

Risk Assessment of Enteric Virus in the Food Supply Chain: A Critical Review

Mohamed Shaheen NF*

Environmental Virology Laboratory, National Research Centre, Egypt

***Corresponding author:** Mohamed Nasr Fathi Shaheen, Environmental Virology Laboratory, Water Pollution Research Department, Environmental Research Division, National Research Centre, Al-Buhouth Street, Dokki, Giza, Egypt, Email: m_nrc2007@yahoo.com

Review Article

Volume 3 Issue 1

Received Date: March 19, 2019

Published Date: March 29, 2019

DOI: 10.23880/vij-16000206

Abstract

Human enteric viruses are one of the most common causes of foodborne diseases in both developed and developing countries. While human hepatitis A virus (HAV) and noroviruses (NoV) lead to the vast majority of outbreaks and diseases, there are handful of Viral enteropathogens that contribute to sporadic outbreaks worldwide including sapovirus, astrovirus, rotavirus, Aichi virus, and enterovirus. In addition, hepatitis E virus is increasingly being identified as an emerging health threat within the food supply. HAV and NoV are highly infectious and may cause widespread outbreaks. The clinical manifestation of infection with NoV, however, is relatively mild. Viruses cannot grow on or in foods but they may reach fresh produce via faecal contamination. This contamination can arise during growth and harvesting region from contact with contaminated water and untreated or inadequately sewage sludge used for fertilization and irrigation. Alternatively, vegetables or fruits handled by an infected individual might become polluted with virus and transmit viral infection. The most frequently documented foodborne viral illness is hepatitis A and viral gastroenteritis: both have been linked to the consumption of vegetables and /or fresh fruit.

Keywords: Enterovirus; Foodborne; Genogroups

Abbreviations: HAV: Hepatitis A Virus; NoV: Noroviruses; WHO: World Health Organization; HEV: Hepatitis E Virus.

Introduction

Food safety has important implications for economies, food security, and public health. Globally, foodborne disease is a major cause of mortality and deaths, it is estimated that about 600 million illnesses and 42,000 deaths are reported due to foodborne disease annually

[1]. Foodborne illness can result in long-term health effects, such as reactive arthritis, irritable bowel syndrome, diabetes, kidney disease, hypertension, and neurological dysfunction [2-4].

Viruses are increasingly identified as significant causes of foodborne illness worldwide in recent years. Viruses are quite different than bacterial pathogens, consisting of nucleic acids enclosed in a protein shell or coat called a capsid. Viruses are not free-living and are unable to replicate in food but only replicate within the

living cells of animals, plants, bacteria, or humans. Most foodborne viruses are extremely stable in the environment as resistant to many common food-processing interventions due to the lack of viral envelopes [5]. Foodborne viruses may be able to survive on hands, in foods, on food contact surfaces, in feces, and on floors for long periods of time [6]. Bacteria was the predominant causative agents of foodborne diseases in previous decades but now it is well known that enteric viruses are one of the main causative pathogens for foodborne disease, resulting in 45% and 13.1% of foodborne outbreaks in the United States and the European Union (EU), respectively [7,8].

Foodborne viral infections are commonly caused by human enteric viruses and these viral infections are spread via virus particles excreted in the vomit or stool of infected peoples. Hepatitis A virus (HAV) and Norovirus (NoV) are the two most well-known foodborne viruses in the United States [9]. Hepatitis E virus (HEV), rotaviruses, sapoviruses, astroviruses, and Aichi viruses are less common causes associated with outbreaks of foodborne illness [7,10-13]. NoV is commonly considered to be the most prevalent cause of viral-associated foodborne gastroenteritis, worldwide [7]. NoV is linked to an estimated 19-21 million cases of acute gastroenteritis annually in the United States [7]. World Health Organization (WHO) estimated that in 2010 NoV caused 677 million illnesses worldwide alone; in addition, NoV was also responsible for the majority of deaths (213 515) as well as the highest occurrence in children less than 5 years of age [14]. The majority of foodborne viruses are recognized enteric due to their fecal-oral mode of transmission. Human enteric viruses are commonly non-enveloped viruses and are stable against environmental conditions.

Diseases due to these viruses are commonly the result of preparing or consuming food with fecal-contaminated water. While these aforementioned viral pathogens are identified to be significant causes of gastroenteritis illness, their role as foodborne causative agents is still being identified as <1% of those cases are often directly linked to food products [15-17]. Outbreaks caused by enteric viruses are most commonly linked to the consumption of ready-to-eat foods, raw oysters, and fresh produce [18]. In the US alone, fresh produce can be further classified into groups such as fruits and leafy greens, which have been cited as responsible for 21% and 30% of NoV outbreaks, respectively [19].

