with the development of dengue and would not be indifferent in the progression to the DS form. Polymorphisms in the IL10-1082 and IL10-592 genes seems to be associated with the pathogenesis of dengue disease and also with progression to the severe form of the disease.

Keywords

Dengue Hemorrhagic Syndrome, Dengue Shock Syndrome, Gene Polymorphism, IL10, Burkina Faso

1. Introduction

Dengue is the most important viral disease transmitted to humans by the bite of the *Aedes aegypti* mosquito in most tropical and subtropical countries of the world [1] [2]. There are an estimated 50 to 100 million cases of dengue fever worldwide every year [3]. Isolated for the first time in 1925 in Burkina Faso, the country recorded 79,867 suspected cases including 34,687 probable cases and 349 deaths in 2023 [4] [5]. Dengue virus (DENV) is a single-stranded RNA virus of positive polarity of 10.7 Kb, belonging to the genus Flavivirus [6].

There are 5 serotypes of the virus which are genetically related but antigenically distinct, classified as DENV 1 to 5 [7]. DENV 1, 2, 3 and 4 are each capable of causing self-limiting fevers to fatal states. Infection with any of the four viral serotypes confers protective immunity against reinfection with the same serotype only, while subsequent infection with other serotypes can result in severe dengue [6]. DENV 5, isolated in 2013, follows a purely sylvatic cycle [7]. The main target cells of DENV *in vitro* are monocytes, macrophages and dendritic cells [8]. Generally, dengue can be asymptomatic or cause mild illness during a primary infection. However, a second infection can be fatal [9] [10]. Environmental factors, the serotype/genotype of the dengue virus, the immune response and the genetic make-up of the host are considered to influence the development of the clinical manifestations of dengue, as well as the severity of the disease [11] [12]. And the host immune response has been highlighted as a genetic marker of the disease with the production and release of several pro-inflammatory, antiviral and immunoregulatory cytokines [11] [13]. The host immune response is primarily determined by the recognition of the virus by the host cells and the subsequent antiviral response. Th1 cells are typically associated with an acute phase of fever, while Th2 cells are expressed throughout the infection, leading to endothelial cell dysfunction and causing plasma leakage. The activation of T lymphocytes triggers a cytokine cascade. During DENV infection, these cytokines can be secreted by macrophages and monocytes [7].

Previous studies shown that cytokines have a significant impact on the severity of dengue fever, including alpha tumor necrosis factor (TNF-α), interleukin 6 (IL6), interleukin 10 (IL10), interleukin 17 (IL17) and gamma interferons (IFN-γ) [10] [12] [14]. In addition, single-nucleotide polymorphisms (SNPs) in the genes for these cytokines have contributed significantly to our understanding of pathophysiology and the role of host genetics in dengue infection [15] [16]. Serum concentrations of TNF-α, IL6, IL2, and INF-γ are elevated during the first three days of infection of DENV, while high levels of IL10, IL5, and IL4 appear later. The interaction of these cytokines, which can either increase or decrease their production, may be responsible for triggering the inflammatory process in infectious diseases [7].

Interleukin 10 (IL10) plays a pleiotropic immune and inflammatory regulatory role in infectious diseases and is produced by monocytes, macrophages, dendritic cells, regulatory T cells (Tregs) [13] [17]. IL10 contains three distinct SNPs in its promoter region, at nucleotide positions 1082, 819, 592 [17]. In the pathogenesis of DENV, IL-10 has immunomodulatory activity, resulting in persistent viral infection, promoting inflammation and disease severity [12]. Polymorphisms of the IL10 gene could influence the Antibody-Dependent Enhancement reaction (ADE) [3].

Given that cytokine production is influenced by genetic polymorphisms and that different genetic variants have been associated with dengue disease, it would be relevant to investigate the potential role of cytokine genetic polymorphisms, in the pathogenesis of dengue.

The aim of our study was to evaluate the role of interleukin 10 (IL-10) gene polymorphisms rs1800872 and rs1800896 in the pathogenesis of dengue fever and to assess their involvement in the progression of infection to severe forms of the disease in a Burkinabe population.

2. Materials and Methods

2.1. Ethics Approval and Consent to Participate

The study was approved by the Health Research Ethics Committee of the Burkina Faso Ministry of Health: No. 2022-02-034 and written informed consent was obtained from participants prior to blood sampling.

