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Population genetics of *Aedes aegypti* collected in suitable development sites in the district of Abidjan (Côte d'Ivoire)

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Abstract

In Côte d'Ivoire, especially in Abidjan, outbreaks of dengue are regularly reported since 2009. Despite the role of *Aedes aegypti*, the primary vector, in dengue transmission, limited information exists on its genetic diversity and population structure. This study aims to describe the dynamics of Aedes populations alongside with genetic data in this region.

Mosquito larvae and pupae were collected from three localties in Abidjan district: Deux-Plateaux, Riviera-Golf, and Bingerville. DNA was extracted, and five microsatellite loci were amplified by PCR. Genetic data were analyzed using Wright's fixation indices (F_{IS}, F_{ST}, and F_{IT}) and software such as Fstat and Micro-Checker to detect null alleles, short allele dominance (SAD), and *stuttering*.

This study revealed 64 alleles and 100% genetic polymorphism of *Aedes aegypti* populations. The number of the alleles per locus ranged from 9 to 15. A highly significant association (70%) among the loci, moderate population differentiation (global F_{ST} = 0.166) and a significant heterozygote deficit (global F_{IS} = 0.733 > 0) were observed. No distinct genetic population structure or geographic clustering was observed.

This study highlights a moderate genetic diversity among *Aedes aegypti* populations in Abidjan that could largely attributed to high levels of gene flow and small geographic distribution. These observations are critical for refining vector control strategies in Côte d'Ivoire.

Keywords: Population genetics; Allelic dropouts; Null alleles; *Stuttering*; Heterozygosity

1. Introduction

Vector-borne diseases, particularly arboviruses, represent a major threat to public health, especially in tropical and subtropical regions [1, 2]. Among these arboviruses, dengue, Zika, chikungunya, and yellow fever affect millions of people each year, causing significant morbidity and mortality [3]. Dengue, also known as « tropical flu » or « breakbone fever » is currently the most widespread re-emerging arboviral disease globally [4]. This viral infection is transmitted through the bites of infected mosquitoes from the genus *Aedes*, particularly *Aedes aegypti* and *Aedes albopictus* [5]. Among the mosquito species identified as vectors, *Aedes aegypti* is considered the primary vector [1].

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In sub-Saharan Africa, *Aedes aegypti* comprises two morphologically and ecologically distinct subspecies, *Aedes aegypti aegypti* and *Aedes aegypti formosus* [6, 7]. The dark, sylvatic form, *Aedes aegypti formosus*, predominantly thrives in natural environments such as tree cavities and preferentially feeds on forest-dwelling animal hosts. The domestic form, *Aedes aegypti aegypti*, develops in anthropogenic habitats. This domesticated form lives near human dwellings and plays a primary role in the transmission of diseases like dengue.

The number of reported dengue cases has significantly increased, reaching 4.2 million in 2019 [3]. This rise is accompanied by a major health impact, with significant mortality [3].

In Côte d'Ivoire, particularly the district of Abidjan, stands out due to conditions highly conducive to the proliferation of *Aedes aegypti*. This region, characterized by high population density, rapid urbanization, and often inadequate infrastructure, creates an ideal environment for the development of this mosquito. Stagnant water, resulting from poor waste management or domestic practices, provides breeding sites that facilitate their multiplication.

However, despite the scale of this issue, detailed scientific data on the genetic diversity and population structure of local *Aedes aegypti* populations, as well as their adaptation to environmental and anthropogenic pressures, remain limited. Such data are essential for better understanding the geographic spread of the vector and improving vector control strategies. The objective of the present study is to explore the dynamic of *Aedes aegypti* populations based on genetic data in Abidjan district in Côte d'Ivoire.

2. Material and methods

2.1. Study Area and sampling

Abidjan, the economic capital of Côte d'Ivoire, is located in the southern part of the country, along the Golf of Guinea [8]. Like many large tropical cities, Abidjan has a hot and humid climate conducive to the proliferation of *Aedes aegypti*, the primary vector of dengue fever. A 2017 WHO study indicated that approximately 80% of cases originated from the Cocody-Bingerville health district [9]. This present study was conducted in three sites: Deux Plateaux, Riviera Golf, and Bingerville (Figure 1). These sites were chosen based on the level of dengue transmission risk [10].

