BIBLIOGRAPHIC INFORMATION SYSTEM

Journal Full Title: Journal of Biomedical Research & Environmental Sciences

Journal NLM Abbreviation: J Biomed Res Environ Sci

Journal Website Link: <https://www.jelsciences.com>

Journal ISSN: 2766-2276

Category: Multidisciplinary

Subject Areas: Medicine Group, Biology Group, General, Environmental Sciences

Topics Summation: 128

Issue Regularity: Monthly

Review Process type: Double Blind

Time to Publication: 7-14 Days

Indexing catalog: [Visit here](#)

Publication fee catalog: [Visit here](#)

DOI: 10.37871 ([CrossRef](#))

Plagiarism detection software: [iThenticate](#)

Managing entity: USA

Language: English

Research work collecting capability: Worldwide

Organized by: [SciRes Literature LLC](#)

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Monitoring of Group A Rotavirus Strains Circulating in the Environment and Among Children with Acute Gastroenteritis

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ABSTRACT

Rotavirus A is the causative agent of 90% of acute gastroenteritis in children under 5, which kills 1 to 3 million children per year. Their strong resistance in the environment, their inter-species transmission as well as their power of genetic recombination can give rise to new reasserting that may be harmful to public health.

The simultaneous search for the presence of rotavirus A in different environmental and clinical biotopes and matrices as well as the monitoring of the seasonal evolution of episodes is of major importance. At cost, genetic monitoring of rotaviruses shows a correlation between the presence of different genotypes of RVA in the environment and the rate of morbidity, Hence the need to monitor the emergence of new circulating strains with a view to integrating them into routine immunization programmes appropriate for each region in order to limit the spread of the disease.

INTRODUCTION

Human enteric viruses pose a major public health risk. This risk is linked to human-to-human, zoonotic, food and water transmission. Infectious agents transported by humans are sometimes asymptomatic or in the incubation phase, those transported by animals, water and food pass as unnoticed also [1]. Epidemics of Acute Gastroenteritis (AGE) are often linked to adenoviruses, noroviruses and rotaviruses [2-5]. Rotaviruses (RVs) are the main causative agents of AGE in children under 5 worldwide [6,7]. They are responsible for high infant mortality, death of 1 to 3 million including more than 800,000 in Africa [8,9] and almost 220,000 hospitalizations per year in industrialized countries, [10]. The prevalence is 35-60% of acute and severe diarrhea, almost every child might went through at least one episode of Rotavirus Gastroenteritis (RVGE) [11], the highest prevalence corresponds to the age range 0-11 months [6,12]. Developing countries [13-15], sub-Saharan Africa and the Indian subcontinent are the most affected [16] (Figure 1). According to surveillance data in 2013 and 2014, there were 215,000 deaths from rotavirus in children under 5 years worldwide, 56% of deaths were reported in sub-Saharan Africa and 22% in India alone [17].

In the same context, faecal contamination of environmental water can play an important role in the transmission and epidemiology of enteric viruses [18]. Indeed, water-borne epidemics pose a significant threat to human health worldwide,

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DOI: 10.37871/jbres1198

Submitted: 15 January 2021

Accepted: 02 March 2021

Published: 03 March 2021

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OPEN ACCESS

Subject: Biology Group

Topic & Subtopic(s): Infectious Diseases;
Antiretrovirology; Microbiology

Keywords

- Rotavirus
- Gastroenteritis
- Environment
- Circulating strains
- Emergence

VOLUME: 2 ISSUE: 3

Children under 5 years, annual estimate (1986-2000)

- 111 million diarrhoea episodes treated at home;
- 25 million consultations;
- 2 million hospitalizations.

440 000 death (●=1000 death)

Figure 1 Geographic distribution of rotavirus mortality in children under 5 years of age according to Parashar, et al. [14].

despite the relevant advances in water and wastewater treatment technologies, particularly in developing countries where access to drinking water is very limited in favour of untreated surface water and where the appropriate outbreak notification and monitoring system is still deficient.

