Journal of Clinical Virology 78 (2016) 111–119

Contents lists available at ScienceDirect

Journal of Clinical Virology

journal homepage: www.elsevier.com/locate/jcv

Review

Human norovirus infection in Latin America

Tatiane da Silva Poló[a,b], Juliana R. Peiró[b], Luiz Cláudio Nogueira Mendes[b], Louisa F. Ludwig[a], Edmilson F. de Oliveira-Filho[a], Filemon Bucardo[c], Pascale Huynen[d], Pierrette Melin[d], Etienne Thiry[a], Axel Mauroy[a,*]

[a] Veterinary Virology and Animal Viral Diseases, Fundamental and Applied Research on Animal and Health centre and Faculty of Veterinary Medicine, University of Liège, Belgium
[b] Department of Clinics, Surgery, and Animal Reproduction, School of Veterinary Medicine, Univ Estadual Paulista (UNESP), Araçatuba, SP, Brazil
[c] Department of Microbiology, National Autonomous University of León, Nicaragua (UNAN-León), Nicaragua
[d] Clinical Microbiology Department, University Hospital of Liège, Liège, Belgium

ARTICLE INFO

Article history:
Received 7 December 2015
Received in revised form 9 March 2016
Accepted 14 March 2016

Keywords:
Norovirus
Gastroenteritis
Epidemiology
Latin america

ABSTRACT

Noroviruses are important enteric pathogens involved in non-bacterial gastroenteritis outbreaks worldwide. Noroviruses mainly occur from person to person via the fecal-oral route but also through contaminated food or water; indirect contamination is also possible due to the resistance of the virus in the environment. Latin American countries as a whole cover a vast North-to-South range, which is highly heterogeneous in terms of climate, ecosystem, human population distribution (urban areas with high human densities versus closed communities), economic development and genetic backgrounds resulting from each particular historical context. This review aims to present epidemiological and clinical patterns of human norovirus infections in Latin American countries. Divergent prevalences were observed depending on the country and the surveyed population. In particular, a shift in rotavirus/norovirus ratio in the etiologies of gastroenteritis was detected in some countries and could be attributed partly to rotavirus vaccine coverage in their infant population. While GII.4 noroviruses were seen to constitute the most common genotype, differences in genotype distribution were observed both in the environment (via sewage sampling proxy) and between genotypes circulating in healthy and diarrheic patients. Due to high climatic discrepancies, different patterns of seasonality were observed. Accordingly, this continent may condense the different particular epidemiological features encountered for HuNoV infections worldwide.

Contents

* Corresponding author.
E-mail address: amauroy@ulg.ac.be (A. Mauroy).

http://dx.doi.org/10.1016/j.jcv.2016.03.016
1386-6532/

1. Introduction

Infectious diarrhea, especially in children, is an important worldwide health issue, particularly in developing countries [1–3]. In most countries, human noroviruses (HuNoVs) are considered as the main viral cause of acute gastroenteritis (AGE) outbreaks in adults and as the second most common viral agent in children after group A rotaviruses (RVA) [4].

HuNoVs belong to the genus *Norovirus*, family *Caliciviridae* and are non-enveloped viruses with a positive-sense, single-stranded RNA genome. NoV strains are currently classified into seven genogroups (G), which are further subdivided into at least thirty genotypes [5–7]. Viruses belonging to GI, GII, and GIV infect humans, while GII, GIII, GIV, GV, GVI and GVII NoVs have been described in animal species [8–12]. Interestingly, few animal species share strains with human within the same genogroups (GII for porcine and GIV for canine and feline).

This review compiles data from Latin American countries and aims at the description of their particular characteristics of regarding HuNoV infections. In particular, the reviews will focus on main transmission routes, clinical aspects and (molecular) epidemiology. Each aspect will be presented from what is known from other regions of the world and confronted to the Latin American situation.

2. Transmission

HuNoVs are resistant in the environment and can be transmitted faecal-orally via different routes, notably by the consumption of contaminated food or water, by contact with contaminated people, objects or surfaces, or even via vomit-derived aerosols [13]. Although NoVs have been detected in animal feces, no evidence of zoonotic transmission has been reported so far [13].

River waters are at risk for HuNoV contamination as they usually receive effluents of wastewater treatment plants, which are more efficient for removal of bacteria rather than viruses [14,15]. Several studies throughout the world have detected HuNoV in river water used for irrigation as well as in drinking water [13]. While in the Amazon region, HuNoVs have been molecularly detected only at low frequencies in stream water samples from the city of Manaus, they have been shown to be responsible for AGE outbreaks following consumption of contaminated water from the river, poor hygienic behavior, or recreational activities in contaminated water [16]. In Guatemala, an AGE outbreak occurred in a student group after a school trip and its origin was traced to contaminated water consumption [17]. HuNoVs have been also detected in water from Argentinean rivers with 7.7% of positive samples from the Luján River and up to 80.8% of positive samples from the Matanza-Riachuelo River in Buenos Aires [18]. This study also showed that faecally contaminated waters frequently involve multiple genetically divergent strains. In the same country, in a study conducted on eight AGE outbreaks due to HuNoVs, three outbreaks were attributed to a waterborne origin [19], confirming that HuNoVs can constitute an issue for safe water supply. Also in Colombia, HuNoVs have been detected in two samples from freshly treated potable water [20]. In the region of Antofagasta, Chile, AGE cases have been reported wherein HuNoVs were found both in stool samples and in environmental samples, leading to a revision of the rules concerning wastewater usage for irrigation of vegetables, especially those habitually consumed raw [21]. In Mexico, the virus was detected during spring in estuary waters [22].

The different stages of the food production (at pre-harvest, harvest and post-harvest levels) are at risk for HuNoV contamination [13]. In Rio de Janeiro, Brazil, HuNoVs were detected in six out of nine lettuce samples collected from supermarkets, food services and restaurants [23], mirroring previous observations in other countries [24–28]. In Chile, foodborne AGE outbreaks often occur following consumption of contaminated seafood products [29]. Seafood constitutes a major food supply in Latin American countries and shellfish, in particular, have been shown to concentrate HuNoVs in their digestive tissues by filtration and concentration through specific binding [30,31]. Moreover, an underestimation of the real burden of foodborne and waterborne HuNoV infections probably exists due to the frequent absence of an active surveillance system to identify and report HuNoV outbreaks in most Latin American countries [32].

Due to their low infectious dose and high environmental stability, HuNoVs are highly transmissible, rendering the person-to-person transmission route the most efficient, especially in (semi)-closed communities [13]. Sharing the same contaminated environment can obviously facilitate the transmission of this virus as exemplified by an AGE outbreaks with highly efficient person-to-person transmission reported in Peru [33], in Argentina [34] and in Brazil [35,36]. Furthermore, the person-to-person transmission route facilitates geographical extension of outbreaks caused by contaminated food as observed in Antofagasta, Chile [21], and in outbreaks involving Latin American travelers [33,37–39]. In Chile, a prevalence of 12.3% for HuNoVs in AGE outbreaks has been found, with positive detection in samples from wastewater treatment plants but negative results in potable water samples collected from households [21]. In this country, several outbreaks caused by HuNoVs have been reported from closed communities such as childcare centers, schools and hospitals, while a smaller fraction was reported to occur in restaurants [29].

3. Clinical aspects

After a short incubation period (1–2 days), the main symptoms reported following HuNoV infection, are vomiting, fever, diarrhea, nausea, abdominal cramps, headache, chills and myalgia [4,32,40–42], which are all usually short-term and self-limiting. Vomiting seems to be the most frequent symptom regardless of the age of patient [42,43]. Severe AGE cases, although less frequent, usually involve infants and elderly or immunocompromised patients, and can sometimes require hospitalization [32,41,44–46]. Besides the classical symptoms associated to AGE, HuNoV infections have in some cases been associated to other non-usual

symptoms such as seizures [47,48], encephalopathy [49], intestinal hemorrhage [50] and necrotizing enterocolitis [41,51,52].

HuNoV infections affect all age groups, being the most common cause of diarrhea in adults [40]. This can be partly explained by the high antigenic diversity in HuNoV strains, evolving to evade herd immunity [53–55], and allowing frequent adult reinfections [40]. Among children, a higher incidence rate is observed in under-two year-old [35,56–62].