2.2. Type and Study Population

This case-control study, involved patients of all ages, including children and blood donors from various professions and social categories in Ouagadougou. Blood samples from dengue cases were collected in the laboratories of the Hopital Saint Camille de Ouagadougou (HOSCO) and the Pietro Annigoni Biomolecular Research Center (CERBA) from October 2021 to June 2022. Blood samples from subjects with no known history of dengue, received during the collection period, served as controls. Patients with well-documented pre-existing pathologies were excluded from this study.

2.3. Genomic DNA Extraction

Peripheral venous blood was collected from each individual in tubes containing EDTA anticoagulant. Genomic DNA was extracted using the FAVORGEN Mini kit, following the protocol provided by the manufacturer. It was then quantified and checked for purity using the BioDrop apparatus. The DNA was stored at −20˚C until analysis.

2.4. Genotyping

Two bi-allelic polymorphisms of the IL-10 promoter were detected by the PCR-RFLP method using primers that amplify a short fragment of DNA containing the polymorphisms. A pair of primers (sense: 5'-GGTGAGCACTACCTGACTAGC-3' and antisense: 5'-CCTAGGTCACAGTGACGTGG) was used to amplify the IL10-592 fragment (C/A; rs1800872) and for the IL10-1082 (G/A; rs1800896), we used the primer pair (sense: 5'-CCAGATATCTGAAGAAGTCCTG-3'; antisense: 5'-CTCTTACCTATCCCTACTTCC-3'). Amplification was performed in a 20 µL reaction volume containing 1 µL of each primer, 100 ng of template DNA and 10 µL of 5X FIREPOL® master mix and 7 µL of water. Polymerase chain reactions were run for 30 cycles according to the following program: initial denaturation for 5 minutes at 95˚C, denaturation for 30 seconds at 95˚C, hybridization for 30 seconds at 55˚C, extension for 30 seconds at 72˚C and final extension for 5 minutes at 72˚C.

2.5. Enzymatic Digestion

The amplification products, either 5 µL of each sample, were digested with the restriction enzymes Fast Digest RSaI (Thermo Scientific TM) and Fast Digest MnlI (Thermo Scientific TM) for the IL-10-592 and IL10-1082 genes respectively (see Table 1) at 37˚C for 30 minutes in a water bath and then subjected to electrophoresis on a 2% agarose gel and stained with ethidium bromide. The enzymatic digestion reaction of each sample was carried out in a total reaction volume of 25 µL containing the enzyme buffer, the enzyme, pure water, and the PCR product. The IL10 A-1082 allele also gave 2 fragments of 134 and 65 bp and the IL10 G-1082 allele gave 3 fragments of 112 bp, 65 bp and 22 bp (Figure 1). The IL10 A-592 allele gave 2 fragments of 236 and 176 bp and the IL10 C-592 allele gave a single fragment of 412 bp (Figure 2).

Table 1. PCR products from enzymatic digestion.

Genes	Polymorphism	Size (bp)	Restriction enzyme	Concentration of electrophoresis gel	Expected digestion products
IL10	IL10-592C/A; (rs1800872)	412	RSAI	2%	AA (264 pb et 176 pb) AC (412 pb, 236 pb et 176 pb) CC (412 pb)

Continued

IL10-1082G/A; (rs1800896)	189	MnLI	AA (134 pb et 65 pb) AG (134 pb~112 pb 55 pb et 22pb) GG (112 pb 55 pb et 22 pb)

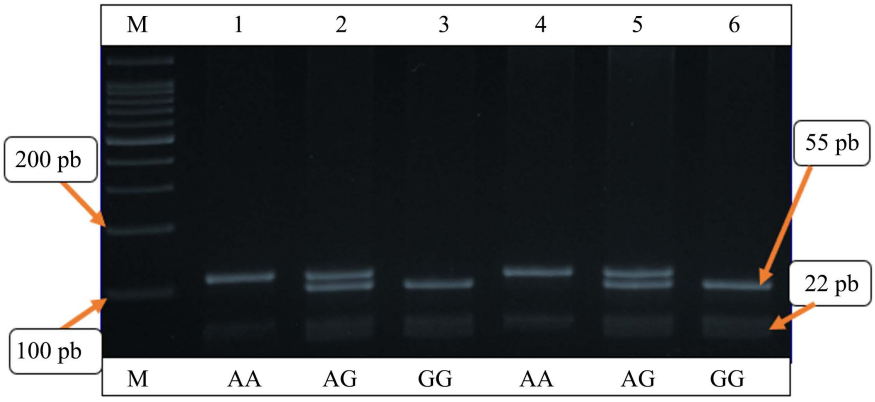


Figure 1. Genotypic illustration of the IL10-1082 G/A polymorphism in agarose gel.

