Biochemistry and Molecular Biology
2026, Vol. 11, No. 2, pp. 36–41
https://doi.org/10.11648/j.bmb.20261102.12

Research Article

Trim5α Genetic Variants and HIV-1 Infection in the North Region of Cameroon

Marie Nicole Ngoufack[1, *], Georges Nguefack-Tsague[2], Celine Nguefeu Nkenfou[3, 4]

[1]Department of Chemistry-Biology, University of Bertoua, Bertoua, Cameroon
[2]Department of Public Health, University of Yaounde I, Yaounde, Cameroon
[3]Laboratory of Systems Biology, Chantal Biya International Reference Centre (CBIRC), Yaounde, Cameroon
[4]Department of Biological Sciences, Higher Teachers Training College, Yaounde, Cameroon

Abstract

The intrinsic host defense system plays an important role in the restriction of retroviral infection. The objective of the study was to determine the role of Trim5α polymorphism in children living in the North region of Cameroon. A case control study was carried out in the health facilities among HIV-1 infected and uninfected children under 15 years, all born from HIV-1 infected mothers. Blood sample was collected to determine HIV status and genotyping conducted by Polymerase Chain Reaction (PCR) followed by Restriction Fragment Length Polymorphism (RFLP). The Chi-squared test was used to assess the Hardy-Weinberg equilibrium. Overall, 25 HIV-1 infected and 88 uninfected children were recruited. We found that the proportion of GG genotype was lower in uninfected children (85.2%) than in infected ones (92.5%). The proportion of GA genotype was higher in uninfected (12.5%) compared to infected children (8.0%). AA genotype was absent among infected children while the proportion in uninfected children was 2.3%. The frequency of Trim5α-136Q allele in uninfected and infected children was 9.0% and 4.0% respectively. The proportion of mutant homozygotes was elevated in uninfected children (14.8%) than in infected ones (8.0%). Moreover, children carrying mutated phenotype were 2 times less likely to be infected compared to those without it. The mutated phenotype of the Trim5α-136Q gene may be protective against HIV-1 acquisition in children. Further investigation in a follow-up cohort considering other polymorphisms in a large population will help in better appreciation of Trim5α role in HIV-1 acquisition and disease progression in children.

Keywords

Genes Variants, HIV-1, MTCT, Restriction Factor, Trim5α

1. Introduction

Acquired Immunodeficiency Syndrome (AIDS) was discovered 43 years ago and was followed by the identification and characterization of the responsible agent, Human Immunodeficiency virus (HIV). During the replication of HIV in the target cell, it interacts with host cellular proteins that facilitate the process. Various cellular proteins with potent antiviral activities have been developed due to the evolutionary pressures applied by viral infections [1]. Tripartite Motif Containing 5

*Correspondence: Marie Nicole Ngoufack (mnngoufack@gmail.com)

Received: 4 May 2026; **Accepted:** 25 May 2026; **Published:** 27 June 2026

alpha (Trim5α), in cells and tissues of the body plays an important role in the immune response against HIV [2, 3]. A number of cellular proteins are known as restriction factors [4-6]. Trim5α, Apolipoprotein B mRNA editing enzyme, catalytic polypeptide-like (APOBEC), and Tetherin proteins are few important restriction factors that have been extensively studied. [7-10]. Trim proteins are defined by an N-terminal RBCC structure, which consists of an N-terminal RING E3 ligase domain (R), one or two Bbox domains (B), and a coiled-coil domain (CC) [7, 11]. Trim5 is a member of tripartite motif family [12] known as one of the factors contributing to intracellular defense mechanisms against HIV-1 infection [13].

Trim5α is the best studied among members of Trim family that inhibits HIV-1. It has species-specific restriction activity against replication of many retroviruses [14-16]. Trim5α is a post-entry restriction factor that confers resistance to HIV [9] through interaction of its PRYSPRY/B30.2 domain with the viral capsid [6] after the entry of the virus but, prior to the integration of viral DNA into the host cell genome [17]. Inhibition is initiated in the cytoplasm through the recognition of viral capsids, leading to their fragmentation and the suppression of reverse transcription [18]. The mechanism of restriction is not well understood, but it's suggested to involve recruitment of components of the autophagy machinery [19].