Epidemiology of rotavirus gastroenteritis

RVs were first described in 1973 by Bishop, et al. [19], although most enteric viruses were discovered in the 1950s and 1960s. They are ubiquitous viruses, very widespread in animal kingdom where viral strains differ from one animal species to another. However, animal-human transmission remains possible and would give new strains in case of co-infection.

RVs can cause diarrhea which is one of the most deadly childhood diseases in Africa [20]. RV infections appear in winter as epidemics on a South-West-North-East gradient in Europe, and on a West-East gradient in the United States [21,22]. In 2013, despite the approval and availability of several RV vaccines on the market, RV caused about 215,000 deaths worldwide [17]. In 2017, New South Wales experienced a major outbreak of rotavirus gastroenteritis [23]. To help control the burden of severe rotavirus diseases, the World Health Organization (WHO) has recommended rotavirus vaccines for routine immunization of all children worldwide [24,25].

Clinical trials of the two RVs vaccines (RotaTeq, Merck and Rotarix, GSK Biologicals) recommended by the WHO for global use since 2009 have successfully demonstrated the

effectiveness of these vaccines in a wide range of countries [26–30]. Based on WHO recommendations, the Moroccan Ministry of Health introduced the single-strain rotavirus vaccine, Rotarix, into its national vaccination program in 2010 to reduce the high rate of rotavirus disease in Morocco [31].

Source of rotavirus contamination and transmission

The transmission of RV is faecal-oral, by direct contact during nosocomial infections in health units. Vomits aerosols also ensure human-to-human transmission of the virus. Man is therefore a natural reservoir of human rotavirus that contaminates its environment through wastewater [32]. RVs are excreted in very large numbers in stool, with more than 10^{11} particles per gram. Faecal contamination of vegetable and fish products such as molluscs and crustaceans by viruses has been reported with a load of between 10^2 and 10^4 viral particles per gram of tissue [33–36]. This mode of transmission to healthy people is ensured by the consumption of raw or undercooked seafood products.

Due to the resistance to protein degradation conferred by the three-layer structure of the protein capsid, ribonucleic acid from rotavirus may remain infectious. RVs are highly resistant to physical inactivation, but remain sensitive to chlorinated derivatives (Bleach) and 70% diluted ethanol. Hence, the virus is highly resistant in the environment and can keep its infectivity for weeks outside the human body [37]. And for several days, up to two months in water at 20°C, in groundwater sources for several months and

several hours on the hands [38]. In addition, rotaviruses are highly contagious, so even small amounts level (10 to 100 particles) of RVs are capable of infecting humans [37]. Abundant diarrhoea promotes the secretion and spread of new viruses in the environment, thereby amplifying the spread of outbreaks.

BIOLOGY OF ROTAVIRUSES

The RVs are non-enveloped viruses, whose mature, wheel-shaped virus particles are about 75 nm in diameter, which can increase up to 100 nm with spicules. Its double-stranded Ribonucleic Acid (RNA) genome contains 11 segments (Figure 2) encoding six structural proteins (VP) and six Non-Structural Proteins (NSP), the size of these segments ranges from approximately 667 (Segment 11) to 3302 base pairs (Segment 1) [39]. One RNA segment codes two proteins, while the other ten segments code one protein each [40]. The capsid is icosahedral with three concentric layers of a protein nature, the innermost VP2 encoded by segment 2, contains the enzymes needed for the viral replication, the outer layer consists of 780 molecules of glycoprotein VP7 encoded by segment 9. This outer layer is surmounted by spicules of a protein nature VP4 (120 molecules) encoded by segment 4 [41]. These last two proteins have antigenic

activity in particular VP7 defining serotype G, and VP4 defining serotype P, both inducing neutralizing antibodies in the host, these 2 proteins define the viral serotype and appear to be a major target of vaccination. The intermediate protein layer VP6 encoded by segment 6 whose conserved sequences at the 5' and 3' ends of the VP6 gene are specific to each rotavirus group (A - G). The VP1 and VP3 proteins, encoded by segments 1 and 3 respectively, are associated with the genome; their enzymatic activity is responsible for the infectivity of the virion. The virulence and pathogenesis of RV diarrhoea is closely related to non-structural NPS proteins in particular the NPS4 encoded by segment 10, which plays the role of intestinal toxin.