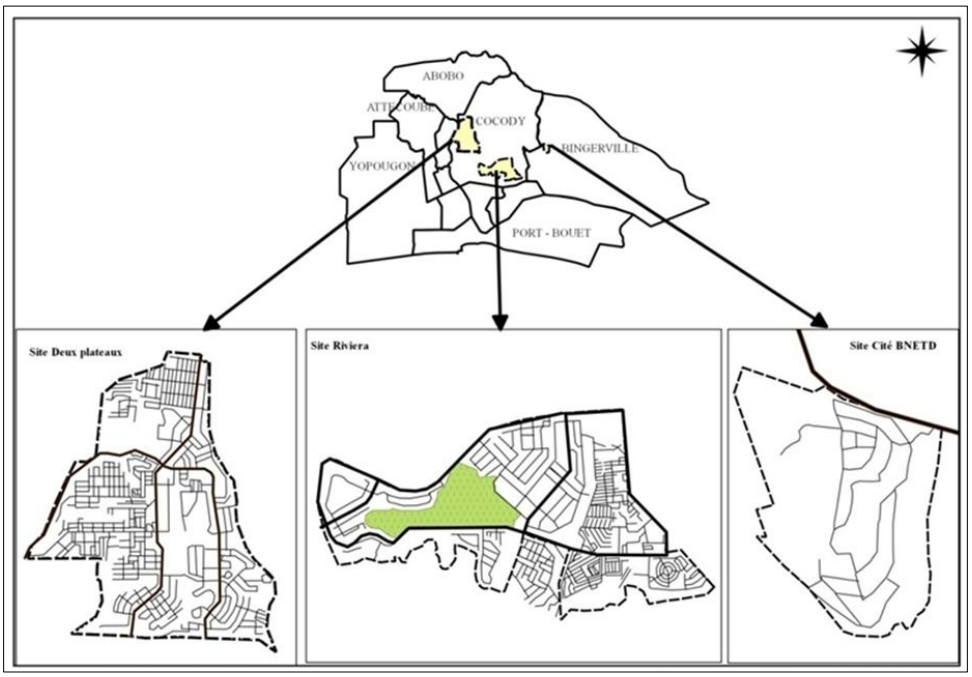

Figure 1 Sampling sites in Abidjan district (source: INS/ Nos enquêtes, 2019)

2.2. DNA extraction and PCR

DNA was extracted from the supernatant of mosquito homogenates using the sodium chloride (NaCl) method adapted from Laure Sonnier [11]. The resulting dry pellet was dissolved in 20 µl of sterile water and stored at -20°C for molecular analyses.

A total of 5 microsatellite loci were used (Table 1). PCR amplification for each locus was performed in a 22.5 µL reaction mixture containing 2.5 µl of 10X buffer (with MgCl), 1.6 µl of dNTPs (400 µM), 1.3 µl of each primer (forward and reverse), 0.2 µl of Taq polymerase, 15.6 µl of molecular-grade water, and 2.5 µl of DNA extract. The PCR amplification program included an initial denaturation step at 95°C for 5 minutes, followed by 30 cycles consisting of denaturation at 94°C for 1 minute, annealing at 58°C for 1 minute and extension at 72°C for 1 minute. A final extension step was performed at 72°C for 10 minutes. The PCR amplicons were then subjected to electrophoresis on a 2% agarose gel containing GelRed. Electrophoresis was carried out for 35 minutes at a voltage of 70 volts. DNA bands were visualized and photographed under UV light.

Table 1 Primer used for genotyping

Primers	Pattern	Primer sequence (forward, reverse)	References
SQM1	CT10(TT) CT	F : AATCGTGACGCGTCTTTTG R : TAACTGCATCGAGGGAAACC	[12]
SQM2	GA15	F : CAAACAACGAACTGCTCACG R : TCGCAATTTCAACAGGTAGG	[12]
SQM3	CAT7	F : ATTGGCGTGAGAACATTTTG R:GAGGAGTGAGCAGATAGGAGTG	[12]
SQM4	TAGA8	F : GCCAAAAACCAACAAACAGG R : AATCGACCCGACCAATAACA	[12]
SQM5	ATA36	F : GGAGCATTCATAGAGAATTGTCA R : GAGATGAACCAGTCATAGGGC	[12]

