ISSN: 2469-567X

Abdullahi et al. Int J Virol AIDS 2024, 11:096
DOI: 10.23937/2469-567X/1510096
Volume 11 | Issue 1
Open Access

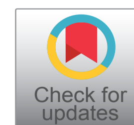
International Journal of
Virology and AIDS

RESEARCH ARTICLE

Circulating Measles Virus Genotypes in Parts of North-eastern Nigeria (2019-2022)

Umar Abdullahi[1], Maryam Aminu[2*], Ahmed Babangida Suleiman[3], Grace N.S. Kia[4], Nnaemeka Darlington Ndodo[5], Richard Ojedeleand[5] and Luc Venance Kouakou[5]

[1]*Departments of Science Laboratory Technology, Modibbo Adama University, Yola, Nigeria*
[2]*Department of Microbiology, Ahmadu Bello University, Zaria, Nigeria*
[3]*Department of Veterinary Public Health, Ahmadu Bello University, Zaria, Nigeria*
[4]*Nigerian Center for Disease Control (NCDC), Genomics and Research Unit, NRL, Abuja, Nigeria*
[5]*Institut Pasteur de Côte d'Ivoire (IPCI), Abidjan, Côte d'Ivoire*



***Corresponding author:** *Maryam Aminu, Department of Microbiology, Ahmadu Bello University, Zaria, Nigeria, Tel: +2348033287031*

Abstract

Background: Measles is a highly contagious viral disease that remains a leading cause of morbidity and mortality in many developing countries. Caused by the measles virus (MeV), a single-stranded RNA virus, it exhibits genetic diversity based on the nucleoprotein gene, with 24 known genotypes. The World Health Organization AFRO region which includes Nigeria targeted the year 2020 as the year to eliminate measles. In 2021, Nigeria had the world's largest number of measles cases than any country: Over 10,000 cases based on data from the Integrated Disease Surveillance and Response (IDSR). Genetic data can be used to track transmission patterns and identify sources of infection. Despite its importance, Nigeria has yet to fully integrate molecular characterization into her measles control program. Data on molecular characterization in the northeastern region of Nigeria are limited, outdated, or fragmented. That is why the present study aimed to describe the molecular epidemiology of measles in parts of Northeast, Nigeria. These results will enable to establish a genetic basis of virological surveillance in Northeast, Nigeria and evaluate the country's efforts to eradicate the disease.

Methods: The study analyzed 558 serum samples for measles-specific IgM among children 0-12 years, out of which, 163 were positive. Of the 163 IgM-positive samples, 100 Clinical specimens (nasopharyngeal swabs) were randomly selected for molecular analysis. Viral RNA was extracted (Qiagen, Germany) and the N-gene of measle virus was detected using one-step RT-PCR (Invitrogen, USA). Amplicons were sequenced for genotype identification using the ABI 3130 Genetic analyzer (Applied Biosystem, USA).

Results: Of the 163 IgM-positive samples, 100 were randomly selected and amplified for the measles virus N gene. Of the 100 samples tested, 27% (27/100) were positive for measles virus N-gene by RT-PCR. Sequencing of 10 positive isolates revealed that they clustered with the WHO reference strain of genotype B3 cluster 1.

Conclusion: This study provides recent data on the circulating measles virus strains in northeast Nigeria. It highlights the need for ongoing surveillance and molecular investigation to better understand outbreak patterns and improve measles control strategies.

Keywords

Measles virus, Genotyping, Northeast Nigeria

Introduction

The World Health Organization AFRO region which includes Nigeria targeted the year 2020 as the year to eliminate measles in Africa [1]. The WHO AFRO region recommends molecular identification as a tool for outbreak investigations and surveillance but Nigeria has yet to integrate molecular surveillance into its measles control program. Despite this effort, Nigeria had the world's largest number of measles cases than any country: Over 10,000 cases in 2021 [2]. Sub-Saharan

Citation: Abdullahi U, Aminu M, Suleiman AB, Kia GNS, Ndodo ND, et al. (2024) Circulating Measles Virus Genotypes in Parts of North-eastern Nigeria (2019-2022). Int J Virol AIDS 11:096. doi.org/10.23937/2469-567X/1510096

Accepted: November 14, 2024: **Published:** November 16, 2024

Copyright: © 2024 Abdullahi U, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

ClinMed International Library

Africa faces a spike of Measles where a total of 17,500 cases of Measles have been reported in the region as of January 2022, signifying an increase of 400% compared to cases reported in 2021 [3]. This outbreak comes as an after-effect of the COVID-19 pandemic, which has resulted in immunization gaps in most parts of the region [4].