In immunocompromised patients, HuNoV infections can constitute an important risk factor of debilitating diarrhea or chronic AGE with prolonged viral excretion [63]. In Venezuela, HuNoVs were equally detected in HIV-positive and HIV-negative adults. However, HIV carrier children had a higher prevalence than HIV uninfected children [64]. In Brazil, a study performed on stem cell recipients reported 60% of HuNoV positive stool specimens after transplantation, associated to fever, diarrhea and vomiting in these patients [65], while another study did not detect HuNoVs in 11-year-old HIV-positive children [66].

4. Epidemiological aspects

In absence of an active surveillance, most data result from epidemiological studies performed in the field [32]. The samples used for diagnosis are usually stool specimens, less frequently rectal swabs and, in rare cases, blood samples. RT-PCR is considered as the gold standard for detection and typing of HuNoVs [67]. Currently, real time RT-PCR is generally used for higher sensitivity and quantification benefits [40]. Enzyme immunoassays (EIA) are commercially available for HuNoV detection, sometimes allowing the differentiation between GI and GII infections, and are widely used in outbreak investigations [67]. Fumian et al. [41] suggest that HuNoVs can spread beyond the intestine to reach the bloodstream, and viral detection in blood could correlate with longer hospitalizations.

In a systematic review on AGE cases, regardless of patients' ages, a global HuNoV prevalence of 18% was estimated [68]. HuNoV prevalences in some Latin American countries, reported in Table 1, appear to lie in the same range, even if higher prevalences have been reported for cities with high population densities and/or poor health and socioeconomic conditions.

4.1. Prevalence in hospitalized children

Young children represent a population where incidence rates of HuNoV infections are elevated. In Ecuador, 66% of children under three years-of age experienced at least one HuNoV infection and 40% have had two [69]. Prevalences of HuNoV infections in children hospitalized for AGE constitute an accurate even slightly biased proxy for comparison between countries or regions in epidemiological studies. Indeed, data are easy to record and samples diagnosed for a large set of pathogens but only the more severe cases are registered. In a systematic review, the proportion of HuNoVs in hospitalized children with AGE ranged from 3% to 31% worldwide [70]. In Latin America, it ranged from 2.2% up to 43%. The highest prevalence (43%) was reported in 2004–2005 in Paraguayan children less than five year-old, hospitalized with AGE and negative for both bacteria and rotavirus [71]. The lowest proportion (2.2%) was reported in Argentina, in Mendoza, among hospitalized children less than two year-old [72]. High prevalences were found in regions with high population densities. In Buenos Aires, 24.2% of children under three years of age, hospitalized with symptoms of AGE were HuNoV positive [60], while in São Paulo, a HuNoV prevalence of 33% was found in a similar group (<3 year-old) from four hospitals of the metropolitan region of São Paulo [73]. Similar prevalences were recorded in the city of Vitória, Brazil, in AGE hospitalized children

Table 1
Apparent prevalences of Human Norovirus infections in Latin America.

Country	Region	Population characteristics	Period	Age group	Type of sample	Type of diagnostic test	Number of analyzed samples (persons)	Number of norovirus positive samples	Apparent prevalence (%)		References
									Symptomatics	Asymptomatics	
Argentina	Mendonza	Children's Hospital	1995–1998	≤2 years	Stools	RT-PCR, EIA	941	8	2.2	–	[71]
	Buenos Aires	Children's Hospital	1997–1998	≤3 years	Stools	RT-PCR	66	16	24.2	–	[59]
Brazil	Rondônia	Children's Hospital	2010–2012	≤6 years	Stools	RT-PCR	591	46	7.8	–	[75]
	Northeastern	Shantytown	1989–1993	≤4 years	Serum	EIA	420 (135)	96	71	–	[104]
	Mato Grosso do Sul	2 hospitals	2000–2004	≤3 years	Stools	RT-PCR	406	31	7.6	–	[76]
	Espírito Santo	Children's Hospital	2003–2004	≤3 years	Stools	RT-qPCR	319	52	17	13	[42]
	Espírito Santo	Quilombola comunity	2007–2009	≤11 years	Stools	PCR	397	16	9.2	1.5	[106]
	São Paulo	4 hospitals	1995–1999	≤3 years	Stools	RT-PCR	234	78	33	36.4	[72]
	São Paulo	Day care unit	2010–2011	Children	Stools	RT-PCR	100	2	4	0	[126]
Chile	Santiago	Metropolitan Region	2006–2008	≤1.5 years	Stools	ELISA, RT-PCR	3106 (198)	26	12.6	57	[81]
Colombia	Chia and Cuidad Bolivar	Day care unit	2009	1–5 years	Stools	RT-qPCR	277	18	9.3	5	[83]
Guatemala	San Juan Sacatepequez	Rural área	1999	≤3 years	Serum	EIA	522	388	72.4	–	[92]
	Santa Rosa, Quetzaltenango	2 hospitals	2007–2010	Children and adults	Stools	RT-qPCR	2403	341	14	–	[73]
Mexico	Mexico	Peri-urban	1989–1991	≤2 years	Stools	RT-PCR	181 (140)	12	12	5	[82]
Nicaragua	León	Community and hospital	2005–2006	children and adults	Stools, serum, saliva	ELISA, RT-PCR, EIA, RT-qPCR	694	73	11	–	[105]
Paraguay	Asuncion	Private hospital	2004–2005	≤5 years	Stools	RT-PCR	378	161	43	–	[70]
Peru	Lima	Peri-urban	2006–2008	2 months–2 years	Stools	RT-PCR	224	39	17.4	–	[88]
	Las Pampas de San Juan de Miraflores	Peri-urban	2007–2011	≤2 years	Stools	RT-qPCR	5185 (498)	607	22.8	13	[78]
Uruguay	Montevideo	Private hospital (high socioeconomic level)	2010–2011	≤5 years	Stools	IC	59	2	3.4	–	[1]
Venezuela	Valencia	Children's Hospital	2003	≤5 years	Stools	EIA, qRT-PCR	480	61	12.7	–	[74]

RT-PCR: Reverse Transcription Polymerase Chain Reaction; EIA: Enzyme-immunoassay; RT-qPCR: Real Time Polymerase Chain Reaction after reverse transcription; qRT-PCR: Real Time Reverse Transcription Polymerase Chain Reaction; IC: Qualitative lateral immunochromatography.

under three years of age (17% in 2003–2004 [42]), in Guatemala (14% in ambulatory patients from the regions of Santa Rosa and Quetzaltenango between 2007 and 2010 [74]), and in children hospitalized with sporadic AGE during a study conducted in Valencia, Venezuela (12.7% in 2003 [75]). Despite regional differences, low detection rates have been reported in children hospitalized with AGE living in households of high socioeconomic levels in Uruguay [1] and in Brazil [76,77].

4.2. Asymptomatic infections

Asymptomatic infections are common in children under five years of age [78]. In asymptomatic Latin American populations, detection rates can vary from 3.5% among Chilean children in 2007–2008 [59], to 13.3% in 2007–2011 in Peru [79], and up to 49.2% in Mexico during a study performed in 1998 [80]. In Brazil, this ratio reached 36.4% in children less than 3 years-old [73] while in Nicaragua, 11.7% of asymptomatic infections were reported among children [81]. In the metropolitan area of Santiago, Chile, the highest HuNoV prevalences were found in asymptomatic patients compared to symptomatic ones HuNoV [82]. In peri-urban areas, prevalences among asymptomatic patients ranged from 5% in Mexico [83] to 13% in Peru [79], however lower than those observed in symptomatic patients from the same region. Similarly, a lower prevalence was observed in asymptomatic than in symptomatic patients from a day care center in Colombia [84] and from a Brazilian children's hospital [42].

4.3. Detection in the environment

An average prevalence of 25.4% was found in Argentinean rivers while regional prevalences ranged from 7.7% to 80.8% [18]. In the same country [18,85] and also in Nicaragua [86], HuNoVs have also been detected in effluents and in both raw and treated sewage. In particular, in Nicaragua, HuNoV prevalence detected in raw sewage and industrial water treatment plants reached 44%, and 32.1%, respectively, suggesting that HuNoVs, or at least their genomes, may persist even after treatment [85,86]. In Mexico, detection rates of 70% have been reported from estuary waters [22]. In Colombia, a low detection rate of 2.9% has been observed in treated water [20]. On the Uruguay River, HuNoVs were detected together with human rotaviruses and astroviruses in sewage dumped directly from four Uruguayan cities, showing the highest detection rate, 51%, among environmental samples from Latin American countries [87].