2.3. Data Analysis

All genetic data were entered into Excel and formatted for the Create software version 1.2 [13]. For population genetics analyses, the samples were divided into three subpopulations: Bingerville, RiveraGolf, and Deux-Plateaux. The genetic structure of the populations was first assessed using linkage disequilibrium (LD) tests and Wright's fixation indices: F_{IS}, F_{ST}, and F_{IT} [14]. Deviations from expected genotypic proportions under local panmixia are measured by F_{IS}, while F_{ST} measures the effect of subdivision (genetic isolation between subsamples), and F_{IT} reflects the combination of both [15]. All these statistical tests were performed with Fstat 2.9.4[16]. The classification of *Aedes* populations was performed by generating a dendrogram using the NJTree (Neighbor-Joining Tree) method [17] based on the Cavalli-Sforza and Edwards distance matrix [18], as recommended by Takezaki and Nei [19].

3. Results

3.1. Genetic diversity

In total, 64 alleles were obtained for five microsatellite loci, with an average allele count of 12.8. The highest number of alleles (15) was found at locus SQM2, and the lowest number (9) was found at locus SQM5. Allele sizes ranged from 098 to 290 bp (Table 2). No allele had a frequency equal to or greater than 95% or 99%; therefore, all loci are polymorphic.

Table 2 Number of alleles and allele size per locus information for genetic markers of *Aedes Aegypti*.

Loci	number of alleles	Allele size
QSM1	14	135 - 270
QSM2	15	098 - 234
QSM3	12	100 - 280
QSM4	14	110 - 290
QSM5	9	098 – 200
Total	**64**	

3.2. Linkage disequilibrium and F-statistics

The proportion of locus pairs in linkage disequilibrium was very high, with 7 significant tests out of the 10 combinations, or 70%. After adjustment using Benjamini and Yekutieli, only two locus pairs remained in linkage disequilibrium (20%) at the 5% threshold. These were the combinations between loci 1 and 4 (QSM1 X QSM4), and between loci 1 and 5 (QSM1 X QSM5). Each locus was involved at least twice in a significant pair of loci in LD (Table 3), except for locus QSM3.

Table 3 Linkage disequilibrium between alleles within and between loci.

Locus pair	*p-value*	p-BY	K	Propsig	PropSigBY
QSM1 X QSM4	0.00053	0.0155235	1	0.7	0.2
QSM1 X QSM5	0.00133	0.0194776	2		
QSM2 X QSM4	0.02293	0.1722652	3		
QSM1 X QSM2	0.02613	0.1722652	4		
QSM4 X QSM5	0.03103	0.1722652	5		
QSM3 X QSM5	0.0355	0.1722652	6		
QSM2 X QSM5	0.04117	0.1722652	7		
QSM3 X QSM4	0.22647	0.8291543	8		
QSM1 X QSM3	0.3573	1	9		
QSM2 X QSM3	0.72607	1	10		

k: number of combinaison; p-BY: *p-adjustment value* of Benjamini and Yekutieli; Prop: proportion.

3.3. Global Heterozygosity

The F_{IS} value (F_{IS} average = 0.733, 95% bootstrap CI = [0.57 - 0.895]) shows a significant and variable deficit in heterozygote (Figure 2). In the different *Aedes aegypti* populations, a significant and variable deficit of heterozygotes was also observed ($F_{IS.\text{Bingerville}}$ = 0.835, $F_{IS.\text{RiveraGolf}}$ = 0.798, and $F_{IS.\text{DeuxPlateaux}}$ = 0.505). A moderate genetic differentiation (F_{ST} average = 0.166, 95% CI = [0.12 - 0.213]) was observed between the *Aedes aegypti* populations (Figure 3).

