ISSN: 2415-038X (Print)

Journal of Preventive and Rehabilitative Medicine

2018; 1(1): 5-11
Published Online:23/03/2018 (http://medicine.unza.zm/research/journal)
doi: 10.21617/jprm.2018.0101.1

Original Paper

Measles Viruses in Zambia: A Review on Circulating Wild-type Genotypes, Complications with Human Immunodeficiency Virus and Control (2006-2016).

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To cite this article:
Kunda Ndashe, Samuel Munjita, Novan Tembo, Sody Musanka, Bernadette Mumba. Measles Viruses in Zambia: A Review on Circulating Wild-type Genotypes and Complications with Human Immunodeficiency Virus. *Journal of Preventive and Rehabilitative Medicine,* Vol. 1, No. 1, 2018, pp. 5-11. doi: 10.21617/jprm.2018.0101.1

ABSTRACT

Measles is a highly contagious disease that most commonly affects children. The disease continues to record morbidity and mortality among infants in Zambia. We searched online databases such as PubMed, Scopus, Google Scholar, and National Center for Biotechnology Information (NCBI) database and ISI Web of Science and critically reviewed appropriate publications to extract consistent findings, the wild-type MeV present in Zambia, the complications of Measles and the Human immunodeficiency Virus and the control of Measles in Zambia. We included 18 research articles and 2 epidemiological bulletins in the synthesis. From the search of the NCBI database a total of 80 nucleotide sequences of 48 MeV isolates were obtained, 34 sequences (25 MeV isolates) from Zambia and 46 sequences (23 MeV isolates) WHO reference strains. Out of the 34 sequences from Zambia, 9 and 25 were H-gene and N-gene nucleotide sequences, respectively. This study identified 3 MeV genotypes in Zambia (B2, B3 and D2) spatially distributed in Lusaka, Ndola, Kitwe, Mwense and Samfya. Infants born from women who are HIV-1 seropositive had lower maternal antibodies and post initial vaccination antibodies to measles in HIV-1-infected infants waned off rapidly. The review re-emphasized the need for supplemental immunisation activities which include second opportunity to immunisation and case-based surveillance.

Key words: Measles virus, genotypes, control, Zambia

1. Introduction

Measles is a highly contagious disease that most commonly affects children. It is caused by an enveloped non-segmented, negative stranded RNA Measles virus (MeV) of the genus *Morbillivirus*, family *Paramyxoviridae* [1]. The MeV genome encodes a total of eight proteins. The six structural proteins are the nucleocapsid protein (N), phosphoprotein (P), matrix protein (M), fusion protein (F), attachment protein (H), and the large error-prone RNA-dependent RNA polymerase protein (L) [2]. Two additional nonstructural proteins (C and V) are encoded in the P transcription unit. While the C protein is translated from an overlapping reading frame within the P gene, the V protein is initiated from the same start codon as P, but a frame-shift is created by mRNA editing [3]. The outcome is that P and V share an N-terminal domain of 231 amino acids, but differ in their C-terminal domains (276 and 69 amino acids, respectively). The N and H gene sequences are most commonly used for genetic characterization of wild-type MeV [4]. The World Health Organisation (WHO) currently recognizes 8 clades, designated A, B, C, D, E, F, G, and H. Within these clades, there are 23 recognized genotypes, designated A, B1, B2, B3, C1, C2, D1, D2, D3, D4, D5, D6, D7, D8,D9, D10, E, F, G1, G2, G3, H1, and H2, and 1 provisional genotype, d11 [5].

In the developed world, measles immunisation programmes have reduced the number of cases reported annually to negligible levels [6]. Nonetheless, measles remains a major health problem in densely populated urban communities in sub-Saharan Africa [7].

In Zambia, measles is endemic with transmission peaks occurring between August and December, despite the relentless efforts of immunisation [8]. Since 1992, MeV has been isolated from children admitted to hospital in Lusaka, Zambia. Between 1992 and 1995, the University Teaching Hospital in Lusaka clinically diagnosed 1066 children with measles of which 203 (19.0%) were less than the 9 months of age which is the recommended time for measles vaccination in Zambia [8]. In another study conducted in Zambia, out of 277 children with clinical measles that were admitted to the University Teaching Hospital, of 149 samples tested, 132 (88.6%) were positive for IgM antibody while 14 (20.9%) of 67 samples, measles viruses were isolated [9]. The latter study highlights the importance of confirmatory tests in the diagnosis of measles to avoid misdiagnosis, since other clinical conditions may cause similar symptoms to measles.