Of the eight polymorphisms that have been identified in the Trim5α gene, two have been reported to have functional consequences with regard to the antiviral activity of Trim5α (H43Y and R136Q). The 43Y variant was less efficient in restricting HIV-1 replication *in vitro*. The single amino acid substitution referring to a shift from arginine to glutamine at codon 136 of Trim5α leads to R136Q polymorphism. The Trim5α-136Q mutation has been associated with a higher anti-HIV-1 activity. Trim5α protein possess a C-terminal capsid binding domain that mediates specific recognition and restriction of certain retroviruses. Trim5α has also been observed to possess functional ubiquitin ligase activity that may play a role in its ability to restrict retroviral infection [20].

In some previous studies, the R136Q polymorphism has been associated with a slightly higher anti-HIV-1 activity. The effect of the Trim5α H43Y and R136Q polymorphisms on the clinical course of HIV-1 infection in participants of the Amsterdam cohort studies revealed a protective effect of the 136Q genotype [21]. A study conducted in Nairobi, Kenya suggested that a shift from arginine to glutamine at codon 136 in the coiled-coil region of Trim5α confers protection against HIV-1 in the Pumwani sex worker cohort [22]. No association of the Trim5α-136Q allele was found neither with the risk of transmission nor the acquisition of HIV in a Cameroonian study [23].

Previous studies in Northern Cameroon concerning Trim5α and HIV focused on people aged 18-70 years [24]. Up to our knowledge, there is no study regarding the polymorphism of Trim5α polymorphism among children in this part of the country. The objective of the study was to determine the role of Trim5α polymorphism in children living in the North region of Cameroon.

2. Materials and Methods

2.1. Design and Period

This was a case-control study conducted in hospitals in the North Region of Cameroon during the period from July 2014-August 2015. The participants were children under 15 years of age all born from HIV-infected mothers. The study protocol received approval from the Cameroon National Ethical Committee for Research on Human Health under the number N °2013/11/375/L/CNERH/SP. Written informed consent was obtained from the mothers as well as parental consent and assent, when indicated.

2.2. HIV Testing

The HIV status of children under 18 months of age was confirmed using a direct detection test for the virus: Abbott qualitative Real-time PCR kits (Abbott TM mSample Preparation System, DNA Promega Corporation, Madison WI 53711USA). For children who were 18 months and above serological tests were used: Alere DetermineTM HIV 1/2 and Oraquick HIV 1/2.

2.3. Genotyping

Human genomic DNA was extracted from buffy coat through several stages using QIAamp®DNA mini kit (Qiagen), following the instructions of the manufacturer. The fragment of interest was amplified from DNA extract for genotyping analyses. Polymerase Chain Reaction (PCR) followed by Restriction Fragment Length Polymorphism (RFLP) were the techniques used for Trim5α-136Q genotyping. The primer sequences used for the amplification were: 5'-ATGGCTTCTGGAATCCTGGTTAATG-3'(forward) and 5'CCCGGGTCTCAGGTCTATCATG-3' (reverse) [25] according to the experimental conditions: initial denaturation at 94 ℃ for 3min, followed by 40 cycles of denaturation at 94 ℃ for 30sec, annealing at 50 ℃ for 30sec, extension at 72 ℃ for 1min and a final extension at 72 ℃ for 10 min. The amplified product was digested with a restriction enzyme (AvaI). To separate fragments issues from digestion, samples were prepared for electrophoresis in the presence of 2 μL ethidium bromide help to visualized DNA using a DNA transilluminator (BIO-RAD, ChemiDocTM XRS+).