Foods may become contaminated with enteric viruses through direct contact with contaminated water sources

or by a person handling food. While outbreaks linked to shellfish are most often associated with fecal-contaminated water, research shows outbreaks linked to ready-to-eat foods and fresh produce usually caused by a person handling food [18]. It is notable to state that foods could be contaminated with microbes at any point from pre- to postharvest and the transmission routes often remain unidentified [18]. Generally, human enteric viruses can be transmitted via the faecal-oral route and they have a low infectious dose and are excreted in the feces of infected individuals in large numbers. This can commonly lead to prolonged outbreaks due to both asymptomatic and symptomatic carriers. In this review, we will focus on the viruses causing most foodborne viral disease, NoV and HAV, as well the emerging foodborne viral hazard, HEV.

Norovirus

Noroviruses, belonging to the family Caliciviridae, are non-enveloped viruses. These viruses are very small particles (28–30 nm) with single-stranded and positive sense (+) RNA genomes, containing three open reading frames [20]. The major presentation of the disease is diarrhea and/or vomiting which is generally self-limiting lasting up to 3 days. NoV has a low infectious dose, and infectious viral particles are excreted in very high amounts from infected individual (109 virions/gram) in stool for prolonged periods ranged from 2 to 4 weeks after infection signs have subsided leading to asymptomatic carriers [21]. Currently, there is identified 7 genogroups (G) of norovirus of which only three genotypes (GI, GII and GIV) infect humans. NoV GI and GII are the primary genogroups which cause diarrheal infection in humans, since GII.4 cluster is the most prevalent of foodborne pathogen [22].

According to present estimates, NoV is the major cause of foodborne disease and responsible for 58% of cases domestically, worldwide [7,23]. Outbreaks commonly occur in cruise ships, longterm care facilities, schools, and catered events. While both NoV GI and GII hNoV have been reported in many outbreak scenarios, there are some apparent trends in source attribution that are group-specific. Matthews et al. [24] reported that 66% of foodborne illness were caused by NoV GII, 13% were due to GI infection, and 20% due to multiple infections (GI/GII) between 1993 and 2011. Moreover, it is reported that GII.4 is the predominant NoV in all outbreaks associated with person-to-person transmission whereas NoV GI.2 and GI.4 genogroups are the most prevalent GI in foodborne disease outbreaks [25].

Hepatitis A Virus

Hepatitis A virus, belonging to the family Picornaviridae, is a small round virus of size ranged from 32 to 37 nm with a positive single stranded RNA. The genome of HAV consists of one an open reading frame (ORF) which is classified into 3 parts that encode for capsid proteins as well as other nonstructural and structural components [7]. There is a total of 7 genogroups (GI-GVII) of which four genogroups (GI, GII, GIII and GVII) infect humans. The incubation period of HAV infection can be from 2-6 weeks with signs presenting in four phases. Unlike most human enteric viruses, the illness related to Hepatitis A also affects the liver [7]. More specifically, early symptoms of infection include vomiting, anorexia, and nausea with general fatigue followed by jaundice presenting 1–14 days after infection with the virus. The disease can last from 2-5 weeks and is typically milder symptoms in children than adults [26].

Globally, there are an estimated 200 million asymptomatic shedders and 1.4 million cases annually [27]. The prevalence of HAV in developed countries is low though there can be large susceptible populations in nonendemic regions where immunization participation is low. Sources of HAV infection within developed countries are commonly from fresh produce (e.g. soft red fruits and green onions) and bivalve mollusks (e.g. clams, oysters, and mussels) imported from areas with poor hygiene conditions or from travel to similar regions [28]. Actual foodborne cases are about 5% of total HAV infections with 41% being travel-related [29]. Globally, in 2010, the WHO reported that 90 000 people died due to hepatitis A illness with 30,000 of those deaths directly occurred due to contaminated food with to HAV [27].

Hepatitis E Virus

Hepatitis E, belonging to the Hepeviridae family, consisting of positive sense (+) and single-stranded RNA genome with a virion of size ranged from 27 to 34 nm [5]. Hepatitis E cannot be distinguished clinically from other forms of viral hepatitis, causing inflammation of the liver tissue leading to sever jaundice syndrome [5]. Hepatitis E virus has 4 genogroups (HEV A–D); however, only group HEV A infects humans, which is further classified into genotypes 1 through 4 are more often in developing areas whereas types 3 and 4 are observed more in developed areas —the latter have been related to zoonotic transmission [30]. The incubation period of HEV is 2 weeks to 2 months with illness lasting anywhere from 4-6 weeks. Although HEV infection is usually a self-

limiting, it poses a high risk to pregnant women and their fetuses where the death rates can be up to 10% [31].