Glycoprotein VP7, and protein VP4 have antigenic properties involved in anti-RV immunity. They are neutralizing antibody targets, which allow the determination of genotypes G and P respectively. VP4 and VP7 proteins in the outer layer of rotavirus particles induce neutralizing antibodies [42]. However, some VP6-oriented antibodies [43,44], and enterotoxin NSP4 [45,46] may also provide effective protection against rotaviruses.

VIRAL REPLICATION OF ROTAVIRUSES

The strong tropism of RVs for mature enterocytes of the small intestine induces infection and lesion of the villi of the intestinal brush border inducing severe diarrhoea responsible for morbidity. Viral replication usually occurs in enterocytes, but in immunocompromised children, the infection can also affect the liver and kidneys. VP7 and VP4 proteins play a specific role in the early stages of the viral cycle by expressing the antigenic properties of the virus [47]. This tissue tropism can be inhibited by neutralizing antibodies that prevent the virus from physically attaching to the cell receptor or stabilize the outer layer, thereby preventing the virus from entering the enterocyte and detaching the capsid [48].

Under the action of proteolytic enzymes, the VP4 protein from the capsid cleaves and the cell membrane becomes permeable which facilitates the penetration of the virus into the cytoplasm, the Rotavirus-specific VP1 polymerase, is activated following partial decapsidation and begins transcription of viral RNA to RNA⁻. The 11 newly formed RNA⁺ segments assemble with the VP1, VP2, VP3, and VP6 proteins in cytoplasmic inclusions (Viroplasm) which bind to the NSP4 protein at the endoplasmic reticulum membrane, which provides them with a transient envelope by budding. Subsequently, the VP4 and VP7 proteins attach to the bud which loses its envelope and the release of the mature viral particle by cell lysis [49] or by destabilization of the brush border [50].

Human RVs can be grown on a wide variety of cells. MA104 monkey kidney cells represent the most classic

Figure 2 RNA segments encoding structural and non-structural proteins of RV SA11 with their molecular sizes.

line in which culture lasts 10–12 hours at 37°C [51] but this diagnostic technique remains difficult compared to animal RV which makes it not suitable for routine amplification technique.

ROTAVIRUS TAXONOMY AND GENETICS

Diversity of rotaviruses and risk of emergence of new strains

RVs belong to the family Reoviridae, the Rotavirus genus consists of 7 groups (A to G), these groups are identified by the antigenic determinant of the internal Protein of the Capsid (VP6). Rotaviruses infect several species where humans are susceptible to groups A, B and C, while rotaviruses from groups D to G have only been found in animals. Group A is responsible for the majority of AGEs in young children. Although rare, group B rotavirus causes sometimes severe epidemics in China [52], while Rotavirus C remains the cause of sporadic cases of intrafamilial epidemics.

There are currently 32 G and 47 P genotypes in Rotavirus group A [53], of which 12 G and 15 P have been reported to cause GEA in humans [54,55]. However 90% of clinically relevant genotypes characterized globally correspond to genotypes G1, G2, G3, G4 and G9 [56]. Other genotypes occur and rarely emerge in areas such as the G5 in South America, the G8 in sub-Saharan Africa and the G12 in south-eastern Asia. Genotype G5 circulating in Brazil since 1982 has been found to be of porcine origin [57], and is often combined with human genotype P[8]. This same G5 genotype was also characterized in Cameroon in 2000 [58] but with phylogenetic characteristics closer to the porcine strain found in Brazil. The G8 and G10 genotypes are known

through human-to-human transmission in Africa and India respectively, while the G6 genotype is common in cattle. The G8 genotype encountered in Malaoui in 1977 and currently in Australia is often associated with genotypes P[4], P[6] or P[8] [59,60].