4.4. Seasonality

In the Northern hemisphere, a clearly defined seasonality for HuNoV has been described with the large majority of the clinical cases occurring in winter [88], which gave rise to the infection's old name of 'Winter Vomiting Disease' [68,88]. Although some studies failed to detect such seasonality [32,42,58,83,89–93], HuNoV infections have been shown to frequently occur mainly from June to October in temperate South American countries, namely Argentina, Chile, Uruguay, Southern Brazil and Paraguay [18,21,43,59,60,85,94–96], similarly to the seasonality observed in Oceania [88,97], and mainly from January to April in temperate countries of the Northern hemisphere [32,44,57,98,99]. These infections tend to predominantly occur in association with the rainy season in tropical Latin American regions [62,74,75,77,79,81,100–102]. Seasonality could be less apparent in cities with high population density [18] and could depend on the geographical area and the ethnic group, e.g. in the Brazilian 'Quilombola' community most HuNoV infections took place during the months of the school holidays (January, February and July) and coincided with family trips out of the village [103]. Environmental samples from Manaus (Brazil) also showed the same pattern [104].

4.5. Prevalence in rural versus urban areas

HuNoV infections can occur continuously in populations from large cities with higher densities, while in rural populations they can be more sporadic [105] and depend on hygienic conditions. A very high prevalence of 71% was observed in children with AGE living in a shantytown in the Northeastern region of Brazil between 1989 and 1993 [106] while peri-urban areas of Mexico and Peru presented a much lower prevalence in children with AGE [79,83,89]. In Guatemala, the HuNoV prevalence reached 72.4% in a rural area with poor sanitary conditions and high AGE rates [93]. In Nicaragua, a prevalence of 11% has been observed in patients with AGE [107], similar to the prevalence found in the metropolitan region of Santiago, Chile [82]. In the North of the Brazilian Espírito Santo state, studies performed on children less than 11 years-old from the 'Quilombola' community and from rural areas, showed lower prevalences for HuNoV infections in symptomatic (9.2%) and asymptomatic patients (1.5%) [108] than studies performed on children under three years from the same community but from urban areas (17% and 13% in symptomatic and asymptomatic patients, respectively) [42]. In Peru, this pattern was less marked, with prevalences of 21.3% and 8% in symptomatic and asymptomatic five year-old children, respectively, in a rural community in the Amazon region [61], and of 17.4% in diarrheic children from a peri-urban community of Lima [89], similar to the prevalences of 22.8% and 13.3% on symptomatic and asymptomatic patients, respectively, from a shantytown community in Lima [79].

4.6. Correlation between increased human norovirus detection and rotavirus vaccination

The burden of pediatric rotavirus infections declined significantly following the implementation of rotavirus vaccine coverage [109] and many studies correlated it with a significant increase in HuNoV detection rates [110–112]. In Nicaragua, prevalence of detected HuNoV infections increased after the onset of rotavirus vaccination, starting from 11–12% in 2005–2006 and reaching 24% in 2009–2010 [62,81,102]. Similarly, HuNoV detection in environmental waters significantly increased at the same time [86]. In Santiago, Chile, these prevalences started from 8% for calicivirus infections (both HuNoV and sapovirus) in 1997–1999 [113] and reached 18% in 2006–2008 [95]. These increased detection rates could be either associated to a true shift in etiologies or associated to the concomitant development of both awareness and diagnosis methods for HuNoVs.

5. Molecular epidemiology

HuNoV genotyping currently relies on sequencing and phylogenetic relationships of both complete (preferentially) or partial sequences of the capsid protein coding gene [114]. HuNoV genogroups GI, GII and GIV have been described in Latin America with GIV detected for the first time in 2004 during an AGE outbreak in Argentina [19] and later in environmental water samples from the city of Belém in Brazil [115]. There is a wide variety in genotypes circulating in Latin America, revealing the viral diversity in this region [32,42,103,116] (Fig. 1).

5.1. Genogroup II genotype 4 viruses

Strains genetically related to GII.4 are the most commonly detected worldwide [78] and have also been reported in Latin America by several studies [21,40,58,117–119]. Despite differences

Fig. 1. Molecular epidemiology of Human Norovirus in Latin America: genotypes circulating in the different countries (references in Supplementary Table 1).

in HuNoV prevalences between rural and urban communities, GII.4 remains the most frequent in both cases. Furthermore, this genotype has frequently been detected in several environmental studies involving sewage, rivers and surface waters in Argentina [85], treated sewage in Rio de Janeiro, Brazil [121], a sewage treatment plant in Florianopolis, Brazil [122], and wastewater in Nicaragua [86]. Additionally, an ability for global spread has been revealed by different studies [21,43,58,86,123–125].

5.2. Other genogroup II viruses

In addition to GII.4, several other GII genotypes have been detected from Latin American hospitals and asymptomatic patients [42,71,74,75,81,99,103]. A study performed on surface waters from Argentinean rivers reported that genotype diversity is directly related to the size of the cities [18]. Similarly, studies have demonstrated a notable genetic diversity of GII HuNoVs in children living in peri-urban and shantytown communities in Mexico and Peru [79,80]. GII.3 has been detected at high frequencies in many regions, in both symptomatic and asymptomatic patients [32,42,43,74,79,118,126] as well as in the environment [86,122]. It is difficult to associate a particular genotype to an exacerbated virulence, with a possible exception for GII.4. Interestingly, if GII.17HuNoVs have been regularly detected in Latin America, outbreaks related to the potentially emerging GII.17 strain [127] has not been yet reported from Latin America.

5.3. Genogroup I viruses

Although GI strains are less frequently detected, they also present a wide genetic diversity in Latin America, with the detection of GI.1–5, 7–8, 11 and 14 [32,56,71,73–75,79–81,103,108,121,122].

5.4. Recombinant viruses

A consequence of the wide genetic diversity in circulating genotypes is the possibility of co-infections, which may further result in recombination events [128,129]. In Latin America, different combinations in recombinant strains have been reported: GII.P7/GII.6 [130]; GII.P7/GII.20 [131], GII.P2/GII.3 and GI.P2/GI.6 [58], GII.P4/GII.1 [123].

6. Conclusion

Latin American countries present a wide diversity in climatic conditions, demography, medical and sanitary development, ethnic and economic conditions. Accordingly, this continent may condense the different particular epidemiological features encountered for HuNoV infections worldwide. Of particular interest, epidemiological patterns seem to be tightly linked to the community type (closed versus high density). This review highlights the need for a coordinated subcontinental surveillance network (molecular data sharing and emerging HuNoV detection) at both the clinical and food safety levels. Even though a huge genetic diversity has been detected, GII.4 remains the most prevalent circulating genotype, echoing the worldwide epidemiological situation. Future emerging genotypes could find their source from either the local genotype collection or from imported ones in a globalization context. If person-to-person is still the main transmission route, the hazard associated to HuNoV distribution in the environment, especially in waters designated for agriculture or consumption, should be particularly considered in the risk assessment for Latin America.

Conflict of interest

None declared

Funding

None

Ethical approval

Not required

Acknowledgements

This work was partly supported by a project '*Coopération bilatérale – aide à la mobilité*' co-funded by the Wallonie-Bruxelles International agency and the Brazilian CAPES foundation (process: 8880/14-0; CAPES/WBI process: 17/2014). The research that yielded these results was also funded by the Belgian Federal Public Service of Health, Food Chain Safety and Environment through the contract [RT 15/8 IQUINOR 2]. With the Support of Fund Leon Fredericq for biomedical research at the University Hospital of Liege.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jcv.2016.03.016.

Bibliography

[1] G. Varela, L. Batthyány, M.N. Bianco, W. Pérez, L. Pardo, G. Algorta, et al., Enteropathogens Associated with Acute Diarrhea in Children from Households with High Socioeconomic Level in Uruguay, Int. J. Microbiol. 2015 (2015) 1–8, http://dx.doi.org/10.1155/2015/592953.

[2] K.L. Kotloff, W.C. Blackwelder, D. Nasrin, J.P. Nataro, T.H. Farag, A. Van Eijk, et al., The Global Enteric Multicenter Study (GEMS) of diarrheal disease in infants and young children in developing countries: epidemiologic and clinical methods of the case/control study, Clin. Infect. Dis. 55 (S4) (2012) S232–S245, http://dx.doi.org/10.1093/cid/cis753.

[3] C.L. Fischer Walker, J. Perin, M.J. Aryee, C. Boschi-Pinto, R.E. Black, Diarrhea incidence in low- and middle-income countries in 1990 and 2010: a systematic review, BMC Public Health 12 (2012) 1–7, http://dx.doi.org/10.1186/1471-2458-12-220.