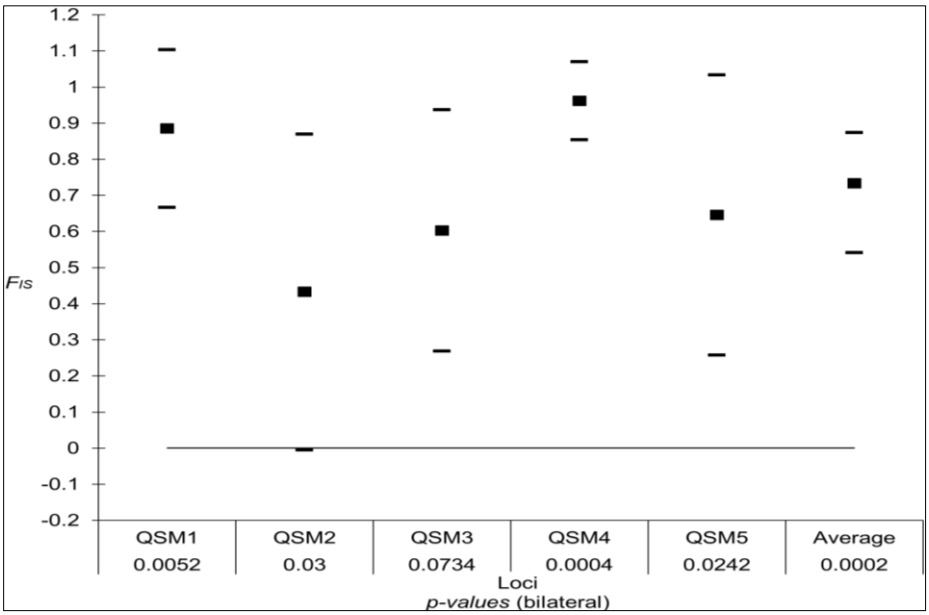

Figure 2 The average F_{IS} values by locus and the 95% confidence interval (CI) obtained using the standard error provided by the jackknife across different populations are shown. The 95% CI for the global value was obtained by

5000 bootstraps on the loci. The different bilateral *p-values* obtained after allele randomization among individuals of each subpopulation are indicated.

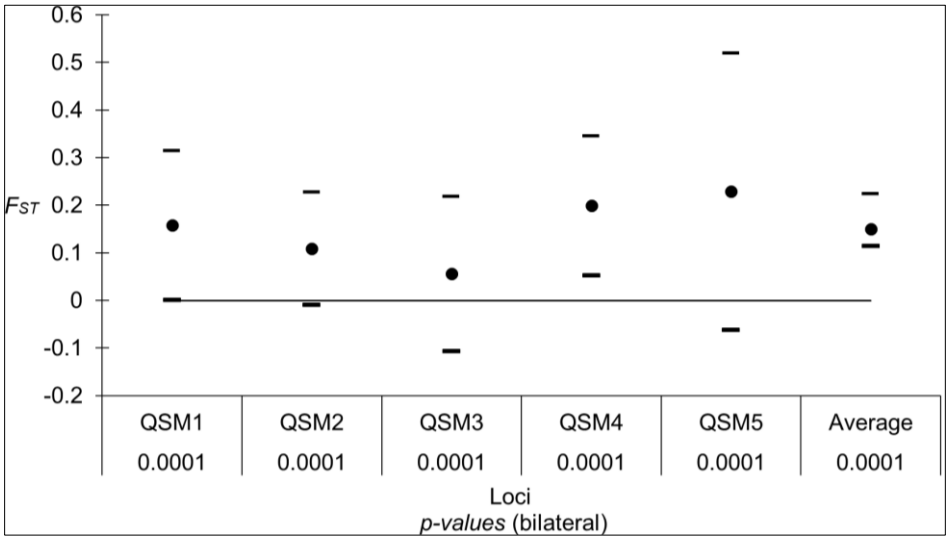

Figure 3 The average F_{IS} values by locus and the 95% confidence interval (CI) obtained using the standard error provided by the jackknife across subpopulations are shown. The 95% CI for the global value was obtained by 5000 bootstraps on the loci. The different bilateral *p-values* obtained after allele randomization among individuals of each subpopulation are indicated.

3.4. Research for Technical Issues behind the Heterozygote deficit and linkage disequilibrium

The ratio between the standard error of F_{IS} (StdrdErrF_{IS} = 0.095) obtained by the jackknife method across all loci and the standard error of F_{ST} (StdrdErrF_{ST} = 0.024) is 3.96. This value of 3.96, which is greater than 2 (3.96 > 2), indicates the presence of null alleles (blanks genotype) and/or probable allele short dominance (SAD) and/or *stuttering*.