The measles virus is an enveloped particle, approximately 100-300 nm in diameter, containing a single-stranded, negative-sense RNA genome. Although the genome size has traditionally been considered highly conserved at 15,894 nucleotides, minor deviations have been observed [5,6]. While the measles virus is monotypic, genetic variability in the viral hemagglutinin (H) and nucleoprotein (N) genes enables classification into 8 clades (A-H) and 24 genotypes (A, B1-B3, C1-C2, D1-D11, E, F, G1-G3, H1-H2) [7]. According to the World Health Organization (WHO), genotype B3 has been the most prevalent worldwide from 2010 to 2015 and remains endemic in Africa [8].

In Africa, measles virus genotypes exhibit distinct geographical distributions. East and Southern Africa predominantly feature genotypes D2 and D4, while West Africa is mainly affected by genotype B3 [9]. Genotypes B3 (clusters B3.1 and B3.2) have also spread to Europe [10], Asia [11,12], and the Americas [13]. Genetic characterization typically involves sequence analysis of the 450 nucleotides encoding the carboxyl terminus of the nucleoprotein (N-450) [14,15].

In 2021, Nigeria reported the highest number of measles cases globally, with over 10,000 reported cases, primarily concentrated in the Northeast due to insurgency affecting vaccine coverage [2]. Molecular characterization can aid in tracking transmission patterns and identifying sources of infection. Given the lack of integration of molecular studies into Nigeria's measles control program, this study aims to establish a genetic basis for virological surveillance in Northeast Nigeria and assess the country's efforts towards measles eradication.

Materials and Methods

Sample collection and processing

A total of 100 nasopharyngeal swabs were collected by a research assistant nurse from children between 0-12 years, the nasal swabs were collected by gently rubbing the nasopharyngeal passage and back of the throat with sterile cotton swabs to collect epithelial cells. The swabs were placed in labelled screw-capped tubes containing a sterile 1:1 viral transport medium (COPAN Diagnostics, USA) and DNA/RNA shield (Zymo Research, USA). The samples were transported on ice packs (4 °C) to the National Reference Laboratory (NRL) of the Nigerian Center for Disease Control and stored at -20 °C until processing.

Extraction of Viral RNA

Viral RNA was extracted from the nasopharyngeal swab samples using the QIAamp Viral RNA Mini Kit (QIAGEN, Hilden, Germany) following the manufacturer's instructions (Spin Protocol).

Revere transcription-PCR and amplification

The RNA extracts were analyzed using MeV genotyping RT-PCR to target a 634-nucleotide region of the N-gene. The primers used were forward primer MeV214 (5'-TAACAATGATGGAGAGGGTAGG-3') and reverse primer MeV216 (5'-TGGAGCTATGCCATGGGAGT-3'). The Superscript™ III One-Step RT-PCR System with Platinum Taq High Fidelity enzyme (Invitrogen) was employed for amplification. The RT-PCR reaction mixture included 9.5 µL DNAse/RNAse-free water, 25 µL 2X buffer, 8 µL $MgSO_4$, 0.5 µL of each primer, 0.5 µL RNase inhibitor, 1 µL Invitrogen enzyme, and 5 µL RNA extract. The RT-PCR conditions were as follows: Reverse transcription at 55 °C for 30 minutes, initial denaturation at 95 °C for 2 minutes, 40 cycles of denaturation at 94 °C for 15 seconds, primer annealing at 55 °C for 30 seconds, and elongation at 72 °C for 30 seconds, followed by a final elongation at 72 °C for 7 minutes and storage at 4 °C.

Detection and identification of PCR products

The PCR products were resolved on l.5% agarose gel prepared by dissolving 1.5g of agarose powder in 100 ml of 1X Tris-borate-EDTA (TBE) buffer solution inside a clean conical flask. The 1.5% agarose solution was heated in a microwave oven for 3 minutes and was observed for clarity which was an indication of complete dissolution. The mixture was then allowed to cool to about 50 °C after which 10 µL of SYBR stain dye was then added. It was allowed to cool further and then poured into a tray sealed at both ends with support to form a mold with special combs placed in it to create wells. The comb was carefully removed after the gel had set and the plate was placed inside the electrophoresis tank which contained 1X TBE solution. 5 µL of amplicon was mixed with 1 µL of loading buffer and the mixture was loaded to the wells of the agarose gel. The power supply was adjusted to 100 volts for 30 minutes. For each run, a 50 base-pair molecular weight DNA standard (size marker) was used to determine the size of each PCR product. The DNA bands were then visualized with a short-wave Axygene ultraviolet trans-illuminator and photographed using a gene gel bio-imaging system. DNA bands were visualized with a short-wave ultraviolet transilluminator (Axygene, Life Sciences, USA).