Genetic analysis of MeV in a region helps document the effectiveness of control measures. In areas that have endemic transmission of measles, virologic surveillance of cases detects a limited number of genotypes while in areas where endemic transmission of virus has been interrupted, a variety of genotypes are detected, reflecting the multiple sources of imported viruses [10]. The virologic surveillance information has shown that vaccination programs can reduce the number of co-circulating chains of transmission and eventually interrupt measles transmission [11]. However, viruses are continually being introduced from external sources, and if the number of susceptible individuals increases, sustained transmission of the newly introduced viral genotype is possible. This results in what appears as a rapid change in the endemic genotype [12, 13]. The genetic stability of MeV is exceptionally high, and it has been observed that it undergoes remarkably little sequence variation over long periods of time, both in laboratory settings and in the field [14]. Therefore, genetic analysis of MeV in endemic areas such as Zambia helps to document the genotype of circulating virus strains, effectiveness of immunisation and possible introduction of new genotypes from other countries or regions.

The article reviews the wild-type MeV present in Zambia, the complications of Measles and the Human immunodeficiency Virus and the control of Measles in Zambia.

2. Methodology

We searched PubMed, Scopus, Google Scholar, and ISI Web of Science (up to November 17, 2017) using the following search terms: “Epidemiology of Measles in Zambia”, “Genotype of Measles Virus in Zambia”, “Measles and Human Immunodeficiency Virus in Zambia”. We supplemented database searches by screening bibliographies of the articles. Two independent reviewers (KN, NT) screened article titles and abstracts to select articles for full-text screening. The reviewers of the current paper assessed full texts independently; in case of disagreement, they consulted a third author (SM), and agreed upon a decision by consensus.

We further searched the National Center for Biotechnology Information (NCBI) database for all available nucleotide sequences of MeV isolated from Zambia and WHO reference strains that are used for genetic analysis. The obtained MeV nucleotide sequences were then analysed using Bioedit and MEGA 6 software. Phylogenetic trees were constructed in MEGA6 using the neighbor-joining method with the Kimura two-parameter evolutionary model [15, 16].

3. Results

The primary search identified 58 papers. We removed 24 duplicates. We screened 34 articles to assess eligibility, and excluded 16 that did not meet the inclusion criteria. We included 18 articles in the synthesis. We also included 2 epidemiological bulletins and alert from WHO and CDC. From the search of the NCBI database a total of 80 nucleotide sequences of 48 MeV isolates were obtained, 34 sequences (25 MeV isolates) from Zambia and 46 sequences (23 MeV isolates) WHO reference strains (Table 1). Out of the 34 sequences from Zambia, 9 and 25 were H-gene and N-gene nucleotide sequences, respectively.

Topologically, the phylogenetic tree of the N-gene, MeV was separated in 8 groups and the Zambian isolates identified in 3 groups (Figure 1). While phylogenetic tree of the H-gene showed that the Zambian isolates to belong to one group (Figure 2). The Zambian MeV isolates were clustered in the genotypes B2, B3 and D2. Geographical distribution of the MeV in Zambia revealed that genotype B3 was found in Lusaka, Ndola, Kitwe, Samfya and Mwense, genotype B2 in Kitwe and genotype D2 in Lusaka (Figure 3).