3.4.1. Detection of Null Alleles

The Spearman correlation coefficient ρ shows a positive but non-significant correlation between F_{IS} and F_{ST} (Spearman ρ = 0.6, *p-value* = 0.35). The Spearman correlation test revealed a positive but non-significant correlation (ρ = 0.7, *p-value* = 0.2333) between the number of missing data (blank genotypes) observed for each locus and the F_{IS} values. (Figure 4). Analysis performed with Micro-Checker indicates that null alleles partially explain the observed heterozygote deficits at loci QSM2 (*p-value* = 0.4711), QSM3 (p-value = 0.1054), and QSM5 (*p-value* = 0.0853). The *p-values* from the binomial test for loci QSM1 and QSM4 (*p-value* = 0.00483 and *p-value* = 0.00215, respectively) indicate that null alleles do not significantly explain the heterozygote deficits at these loci (Table 4).

3.4.2. Detection of Stuttering

The Micro-Checker analysis did not detect any locus *stuttering* in the different *Aedes* populations studied (Table 4).

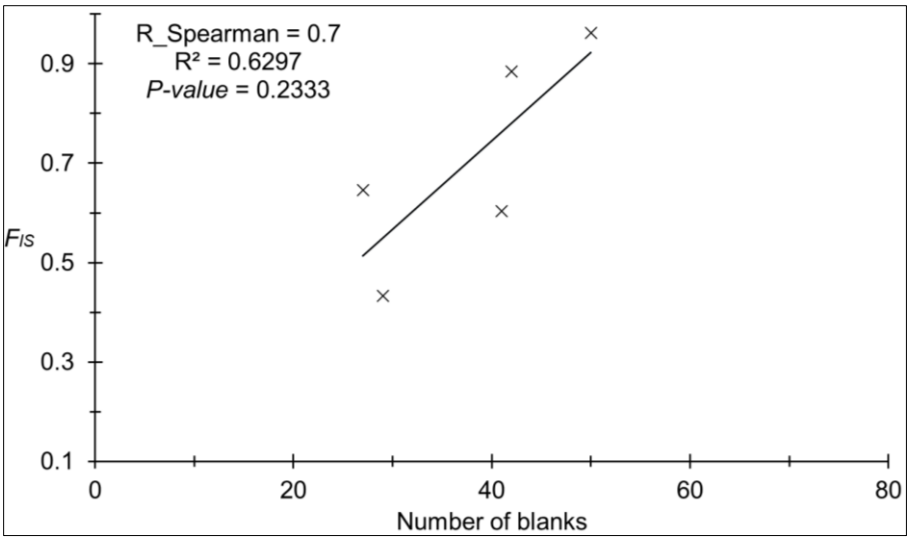

Figure 4 Correlation between the number of missing genotypes observed and the F_{IS} value per locus and across all subpopulations.

Table 4 Synthesis of Micro-Checker results for each locus and across all different Aedes populations.

Loci	Observed blanks	Expected blanks	N'	P_{B2}	*p-value*	Stuttering
QSM1	42	54.60	90	0.61	0.0048	No
QSM2	29	29.86	90	0.33	0.4711	No
QSM3	41	47.39	90	0.53	0.1054	No
QSM4	50	63.29	90	0.7	0.0022	No
QSM5	27	33.78	90	0.38	0.0853	No

Brookfield 2: Brookfield method 2; P_{B2}: Expected frequency of blanks; N: Genotyped individuals; N': Sum of N and observed blanks; Populations (Deux Plateaux, Bingerville, and RiveraGolf); *p-value*: *p-value* (binomial test).

3.4.3. Detection of Short Allele Dominance (SAD)

The regression approach between the allele size of a locus and the F_{IT} to detect SAD showed that only the locus QSM4 exhibited short allele dominance (ρ = -0.61, *p-value* = 0.0103). This approach did not highlight any significant SAD at the loci QSM1, QSM2, QSM3 and QSM5. The level of short allele dominance at the QSM4 locus was confirmed by testing the F_{IS} by allele. The result of this test was highly significant (*p-value* = 0.000262), confirming the phenomenon of short allele dominance.

The results of the panmictic tests, short allele dominance tests, *stuttering* detection, and possible null allele frequencies are provided in figure 5.