[4] M.K. Estes, B.V. Prasad, R.L. Atmar, Noroviruses everywhere: has something changed? Curr. Opin. Infect. Dis. 19 (2006) 467–474, http://dx.doi.org/10.1097/01.qco.0000244053.69253.3d.

[5] K.Y. Green, Caliciviridae: the noroviruses, in: David M. Knipe, P. Howley (Eds.), Fields Virology, 5th ed., Lippincott Williams and Wilkins, Philadelphia, 2007, pp. 949–979.

[6] J. Vinjé, Advances in laboratory methods for detection and typing of norovirus, J. Clin. Microbiol. (2014) 373–381, http://dx.doi.org/10.1128/JCM.01535-14.

[7] D.P. Zheng, T. Ando, R.L. Fankhauser, R.S. Beard, R.I. Glass, S.S. Monroe, Norovirus classification and proposed strain nomenclature, Virology 346 (2006) 312–323 http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=16343580.

[8] V. Martella, M. Campolo, E. Lorusso, P. Cavicchio, M. Camero, A.L. Bellacicco, et al., Norovirus in captive lion cub (Panthera leo), Emerging Infect. Dis. 13 (2007) 1071–1073, http://dx.doi.org/10.3201/eid1307.070268.

[9] V. Martella, E. Lorusso, N. Decaro, G. Elia, A. Radogna, M. D'Abramo, et al., Detection and molecular characterization of a canine norovirus, Emerging Infect. Dis. 14 (2008) 1306–1308, http://dx.doi.org/10.3201/eid1408.080062.

[10] A. Scipioni, A. Mauroy, J. Vinjé, E. Thiry, Animal noroviruses, Vet. J. 178 (2008) 32–45.

[11] G.N. Woode, J.C. Bridger, Isolation of small viruses resembling astroviruses and caliciviruses from acute enteritis of calves, J. Med. Microbiol. 11 (1978) 441–452 http://jmm.sgmjournals.org/content/11/4/441.full.pdf.

[12] E. Di Felice, A. Mauroy, F.D. Pozzo, D. Thiry, C. Ceci, B. Di Martino, et al., Bovine noroviruses: a missing component of calf diarrhoea diagnosis, Vet. J. (2015), http://dx.doi.org/10.1016/j.tvjl.2015.10.026.

[13] E. Mathijs, A. Stals, L. Baert, N. Botteldoorn, S. Denayer, A. Mauroy, et al., A review of known and hypothetical transmission routes for noroviruses, Food Environ. Virol. 4 (2012) 131–152.

[14] L. Maunula, Waterborne norovirus outbreaks, Future Virol. 2 (2007) 101–112, http://dx.doi.org/10.2217/17460794.2.1.101.

[15] A.K. da Silva, J.-C. Le Saux, S. Parnaudeau, M. Pommepuy, M. Elimelech, F.S. Le Guyader, Evaluation of removal of noroviruses during wastewater treatment, using real-Time reverse transcription-PCR: different behaviors of genogroups I and II, Appl. Environ. Microbiol. 73 (2007) 7891–7897, http://dx.doi.org/10.1128/AEM.01428-07.

[16] M.P. Miagostovich, F.F.M. Ferreira, F.R. Guimaraes, T.M. Fumian, L. Diniz-Mendes, S.L.B. Luz, et al., Molecular detection and characterization of gastroenteritis viruses occurring naturally in the stream waters of manaus, central amazonia, Brazil, Appl. Environ. Microbiol. 74 (2008) 375–382, http://dx.doi.org/10.1128/AEM.00944-07.

[17] W. Arvelo, S.M. Sosa, P. Juliao, M.R. López, A. Estevéz, B. López, et al., Norovirus outbreak of probable waterborne transmission with high attack rate in a Guatemalan resort, J. Clin. Virol. 55 (2012) 8–11, http://dx.doi.org/10.1016/j.jcv.2012.02.018.

[18] M.D.B. Fernández, C. Torres, H.R. Poma, G. Riviello-López, L.C. Martínez, D.M. Cisterna, et al., Environmental surveillance of norovirus in Argentina revealed distinct viral diversity patterns, seasonality and spatio-temporal diffusion processes, Sci. Total Environ. 437 (2012) 262–269, http://dx.doi.org/10.1016/j.scitotenv.2012.08.033.

[19] K.A. Gomes, J.A. Stupka, J. Gómez, G.I. Parra, Molecular characterization of calicivirus strains detected in outbreaks of gastroenteritis in Argentina, J. Med. Virol. 79 (2007) 1703–1709, http://dx.doi.org/10.1002/jmv.20989.

[20] M.F. Gutiérrez, M.V. Alvarado, E. Martínez, N.J. Ajami, Presence of viral proteins in drinkable water—sufficient condition to consider water a vector of viral transmission? Water Res. 41 (2007) 373–378, http://dx.doi.org/10.1016/j.watres.2006.09.022.

[21] T.J. Díaz, G.V. Solari, C.O. Cáceres, A.J. Mena, P.S. Baeza, U.X. Muñoz, et al., Brote de gastroenteritis aguda en la Región de Antofagasta, Chile. 2010, Rev. Chilena Infectol. 29 (2012) 19–25, http://dx.doi.org/10.4067/S0716-10182012000100003.

[22] J. Hernandez-Morga, J. Leon-Felix, F. Peraza-Garay, B.G. Gil-Salas, C. Chaidez, Detection and characterization of hepatitis A virus and norovirus in estuarine water samples using ultrafiltration – RT-PCR integrated methods, J. Appl. Microbiol. 106 (2009) 1579–1590, http://dx.doi.org/10.1111/j.1365-2672.2008.04125.x.

[23] M.L.L. Brandão, D.O. Almeida, F.C.P. Bispo, S.M.L. Bricio, V.A. Marin, M.P. Miagostovich, Assessment of microbiological contamination of fresh, minimally processed, and ready-to-eat lettuces (Lactuca sativa), Rio de Janeiro State, Brazil, J. Food Sci. 79 (2014) M961–M966, http://dx.doi.org/10.1111/1750-3841.12459.

[24] S.F. Grove, A. Suriyanarayanan, B. Puli, H. Zhao, M. Li, D. Li, et al., Norovirus cross-contamination during preparation of fresh produce, Int. J. Food Microbiol. 198 (2015) 43–49, http://dx.doi.org/10.1016/j.ijfoodmicro.2014.12.023.

[25] A. Stals, L. Jacxsens, L. Baert, E. Van Coillie, M. Uyttendaele, A quantitative exposure model simulating human norovirus transmission during preparation of deli sandwiches, Int. J. Food Microbiol. 196 (2015) 126–136, http://dx.doi.org/10.1016/j.ijfoodmicro.2014.12.004.

[26] J.Y. Bae, J.S. Lee, M.H. Shin, S.H. Lee, I.G. Hwang, Effect of wash treatments on reducing human norovirus on iceberg lettuce and perilla leaf, J. Food Prot. 74 (2011) 1908–1911, http://dx.doi.org/10.4315/0362-028X.JFP-11-063.

[27] H.F. Mok, S.F. Barker, A.J. Hamilton, A probabilistic quantitative microbial risk assessment model of norovirus disease burden from wastewater irrigation of vegetables in Shepparton, Australia, Water Res. 54 (2014) 347–362, http://dx.doi.org/10.1016/j.watres.2014.01.060.

[28] J. Loutreul, C. Cazeaux, D. Levert, A. Nicolas, S. Vautier, A.L. Le Sauvage, et al., Prevalence of human noroviruses in frozen marketed shellfish, red fruits and fresh vegetables, Food Environ. Virol. 6 (2014) 157–168, http://dx.doi.org/10.1007/s12560-014-9150-8.

[29] R. Vidal, V. Solari, N. Mamani, X. Jiang, J. Vollaire, P. Roessler, et al., Caliciviruses and foodborne gastroenteritis, Chile, Emerg. Infect. Dis. 11 (2005) 1134–1137, http://dx.doi.org/10.3201/eid1107.041062.

[30] H. Maalouf, J. Schaeffer, S. Parnaudeau, J. Le Pendu, R.L. Atmar, S.E. Crawford, et al., Strain-dependent norovirus bioaccumulation in oysters, Appl. Environ. Microbiol. 77 (2011) 3189–3196, http://dx.doi.org/10.1128/AEM.03010-10.

[31] H. Maalouf, M. Zakhour, J. Le Pendu, J.C. Le Saux, R.L. Atmar, F.S. Le Guyader, Distribution in tissue and seasonal variation of norovirus genogroup I and II ligands in oysters, Appl. Environ. Microbiol. 76 (2010) 5621–5630, http://dx.doi.org/10.1128/AEM.00148-10.