Cleanup of PCR

Post-PCR amplicons were purified using ExoSAP-IT™ reagent. Ten microliters (10 µL) of the reaction product were combined with 4 µL of ExoSAP-IT™ reagent for a total volume of 14 µL. The mixture was incubated at 37 °C for 15 minutes to degrade residual primers and

nucleotides, then at 80 °C for 15 minutes to inactivate the reagent. The purified PCR products were stored at -20 °C.

Sequencing and cleanup

Sequencing was performed using the BigDye Terminator v3.1 Cycle Sequencing Kit (Applied Biosystems, Nieuwerkerk, Netherlands). The reaction mixture included 9 µL nuclease-free water, 8 µL BigDye 3.1v Reaction Mix, 1 µL of each forward and reverse primer (3.2 µM), and 2 µL of the template DNA. The sequencing primers were MeV214 (5'-TAACAATGATGGAGAGGGTAGG-3') and MeV216 (5'-TGGAGCTATGCCATGGGAGT-3') targeting the N-450 region. The sequencing conditions were denaturation at 96 °C for 30 seconds, primer annealing at 50 °C for 15 seconds, elongation at 60 °C for 4 minutes, and storage at 4 °C.

Sequencing products were purified using the CleanSEQ Dye-Terminator Removal Kit (BECKMAN COULTER Life Sciences, USA) according to the manufacturer's instructions. Sequencing was performed on an ABI 3130 Genetic Analyzer (Applied Biosystems, USA). Nucleotide sequences were aligned and edited using MEGA software (version 11.0.6).

Phylogenetic analysis

Phylogenetic analysis was conducted using MEGA software (version 11.0.6). Maximum likelihood methods were employed to compare the N-gene sequences obtained in this study with WHO reference strains of known measles virus genotypes. The robustness of the phylogenetic groupings was assessed using bootstrap resampling with 1000 replicates. The resulting phylogenetic tree was visualized using MEGA software.

Results

Molecular identification of measles virus

Among the 100 nasopharyngeal swabs tested for the measles virus, 27 samples (27%) were positive for the N-gene, as evidenced by a 634 bp amplicon. The distribution of positive samples was as follows: 8 (29.6%) from Adamawa, 10 (37.1%) from Borno, and 9 (33.3%) from Yobe states (Plate 1, Plate 2 and Plate 3).

All 27 PCR-positive samples were subjected to sequencing, however, only 10 sequences were selected for detailed analysis: 4 from Adamawa, 3 from Borno, and 3 from Yobe states. The nucleotide sequences of the nucleoprotein (N) gene of these measles virus isolates are available in the GenBank database under accession numbers OR634810.1 through OR634819.1. A BLAST search of these sequences revealed that the isolates belong to the measles virus genotype B3, with an average sequence identity of 92% and an E-value of 0.0, indicating a high degree of similarity to known B3 strains (Table 1).

Phylogenetic analysis

The phylogenetic analysis of the measles virus sequences, using neighbour-joining methods, is illustrated in Figure 1. The phylogenetic tree includes sequences of genotype B3 (accession numbers OR634810-OR634819), alongside two WHO reference strains of genotype B3: Ibadan. Nie./97/1 (repseqid 4) and New York. USA/94 (repseqid 5), as well as 13 other WHO-named strains.