Table 1: Measles Virus isolates from Zambia and WHO reference strains

S/N	Virus name	Location	Year	Author(s)/Year	GenBank accession No.	
					H-gene	N-gene
1	MVi/Lusaka.ZAM/4.02	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845414	DQ839364
2	MVi/Lusaka.ZAM/44.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845413	DQ839363
3	MVi/Lusaka.ZAM/39.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845412	DQ839362
4	MVi/Lusaka.ZAM/38.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845411	DQ839361
5	MVi/Lusaka.ZAM/36.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845410	DQ839360
6	MVi/Lusaka.ZAM/5.02	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845415	DQ839365
7	Mvi/Lusaka.ZAM/34.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845409	DQ839359
8	Mvi/Lusaka.ZAM/23.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845408	DQ839358
9	MVi/Lusaka.ZAM/19.01	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007	DQ845407	DQ839357
10	929_PBMC	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007		DQ839370
11	916_PBMC	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007		DQ839369
12	797_PBMC	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007		DQ839368
13	685_NP	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007		DQ839367
14	658_PBMC	Lusaka, Zambia	2006	Riddell *et al*., 2006; Riddell *et al*., 2007		DQ839366
15	MVs/Ndola.ZMB/40.10/4[B3]	Ndola, Zambia	2011	Smit, 2011		JF973136
15	MVs/Ndola.ZMB/40.10/3[B3]	Ndola, Zambia	2011	Smit, 2011		JF973135
16	MVs/Ndola.ZMB/40.10/2[B3]	Ndola, Zambia	2011	Smit, 2011		JF973134
17	MVs/Ndola.ZMB/40.10/1[B3]	Ndola, Zambia	2011	Smit, 2011		JF973133
18	MVs/Kitwe.ZMB/40.10/[B3]	Kitwe, Zambia	2011	Smit, 2011		JF973132
19	MVs/Lusaka.ZMB/16.10/2[B3]	Lusaka, Zambia	2011	Smit, 2011		JF973131
20	MVs/Kitwe.ZMB/5.09/[B2]	Kitwe, Zambia	2011	Smit, 2011		JF973062
21	MVs/Kitwe.ZMB/3.09/[B2]	Kitwe, Zambia	2011	Smit, 2011		JF973061
22	MVs/Mwense.ZMB/17.12/[B3]	Mwense, Zambia	2014	Smit, 2014		KM581241
23	MVs/Samfya.ZMB/10.12/[B3]	Samfya, Zambia	2014	Smit, 2014		KM581240
24	MVs/Lusaka.ZMB/17.10/[B3]	Lusaka, Zambia	2012	Smit, 2012		JQ627709
25	MVs/Lusaka.ZMB/16.10/3[B3]	Lusaka, Zambia	2012	Smit, 2012		JQ627708
26	Edmonston-wt.USA/54	USA	1994	Rota *et al*., 1994	U03669	U01987
27	Yaounde.CAE/12.83"Y-14"	Cameroon	1998	Rota *et al*., 1993	AF079552	U01998
28	Libreville.GAB/84 "R-96"	Gabon	1998	Rota *et al*., 1994	AF079551	U01994
29	Ibadan.NIE/97/1	Nigeria	1998	Hanses *et al*., 1999	AJ239133	AJ232203
30	Tokyo.JPN/84/K	Japan	2001	Rota, 2001	AY047365	AY043459
31	Maryland.USA/77 "JM"	USA	2001	Baczko *et al*., 1992	M81898	M89921
32	Bristol.UNK/74 (MVP)	UK	1996	Rozenblatt *et al*., 1985	Z80805	D01005
33	Johannesburg.SOA/88/1	RSA	1998	Kreis and Whistler, 1996	AF085198	U64582
34	Illinois.USA/89/1 "Chicago-1"	USA	1993	Rota *et al*., 1994	M81895	U01977
35	Montreal.CAN/89	Canada	1998	Rota *et al*., 1994	AF079554	U01976
36	Bangkok.THA/93/1	Thailand	1997	Rota, 1998	AF009575	AF079555
37	New Jersey.USA/94/1	USA	2001	Rota *et al*., 1996	L46749	L46750
38	Victoria.AUS/16.85	Australia	2000	Chibo *et al*., 2000	AF247202	AF243450
39	Manchester.UNK/30.94	UK	1998	Nigatu et al., 2001	U29285	AF280803
40	Victoria.AUS/12.99	Australia	2002	Chibo *et al*., 2003	AY127853	AF481485
41	Kampala.UGA/51.00/1	Uganda	2005	Muwonge *et al*., 2005	AY923213	AY923185
42	Goettingen.DEU/71 "Braxator"	Germany	1996	Rima *et al*., 1995	Z80797	X84879
43	MVs/Madrid.SPA/94 SSPE	Spain	1996	Rima *et al*., 1995	Z80830	X84865
44	Berkeley.USA/83	USA	1998	Rota, 1993	AF079553	U01974
45	Amsterdam.NET/49.97	Indonesia	1999	Truong *et al*., 1999	AF171231	AF171232
46	Gresik.INO/17.02	Indonesia	2002	Rota and Liffick, 2002	AY184218	AY184217
47	Hunan.CHN/93/7	China	1998	Xu *et al*., 1998	AF045201	AF045212
48	Beijing.CHN/94/1	China	1998	Xu *et al*., 1998	AF045203	AF045217