[32] M.S.R. Ferreira, M. Victoria, F.A. Carvalho-Costa, C.B. Vieira, M.P.T.P. Xavier, J.M. Fioretti, et al., Surveillance of norovirus infections in the state of Rio De Janeiro, Brazil 2005–2008, J. Med. Virol. 82 (2010) 1442–1448, http://dx.doi.org/10.1002/jmv.21831.

[33] V.E. Gonzaga, M. Ramos, R.C. Maves, R. Freeman, J.M. Montgomery, Concurrent outbreak of norovirus genotype I and enterotoxigenic escherichia coli on a U.S Navy Ship following a visit to Lima, Peru, PLoS One 6 (2011) e20822, http://dx.doi.org/10.1371/journal.pone.0020822.

[34] K.A. Gomes, J.A. Stupka, A. Diana, G.I. Parra, Caracterización molecular de calicivirus aislados de brotes de gastroenteritis ocurridos en la Argentina durante los años 2005 y 2006, Rev. Argent. Microbiol. 40 (2008) 222–228.

[35] M.S.R. Ferreira, M.P.T.P. da Xavier, A.C.C. de Tinga, T.L. Rose, T.M. Fumian, A.M. Fialho, et al., Assessment of gastroenteric viruses frequency in a children's day care center in Rio De Janeiro, Brazil: a fifteen year study (1994–2008), PLoS One 7 (2012) e33754, http://dx.doi.org/10.1371/journal.pone.0033754.

[36] D.M.M. Oliveira, M. Souza, F.S. Fiaccadori, H.C.P. Santos, D. das D. de P. Cardoso, Monitoring of Calicivirus among day-care children: evidence of asymptomatic viral excretion and first report of GI.7 norovirus and GI.3 sapovirus in Brazil, J. Med. Virol. 86 (2014) 1569–1575, http://dx.doi.org/10.1002/jmv.23791.