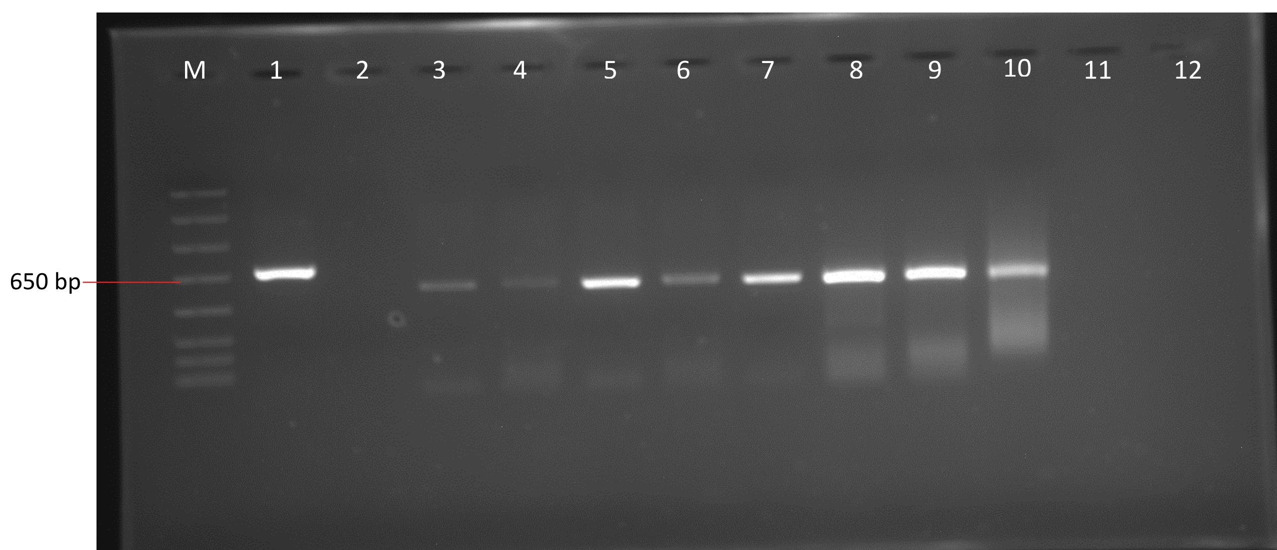


Plate 1: A gel photo of amplified measles virus.
Lane M = 50 bp ladder
Lane 1 = Positive control
Lane 2 = Negative Control
Lane 3-12 = Measles virus samples
Lane 3-10 = Positive samples for measles virus at 634 bp.

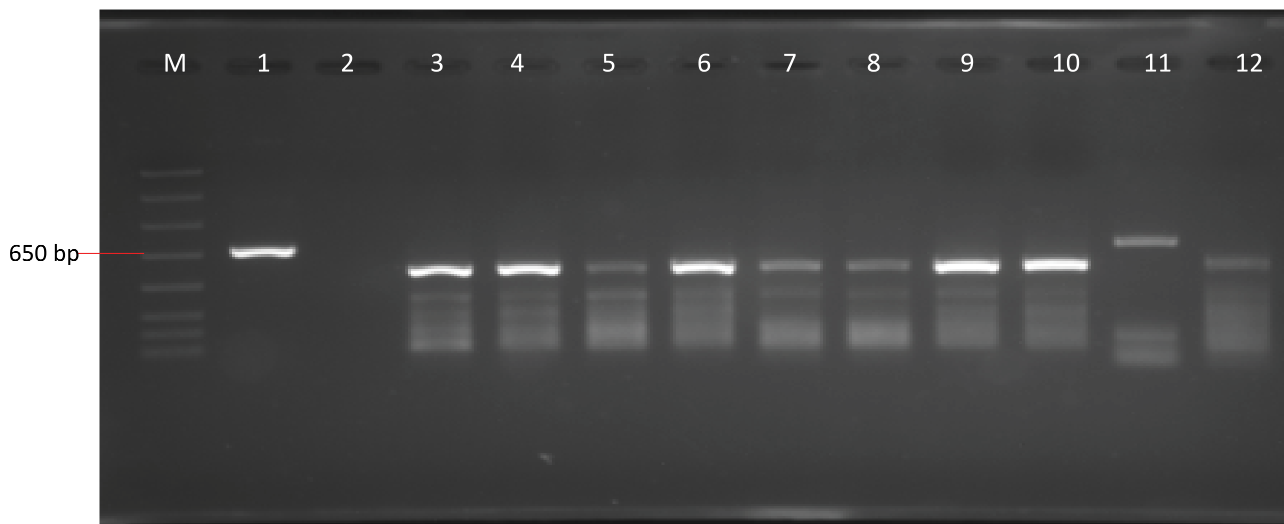


Plate 2: A gel photo of amplified measles virus.
Lane M = 50 bp ladder
Lane 1 = Positive control
Lane 2 = Negative Control
Lane 3-12 = Measles virus samples
Lane 3-12 = Positive samples for measles virus at 634 bp.