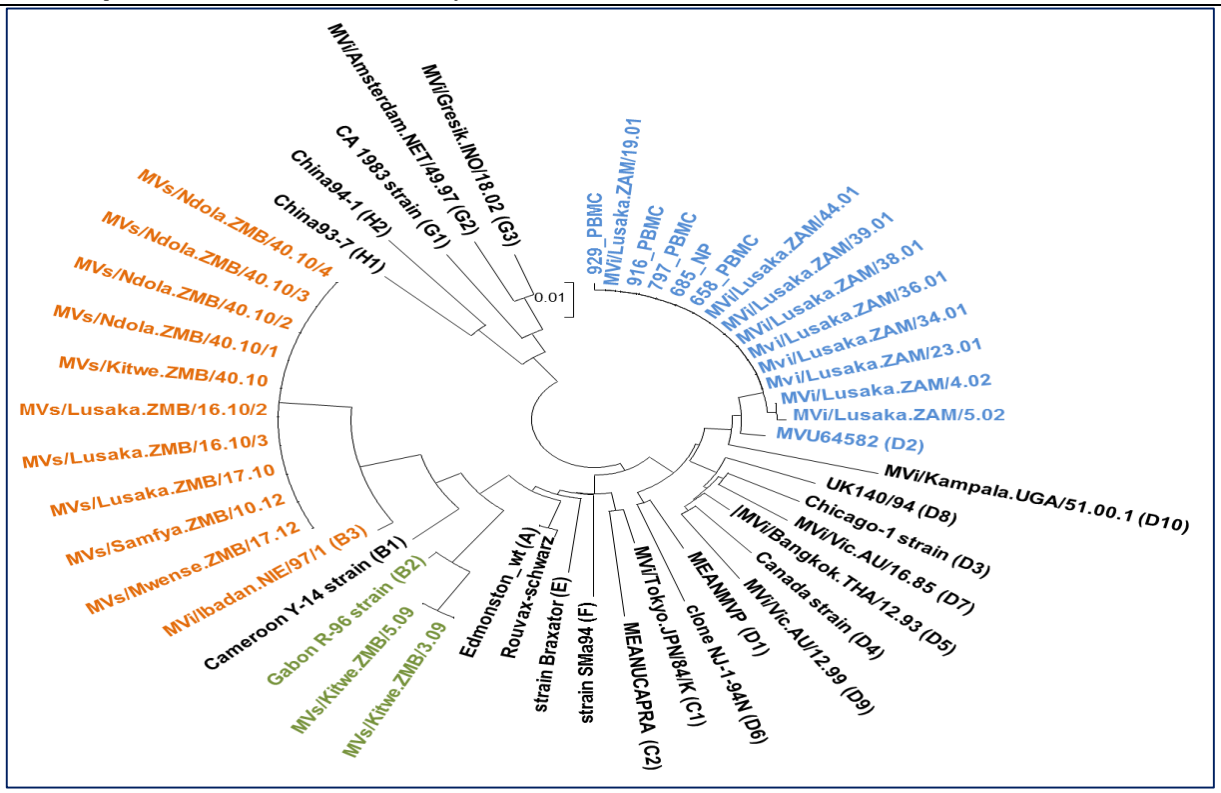

Figure 1: Phylogenetic relationships of the N-gene of MeV detected in clinical patients in Zambia and the WHO reference strains. Phylogenetic analysis was based on 456 bp of the N-gene. Isolate names for nucleotide sequences included in the analyses are given in parentheses