[37] H.L. Koo, N.J. Ajami, Z.D. Jiang, F.H. Neill, R.L. Atmar, C.D. Ericsson, et al., Noroviruses as a cause of diarrhea in travelers to Guatemala, India, and Mexico, J. Clin. Microbiol. 48 (2010) 1673–1676, http://dx.doi.org/10.1128/JCM.02072-09.
[38] G. Ko, C. Garcia, Z. Jiang, C. Pablo, J. Belkind-gerson, R.I. Glass, et al., Noroviruses as a cause of traveler's diarrhea among students from the United States visiting Mexico, J. Clin. Microbiol. 43 (2005) 6126–6129, http://dx.doi.org/10.1128/JCM.43.12.6126.
[39] A.R. Chapin, C.M. Carpenter, W.C. Dudley, L.C. Gibson, R. Pratdesaba, O. Torres, et al., Prevalence of norovirus among visitors from the United States to Mexico and Guatemala who experience traveler's diarrhea, J. Clin. Microbiol. 43 (2005) 1112–1117, http://dx.doi.org/10.1128/JCM.43.3.1112.
[40] R.I. Glass, U.D. Parashar, M.K. Estes, Norovirus gastroenteritis, N. Engl. J. Med. 361 (2009) 1776–1785, http://dx.doi.org/10.1056/NEJMra0804575.
[41] T.M. Fumian, M.C. Justino, J. D'Arc Pereira Mascarenhas, T.K. Reymão, E. Abreu, L. Soares, et al., Quantitative and molecular analysis of noroviruses RNA in blood from children hospitalized for acute gastroenteritis in Belém, Brazil, J. Clin. Virol. 58 (2013) 31–35, http://dx.doi.org/10.1016/j.jcv.2013.06.043.
[42] D.M.P.G. Barreira, M.S.R. Ferreira, T.M. Fumian, R. Checon, A.D.I. de Sadovsky, J.P.G. Leite, et al., Viral load and genotypes of noroviruses in symptomatic and asymptomatic children in Southeastern Brazil, J. Clin. Virol. 47 (2010) 60–64, http://dx.doi.org/10.1016/j.jcv.2009.11.012.
[43] J.D.S.R. de Andrade, M.S. Rocha, F.A. Carvalho-Costa, J.M. Fioretti, M.D.P.T.P. Xavier, Z.M.A. Nunes, et al., Noroviruses associated with outbreaks of acute gastroenteritis in the State of Rio Grande do Sul, Brazil 2004–2011, J. Clin. Virol. 61 (2014) 345–352, http://dx.doi.org/10.1016/j.jcv.2014.08.024.
[44] A.L. Gutiérrez-Escolano, F.R. Velázquez, J. Escobar-Herrera, C.L. Saucedo, J. Torres, T. Estrada-García, Human caliciviruses detected in Mexican children admitted to hospital during 1998–2000, with severe acute gastroenteritis not due to other enteropathogens, J. Med. Virol. 82 (2010) 632–637, http://dx.doi.org/10.1002/jmv.21743.
[45] C. Roddie, J.P.V. Paul, R. Benjamin, C.I. Gallimore, J. Xerry, J.J. Gray, et al., Allogeneic hematopoietic stem cell transplantation and norovirus gastroenteritis: a previously unrecognized cause of morbidity, Clin. Infect. Dis. 49 (2009) 1061–1068, http://dx.doi.org/10.1086/605557.
[46] J.P. Harris, W.J. Edmunds, R. Pebody, D.W. Brown, B.A. Lopman, Deaths from norovirus among the elderly, England and Wales, Emerging Infect. Dis. 14 (2008) 1546–1552, http://dx.doi.org/10.3201/eid1410.080188.
[47] S. Chen, C. Tsai, M. Lai, C. Chen, K. Lin, T. Lin, et al., Norovirus infection as a cause of diarrhea- associated benign infantile seizures, Clin. Infect. Dis. 48 (2009) 849–855, http://dx.doi.org/10.1086/597256.
[48] M.C. Medici, L.A. Abelli, I. Dodi, G. Dettori, C. Chezzi, Norovirus RNA in the blood of a child with gastroenteritis and convulsions—a case report, J. Clin. Virol. 48 (2010) 147–149, http://dx.doi.org/10.1016/j.jcv.2010.03.001.
[49] S. Ito, S. Takeshita, A. Nezu, Y. Aihara, S. Usuku, Y. Noguchi, et al., Norovirus associated encephalopathy, Pediatr. Infect. Dis. J. 25 (2006) 651–652, http://dx.doi.org/10.1097/01.inf.0000225789.92512.6d.
[50] S.-Y. Chen, Y. Feng, H.-C. Chao, M.-W. Lai, W.-L. Huang, C.-Y. Lin, et al., Emergence in Taiwan of novel norovirus GII.4 variants causing acute gastroenteritis and intestinal haemorrhage in children, J. Med. Microbiol. 64 (2015) 544–550, http://dx.doi.org/10.1099/jmm.0.000046.
[51] R.M. Turcios-Ruiz, P. Axelrod, K.S. John, E. Bullitt, J. Donahue, N. Robinson, et al., Outbreak of necrotizing enterocolitis caused by norovirus in a neonatal intensive care unit, J. Pediatr. 153 (2008) 339–344, http://dx.doi.org/10.1016/j.jpeds.2008.04.015.
[52] R.L. Stuart, K. Tan, J.E. Mahar, C.D. Kirkwood, C.A. Ramsden, N. Andrianopoulos, et al., An outbreak of necrotizing enterocolitis associated with norovirus genotype GII.3, Pediatr. Infect. Dis. J. 29 (2010) 644–647, http://dx.doi.org/10.1097/INF.0b013e3181d824e1.
[53] L.C. Lindesmith, V. Costantini, J. Swanstrom, K. Debbink, E.F. Donaldson, J. Vinjé, et al., Emergence of a norovirus GII.4 strain correlates with changes in evolving blockade epitopes, J. Virol. 87 (2013) 2803–2813, http://dx.doi.org/10.1128/JVI. 03106-03112.
[54] L.C. Lindesmith, E.F. Donaldson, R.S. Baric, Norovirus GII.4 strain antigenic variation, J. Virol. 85 (2011) 231–242 (JVI. 01364-10 [pii] 10.1128/JVI. 01364-10.).
[55] L.C. Lindesmith, E.F. Donaldson, A.D. LoBue, J.L. Cannon, D.-P. Zheng, J. Vinje, et al., Mechanisms of GII.4 norovirus persistence in human populations, PLoS Med. 5 (2008) e31, http://dx.doi.org/10.1371/journal.pmed.0050031.
[56] G.C. Aragão, D. de, S. Oliveira, M.C. dos Santos, J.D.P. Mascarenhas, C.S. de Oliveira, A. da, C. Linhares, et al., Molecular characterization of norovirus, sapovirus and astrovirus in children with acute gastroenteritis from Belém, Pará Brazil, Rev. Pan-Amazônica Saúde 1 (2010) 81–86, http://dx.doi.org/10.5123/S2176-62232010000100021.
[57] L.R. Ribeiro, R.S.D.O. Giuberti, D.M.P.G. Barreira, K.W. Saick, J.P.G. Leite, M.P. Miagostovich, et al., Hospitalization due to norovirus and genotypes of rotavirus in pediatric patients, state of Espírito Santo, Mem. Inst. Oswaldo Cruz 103 (2008) 201–206, http://dx.doi.org/10.1590/S0074-02762008000200013.
[58] M. Victoria, F.A. Carvalho-Costa, M.B. Heinemann, J.P. Leite, M.P. Miagostovich, Prevalence and molecular epidemiology of noroviruses in hospitalized children with acute gastroenteritis in Rio de Janeiro, Brazil 2004, Pediatr. Infect. Dis. J. 26 (2007) 602–606, http://dx.doi.org/10.1097/INF.0b013e3180618bea.
[59] S. Montenegro, S. Pineda, I. Enríquez, N. Enríquez, N. Rivera, Detección de norovirus en niños con diarrea adquirida en la comunidad o nosocomial en el Hospital Guillermo Grant Benavente de Concepción Chile, Rev. Chilena Infectol. 31 (2014) 298–304.
[60] A. Bereciartu, K. Bok, J. Gómez, Identification of viral agents causing gastroenteritis among children in Buenos Aires, Argentina, J. Clin. Virol. 25 (2002) 197–203, http://dx.doi.org/10.1016/S1386-6532(02)00010-0.
[61] P.P. Yori, K. Schwab, R.H. Gilman, S. Nappier, D.V. Portocarrero, R.E. Black, et al., Norovirus highly prevalent cause of endemic acute diarrhea in children in the peruvian amazon, Pediatr. Infect. Dis. J. 28 (2009) 844–847, http://dx.doi.org/10.1097/INF.0b013e3181a24730.
[62] F. Bucardo, Y. Reyes, L. Svensson, J. Nordgren, Predominance of norovirus and sapovirus in Nicaragua after implementation of universal rotavirus vaccination, PLoS One. 9 (2014) e98201, http://dx.doi.org/10.1371/journal.pone.0098201.
[63] K.Y. Green, Norovirus infection in immunocompromised hosts, Clin. Microbiol. Infect. 20 (2014) 717–723, http://dx.doi.org/10.1111/1469-0691.12761.
[64] L. Rodríguez-Guillén, E. Vizzi, A.C. Alcalá, F.H. Pujol, F. Liprandi, J.E. Ludert, Calicivirus infection in human immunodeficiency virus seropositive children and adults, J. Clin. Virol. 33 (2005) 104–109, http://dx.doi.org/10.1016/j.jcv.2004.09.031.
[65] L.G.N. Lemes, T.S. Corrêa, F.S. Fiaccadori, D.D.D.D.P. Cardoso, A.D.M. Arantes, K.M.C. Souza, et al., Prospective study on norovirus infection among allogeneic stem cell transplant recipients: prolonged viral excretion and viral RNA in the blood, J. Clin. Virol. 61 (2014) 329–333, http://dx.doi.org/10.1016/j.jcv.2014.08.004.
[66] R.C. Silva, F.J. Benati, G.P.A. Pena, N. Santos, Molecular characterization of viruses associated with gastrointestinal infection in HIV-positive patients, Braz. J. Infect. Dis. 14 (2010) 549–552, http://dx.doi.org/10.1016/S1413-8670(10)70110-0.
[67] E. Robilotti, S. Deresinski, B.A. Pinsky, Norovirus, Clin. Microbiol. Rev. 28 (2015) 134–164, http://dx.doi.org/10.1128/CMR.00075-14.
[68] M.M. Patel, A.J. Hall, J. Vinje, U.D. Parashar, Noroviruses: a comprehensive review, J. Clin. Virol. 44 (2009) 1–8, http://dx.doi.org/10.1016/j.jcv.2008.10.009.
[69] B.A. Lopman, T. Trivedi, Y. Vicuna, V. Costantini, N. Collins, N. Gregoricus, et al., Norovirus infection and disease in an Ecuadorian birth cohort: association of certain norovirus genotypes with host FUT2 secretor status, J. Infect. Dis. 211 (2014) 1–9, http://dx.doi.org/10.1093/infdis/jiu672.
[70] M.M. Patel, M.A. Widdowson, R.I. Glass, K. Akazawa, J. Vinjé, U.D. Parashar, Systematic literature review of role of noroviruses in sporadic gastroenteritis, Emerging Infect. Dis. 14 (2008) 1224–1231, http://dx.doi.org/10.3201/eid1408.071114.
[71] M.E. Galeano, M. Martinez, A.A. Amarilla, G. Russomando, M.P. Miagostovich, G.I. Parra, et al., Molecular epidemiology of norovirus strains in Paraguayan children during 2004–2005: description of a possible new GII.4 cluster, J. Clin. Virol. 58 (2013) 378–384, http://dx.doi.org/10.1016/j.jcv.2013.07.008.
[72] N. Martnez, C. Espul, H. Cuello, W. Zhong, X. Jiang, D.O. Matson, Sequence diversity of human caliciviruses recovered from children with diarrhea in Mendoza, Argentina, 1995–1998, J. Med. Virol. 67 (2002) 289–298, http://dx.doi.org/10.1002/jmv.2220.
[73] J.G. Castilho, V. Munford, H.R. Resque, U. Fagundes-Neto, J. Vinjé, M.L. Rácz, Genetic diversity of norovirus among children with gastroenteritis in São Paulo State, Brazil, J. Clin. Microbiol. 44 (2006) 3947–3953, http://dx.doi.org/10.1128/JCM.00279-06.
[74] A. Estévez, W. Arvelo, A.J. Hall, M.R. López, B. López, L. Reyes, et al., Prevalence and genetic diversity of norovirus among patients with acute diarrhea in Guatemala, J. Med. Virol. 85 (2013) 1293–1298, http://dx.doi.org/10.1002/jmv.23578.
[75] G.G. González, F. Liprandi, J.E. Ludert, Molecular epidemiology of enteric viruses in children with sporadic gastroenteritis in Valencia, Venezuela, J. Med. Virol. 83 (2011) 1972–1982, http://dx.doi.org/10.1002/jmv.22185.
[76] M.S.C. Amaral, G.K. Estevam, M. Penatti, R. Lafontaine, I.C.G. Lima, P.K.P. Spada, et al., The prevalence of norovirus, astrovirus and adenovirus infections among hospitalised children with acute gastroenteritis in Porto Velho, state of Rondônia, western Brazilian Amazon, Mem. Inst. Oswaldo Cruz 110 (2015) 215–221, http://dx.doi.org/10.1590/0074-02760140381.
[77] M.S.A. Andreasi, D.D.D.D.P. Cardoso, S.M. Fernandes, I.A. Tozetti, A.M.T. Borges, F.S. Fiaccadori, et al., Adenovirus, calicivirus and astrovirus detection in fecal samples of hospitalized children with acute gastroenteritis from Campo Grande, MS Brazil, Mem. Inst. Oswaldo Cruz 103 (2008) 741–744, http://dx.doi.org/10.1590/S0074-02762008000700020.
[78] E. Robilotti, S. Deresinski, B.A. Pinsky, Norovirus, Clin. Microbiol. Rev. 28 (2015) 134–164, http://dx.doi.org/10.1128/CMR.00075-14.
[79] M. Saito, S. Goel-Apaza, S. Espetia, D. Velasquez, L. Cabrera, S. Loli, et al., Multiple norovirus infections in a birth cohort in a peruvian periurban community, Clin. Infect. Dis. 58 (2014) 483–491, http://dx.doi.org/10.1093/cid/cit763.
[80] C. García, H.L. DuPont, K.Z. Long, J.I. Santos, G. Ko, Asymptomatic norovirus infection in Mexican children, J. Clin. Microbiol. 44 (2006) 2997–3000, http://dx.doi.org/10.1128/JCM.00065-06.
[81] F. Bucardo, J. Nordgren, B. Carlsson, E. Kindberg, M. Paniagua, R. Möllby, et al., Asymptomatic norovirus infections in Nicaraguan children and its association with viral properties and histo-blood group antigens, Pediatr.