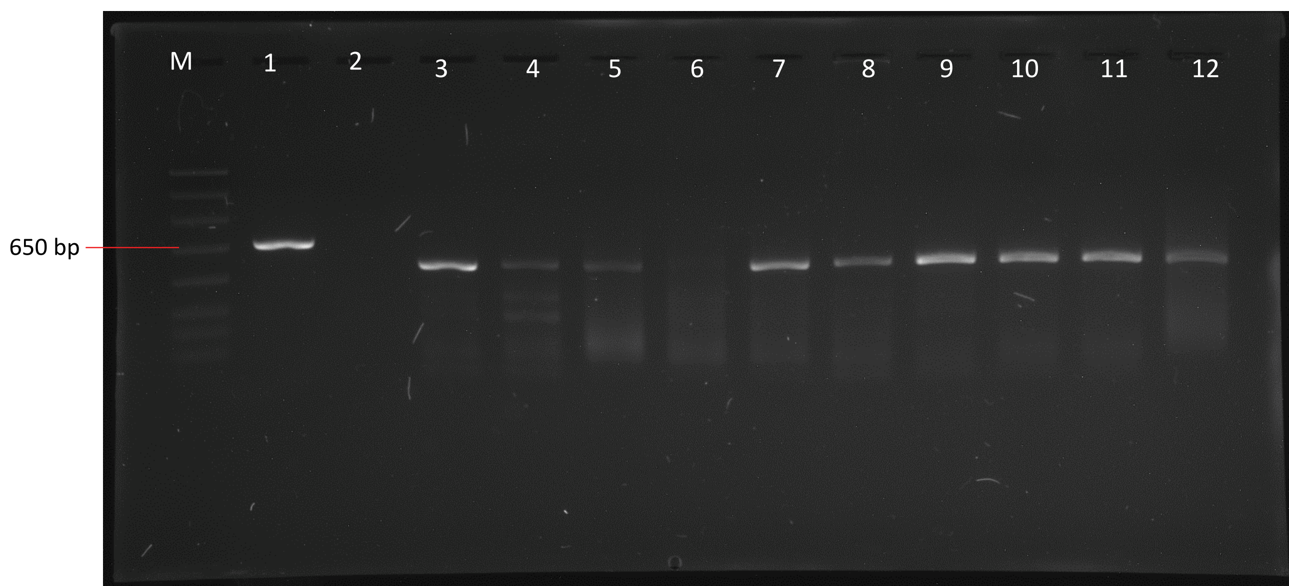


Plate 3: A gel photo of amplified measles virus.
Lane M = 50 bp ladder
Lane 1 = Positive control
Lane 2 = Negative Control
Lane 3-12 = Measles virus samples
Lanes 3, 4, 5, 7-12 = Positive samples for measles virus at 634 bp.

Analysis of the C-terminal 450 nucleotides of the N gene revealed that the 10 sequenced strains exhibited 90% nucleotide identity with the WHO reference strains Ibadan. Nie./97/1 and New York. USA/94. Notably, isolate AD2-OR634812.1 demonstrated 94% nucleotide identity with a strain from Iran (Accession number: OP245161). In contrast, isolates AD5-OR634810.1 and AD3-OR634811.1 showed 96% genetic similarity with a strain from Côte d'Ivoire (Accession number: MH752168). The phylogenetic tree indicates that the measles virus strains sequenced in this study are closely related to wild-type strains circulating in West Africa and belong to Clade B3, specifically Cluster 1 (B3.1).

Discussion

The study analyzed 558 serum samples for measles-

Table 1: Confirmation of isolates of measles virus with sequences in GenBank Database.

S/N	Samples	WHO Nomenclature	Max. Score	Total Score	Query Cover (%)	E-Value	% ID	Accession Number
	AD1	MVs/Adamawa.NGA/05.21/1[B3] nucleoprotein	643	643	100	0.0	92.46	OR634813.1
	AD2	MVs/Adamawa.NGA/15.21/2[B3] nucleoprotein	627	627	100	0.0	91.80	OR634812.1
	AD3	MVs/Adamawa.NGA/11.20/3[B3] nucleoprotein	643	643	100	0.0	92.46	OR634811.1
	AD5	MVs/Adamawa.NGA/22.20/5[B3] nucleoprotein	632	632	100	0.0	92.02	OR634810.1
	B2	MVs/Borno.NGA/06.20/2[B3] nucleoprotein	643	643	100	0.0	92.46	OR634819.1
	B12	MVs/Borno.NGA/12.20/12[B3] nucleoprotein	638	638	100	0.0	92.24	OR634817.1
	B20	MVs/Borno.NGA/21.21/20[B3] nucleoprotein	638	638	100	0.0	92.24	OR634815.1
	Y19	MVs/Yobe.NGA/19.21/19[B3] nucleoprotein	643	643	100	0.0	92.46	OR634816.1
	Y21	MVs/Yobe.NGA/10.21/21[B3] nucleoprotein	643	643	100	0.0	92.46	OR634814.1
	Y3	MVs/Yobe.NGA/09.21/3[B3] nucleoprotein	643	643	100	0.0	92.46	OR634818.1