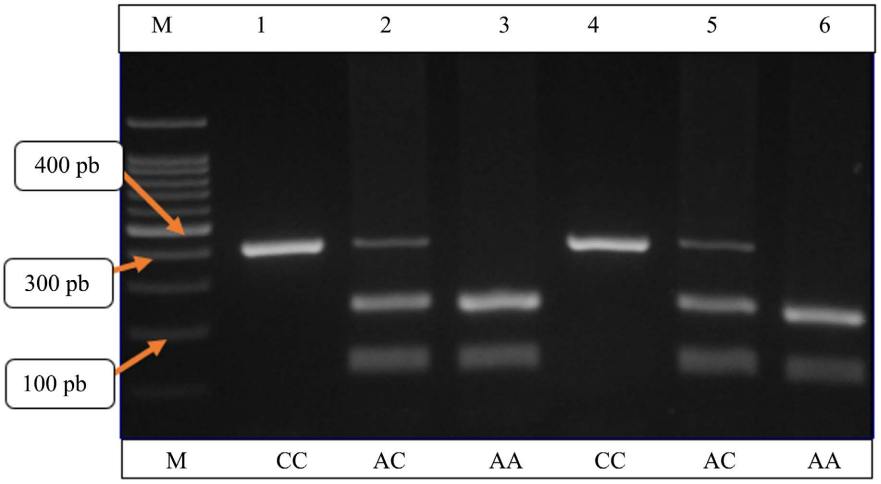


Figure 2. Illustration of IL10-592 C/A genotypic polymorphisms in agarose gel.

2.6. Statistical Analysis

The data were analysed using R software. The chi-square (χ^2) test was used for frequency comparisons. Odds ratios (OR) and 95% confidence intervals (CI) were calculated. Results were considered statistically significant for p value less than 0.05.

3. Results

3.1. Study Population

A total of 246 peoples were recruited. These included 129 people with clinical signs suggestive of dengue fever, confirmed by diagnostic tests at the medical bi-

ology laboratory, and 117 peoples who, based on biological tests, had not been in contact with DENV (Table 2). The study population consisted of 43.09% men infected with DENV compared to 56.91% women. Table 2 also shows that the age group most affected by DENV infection was over 40, with a DF infection rate of 66.89%. The criteria used for cases of severe dengue fever included, plasma leakage characterized by a rise in hematocrit, severe thrombocytopenia, and symptoms noted at the time of consultation, such as abdominal pain with vomiting and splenomegaly. The majority of the population (68.70%, (169/246)) was over 40. This age group also had the highest proportion of cases of dengue fever (65.89%), compared with 12.40% in the least active age group.

Table 2. Socio-demographic data of the study population.

Variables	DF *n* (%)	DS *n* (%)	DF + DS *n* (%)	Controls *n* (%)	Total *n* (%)	OR	95% CI	*p-value*
N	**98**	**31**	**129**	**117**	**246**			
Gender, n (%)								
Male	41 (41.84)	15 (48.39)	56 (43.41)	50 (42.74)	106 (43.09)	-	-	-
Female	57 (58.16)	16 (51.61)	73 (56.59)	67 (57.26)	140 (56.91)	1.303	[0.579 - 2.932]	**0.5397**
Year								
0 - 19	14 (14.29)	2 (6.45)	16 (12.40)	11 (9.40)	27 (10.98)	-	-	-
20 - 39	19 (19.39)	9 (29.03)	28 (21.71)	22 (18.80)	50 (20.33)	0.309	[0.0283 - 1.8449]	**0.1621**
≥40	65 (66.33)	20 (64.52)	85 (65.89)	84 (71.79)	169 (68.70)	0.3016	[0.0562 - 1.6190)	**0.2775**

3.2. Serological Characteristics of the Study Population

Table 3 shows the proportions of DF and DS according to the stage of infection. Primary DENV infection was 46.94% (46/98) and 12.90% (4/31), respectively. Among severe dengue cases, 45.16% (14/31) had at least one previous infection, with 22.58% in the acute phase. The proportion of the population (42.86% (42/98)) had antibody serology corresponding to a phase of infection secondary to another type of DENV or in the recovery phase.

Table 3. Serological data specific to DENV infection in the study population.