The most common P genotypes worldwide are P[4] and P[8], the least common are P[6] and P[9] and the very few are P[3], P[11], P[12] and P[14]. The latter genotypes are marked by zoonotic or inter-species transmission, which results in human-animal reassortments. The transmission of certain Rotaviruses from animals (Bovine or porcine) to humans or co-infection by several strains could lead to reassortments (Figure 3), by gene exchange and generate unusual genotypes or new emerging strains [61]. Phylogenetic analysis of the recently described G8P[7] and G6P[7] bovine strains in Korea has shown that the genomic segments have bovine but also human and porcine origins [62].

Diversity, the result of possible combinations, is at the origin of the binary antigenic classification linked to two segments 4 and 9 of the Rotavirus genome. This classification should theoretically generate from 32 G and 47 P a number of 1504 combinations. Nevertheless, the majority of epidemiological studies worldwide show that 75% of RVs infections in humans are related to G1[P8], G2[P4], G3[P8], G4[P8], and G9[P8] [22,63,64,56]. The G1[P8] strains represent 40% of the strains characterized worldwide; this predominance could be explained by the emergence of new strains. Strains G2[P4], G3[P8] and G4[P8] each account for approximately 10%.

Sequencing of the whole genome of the different rotavirus strains has become the tool of choice for classifying the 11 genome segments and therefore determining the origin of the strain [65].

Figure 3 Example of reassortment between Rotavirus strains.

SPATIOTEMPORAL VARIABILITY

The distribution patterns of rotavirus outbreaks in different geographical regions of the world are closely linked to the socio-economic development of countries [16,66] and climate parameters. Temperate countries experience winter peaks of Rotavirus-related epidemics while these epidemics occur throughout the year in equatorial countries and a less pronounced seasonality in tropical regions. Peaks of infection may vary from province to province and month to month depending on surveillance data [22,56].

Global data [56] support this geographic and temporal variability in Rotavirus genotypes and provide a prevalence profile of the virus genotypes for each country (Figures 4-6).

MEDICAL PROPHYLAXIS OF ROTAVIRUS

AGE occurs in children under 5 years of age in general and in infants from 3 to 11 months in particular. However, at 3 months of age and younger may not develop symptoms of diarrhea when infected with RV since they are protected in the first months of life by transplacental maternal antibodies [67].

The predominantly oral-faecal mode of transmission is a major factor in the prevention and control of rotavirus AGE. The non-specific prevention of viral AGE is based on compliance with universal hygiene rules and the consumption of sufficiently cooked foods, in particular shellfish dishes. Specific prevention against VRs is,

Figure 4 Variation in the distribution of Rotavirus genotypes worldwide 1996 and 2007 (Adapted by Hatib [1]).

Figure 5 Overview of WHO regions that provided strain monitoring data from 1996 to 2007 respectively: Africans (14/46, 10/46, 15/46), Americans (3/36, 10/36, 20/36), Europeans (9/52, 13/52, 30/52), Eastern Mediterranean (2/21, 5/21, 9/21), South-East Asia (3/11, 2/11, 7/11) and the Western Pacific (4/27, 6/27, 12/27).

Figure 6 Geographic distribution of common RVA strains over the period (1996-2007).

according to WHO recommendations, based on vaccination [68]. Moreover, the implementation of the RV eradication program is only possible thanks to the existence of effective vaccines. The current strategy is based on the use of live and attenuated antiviral vaccines designed to achieve immunity comparable to that induced by natural rotavirus infections by providing homotypic or heterotypic protection against severe diarrhoea caused by major circulating rotavirus serotypes [69].