Genotypes were determined based on the number and length of fragments on the gel (Figure 1). A PCR product containing Q at position 136 will result in a 526bp undigested product and the person is a mutant homozygous (genotype AA). The GA genotype is characterized by the presence of the AvaI restriction site on one allele. Therefore, there will be

three fragments on the gel at 526bp, 405bp and 121bp respectively, and the person will be heterozygous. The GG genotype is characterized by the presence of a restriction site. The AvaI digestion will produce two fragments of 405 and 121bp respectively. The person in this case is a wild type.

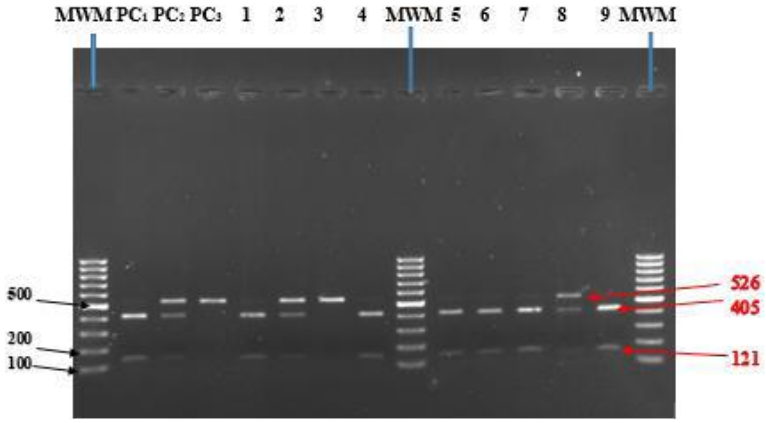

Figure 1. *Electrophoregram of the digested products of Trim5α-136Q gene with AvaI enzyme.*

MWM: Molecular Weight Markers (Ladder: 100bp).
Numbers 1 to 6 are samples. PC 1, PC 2 and PC3 are controls: PC1: GG genotype; PC2: GA genotype; PC3: AA genotype. Samples showing a single band at 526bp are mutant homozygous (3): AA genotype. Samples showing three bands at 526, 405 and 121bp are heterozygous (2, and 8): GA genotype. Samples with two bands at 405 and 121bp are wild type (1, 4, 5, 6, 7, and 9): GG genotype

2.4. Statistical Methods

Allelic frequencies were calculated using the formula: F= (h + 2H)/2N, where F is the frequency, H is the number of samples with a homozygous mutation genotype, h is the number with a heterozygous mutation genotype and N the total number of samples. The Hardy-Weinberg Equilibrium (HWE) was verified by the Chi-square test. To find associations between polymorphism and child HIV status, Pearson's Chi-square test (Fisher exact test where relevant) was used to establish relationships. Odd Ratios (OR) with 95% Confidence Interval (CI) were used as the measure of association. Trim5α-136Q and Trim5α-R136 allelic frequencies were compared between the HIV-1 infected and HIV-1 uninfected children.

3. Results

Overall, 113 children under 15 years of age participated in the study. Among them 49.6% were female, 25 (22.1%) were HIV-1 infected and 88 (77.9%) were HIV-1 uninfected.

3.1. Distribution of Trim5α Genotypes and Allelic Frequencies in the Study Population

The proportion of GG genotype was lower in HIV-1 uninfected children (85.2%) than in HIV-1 infected ones (92.5%) with the difference not statistically significant (p=0.58, 95% CI: [-0.22; 0.09]). The proportion of GA genotype was elevated in HIV-1 uninfected children (12.5%) compared to HIV-1 infected children (8.0%) but the difference was not statistically significant (p=0.58, 95% CI: [-0.22; 0.09]). AA genotype was completely absent in HIV-1 infected children while the proportion in HIV-1 uninfected children was 2.3%. The frequency of Trim5α-136Q allele in HIV-1 uninfected and HIV-1 infected children was 9.0% and 4.0% respectively, meaning that the protective Trim5α-136Q allele was more frequent in uninfected children. The Hardy-Weinberg Equilibrium (HWE) analysis showed that all genetic variants for Trim5α in the studied population were in equilibrium (p=0.07).