Serological markers DENV	Controls		Dengue fever (DF)		Severe dengue (DS)		Total	
	N	%	N	%	N	%	N	%
AgNS1	0	0	61	62.24	17	54.84	78	31.71
Ac-IgM−/IgG−	117	100	46	**46.94**	04	**12.90**	167	67.89
Ac-IgM−/IgG+	0	0	42	**42.86**	14	**45.16**	56	22.76
Ac-IgM+/IgG−	0	0	01	**01.02**	07	**22.58**	08	03.25

Continued

Ac-IgM+/IgG+	0	0	09	**09.18**	06	19.35	15	06.10
TOTAL	**117**	**100**	**98**	**100**	**31**	**100**	**246**	**100**

3.3. Relationship between Hematological Parameters and Dengue Acquisition

The following figures show three blood cell count (CBC) parameters in a whisker plot to assess their involvement in DF. Figure 3 and Figure 4 show the median lymphocyte and hematocrit levels, which are almost identical in both groups. Figure 5, on the other hand, shows the platelet count in DF cases versus controls. From these graphs, we can see that the drop in platelet count is linked to dengue fever (p = 0.00).

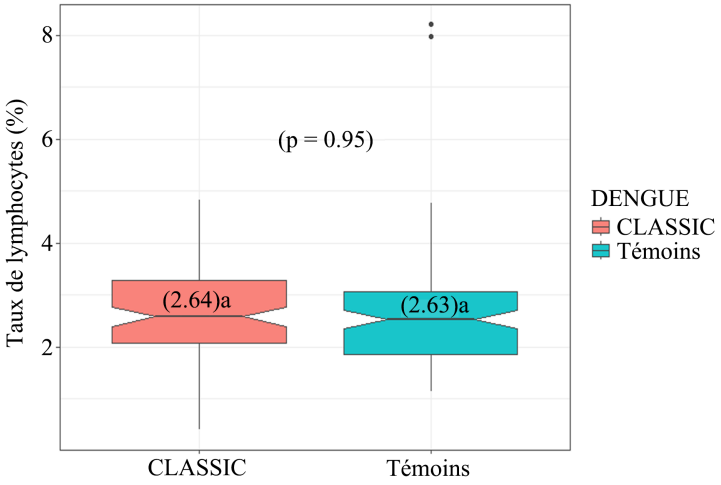


Figure 3. Moustache plot of lymphocyte counts in the study population.

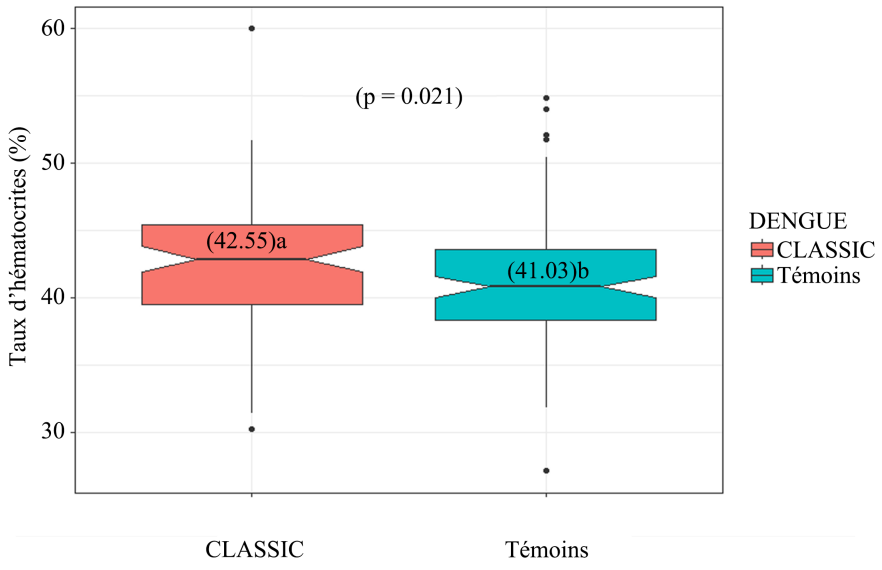


Figure 4. Graph of haematocrit levels in the study population.