Key: %ID- percentage identity

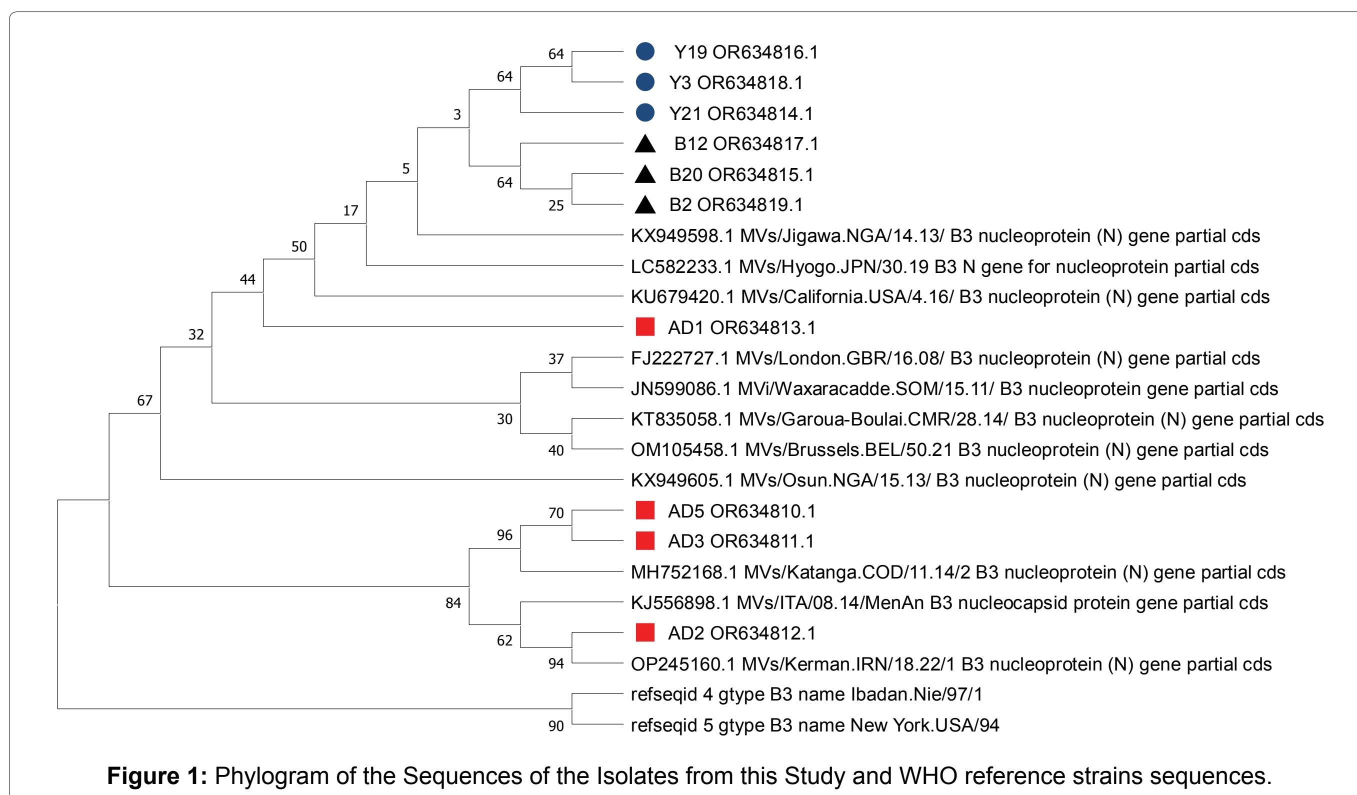


Figure 1: Phylogram of the Sequences of the Isolates from this Study and WHO reference strains sequences.

specific IgM among children 0-12 years, out of which, 163 were positive. Of the 163 IgM-positive isolates, 100 were randomly selected for RT-PCR. Of the 100, 27 were positive for measles virus N-gene. Ten of the 27 measles virus isolates were readable after sequencing. All isolates originated from children whose serum samples were measles virus IgM positive. The observed discrepancy between the low positivity rate by RT-PCR and the higher rate of specific IgM antibodies could be attributed to the thermolability of measles virus RNA, which is prone to degradation. This phenomenon has been previously reported in studies from Northwest Nigeria [16] and Gabon [17].

Phylogenetic analysis of the nucleotide sequences from the ten isolates, compared with WHO reference strains, revealed that all isolates clustered with the genotype B3, specifically in cluster 1. This clustering indicates that the isolates belong to genotype B3, cluster 1. The persistent circulation of genotype B3 in Nigeria suggests ongoing endemic transmission of this virus strain in the country.