In HEV-endemic regions (Asia and Africa), there are an estimated 20 million cases of HEV leading to 3.4 million symptomatic cases and 70 000 deaths [32]. Similar detection rates of infection are not documented in developed areas; however, HEV is currently identified as an emerging foodborne virus [30]. Similar to other enteric viruses, filter-feeding bivalve molluscan shellfish produced in regions susceptible to pollution with untreated animal or human sewage may present a risk for HEV transmission through this route has not been approved. Also of concern is the contamination of certain crops through polluted irrigation water with untreated sewage water in HEV-endemic regions—again, this route has not been directly substantiated although HEV genome has been found in irrigation water [33]. King, et al. [30] stated that there are only five studies that provided molecular and epidemiological evidence for the foodborne transmission of hepatitis E virus to humans. All of the reported cases were related to wild boar meat, wild venison meat, or other pork products and documented in either Japan or the EU between 2003 and 2017. Also, HEV outbreak was documented in 2014 in Australia due to locally acquired HEV infections linked to consumption of contaminated pork products [34].

References

1. SHavelaar AH, Kirk MD, Torgerson PR, Gibb HJ, Hald T, et al. (2015) World Health Organization global estimates and regional comparisons of the burden of foodborne disease in 2010. *PLoS Med* 12(12): e1001923.
2. Batz MB, Henke E, Kowalczyk B (2013) Long-term consequences of foodborne infections. *Infect Dis Clin N Am* 27(3): 599-616.
3. Porter CK, Tribble DR, Aliaga PA, Halvorson HA, Riddle MS (2008) Infectious gastroenteritis and risk of developing inflammatory bowel disease. *Gastroenterology* 135(3): 781-786.
4. Roberts T, Kowalczyk B, Buck P, Blaser MJ, Frenkel JK, et al. (2009) The long-term health outcomes of selected foodborne pathogens. The Center for Foodborne Illness Research & Prevention.
5. Yeargin T, Gibson KE (2018) Key characteristics of foods with an elevated risk for viral enteropathogen