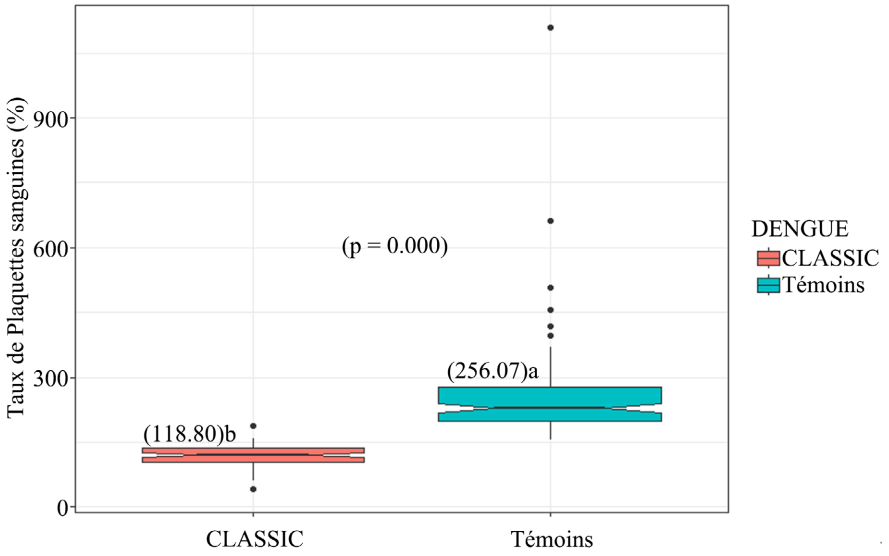


Figure 5. Involvement of thrombocytopenia in dengue disease.

3.4. Genotypic Distribution of IL10-592 and IL10-1082 Genes

The frequencies of IL10-592 and IL10-1082 gene polymorphisms were assessed, and the genotypic distribution was in Hardy-Weinberg equilibrium in both patients and controls (p-value < 0.05) (see **Table 4**). Statistical analysis of IL10-592 and IL10-1082 polymorphisms indicates their involvement in DF infection. The AA genotype (48.36%, p < 0.05) of the IL10-592 SNP is associated with the risk of developing the DS form, while the CA heterozygote (21.31%) tends not to promote DF infection and even slows its progression to the DS form.

Table 4. IL10 genotype frequencies in different forms of dengue fever.

SNP	Genotypes	DF/Control n (%)	DS/Controls n (%)	Controls n (%)	DS/DF n (%)
IL-10-592 (rs1800872)	**CC**	19 (20.21)	7 (25.00)	49 (41.88)	26 (21.31)
	CA	31 (32.98)	6 (21.43)	41 (35.04)	37 (30.33)
	AA	44 (46.81)	15 (53.57)	27 (23.08)	59 (48.36)
	HWE p-value	**0.007019**	**0.009695**	**0.003803**	**0.0001697**
IL10-1082 (rs1800896)	**AA**	18 (19.35)	6 (19.35)	29 (25.66)	24 (19.35)
	AG	26 (27.96)	7 (22.58)	38 (33.63)	33 (26.61)
	GG	49 (52.69)	18 (58.06)	46 (40.71)	67 (54.03)
	HWE p-value	**0.00042**	**0.01144**	**0.001011**	**0.00001418**

As for the SNP of IL10-1082, its GG genotype would be associated with a predisposition to progression from DF to DS, while its AG genotype would confer greater protection against progression to the severe form of dengue.

3.5. IL10 Genotype Frequencies According to Circulating Dengue Serotypes in Burkina Faso

In the present study, DENV1 was the most prevalent, and DENV4 was the least frequent (Table 5). However, this difference was not statistically significant in terms of the relationship between the genotypes of the genes studied and the different DENV serotypes circulating in Burkina Faso.

Table 5. IL10 genotype frequencies by DENV serotype.

SNP	Genotypes	DENV 1 n (%)	DENV 2 n (%)	DENV 3 n (%)	DENV 4 n (%)
IL10-592 (rs1800872)	CC	0 (0.00)	2 (1.55)	0 (0.00)	0 (0.00)
	CA	7 (24.14)	4 (13.19)	1 (3.45)	0 (0.00)
	AA	7 (24.14)	5 (17.24)	4 (13.19)	1 (3.45)
	HWE p-value	**0.513**	**0.5377**	**1.00**	**NA**
IL10-1082 (rs1800896)	AA	2 (6.90)	0 (0.00)	1 (3.45)	0 (0.00)
	AG	3 (10.34)	4 (13.19)	0 (0.00)	0 (0.00)
	GG	8 (6.20)	5 (17.24)	5 (17.24)	1 (3.45)
	HWE p-value	**0.1652**	**1.00**	**0.09091**	**NA**

3.6. Genotypic and Allelic Polymorphisms of IL10 Genes and Dengue Pathogenesis

The allelic distribution shows a higher frequency of the allele A of both genes in controls than in DS and DF patients (59.40% for IL10-592 and 42.48% for IL10-1082). Indeed, with p-value < 0.05, we could suggest that the allele A would confer less risk of infection in DF and would not favor progression from DF to the DS form. The C allele of the IL10-592 gene and the G allele of the IL10-1082 genotype had higher frequencies in patients with DF compared with DS (63.30% and 66.67%) (Table 6).