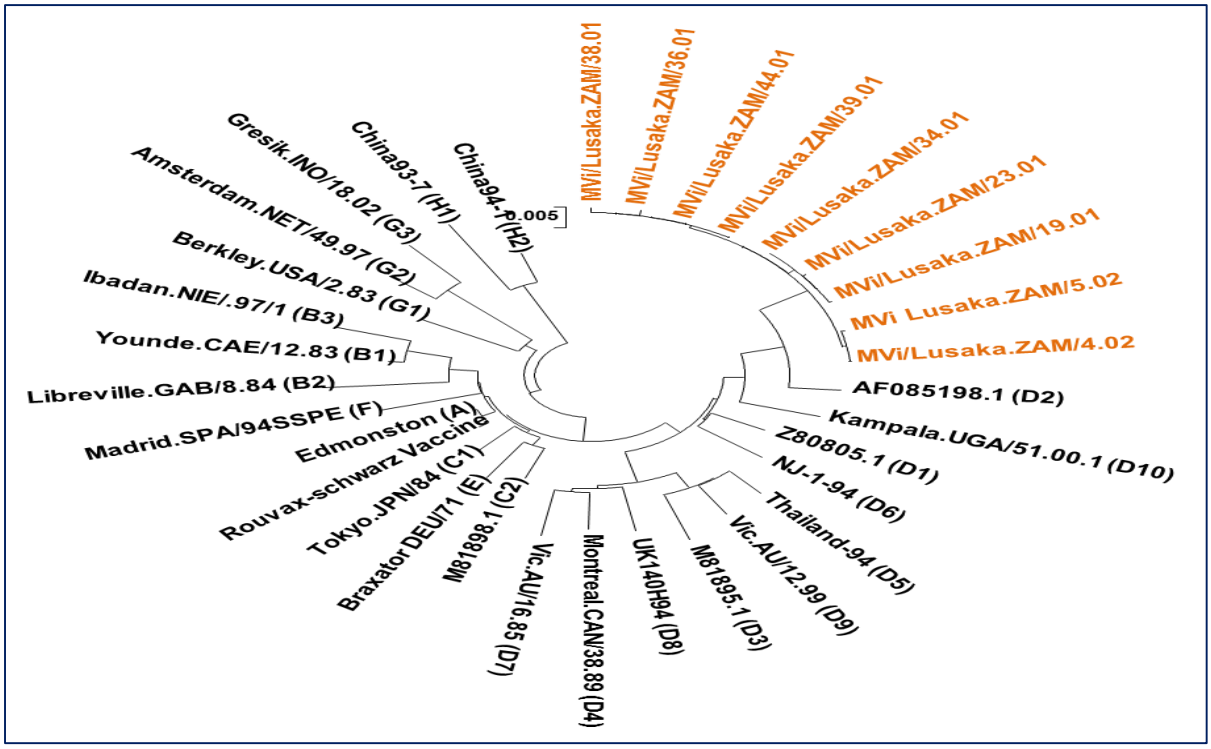

Figure 2: Phylogenetic relationships of the H-gene of MeV detected in clinical patients in Zambia and the WHO reference strains. Phylogenetic analysis was based on 1504 bp of the H-gene. Isolate names for nucleotide sequences included in the analyses are given in parenthese