Globally, the B3 genotype was the most common between 2010 and 2015, with high prevalence reported across the African continent [7]. Studies from Nigeria and Ghana have also highlighted the dominance of clade B3 in sub-Saharan Africa [18]. Consistent with findings from previous studies [18,19], our results confirm the continued presence of genotype B3 in Nigeria, with the B3.1 strain circulating for over two decades. This study is notable as it is the first to characterize measles virus strains in Northeast Nigeria, where molecular data have been either lacking or outdated.

Although this study did not identify any imported measles strains, it is important to consider the potential for cross-contamination with neighbouring regions, as genotype B3 is also reported in Southwest [20] and Northwest [16] Nigeria, as well as throughout sub-Saharan Africa [21]. A global review indicated that genotype B3 is frequently reported in Africa, detected in 197 of 220 sequences from 21 countries [22].

In this study, 16.7% (x/y) of the sample population had been vaccinated, yet no vaccine strains were detected. Genotype B3 is endemic across most of Africa, except Northern African countries and Ethiopia, where genotype D4 predominates [22-25]. Cluster 1 of genotype B3 has been previously identified in several African countries, including Cameroon, Ghana, Kenya, and Tanzania [22]. In contrast, cluster 2, which was not observed in our study, appears to be more localized to Western Africa [26], while cluster 3 has been found in Northern Africa [27] and Cameroon [28].

Genotype B3, including clusters B3.1 and B3.2, has spread to other continents such as Europe [9], China [10], Afghanistan [11], and the United States [12]. Despite our lack of evidence for imported cases, the genetic diversity observed among isolates could be attributed to frequent transmission between Nigeria and neighbouring countries, as supported by studies from Ghana and Congo [19] and among refugees in Cameroon [29].

The exclusive presence of cluster B3.1 in this study suggests the possible elimination of other circulating strains in Northeast Nigeria. This finding implies that current efforts to control measles are effective but should be reinforced, given that the B3.1 genotype has been in circulation for over 20 years. This study provides updated data on circulating strains in Northeast Nigeria and underscores the need for ongoing surveillance and molecular investigation to understand outbreak patterns better.

Study Limitation

Limitations of the study include the inability to amplify the H-gene and the restricted sample size due to COVID-19 and regional insecurity.

Acknowledgements

We would like to express our gratitude to the Ministries of Health of Adamawa, Borno, and Yobe States for granting ethical approval for sample collection. Our sincere appreciation goes to the Nigerian Center for Disease Control and Prevention (NCDC) and the Pasteur Institute, Abidjan, for providing laboratory space for the serological and molecular aspects of this research. We also extend our heartfelt thanks to all the parents who consented to participate, making this study possible.

References

1. WHO (2011) Regional committee for Africa, 61st session. Measles elimination by 2020. A Strategy for the African Region.
2. Ryoko S, Olusesan AM, Koeloengan CD, Bola L (2022) Geographical and time trends of measles incidence and measles vaccination coverage and their correlation in Nigeria. Hum Vaccin Immunother 18: e2114697.
3. WHO (2020) Vaccine-preventable disease outbreaks on the rise in Africa.
4. Rigby J (2022) Measles cases jumps 79% in 2022 after COVID hit vaccination campaigns.
5. Bankamp B, Liu C, Rivailler P, Bera J, Shrivastava S, et al. (2014) Wild-type measles viruses with non-standard genome lengths. PLoS One 9: e95470.
6. Gil H, Fernandez-Garcia A, Mosquera MM, Hubschen JM, Castellanos AM, et al. (2018) Measles virus genotype D4 strains with non-standard length M-F non-coding region circulated during the major outbreaks of 2011-2012 in Spain. PLoS One 13: e0199975.
7. World Health Organization (2003) Update of the nomenclature for describing the genetic characteristics of wild-type measles viruses: New genotypes and reference strains. Weekly Epidemiological Record 78: 229-232.
8. World Health Organization (2016) Policy statement on data sharing by WHO in the context of public health emergencies (as of 13 April 2016). Weekly Epidemiological Records 91: 237-240.
9. World Health Organization (2015) Genetic diversity of wild-type measles nucleotide surveillance database (MeaNS). Weekly Epidemiological Record 90: 373-380.
10. Kremer JR, Brown KE, Jin L, Santibanez S, Shulga SV, et al. (2008) High genetic diversity of measles virus, World Health Organization European Region, 2005-2006. Emerging Infectious Disease 14: 107-114.
11. Wang H, Zhang Y, Mao N, Zhu Z, Cui A, et al. (2019) Molecular characterization of measles viruses in China: Circulation dynamics of the endemic H1 genotype from 2011 to 2017. PLoS One 14: e0218782.
12. Muhammad SR, Usman M, Ikram A, Alam MM, Salman M, et al. (2022) Molecular identification and characterization of measles virus genotypes circulating in Afghanistan, 2008-2018. J Med Virol 94: 3448-3452.
13. Rota J, Lowe L, Rota P, Bellini W, Redd S, et al. (2006) Identical genotype B3 sequences from measles patients in 4 countries, 2005. Emerging Infectious Diseases 12: 1779-1781.
14. World Health Organization (2001) Nomenclature for describing the genetic characteristics of wild-type measles viruses (update). Part I. Weekly Epidemiological Record 76: 242-247.
15. World Health Organization (2012) Measles virus nomenclature update: 2012. Wkly Epidemiol Rec 87: 73-81.
16. Abdulfatai K, Olonitola OS, Aminu M, Jatau ED (2017) Seroprevalence of measles virus among children 0-12 years of age in some states in NorthWestern Nigeria. Int J Curr Microbiol App Sci 6: 2584-2594.
17. Lekana-Douki SE, Sir-Ondo-Enguier PN, Banga-Mve-Ella O, Imboumy-Limoukou RK, Maganga GD, et al. (2018) Epidemiology and molecular characterization of the re-emerging measles virus among children and adults in the Haut-Ogooue, Gabon. BMC Infectious Diseases 19: 90.
18. Hanses F, Truong AT, Ammerlaan W, Ikusika O, Adu F, et