Two safe and effective RVA vaccines have been approved in approximately 100 countries worldwide since 2006 [70] in particular (RotaTeq, Merck Inc., USA and Rotarix, GlaxoSmithKline Biologicals, Belgium). Another live oral

monovalent vaccine (ROTAVAC®) was found to be effective in a large clinical trial in India. This vaccine is given in three doses to infants [71]. Injectable vaccines among other RV vaccines are also being studied [72]. The vaccine against pentavalent rotavirus was effective against severe RVA gastroenteritis in the first 2 years of life in high-mortality African countries in infants under 5 years of age [29]. In 2014, more than 65 countries have already introduced RVA vaccines into their national vaccination programs, including more than 20 countries in Africa [73]. Thus the mortality rate per 1 000 live births among children under the age of 5 has increased from 73 to 57 [74], which shows an improvement in child survival worldwide over the past decade. Other countries, such as South Africa, have seen a

remarkable decrease in the rate of hospitalizations due to RVA, from 69% to 54% after two years of the introduction of the vaccine in 2009 [75].

Vaccination of young children has shown high efficacy against severe diarrhea and hospitalization due to RVA in high- and middle-income countries [27]. However, both vaccines showed significantly lower immunogenicity and efficacy in low-income countries in Africa and Asia [76], compared to developed countries in this case Australia where two-dose vaccine efficacy estimates are 88.4%, 83.7% and 78.7% in individuals 6-11 months of age, 1-3 years of age, and 4-9 years of age, respectively [23]. However, in Finland after the introduction of the vaccine in children under 5 years of age, the infection became more frequent in unvaccinated children aged 6 to 16 and people over 70 years of age [77].

INFECTION CONTROL MEASURES

Infection control measures begin with isolation of infected children; hygiene and hand cleaning with alcohol-containing agents, to be used after contact with infected children; improved water quality; the disinfection of environmental surfaces with appropriate detergents and finally ensure the widespread access to sanitation [78], [79].

The monitoring of the AGE is of particular interest, in order to comply with the directives of the international bodies advocating the monitoring of the genetic dynamism of the RV in particular in species living close to humans such as cattle, pigs, cats, horses, chickens etc... Second, direct RV vaccine prophylaxis towards the use of a regional product that includes contemporary strains, which could help determine the strains that may not be covered by the vaccine.

In addition, phylogenetic analysis of strains isolated from the world makes it possible to determine their belonging to which phase and which lineage. The installation of a regional or national network of epidemics-surveillance of the viral etiology AGE makes it possible, undoubtedly, to orientate towards the answers and thus offer the researchers a platform for carrying out virological investigations later.

CONCLUSIONS

The surveillance of RVs, that are responsible for AGE in young children, known by their ability for adaptation and the emergence of new reassorting strains, must be continuously ensured in parallel with vaccination. In order to assess the risk of contamination, the establishment of a surveillance program to monitor RV infection and the burden of related diseases is needed. Hence, by establishing sentinel sites as well as environmental monitoring, characterization of VR strains circulating in different biotopes, as a retrospective procedure. The persistence of RV and other enteric viruses

in the environment must also be addressed by adapting the wastewater treatment method in wastewater treatment plants to manage the reduction of the viral load.

Documentation of all clinical presentations and RV-related environmental analysis results should be maintained in a systematic manner to correlate the seasonal distribution of VR-related hospitalizations, while identification of circulating RV strains, as a platform for epidemiological and environmental studies, and to identify any potential emergence of new strains related, may be, at vaccine-induced selective pressure. However, further strain-specific vaccine efficiency studies are required to follow-up period and accompanying genotypic analysis to assess genotypic variation and to understand the complexities of RV epidemiology.

The intensity of this surveillance should be reduced only when the impact of the vaccine is demonstrated. The adoption of this policy is the most effective way to anticipate any possible intervention when new strains or outbreaks emerge.

ACKNOWLEDGEMENTS

We are grateful to the University Hassan II of Casablanca, and to the members of the Laboratory of Virology, Microbiology, Quality and Biotechnology/ Eco Toxicology and Biodiversity. We appreciate the efforts of the staff of the molecular biology laboratory at the Institute Pasteur Morocco of Casablanca.

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How to cite this article: Hatib A, Hassou N, Ennaji MM. Monitoring of Group A Rotavirus Strains Circulating in the Environment and Among Children with Acute Gastroenteritis. *J Biomed Res Environ Sci*. 2021 Mar 03; 2(3): 104-113. doi: 10.37871/jbres1198, Article ID: JBRES1198