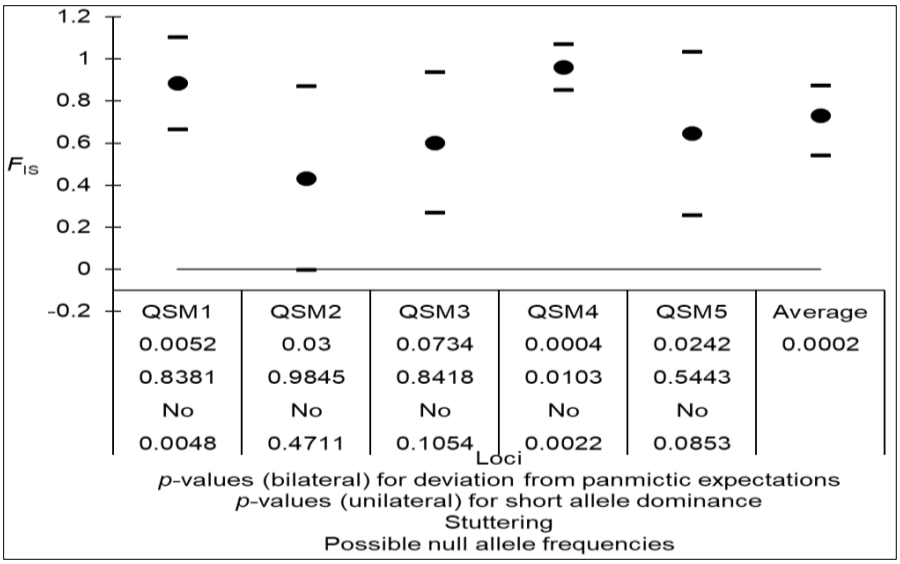

Figure 5 Observed F_{IS} in different *Aedes* populations with 95% confidence intervals from the jackknife method for each locus and the 95% bootstrap confidence interval for the average across loci. The results of the panmictic tests, short allele dominance tests, stuttering detection, and possible null allele frequencies are also provided.

3.5. Neighbor joining tree

Out of the 90 Aedes DNA samples, 33 had more than 50% amplification failures. For better resolution of the tree, the 57 *Aedes* samples with the best amplification rates were considered for this analysis. The NJTree obtained with these samples is shown in Figure 6. The tree does not exhibit a true population structure of *Aedes.* The different populations appear in a scattered order in the tree, with very long branches.

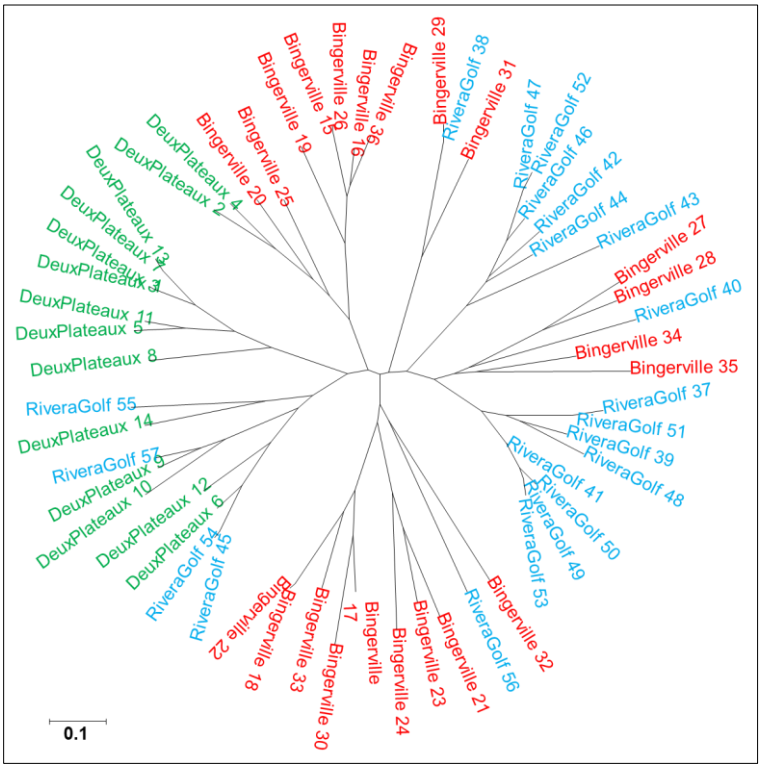

Figure 6 Neighbor-Joining Tree (NJTree) of *Aedes* from a Cavalli-Sforza and Edwards chord distance matrix with the 57 samples that provided an interpretable profile. Biological sample are in blue (Rivera Golf), green (Deux Plateaux) and red (Bingerville)

4. Discussion

In this study, the genetic diversity of *Aedes aegypti* was assessed using five microsatellite loci. All loci were found to be polymorphic at the 95% threshold. This result highlights the effectiveness of the microsatellite loci used for analyzing the genetic diversity of the studied populations. The average number of alleles obtained (12.8) indicates significant allelic richness within the populations.