Table 6. Allelic frequency and involvement of IL10-592 and IL10-1082 genes in dengue pathogenesis.

SNP	Genotypes and alleles	DF n (%)	DS n (%)	Controls n (%)	OR	CI (95%)	*p-value*
IL10-592 (rs1800872)	CA vs CC	31 (62.00)	6 (46.15)	41 (45.56)	0.591	[0.305 - 1.132]	0.108
	AA vs CC	44 (69.84)	15 (68.18)	27 (35.53)	0.246	[0.125 - 0.471]	0.000
	CA + AA vs CC	75 (79.79)	21 (75.00)	68 (58.12)	0.378	[0.212 - 0.664]	0.001
	C (%)	119 (63.30)	36 (64.29)	95 (40.60)	-	-	-
	A (%)	69 (36.70)	20 (35.71)	139 (59.40)	2.523	[1.747 - 3.662]	0.000
IL10-1082 (rs1800896)G/A	AG vs GG	26 (34.67)	7 (28.00)	38 (45.24)	1.671	[0.918 - 3.062]	0.089
	AA vs GG	18 (26.87)	6 (25.00)	29 (38.67)	1.752	[0.906 - 3.418]	0.090
	AG + AA vs GG	44 (47.31)	13 (41.94)	67 (59.29)	1.707	[1.020 - 2.873]	0.040
	G (%)	124 (66.67)	43 (69.36)	130 (57.52)	-	-	-
	A (%)	62 (33.33)	19 (30.64)	96 (42.48)	1.521	[1.046 - 2.215]	0.027

4. Discussion

IL-10 is a major anti-inflammatory cytokine, essential in controlling the host immune response by regulating the production of several pro-inflammatory cytokines. It has been associated with several diseases and is considered an important immunoregulatory mediator produced by monocytes, dendritic cells and T and B lymphocytes [12] [14]. IL-10 is also likely to be regulated at transcriptional level by several polymorphisms, including 819 and 1082 [12].

The aim of our study was to evaluate the involvement of IL10 gene polymorphisms in the pathogenesis of dengue disease and its progression to severe forms in a Burkinabe population. In the present study, the different IL10 polymorphisms showed that IL10-592 and IL10-1082 were statistically associated with DF and even progression from DF to the severe DS form.

Examining the genotypic distribution of IL10-592 polymorphisms, we found that the AA genotype (46.81% of DF cases and 53.57% in DS cases, with a p-value < 0.001) could be associated with the risk of developing DS, while the CA heterozygote (32.98%s of DF cases and 21.43% in DS cases, with a p-value < 0.001) could confer resistance in the progression to the DS form; and homozygous CC (20.21% of DF cases and 25.00% in DS cases, with a p-value < 0.001) would tend not to favor DF infection.

The GG genotype of the IL10-1082 gene could be a predisposing factor for DF and even progression to DS (52.69% DF and 58.06% DS). Whereas the AA genotype (19.35% DS/DF with p-value < 0.001) could be a protective factor against progression to DS. Gyeltshen *et al.* obtained a frequency of GG genotype associated with higher plasma levels of IL10-1082 [16]. According to the virus serotypes circulating in the study population, we obtained equal frequencies of AA and CA genotypes (24.14%) in serotype DENV1, which was the most frequently encountered. Serotype DENV4 was also present in the Burkinabe population, and the AA genotype of IL10-592 and the GG genotype of IL10-1082 were present at 3.45%. The study by Féitosa *et al.* in Brazil also shows an association of high IL10 levels with DENV1 infections [18]. The association of AG + AA mutant genotypes (47.31% of DF cases and 41.94% of DS cases; OR = 1.707; CI [1.020 - 1.873] with p-value = 0.040) of the IL10-1082 gene could confer slight protection against DS.