Table 1. *Distribution of Trim5α genotypes and Trim5α-136Q allele in HIV-1 infected and uninfected children.*

Genes variants	Uninfected children N (%)	Infected children N (%)	P-value	95%CI	X^2	Total N (%)
G/G	75 (85.2)	23 (92.0)	0.58	-0.22; 0.09	0.30	98 (86.7)
G/A	11 (12.5)	2 (8.0)	0.79	-0.11; 0.20	0.07	13 (11.5)

Genes variants	Uninfected children N (%)	Infected children N (%)	P-value	95%CI	X^2	Total N (%)
A/A	2 (2.3)	0 (0.0)	0.60	NA	NA	2 (1.8)
total	88 (100.0)	25 (100.0)				113
Trim5α -136Q frequencies	9.0	4.0				8.0
X^2-HWE	3.46	0.04				3.39
P-HWE	0.06	0.83				0.07

G/G: Homozygous wild type genotype
G/A: Heterozygous genotype
A/A: homozygous mutant genotype
X^2- HWE: Chi-squared for the Hardy Weinberg Equilibrium
P-HWE: Statistical value for the Hardy Weinberg Equilibrium
n: Number of participants presenting the genotype

3.2. Trim5α Phenotypes in HIV-1 Infected and Uninfected Children

The proportion of mutant was higher in HIV-1 uninfected children (14.8%) than in HIV-1 infected ones (8.0%). Moreover, children carrying mutated phenotype were 2 times less likely to be HIV-infected compared to those without it (OR= 2.0, 95% CI: [0.42, 9.50]). This implies that the mutated phenotype of the Trim5α-136Q gene may protect children against HIV-1 acquisition.

Table 2. *Children's phenotypes and their HIV status.*

children phenotypes	Uninfected children N (%)	Infected children N (%)	OR	P
Mutant	13 (14.8)	2 (8.0)	2.0 (0.42-9.50)	0.29
Wild type	75(85.2)	23(92.0)	1	

4. Discussion

Our objective was to determine the role of Trim5α polymorphism in children living in the North region of Cameroon. This is the first study carried particularly on Trim5α gene in children in this part of the country. Trim5α gene encoded a cytoplasmic protein that blocks HIV-1 infection after the virus enters the target cell cytoplasm. The polymorphism of trim5α and the differences in expression modifies the rate of HIV acquisition and disease progression [14].

R136Q polymorphism that is studied in this work correspond to rs10838525 SNP in exon 2 results in the amino acid change from Arginine to Glutamine at codon 136. It has been reported that this polymorphism confer protection against HIV in individuals with high risk and slow disease progression [3]. Trim5α binds retroviruses via its C-terminal PRY/SPRY domain and rapidly recruits them to the proteasome before significant viral DNA synthesis can occur [26]. Trim5α is ubiquitinylated within cells and is rapidly turned over by the proteasome in a RING domain. The suggestion is that autoubiquitinylation might drive this process [27].

In the present study, 113 children under 15 years of age participated. Among them 49.6% were female, 25 (22.1%) were HIV-1 infected and 88 (77.9%) were HIV-1 uninfected. The observed Trim5α-136Q allelic frequencies in HIV-1 uninfected (9.0%) and HIV-1 infected (4.0%) children was comparable with the frequency reported in Cameroon (11.3%) [23], but lower than what was found in Americans and Caucasians [10, 21]. Hence, the mutated allele Trim5α-136Q is not common in this population. Children carrying mutated phenotype were 2 times less likely to be HIV-infected compared to those without it. Trim5α-136Q polymorphism seems to confer protection against HIV acquisition although the Trim5α-136Q allele is not frequent. Another study also carried in Cameroon failed to find any association between Trim5α-136Q polymorphism and MTCT or acquisition of HIV by a child from his mother [23]. AA genotype was completely absent in HIV-1 infected children while the proportion in HIV-1 uninfected

children was 2.3%. This result means that the mutated homozygous genotype did not protect against acquisition of HIV-1 by children. Contrarily, a cohort study among the participants in Amsterdam found a protective effect of the 136QQ genotype [21]. In African Americans, the mutated allele Trim5α-136Q was relatively elevated in uninfected individuals, suggesting a possible protective effect [28]. Concerning HIV-1 disease progression, the trim5α has no effect or modest effect [29].