The linkage disequilibrium tested between locus pairs suggests a highly significant association among these loci, as indicated by the remarkable proportion of associations (70%). Our results indicate that all markers are statistically independent [20].

In both the overall population and the different populations of *Aedes aegypti* studied, a significant and highly pronounced deficit in heterozygosity (F_{IS} *global* > 0 and F_{IS} per population > 0) was observed, contrary to the excess heterozygosity expected in dioecious populations that mate randomly [21]. In the present study, two factors are likely responsible for the observed heterozygote deficits (i) the presence of null alleles and (ii) short allele dominance [22]. The positive correlation between F_{IS} and F_{ST} (ρ = 0.6), as well as the ratio between the standard errors of F_{IS} and F_{ST} (*StdrdErr*F_{IS} = 0.095 / *StdrdErr*F_{ST} = 0.024), which is significantly greater than 2, indicated the presence of null alleles and short allele dominance, as suggested by some authors [21, 22]. Indeed, null alleles and allelic dropouts are specific to certain loci and are likely to produce significant variance in Wright's F_{IS} across loci, similar to what would be observed in cases of rare sexual reproduction [22]. The correlation between F_{IS} and F_{ST} suggests that a hidden genetic structure might exist within the studied population, even though this correlation is not statistically significant (*p-value* = 0.2333). These results do not point to null alleles as the primary explanation for the observed heterozygote deficit in this study. However, they could reflect the possibility of a genetic structure related to a Wahlund effect [21].

The positive but non-significant correlation between the number of missing data points and F_{IS} (ρ = 0.7, *p-value* = 0.2333) indicates that a high number of missing data is associated with elevated F_{IS} values, thus contributing to the observed heterozygote deficit. This correlation could be due to the presence of null alleles [22].

The regression results between allele size at a locus and F_{IT}, as well as the confirmation of the level of SAD using F_{IS} per allele, highlight the phenomenon of short allele dominance at locus QSM4. Specifically, the negative but significant correlation (ρ = -0.61, *p-value* = 0.0103) indicates that allele size and F_{IT} values are inversely related. The correlation between F_{IS} of the alleles at locus QSM4 and their size was highly significant (*p-value* = 0.00026). This result confirms the presence of short allele dominance at this locus (QSM4).

The absence of SAD at loci QSM1, QSM2, QSM3, and QSM5 indicates that these loci do not exhibit technical issues related to allele size. These results suggest that the four aforementioned loci appear to be reliable for population genetic analyses.

The absence of *stuttering* in this study indicates that the observed heterozygote deficits or linkage disequilibrium are not caused by incorrect amplification of certain alleles.

The value average of genetic differentiation between the different populations of *Aedes aegypti* (F_{ST} = 0.166) indicates moderate genetic differentiation among these populations, reflecting the origin of the total genetic variation. These results demonstrate the existence of geographic and genetic structuring among the different populations. The average F_{ST} *value* of 0.166 indicates that a large proportion (83.4%) of the total genetic variability is explained by the sous-population variation, while 16.6% of this variability is attributed to differences between populations.

The analysis of the total populations genetic structure, presented in Figure 7, showed no true structuring of *Aedes aegypti* populations. However, the presence of individuals from diverse origin within the same group could be attributed to human population dynamics, which may transport these vectors from one site to another. It could also be argued that genetic clustering of individuals does not always positively correlate with geographic clustering.

5. Conclusion

This study highlights the moderate genetic diversity of *Aedes aegypti* populations in Abidjan, Côte d'Ivoire, while emphasizing the impact of technical issues such as null alleles and short allele dominance (SAD) in genetic analysis. The results reveal a significant heterozygote deficit (global F_{IS} = 0.733), as well as moderate differentiation between populations (global F_{ST} = 0.166). The lack of clear genetic structure among the populations indicates high genetic

dispersion, with no marked geographic clustering. These data are crucial for refining vector control strategies in Côte d'Ivoire, particularly by taking into account the genetic mechanisms that influence arbovirus transmission.

Compliance with ethical standards

Disclosure of conflict of interest

There is no conflict of interest.

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