The allele C allele of IL10-592 is proportionally predominant in dengue cases with all CA+AA mutated genotypes, compared with the wild-type AA genotype, which would be a factor slowing down progression to DS (79.79% of DF cases and 75.00% in DS cases; OR = 0.378; CI [0.212 - 0.664] with a p-value = 0.001). Also, the A allele of IL10-1082 (OR = 1.521 CI [1.046 - 2.215], p-value = 0.027) was associated with the development of dengue and would not be indifferent in the progression to the DS form; this may be in line with the study of cytokine polymorphism frequencies in a population from Western Europe, Africa, Asia, the Middle East and South America, which obtained high frequencies of the IL10-1082 A allele, and also with the study on a Bhutanese population which found a frequency of the IL10-592 C allele at 53.86% [16] [17].

Our results are in line with a large number of studies that have shown the association of the IL10 gene with dengue fever. Such as those obtained in Indonesia, which obtained higher IL10 levels in DF than in DS [10]; in Cuba, which found that SNPs-1082 (G/A), and -592 (C/A) were associated with DHF risk [14]; in Sri Lanka, which confirmed the association between the same genotypes and DHF, suggesting a risk factor for the development of DS, while alternative combinations were associated with protection against dengue hemorrhagic fever [11]; and also in Brazil, which obtained a distribution of IL10-1082 genotypes [18].

Knowing that one study had associated these genotypes with low IL10-expression we can suggest with other authors that dengue-associated genotypes could be responsible for the ineffective immune response and resulting severity [7] [11] [14].

5. Conclusion

The results of this study showed a statistically significant association of IL10-592, IL-1082 gene polymorphisms in dengue virus infection in the Burkinabe population, and also their implications in disease progression to severe forms of the disease. Despite the studies carried out to understand the pathogenesis of dengue disease, questions remain as to how many genes contribute to susceptibility to dengue, and to what extent these genes interact with each other to cause severe disease. Further studies with other genes and also with the involvement of other arboviruses, particularly in cases of co-infections, in different population groups are essential for a complete understanding of the genetic associations with dengue infections.

Acknowledgements

We would like to thanks The World Academy of Sciences and the Swedish International Development Cooperation Agency (Sida) for funding this research through the grant No. 17-403 RG/BIO/AF/AC_I–FR3240297757. We thank also the UNESCO chaire in "Génie génétique et Biologie Moléculaire" for the technical support.

Informed Consent Statement

See ethical approval.

Data Availability

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

Author Contributions

Conceptualization, data curation, formal analysis, methodology, writing—original draft, writing—review and editing by ASAT, FT, JS and FWD.

FWD, ASAT, FT, LT, SOTB, TWCO, RAO, PDI, MS, PSD and JS contributed for the samples analysis, validation, review and editing the manuscript.

FWD mobilized the funding for the study.

All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Wagenaar, J.F.P., Mairuhu, A.T.A. and van Gorp, E.C.M. (2004) Genetic Influences on Dengue Virus Infections. *Dengue Bulletin*, **28**, 126-134.

[2] Cordeiro, C.A., Moreira, P.R., Andrade, M.S., Dutra, W.O., Campos, W.R., Orefice, F., *et al.* (2008) Interleukin-10 Gene Polymorphism (−1082G/A) Is Associated with Toxoplasmic Retinochoroiditis. *Investigative Opthalmology & Visual Science*, **49**, 1979-1982. https://doi.org/10.1167/iovs.07-1393

[3] Kaur, G., Gupta, K., Singh, A., Kumar, N. and Banerjee, I. (2021) Effect of Ifn-Γ +874 T/A Polymorphism on Clinical Manifestations of Dengue: A Meta-Analysis. *Journal of Genetics*, **100**, Article No. 91. https://doi.org/10.1007/s12041-021-01344-9

[4] Gonzalez, J.P., Du Saussay, C., *et al.* (1985) Dengue in Burkina Faso (ex-Upper Volta): Seasonal Epidemics in the Urban Area of Ouagadougou. *Bulletin de la Société de pathologie exotique et de ses filiales*, **78**, 7-14.

[5] Sondo, K.A., Diendéré, E.A., *et al.* (2022) Descriptive Study of Complications of Dengue during the 2016 Outbreak in Ouagadougou, Burkina Faso. *Pan African Medical Journal One Health*, **7**, 27.