5. Conclusions

Children carrying mutated phenotype of the Trim5α were 2 times less likely to be HIV-infected compared to those without. Hence, the mutated phenotype of the Trim5α-136Q gene may be protective against HIV-1 acquisition in children. However, the trim5α-136Q allelic frequency is not common in the North Region of Cameroon. Further investigation in a follow up cohort considering other polymorphisms and, a large number of samples will help in better appreciation of the role of Trim5α polymorphisms in HIV acquisition and disease progression.

Abbreviations

AIDS	Acquired Immunodeficiency Syndrome
DNA	Deoxyribonucleic Acid
HIV-1	Human Immunodeficiency Virus Type 1
HWE	Hardy-Weinberg Equilibrium
mRNA	Messenger Ribonucleic Acid
MTCT	Mother-to-child Transmission
PCR	Polymerase Chain Reaction
RFLP	Restriction Fragment Length Polymorphism
Trim5α	Tripartite Motif Containing 5 Alpha

Acknowledgments

The authors wish to thank all the participants of the study.

Author Contributions

Marie Nicole Ngoufack: Conceptualization, Data curation, Investigation, Methodology, Writing – original draft

Georges Nguefack-Tsague: Conceptualization, Data curation, Formal analysis, Methodology, Software

Celine Nguefeu Nkenfou: Conceptualization, Investigation, Methodology, Project administration, Supervision, Validation, Visualization

Conflicts of Interest

The authors declare that they have no competing of interest.

References

[1] Spada, S. J., Grigg, M. E., Bouamr, F., Best, S. M., Zhang, P. TRIM5α: A Protean Architect of Viral Recognition and Innate Immunity. *Viruses*. 2024, 16(7), 997. https://doi.org/10.3390/v16070997

[2] Wang, T., Becker, D., Twizerimana, A. P., Luedde, T., Gohlke, H., Münk, C. Cyclophilin ARegulates Tripartite Motif 5 Alpha Restriction of HIV-1. *International Journal of Molecular Sciences*. 2025, 1-28. https://doi.org/10.3390/ijms26020495

[3] Amanya, S. B., Nyiro, B., Waswa, F., Obura, B., Nakaziba, R., Nabulime, E., Katabazi, A. F., Nabatanzi, R., Bayiyana, A., Mboowa, G., Kayongo, A., Wayengera, M., Sande, O. J. Variations in Trim5α and Cyclophilin A genes among HIV-1 elite controllers and non controllers in Uganda: a laboratory-based cross-sectional study. *Retrovirology* 17(2020): 1-11. https://doi.org/10.1186/s12977-020-00527-z

[4] Ghimire, D., Rai, M., Gaur, R. Novel host restriction factors implicated in HIV-1 replication. *J Gen Virol*. 2018, 99(4): 435-446. https://doi.org/10.1099/jgv.0.001026

[5] Malim, M. H., Bieniasz, P. D. HIV Restriction Factors and Mechanisms of Evasion. *Cold Spring Harb Perspect Med 2*. 2012, 99(4): 435-446. https://doi.org/10.1099/jgv.0.001026

[6] Blanco-Melo, D., Venkatesh, S., Bieniasz, P. D. Intrinsic cellular defenses against human immunodeficiency viruses. *Immunity*. 2012, 37(3): 399-411. https://doi.org/10.1016/j.immuni.2012.08.013

[7] Colomer-Lluch, M., Ruiz, A., Moris, A., Prado, J. G. Restriction Factors: From Intrinsic Viral Restriction to Shaping Cellular Immunity Against HIV-1. *Front Immunol*. 2018, 175-182. https://doi.org/10.3389/fimmu.2018.02876

[8] Puvvada, M. P., Patel, S. S. Role of trim5α in the suppression of cross-species transmission and its defence against human immunodeficiency virus. *Curr HIV Res*. 2013, 11(8): 601-9. https://doi.org/10.2174/1570162x12666140306124543