al. (1999) Molecular epidemiology of Nigerian and Ghanaian measles virus isolates reveals a genotype circulating widely in western and Central Africa. J Gen Virol 80: 871-877.

19. Kremer JR, Nkwembe E, Bola Oyefolu AO, Smit SB, Pukuta E, et al. (2010) Measles virus strain diversity, Nigeria and Democratic Republic of the Congo. Emerg Infect Dis 16: 1724-1730.

20. Faneye AO, Motayo BO, Adeniji JA (2019) Molecular characterization and evolutionary dynamics of measles virus sequences isolated from children in Lagos and Ibadan, Southwestern, Nigeria. J Infect Public Health 13: 309-312.

21. El Mubarak HS, Van De Bildt MWG, Mustafa OA, Vos HW, Mukhtar MM, et al. (2002) Genetic characterization of wild-type measles viruses circulating in suburban Khartoum, 1997-2000. J Gen Virol 83: 1437-1443.

22. Rota PA, Brown K, Mankertz A, Santibanez S, Shulga S, et al. (2011) Global distribution of measles genotypes and measles molecular epidemiology. J Infect Dis 204: 514-523.

23. Nigatu W, Jin L, Cohen BJ, Nokes D J, Etana M, et al. (2001) Measles virus strains circulating in Ethiopia in 1998-1999: molecular characterization using oral fluid samples and identification of a new genotype. J Med Virol 65: 373-380.

24. Kouomou DW, Nerrienet E, Mfoupouendoun J, Tene G, Whittle H, et al. (2002) Measles virus strains circulating in Central and West Africa: geographical distribution of two B3 genotypes. J Med Virol 68: 433-440.

25. Gouandjika-Vasilache I, Waku-Kouomou D, Ménard D, Beyrand C, Guye F, et al. (2006) Co-circulation of measles virus genotype B2 and B3.1 in Central African Republic during the 2000 measles epidemic. J Med Virol 78: 964-970.

26. Mulders MN, Nebie YK, Fack F, Kapitanyuk T, Sanou O, et al. (2003) Limited diversity of measles field isolates after a national immunization day in Burkina Faso: progress from endemic to epidemic transmission? J Infect Dis 187: 277-782.

27. Haddad-Boubaker S, Rezq M, Smeo MN, Ben-Yahia A, Abudher A, et al. (2010) Genetic characterization of clade B measles viruses isolated in Tunisia and Libya 2002-2009 and a proposed new subtype within the B3 genotype. Virus Res 153: 258-264.

28. Demanou M, Ratsitoharana R, Yonga M, Dosseh A, Anya B, et al. (2013) Molecular characterization of measles viruses that circulated in Cameroon between 2010 and 2011. Virol J 10: 71.

29. Ndombo PK, Ndze VNN, Mbarga FD, Anderson R, Acho A, et al. (2018) Molecular characterisation of measles virus strains among refugees from the Central African Republic in Cameroon in 2014. Epidemiol Infect 146: 319-323.