Infect. Dis. J. 29 (2010) 934–939, http://dx.doi.org/10.1097/INF.0b013e3181ed9f2f.
[82] M.L. O'Ryan, Y. Lucero, V. Prado, M.E. Santolaya, M. Rabello, Y. Solis, et al., Symptomatic and asymptomatic rotavirus and norovirus infections during infancy in a Chilean birth cohort, Pediatr. Infect. Dis. J. 28 (2009) 879–884, http://dx.doi.org/10.1097/INF.0b013e3181a4bb60.
[83] T. Farkas, X. Jiang, M.L. Guerrero, W. Zhong, N. Wilton, T. Berke, et al., Prevalence and genetic diversity of human caliciviruses (HuCVs) in Mexican children, J. Med. Virol. 62 (2000) 217–223, 10.1002/1096-9071(200010)62:2<217:AID-JMV13>3.0.CO;2-F[pii].
[84] G. Solano-Aguilar, K.P. Fernandez, H. Ets, A. Molokin, B. Vinyard, J.F. Urban, et al., Characterization of fecal microbiota of children with diarrhea in 2 locations in Colombia, J. Pediatr. Gastroenterol. Nutr. 56 (2013) 503–511, http://dx.doi.org/10.1097/MPG.0b013e318282aa12.
[85] M.D.B. Fernández, C. Torres, L.C. Martínez, M.O. Giordano, G. Masachessi, P. a. Barril, et al., Genetic and evolutionary characterization of norovirus from sewage and surface waters in Córdoba City, Argentina, Infect. Genet. Evol. 11 (2011) 1631–1637, http://dx.doi.org/10.1016/j.meegid.2011.06.005.
[86] F. Bucardo, P.E. Lindgren, L. Svensson, J. Nordgren, Low prevalence of rotavirus and high prevalence of norovirus in hospital and community wastewater after introduction of rotavirus vaccine in Nicaragua, PLoS One 6 (2011) e25962, http://dx.doi.org/10.1371/journal.pone.0025962.
[87] M. Victoria, L.F.L. Tort, M. García, A. Lizasoain, L. Maya, J.P.G. Leite, et al., Assessment of gastroenteric viruses from wastewater directly discharged into Uruguay river, Uruguay, Food Environ. Virol. 6 (2014) 116–124, http://dx.doi.org/10.1007/s12560-014-9143-7.
[88] S.M. Ahmed, B.A. Lopman, K. Levy, A systematic review and meta-analysis of the global seasonality of norovirus, PLoS One. 8 (2013) e75922, http://dx.doi.org/10.1371/journal.pone.0075922.
[89] F.P. Rivera, T.J. Ochoa, J. Ruiz, A.M. Medina, L. Ecker, E. Mercado, et al., Norovirus prevalence in pathogen negative gastroenteritis in children from periurban areas in Lima, Peru, Trans. R. Soc. Trop. Med. Hyg. 105 (2011) 734–736, http://dx.doi.org/10.1016/j.trstmh.2011.08.009.
[90] C.C. Soares, N. Santos, R.S. Beard, M.C.M. Albuquerque, A.G. Maranhão, L.N. Rocha, et al., Norovirus detection and genotyping for children with gastroenteritis, Brazil, Emerging Infect. Dis. 13 (2007) 1244–1246, http://dx.doi.org/10.3201/eid1308.070300.
[91] A.C.C. Sá, M.M. Gómez, I.F.N. Lima, J.S. Quetz, A. Havt, R.B. Oriá, et al., Group a rotavirus and norovirus genotypes circulating in the northeastern Brazil in the post-monovalent vaccination era, J. Med. Virol. 87 (2015) 1480–1490, http://dx.doi.org/10.1002/jmv.24144.
[92] M.S.R. Ferreira, R.D.C.C. Garcia, M.D.P.T.P. Xavier, R.L. Ribeiro, R.M. Assis, M.D.C.M. Mota, et al., Genotyping of gastroenteric viruses in hospitalised children: first report of norovirus GII.21 in Brazil, Mem. Inst. Oswaldo Cruz 107 (2012) 1064–1067, http://dx.doi.org/10.1590/S0074-02762012000800017.
[93] E.B. Steinberg, C.E. Mendoza, R. Glass, B. Arana, M.B. Lopez, M. Mejia, et al., Prevalence of infection with waterborne pathogens: a seroepidemiologic study in children 6–36 months old in San Juan Sacatepequez Guatemala, Am. J. Trop. Med. Hyg. 70 (2004) 83–88.
[94] S.M. Raboni, G.A.C. Damasio, C.E.O. Ferreira, L.A. Pereira, M.B. Nogueira, L.R. Vidal, et al., Acute gastroenteritis and enteric viruses in hospitalised children in southern Brazil: aetiology, seasonality and clinical outcomes, Mem. Inst. Oswaldo Cruz 109 (2014) 428–435, http://dx.doi.org/10.1590/0074-0276140066.
[95] M.L. O'Ryan, A. Peña, R. Vergara, J. Díaz, N. Mamani, H. Cortés, et al., Prospective characterization of norovirus compared with rotavirus acute diarrhea episodes in chilean children, Pediatr. Infect. Dis. J. 29 (2010) 855–859, http://dx.doi.org/10.1097/INF.0b013e3181e8b346.
[96] H.R. Poma, D.G. Cacciabue, B. Garcé, E.E. Gonzo, V.B. Rajal, Towards a rational strategy for monitoring of microbiological quality of ambient waters, Sci. Total Environ. 433 (2012) 98–109, http://dx.doi.org/10.1016/j.scitotenv.2012.06.019.
[97] J.S. Eden, J. Hewitt, K.L. Lim, M.F. Boni, J. Merif, G. Greening, et al., The emergence and evolution of the novel epidemic norovirus GII.4 variant Sydney 2012, Virology 450-451 (2014) 106–113, http://dx.doi.org/10.1016/j.virol.2013.12.005.
[98] S. Georgiadis, D.A. Pilger, F. Pereira, V. Cantarelli, Avaliação molecular de norovírus em pacientes com gastroenterite aguda, Rev. Soc. Bras. Med. Trop. 43 (2010) 277–280.
[99] F. Gómez-Santiago, R.M. Ribas-Aparicio, H. García-Lozano, Molecular characterization of human calicivirus associated with acute diarrheal disease in mexican children, Virol. J. 9 (2012) 1–9, http://dx.doi.org/10.1186/1743-422X-9-54.
[100] U.D. Parashar, J.F. Li, R. Cama, M. DeZalia, S.S. Monroe, D.N. Taylor, et al., Human caliciviruses as a cause of severe gastroenteritis in Peruvian children, J. Infect. Dis. 190 (2004) 1088–1092, http://dx.doi.org/10.1086/423324.
[101] J.A.M. Siqueira, A. da, C. Linhares, M. dos, S. Gonçalves, T.C.N. de Carvalho, M.C.A. Justino, J.D.P. Mascarenhas, et al., Group A rotavirus and norovirus display sharply distinct seasonal profiles in Belém, northern Brazil, Mem. Inst. Oswaldo Cruz 108 (2013) 661–664, http://dx.doi.org/10.1590/0074-0276108052013020.
[102] F. Bucardo, J. Nordgren, B. Carlsson, M. Paniagua, P.E. Lindgren, F. Espinoza, et al., Pediatric norovirus diarrhea in Nicaragua, J. Clin. Microbiol. 46 (2008) 2573–2580, http://dx.doi.org/10.1128/JCM.00505-08.
[103] G.C. Aragão, J.D.A.P. Mascarenhas, J.H.L. Kaiano, M.S.S. de Lucena, J.A.M. Siqueira, T.M. Fumian, et al., Norovirus diversity in diarrheic children from an african-descendant settlement in Belém, northern Brazil, PLoS One 8 e56608 (2013), http://dx.doi.org/10.1371/journal.pone.0056608.
[104] C.B. Vieira, A. de Abreu Corrêa, M.S. de Jesus, S.L. Luz, P. Wyn-Jones, D. Kay, et al., Viruses surveillance under different season scenarios of the Negro River Basin, Amazonia, Brazil, Food Environ Virol. 8 (2016) 57–69, http://dx.doi.org/10.1007/s12560-016-9226-8.
[105] J. Hewitt, M. Leonard, G.E. Greening, G.D. Lewis, Influence of wastewater treatment process and the population size on human virus profiles in wastewater, Water Res. 45 (2011) 6267–6276, http://dx.doi.org/10.1016/j.watres.2011.09.029.
[106] A.H. Talal, C.L. Moe, A.A.M. Lima, K.A. Weigle, L. Barrett, S.I. Bangdiwala, et al., Seroprevalence and seroincidence of Norwalk-like virus infection among brazilian infants and children, J. Med. Virol. 61 (2000) 117–124, 10.1002/(SICI)1096-9071(200005)61:1<117:AID-JMV19>3.0.CO;2-N.
[107] F. Bucardo, E. Kindberg, M. Paniagua, M. Vildevall, L. Svensson, Genetic susceptibility to symptomatic norovirus infection in Nicaragua, J. Med. Virol. 81 (2009) 728–735, http://dx.doi.org/10.1002/jmv.21426.
[108] F. Vicentini, W. Denadai, Y.M. Gomes, T.L. Rose, M.S.R. Ferreira, B. Le Moullac-Vaidye, et al., Molecular characterization of noroviruses and HBGA from infected quilombola children in Espirito Santo state, Brazil, PLoS One. 8 (2013) e69348, http://dx.doi.org/10.1371/journal.pone.0069348.
[109] M.M. Patel, D. Steele, J.R. Gentsch, J. Wecker, R.I. Glass, U.D. Parashar, Real-world impact of rotavirus vaccination, Pediatr. Infect. Dis. J. 30 (2011) 1–5, http://dx.doi.org/10.1097/INF.0b013e3181fefa1f.
[110] H.L. Koo, F.H. Neill, M.K. Estes, F.M. Munoz, A. Cameron, H.L. DuPont, et al., Noroviruses: the most common pediatric viral enteric pathogen at a large university hospital after introduction of rotavirus vaccination, J. Pediatr. Infect. Dis. Soc. 2 (2013) 57–60, http://dx.doi.org/10.1093/jpids/pis070.
[111] D.C. Payne, J. Vinjé, P.G. Szilagyi, K.M. Edwards, M.A. Staat, G.A. Weinberg, et al., Norovirus and medically attended gastroenteritis in U.S children, N. Engl. J. Med. 368 (2013) 1121–1130, http://dx.doi.org/10.1056/NEJMsa1206589.
[112] M. Hemming, S. Räsänen, L. Huhti, M. Paloniemi, M. Salminen, T. Vesikari, Major reduction of rotavirus, but not norovirus, gastroenteritis in children seen in hospital after the introduction of RotaTeq vaccine into the National Immunization Programme in Finland, Eur J. Pediatr. 172 (2013) 739–746, http://dx.doi.org/10.1007/s00431-013-1945-3.
[113] M.L. O'Ryan, N. Mamani, A. Gaggero, L.F. Avendaño, S. Prieto, A. Peña, et al., Human caliciviruses are a significant pathogen of acute sporadic diarrhea in children of Santiago, Chile, J. Infect. Dis. 182 (2000) 1519–1522, http://dx.doi.org/10.1086/315874.
[114] A. Stals, E. Mathijs, L. Baert, N. Botteldoorn, S. Denayer, A. Mauroy, et al., Molecular detection and genotyping of noroviruses, Food Environ. Virol. 4 (2012) 153–167.
[115] D.M. Teixeira, J.M. Hernandez, L.D. Silva, D. de, S. Oliveira, P.K. de, P. Spada, T.C.M. Gurjão, et al., Occurrence of norovirus GIV in environmental water samples from belém city, amazon region, Brazil, Food Environ. Virol. (2015), http://dx.doi.org/10.1007/s12560-015-9220-6.
[116] J.M. Fioretti, M.S.R. Ferreira, M. Victoria, C.B. Vieira, M.D.P.T.P. Xavier, J.P.G. Leite, et al., Genetic diversity of noroviruses in Brazil, Mem. Inst. Oswaldo Cruz 106 (2011) 942–947, http://dx.doi.org/10.1590/S0074-02762011000800008.
[117] A. Cilli, A. Luchs, S.G. Morillo, F.F. Costa, R.D.C.C. Carmona, M.D.C.S.T. Timenetsky, Characterization of rotavirus and norovirus strains: a 6-year study (2004–2009), J. Pediatr. (Rio. J) 87 (2011) 445–449, http://dx.doi.org/10.2223/JPED.2122.
[118] G.S. Campos, V.H. Moreau, A. Bandeira, G. Barberino, P.F. Almeida, D.M. Amador, et al., Molecular detection and genetic diversity of norovirus in hospitalized young adults with acute gastroenteritis in Bahia, Brazil, Arch. Virol. 153 (2008) 1125–1129, http://dx.doi.org/10.1007/s00705-008-0078-x.
[119] M.S.R. Ferreira, M.P.T.P. Xavier, T.M. Fumian, M. Victoria, S.A. Oliveira, L.H.A. Pena, et al., Acute gastroenteritis cases associated with noroviruses infection in the state of Rio de Janeiro, J. Med. Virol. 80 (2008) 338–344, http://dx.doi.org/10.1002/jmv.21059.
[121] M. Victoria, F.R. Guimaraes, T.M. Fumian, F.F. Ferreira, C.B. Vieira, T. Shubo, et al., One year monitoring of norovirus in a sewage treatment plant in Rio de Janeiro, Brazil, J. Water Health 8 (2010) 158–165, http://dx.doi.org/10.2166/wh.2009.012.
[122] M. Victoria, C. Rigotto, V. Moresco, A. de, A. Corrêa, C. Kolesnikovas, J.P.G. Leite, et al., Assessment of norovirus contamination in environmental samples from Florianópolis City, Southern Brazil, J. Appl. Microbiol. 109 (2009) 231–238, http://dx.doi.org/10.1111/j.1365-2672.2009.04646.x.
[123] R. Vidal, P. Roessler, V. Solari, J. Vollaire, X. Jiang, D.O. Matson, et al., Novel recombinant norovirus causing outbreaks of gastroenteritis in Santiago, Chile, J. Clin. Microbiol. 44 (2006) 2271–2275, http://dx.doi.org/10.1128/JCM.01890-05.
[124] C.I. Gallimore, M.A. Barreiros, D.W. Brown, J.P. Nascimento, J.P. Leite, Noroviruses associated with acute gastroenteritis in a children's day care facility in Rio de Janeiro, Brazil, Braz. J. Med. Biol. Res. 37 (2004) 321–326.
[125] S.G. Morillo, A. Luchs, A. Cilli, M. do C.S.T. Timenetsky, Rapid detection of norovirus in naturally contaminated food: foodborne gastroenteritis outbreak on a cruise ship in Brazil 2010, Food Environ. Virol. 4 (2012) 124–129, http://dx.doi.org/10.1007/s12560-012-9085-x.