[9] Zhang, X., Kondo, M., Chen, J., Miyoshi, H., Suzuki, H., Ohashi, T., Shida, H. Inhibitory effect of human TRIM5alpha on HIV-1 production. *Microbes Infect*. 2010, 12(10): 768-77. https://doi.org/10.1016/j.micinf.2010.05.004

[10] Speelmon, E. C., Livingston-Rosanoff, D., Li, S. S., Vu, Q., Bui, J., Geraghty, D. E., Zhao, L. P., McElrath, M. J. Genetic Association of the Antiviral Restriction Factor TRIM5α with Human Immunodeficiency Virus Type 1 Infection. *J Virol*. 2006, 2463-2471. https://doi.org/10.1128/jvi.80.5.2463-2471.2006

[11] Perez-Caballero, D., Hatziioannou, T., Yang, A., Cowan, S., Bieniasz, P. D. Human tripartite motif 5alpha domains responsible for retrovirus restriction activity and specificity. *J Virol*. 2005, 79(14): 8969-78. https://doi.org/10.1128/JVI.79.14.8969-8978.2005

[12] Sokolskaja, E., Berthoux, L., Luban, J. Cyclophilin A and TRIM5alpha independently regulate human immunodeficiency virus type 1 infectivity in human cells. *J Virol*. 2006, 80(6): 2855-62. https://doi.org/10.1128/JVI.80.6.2855-2862.2006

[13] Nakajima, T., Nakayama, E. E., Kaur, G., Terunuma, H., Mimaya, J. I., Ohtani, H., Mehra, N., Shioda, T., Kimura, A. Impact of novel TRIM5alpha variants, Gly110Arg and G176del, on the anti-HIV-1 activity and the susceptibility to HIV-1 infection. *AIDS*. 2009, 23(16): 2091-100. https://doi.org/10.1097/QAD.0b013e328331567a

[14] Nakayama, E. E., Nakajima, T., Kaur, G., Mimaya J., Terunuma, H., Mehra, N., Kimura, A., Shioda, T. A Naturally Occurring Single Amino Acid Substitution in Human TRIM5a Linker Region Affects Its Anti-HIV Type 1 Activity and Susceptibility to HIV Type 1 Infection. *Aids research and human retroviruses*, 2013. 29(6): 919-24. https://doi.org/10.1089/AID.2012.0369

[15] Feng-Liang, L., Yu-Qing, Q., Hong, L., Yi-Qun, K., Xia, T., Guang, C., Nelson Leung, S. T., Yong-Tang, Z. An HIV-1 Resistance Polymorphism in TRIM5α Gene Among Chinese Intravenous Drug Users. *JAIDS Journal of Acquired Immune Deficiency Syndromes*. 2011, 306-311. https://doi.org/10.1097/QAI.0b013e318205a59b

[16] Cloherty, A. P. M., Rader, A. G., Compeer, B., Ribeiro, C. M. S. Human TRIM5α: Autophagy Connects Cell-Intrinsic HIV-1 Restriction and Innate Immune Sensor Functioning. *Viruses*. 2021, 13(2): 1-32. https://doi.org/10.3390/v13020320

[17] Saha, B., Salemi, M., Williams, G. L, Oh, S., Paffett, M. L., Phinney, B., Mandell, M. A. Interactomic analysis reveals a homeostatic role for the HIV restriction factor TRIM5α in mitophagy. *Cell Rep*. 2022, 39(6): 1-38. https://doi.org/10.1016/j.celrep.2022.110797

[18] Jimenez-Guardeño, J. M., Apolonia, L., Betancor, G., Malim, M. H. Immunoproteasome activation enables human TRIM5α restriction of HIV-1. *Nat Microbiol*. 2019, 4(6): 933-940. https://doi.org/10.1038/s41564-019-0402-0