- contamination. *Journal of applied microbiology* 126(4): 996-1010.
6. Miranda RC, Schaffner DW (2019) Virus Risk in the Food Supply Chain. *Current Opinion in Food Science* 30: 43-48.
 7. Scallan E, Hoekstra RM, Angulo FJ, Tauxe RV, Widdowson MA, et al. (2011) Foodborne illness acquired in the United States- major pathogens. *Emerg Infect Dis* 17(1): 7-15.
 8. Shukla S, Cho H, Kwon OJ, Chung SH, Kim M (2018) Prevalence and evaluation strategies for viral contamination in food products: risk to human health-a review. *Crit Rev Food Sci Nutr* 58(3): 405-419.
 9. Centers for Disease Control and Prevention (2013) Surveillance for Foodborne Disease Outbreaks United States, 2013: Annual Report. US Department of Health and Human Services, CDC, 2015 Atlanta, Georgia, US.
 10. Centers for Disease Control and Prevention (2012) Surveillance for Foodborne Disease Outbreaks United States, 2012: Annual Report. US Department of Health and Human Services, CDC, 2014, Atlanta, Georgia, US.
 11. Matussek A, Dienus O, Djeneba O, Simporé J, Nitiema L (2015) Molecular characterization and genetic susceptibility of sapovirus in children with diarrhea in Burkina Faso. *Infect Genet Evol* 32: 396-400.
 12. Pacilli M, Cortese MM, Smith S, Siston A, Samala U, et al. (2015) Outbreak of Gastroenteritis in Adults Due to Rotavirus Genotype G12P[8]. *Clin Infect Dis* 61(4): e20-25.
 13. Le Guyader FS, Le Saux J-C, Ambert-Balay K, Krol J, Serais O, et al. (2008) Aichi virus, norovirus, astrovirus, enterovirus and rotavirus all involved in clinical cases from a French oyster-related gastroenteritis outbreak. *Journal of Clinical Microbiology* 46(12): 4011-4017.
 14. Pires SM, Fischer-Walker CL, Lanata CF, Devleeschauwer B, Hall AJ, et al. (2015) Aetiology-specific estimates of the global and regional incidence and mortality of diarrhoeal diseases commonly transmitted through food. *PLoS ONE* 10(12): e0142927.
 15. Todd EC, Grieg JD (2015) Viruses of foodborne origin: a review. *Virus Adapt Treatment* 7: 25-45.
 16. Greening GE, Cannon JL (2016) Human and animal viruses in food (including taxonomy of enteric viruses). In *Viruses in Foods* ed. Goyal, S.M. and Cannon, New York, pp: 5-57.
 17. Smits SL, Koopmans MP (2017) Genomics and foodborne viral infections. In *Applied Genomics of Foodborne Pathogens* ed. Deng, X., den Bakker HC and Hendrikson, New York, pp: 145-166.
 18. Marsh Z, Shah MP, Wikswo ME, Barclay L, Kisselburgh H, et al. (2018) Epidemiology of foodborne norovirus outbreaks- United States, 2009-2015. *Food Safety* 6: 58-66.
 19. Li D, De Keuckelaere A, Uyttendaele M (2015) Fate of foodborne viruses in the "farm to fork" chain of fresh produce. *Comp Rev Food Sci Food Safety* 14(6): 755-770.
 20. Vinje J (2015) Advances in laboratory methods for detection and typing of norovirus. *J Clin Microbiol* 53(2): 373-381.
 21. Robilotti E, Deresinski S, Pinsky BA (2015) Norovirus. *Clin Microbiol Rev* 28(1): 134-164.
 22. Cannon JL, Barclay L, Collins NR, Wikswo ME, Castro CJ, et al. (2017) Genetic and Epidemiologic Trends of Norovirus Outbreaks in the United States from 2013 to 2016 Demonstrated Emergence of Novel GII.4 Recombinant Viruses. *J Clin Microbiol* 55(7): 2208-2221.
 23. Bartsch SM, Lopman BA, Ozawa S, Hall AJ, Lee BY (2016) Global economic burden of norovirus gastroenteritis. *PLoS ONE* 11(4): e0151219.
 24. Matthews J, Dickey B, Miller R, Felzer J, Dawson B, et al. (2012) The epidemiology of published norovirus outbreaks: a review of risk factors associated with attack rate and genogroup. *Epidemiol Infect* 140(7): 1161-1172.
 25. Vega E, Barclay L, Gregoricus N, Shirley SH, Lee D et al. (2013) Genotypic and epidemiologic trends of norovirus outbreaks in the United States, 2009 to 2013. *J Clin Microbiol* 52(1): 147-155.
 26. Shouval D (2014) Hepatitis A virus. In *Viral Infections of Humans: Epidemiology and Control* ed. Kaslow, R.A., Stanberry, L.R. and Le Duc, New York, pp: 417-438.

27. Kirk MD, Pires SM, Black RE, Caipo M, Crump JA, et al. (2015) World Health Organization estimates of the global and regional disease burden of 22 foodborne bacterial, protozoal, and viral diseases, 2010: a data synthesis. *PLoS Med* 12(12): e1001921.
28. Jacobsen KH (2018) Globalization and the changing epidemiology of hepatitis A virus. *Cold Spring Harb Perspect Med* 8(10): a031716. <https://www.ncbi.nlm.nih.gov/pubmed/29500305>
29. Maunula L, von Bonsdorff CH (2016) Chapter 4 - Foodborne viruses in ready-to-eat foods. In *Food Hygiene and Toxicology in Ready-to-Eat Foods* ed. Kotzekidou, San Diego, pp: 51-68.
30. King NJ, Hewitt J, Perchee-Merien AM (2018) Hiding in plain sight? It's time to investigate other possible transmission routes for hepatitis E virus (HEV) in developed countries. *Food Environ Virol* 10(3): 225-252.
31. Perez-Gracia MT, Suay-Garc B, Mateos-Lindemann ML (2017) Hepatitis E and pregnancy: current state. *Rev Med Virol* 27: e1929.
32. Rein DB, Stevens GA, Theaker J, Wittenborn JS, Wiersma ST (2012) The global burden of hepatitis E virus genotypes 1 and 2 in 2005. *Hepatology* 55(4): 988-997.
33. Kokkinos P, Kozyra I, Lazic S, Söderberg K, Vasickova P, et al. (2017) Virological quality of irrigation water in leafy green vegetables and berry fruits production chains. *Food Environ Virol* 9(1): 72-78.
34. Yapa CM, Furlong C, Rosewell A, Ward KA, Adamson S, et al. (2016) First reported outbreak of locally acquired hepatitis E virus infection in Australia. *Med J Aust* 204(7): 274.