[6] Patro, A.R.K., Mohanty, S., Prusty, B.K., Singh, D.K., Gaikwad, S., Saswat, T., *et al.* (2019) Cytokine Signature Associated with Disease Severity in Dengue. *Viruses*, **11**, Article 34. https://doi.org/10.3390/v11010034

[7] Cansanção, I.F., do Carmo, A.P.S., Leite, R.D., Portela, R.D.P., de Sá Leitão Paiva Júnior, S., de Queiroz Balbino, V., *et al.* (2016) Association of Genetic Polymorphisms of Il1β-511 C>t, IL1RN VNTR 86 Bp, IL6 -174 G>c, IL10 -819 C>t and Tnfα-308 G>a, Involved in Symptomatic Patients with Dengue in Brazil. *Inflammation Research*, **65**, 925-932. https://doi.org/10.1007/s00011-016-0975-5

[8] Mathew, A. and Rothman, A.L. (2008) Understanding the Contribution of Cellular Immunity to Dengue Disease Pathogenesis. *Immunological Reviews*, **225**, 300-313. https://doi.org/10.1111/j.1600-065x.2008.00678.x

[9] Green, S., Pichyangkul, S., Vaughn, D.W., Kalayanarooj, S., Nimmannitya, S., Nisalak, A., *et al.* (1999) Early CD69 Expression on Peripheral Blood Lymphocytes from Children with Dengue Hemorrhagic Fever. *The Journal of Infectious Diseases*, **180**, 1429-1435. https://doi.org/10.1086/315072

[10] Masyeni, S., Wardhana, I.M.W. and Nainu, F. (2024) Cytokine Profiles in Dengue Fever and Dengue Hemorrhagic Fever: A Study from Indonesia. *Narra J*, **4**, e309. https://doi.org/10.52225/narra.v4i1.309

[11] De Silva, A., Fernando, A., Malavige, G., Perera, K.L., Premawansa, S. and Ogg, G. (2015) Polymorphisms of Transporter Associated with Antigen Presentation, Tumor Necrosis Factor-*A* and Interleukin-10 and Their Implications for Protection and Sus-

ceptibility to Severe Forms of Dengue Fever in Patients in Sri Lanka. *Journal of Global Infectious Diseases*, **7**, 157-164. https://doi.org/10.4103/0974-777x.170501

[12] Santos, A.C.M.D., de Moura, E.L., Ferreira, J.M., Santos, B.R.C.D., Alves, V.D.M., de Farias, K.F., *et al.* (2016) Meta-Analysis of the Relationship between TNF-α (−308G/A) and IL-10 (−819C/T) Gene Polymorphisms and Susceptibility to Dengue. *Immunological Investigations*, **46**, 201-220. https://doi.org/10.1080/08820139.2016.1248560

[13] Srikiatkhachorn, A., Mathew, A. and Rothman, A.L. (2017) Immune-Mediated Cytokine Storm and Its Role in Severe Dengue. *Seminars in Immunopathology*, **39**, 563-574. https://doi.org/10.1007/s00281-017-0625-1

[14] Perez, A.B., Sierra, B., Garcia, G., Aguirre, E., Babel, N., Alvarez, M., *et al.* (2010) Tumor Necrosis Factor–Alpha, Transforming Growth Factor-β1, and Interleukin-10 Gene Polymorphisms: Implication in Protection or Susceptibility to Dengue Hemorrhagic Fever. *Human Immunology*, **71**, 1135-1140. https://doi.org/10.1016/j.humimm.2010.08.004

[15] Fernández-Mestre, M.T., Gendzekhadze, K., Rivas-Vetencourt, P. and Layrisse, Z. (2004) TNF-α-308a Allele, a Possible Severity Risk Factor of Hemorrhagic Manifestation in Dengue Fever Patients. *Tissue Antigens*, **64**, 469-472. https://doi.org/10.1111/j.1399-0039.2004.00304.x

[16] Gyeltshen, S., Na-Bangchang, K. and Chaijaroenkul, W. (2020) Genertic Polymorphism of Dengue Susceptibility Genes among Bhutanese Population. *Science and Technology Asia*, **25**, 125-134. https://doi.org/10.14456/scitechasia.2020.54

[17] Meenagh, A., Williams, F., Ross, O.A., Patterson, C., Gorodezky, C., Hammond, M., *et al.* (2002) Frequency of Cytokine Polymorphisms in Populations from Western Europe, Africa, Asia, the Middle East and South America. *Human Immunology*, **63**, 1055-1061. https://doi.org/10.1016/s0198-8859(02)00440-8

[18] Feitosa, R.N.M., Vallinoto, A.C.R., Vasconcelos, P.F.D.C., Azevedo, R.D.S.D.S., Azevedo, V.N., Machado, L.F.A., *et al.* (2016) Gene Polymorphisms and Serum Levels of Pro and Anti-Inflammatory Markers in Dengue Viral Infections. *Viral Immunology*, **29**, 379-388. https://doi.org/10.1089/vim.2016.0026