[19] Keown, J. R., Black, M. M., Ferron, A., Yap, M., Barnett, M. J., Pearce, F. G., Stoye, J. P., Goldstone, D. C. A helical LC3-interacting region mediates the interaction between the retroviral restriction factor Trim5α and mammalian autophagy-related ATG8 proteins. *J Biol Chem*. 2018, 293(47): 18378-18386. https://doi.org/10.1074/jbc.RA118.004202

[20] Sastri, J., Campbell, E. M. Recent insights into the mechanism and consequences of trim5a retroviral restriction. *AIDS Res Hum Retroviruses*, 2011, 27(3): 231-8. https://doi.org/10.1089/AID.2010.0367

[21] van Manen, D., Rits, M. A. N., Beugeling, C., van Dort, K., Schuitemaker, H., Kootstra, N. A. The Effect of Trim5 polymorphisms on the clinical course of HIV-1 infection. *PLoS Pathogens*. 2008, 4(2): e18. https://doi.org/10.1371/journal.ppat.0040018

[22] Price, H., Lacap, P., Tuff, J., Wachihi, C., Kimani, J., Ball, T. B et al. A TRIM5 alpha exon 2 polymorphism is associated with protection from HIV-1 infection in the Pumwani sex worker cohort. *AIDS*. 2010, 24(12): 1813-21. https://doi.org/10.1097/QAD.0b013e32833b5256

[23] Mekue, L. M., Nkenfou, C. N., Dambaya, B., Fotso, I., Nguefack, F., Fainguem, N., Lobe, E. E., Kuiate J. R., Ndjolo., A. Implication of five AIDS related genes in mother-to-child transmission and acquisition of human immunodeficiency virus 1 in Cameroon. *Afr j infect dis* 13(2018): 1-10. https://doi.org/10.21010/ajid.v13i1.1

[24] Djataou, P., Djuidje Ngounoue, M., Nkenfou-Tchinda, C. N., Ngoufack, M. N., Elong, E., Tiga, A., Muluh, C., Kadji Kameni, J., Djaouda, M., Ndjolo, A., Nkenfou, C. N. Low prevalence of HIV in the northern Cameroon: contribution of some AIDS restriction genes and potential implications for gene therapy. *Frontiers in genetics*. 2024, 01-11. https://doi.org/10.3389/fgene.2024.1447971

[25] Dambaya, B., Nkenfou, C. N., Mekue, L., Teto, G., Ngoufack, N., Ambada, G., Flobert, N., Colizzi, V., Alexis, N. TRIM5α 136Q, CCR5 Promoter 59029G And CCR264I Alleles Impact The Progression Of HIV In Children And Adolescents. *Appl Clin Genet*. 2019, 12: 203-211. https://doi.org/10.2147/TACG.S205335

[26] Huthoff, H., Towers, G. J. Restriction of retroviral replication by APOBEC3G/F and TRIM5alpha. *Trends Microbiol*. 2008, 16(12): 612-9. https://doi.org/10.1016/j.tim.2008.08.013

[27] Towers, G. J. The control of viral infection by tripartite motif proteins and cyclophilin A. *Retrovirology*. 2007, 12; 4: 40. https://doi.org/10.1186/1742-4690-4-40

[28] Javanbakht, H., An, P., Gold, B., Petersen, D. C, O'Huigin, C., Nelson, G. W, O'Brien, S. J., Kirk, G. D., Detels, R., Buchbinder, S., Donfield, S., Shulenin, S., Song, B., Perron, M. J, Stremlau, M., Sodroski, J., Dean, M., Winkler, C. Effects of human TRIM5alpha polymorphisms on antiretroviral function and susceptibility to human immunodeficiency virus infection. *Virology*. 2006, 354(1): 15-27. https://doi.org/10.1016/j.virol.2006.06.031

[29] Goldschmidt, V., Bleiber, G., May, M., Martinez, R., Ortiz, M., Telenti, A. Swiss HIV Cohort Study. Role of common human TRIM5alpha variants in HIV-1 disease progression. *Retrovirology*. 2006, 1-17. https://doi.org/10.1186/1742-4690-3-54