[126] M.P.T.P. Xavier, S.A. Oliveira, M.S.R. Ferreira, M. Victoria, V. Miranda, M.F.M. Silva, et al., Detection of caliciviruses associated with acute infantile gastroenteritis in Salvador, an urban center in northeast Brazil, Brazilian, J. Med. Biol. Res. 42 (2009) 438–444, 10.1590/S0100-879 × 2009000500007.
[127] M. de Graaf, J. van Beek, H. Vennema, A.T. Podkolzin, J. Hewitt, F. Bucardo, et al., Emergence of a novel GII.17 norovirus –End of the GII.4 era? Euro Surveill Bull. 20 (2015).
[128] R.A. Bull, M.M. Tanaka, P.A. White, Norovirus recombination, J. Gen. Virol. 88 (2007) 3347–3359, http://dx.doi.org/10.1099/vir.0.83321-0.
[129] E. Mathijs, B. Muylkens, A. Mauroy, D. Ziant, T. Delwiche, E. Thiry, Experimental evidence of recombination in murine noroviruses, J. Gen. Virol. 91 (2010) 2723–2733.
[130] Á. Fajardo, F.L. Tort, M. Victoria, T.M. Fumian, M.P. Miagostovich, J.P.G. Leite, et al., Phylogenetic analyses of norovirus strains detected in Uruguay reveal the circulation of the novel GII.P7/GII.6 recombinant variant, Infect. Genet. Evol. 28 (2014) 328–332, http://dx.doi.org/10.1016/j.meegid.2014.10.026.
[131] T.M. Fumian, G.C. Aragão, J.D.A.P. Mascarenhas, J.H. Kaiano, J.A.M. Siqueira, L.S. Soares, et al., Detection of a novel recombinant strain of norovirus in an african-descendant community from the Amazon region of Brazil in 2008, Arch. Virol. 157 (2012) 2389–2392, http://dx.doi.org/10.1007/s00705-012-1428-2.