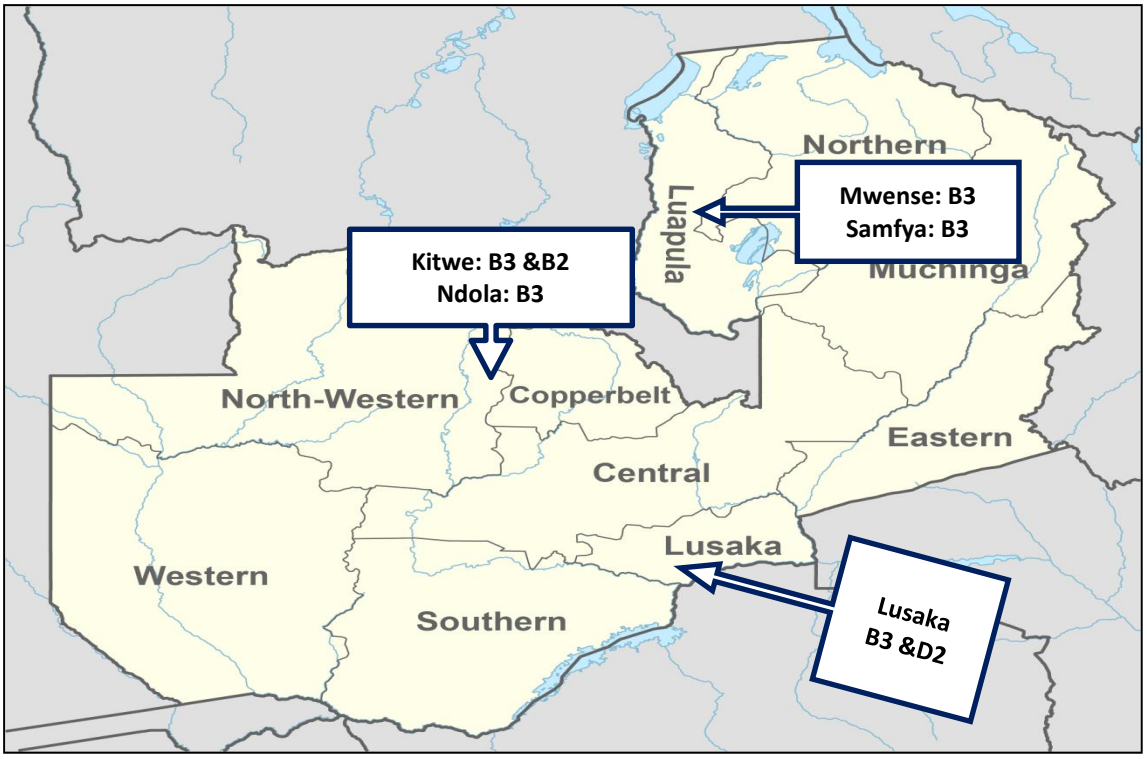

Figure 3: Spatial distribution of MeV genotypes in Zambia, isolated from clinically confirmed cases. Lusaka, Ndola, Kitwe, Mwense and Samfya all had the genotype B3 while B2 and D2 were found in Lusaka and Kitwe respectively.

4. Discussion

The wild type measles virus genotypes circulating in Zambia

Molecular analysis of MeV serves as an important tool to understand the circulating strains of the virus in a region and efforts made in controlling outbreaks through immunisation. This study identified 3 MeV genotypes in Zambia. The genotypes B2, B3 and D2 were isolated from patients clinically diagnosed with measles in Lusaka, Ndola, Kitwe, Mwense and Samfya. The genotype B3 was common in all the 5 districts while B2 and D2 genotypes were unique to Kitwe and Lusaka, respectively. The finding of this review agrees with other workers that have reported MeV in Zambia. Rota and Bellini (2003) and Riddell *et al* (2005) reported that the genotype D2 was circulating in Zambia and South Africa while Rota *et al* (2011) further revealed that between 2007 and 2009, 21 genotype B2 sequences were reported from the Democratic Republic of the Congo, Zambia, and Angola [11, 17, 18].

Results of the study further revealed that the genotypes were identified between 2006 and 2014 after Zambia had adopted strategies to accelerate measles control, which included conducting case based surveillance [19].

The complications of Measles and the Human immunodeficiency Virus

The co-infection of measles and Human immunodeficiency Virus (HIV) has resulted in complications in the immunisation of the former. It has been reported that infants born to women infected with HIV have lower titres of maternal antibodies to MeV and are at higher risk of contracting measles before the mandatory age of vaccination which is at 9 months in most sub-Saharan countries [20, 21]. Moss *et al* (2007) in a study in Lusaka reported that HIV-1–infected Zambian children developed antibody levels considered to be protective after measles vaccination at approximately 9 months of age, with comparable frequency to that achieved by HIV-1–uninfected children [22]. The research further revealed that antibody levels to measles vaccine in HIV-1-infected children waned off rapidly surviving up to 2 to 3 years. Scott *et al* (2007) also reported that levels of maternal antibodies to MeV were lower during the first 9 months of life in Zambian infants born to HIV-1–infected women than in infants born to uninfected women furthermore these levels were lower in HIV-1–infected infants than in HIV-seropositive but uninfected infants [23]. Therefore, the HIV-1- infected infants are at increased risk of measles

before the mandatory age of routine vaccination at 9 months but are also less likely to have levels of maternal antibodies that would neutralize measles vaccine virus. The World Health Organisation (WHO) recommends a second measles vaccination for all children, either through repeated campaigns or a routine second dose [24].

Measles Control in Zambia

Before 2003, Zambia controlled measles through single dose administration of the measles containing vaccine (MCV) to infants at age of 9 months [19]. Between 1992 and 1999, an average of 11,787 suspected measles case were reported annually, ranging 5, 983 in 1998 to 23, 518 in 1999 [19, 26]. During the same period the national measles immunisation coverage ranged from 61% in 1993 to 93% in 1996. In the quest of controlling measles outbreaks, in 2003, Zambia adopted a strategy of supplemental immunisation activities (SIA) which included strengthening routine vaccination, providing a second opportunity for measles immunisation for all children between 9 months and 4 years, and conducting case-based surveillance [25]. Since it was reported by Moss *et al* (2007) that MeV antibody titres wane off rapidly in HIV-1-infected children, the second opportunity for measles immunisation offers booster vaccination for prolonged protection against the disease.

Lowther *et al* (2009) reported that 3 years after a successful SIA that markedly decreased incidence and mortality of measles in Zambia, 84% of children within the study townships had a history of measles immunisation and only 67% had detectable antibodies to MeV in oral fluid samples [27]. This result suggested a build-up of susceptible children and a population at risk for measles outbreaks. It was observed that HIV-1-infected children did not contribute substantially to the pool of susceptible children. In 2016 the Ministry of Health (MoH) in its continued efforts to improve child health introduced the Measles Rubella vaccine (MR) in the national routine immunisation system. The vaccine was given to children at the same age as measles vaccine for the first and second doses at 9 months and 18 months respectively during routine immunisation [28]. The introduction of MR vaccine was a necessary step to accelerate progress towards achieving the global goal of measles and rubella elimination by the year 2020 set by the Measles and Rubella Initiative (M&R) Initiative [28]. Zambia reported improvements in under 5 mortality declining from 168 deaths per 1000 live births in 2002 to 75 deaths per 1000 live births in 2014 and are directly attributed to sustained immunisation coverage and other child health interventions [29].

5. Conclusion

Continued monitoring of the MeV genotypes in clinically diagnosed cases is necessary to document the circulating wild-types in order to monitor the efforts of immunisation campaigns. Zambia is well vested with human resource and laboratory capacity to conduct the routine MeV surveillance. Infants born from women infected with HIV-1 should be given the first MeV vaccine at 6 months of age because they have lower levels of MDA to Measles. As recommended by WHO a second opportunity for measles vaccination for all children is necessary, because of the reported waning immunity among HIV-1–infected children. Therefore, sufficient resources ought to be allocated towards surveillance and vaccination campaigns by the Ministry of Health (MoH) in Zambia.

Recommendations

Knowledge gaps in the epidemiology of measles over an extended period need to be addressed in the elimination of the disease in Zambia. This information is incredibly valuable as predictable epidemiological patterns emerge as measles elimination is approached and achieved. These critical features, including the source, size and duration of outbreaks, the seasonality and age-distribution of cases, genotyping pointers and effective reproduction rate shall be necessary in the control of the disease.

Author contributions

K.N conceived of the research idea. K.N and SM1 developed the theory and performed the computations. N.T and SM2 verified the analytical methods. BM. encouraged K.N. to investigate [control of measles] and supervised the findings of this work. All authors discussed the results and contributed to the final manuscript.

Acknowledgements

We would like to thank Drs. Oswell Khondowe and Margaret Mweshi for the constructive criticism and guidance during the preparation of the manuscript.

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