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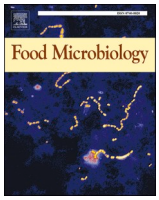

Exploring the potential of foodborne transmission of respiratory viruses

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ABSTRACT

The ongoing pandemic involving severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has raised the question whether this virus, which is known to be spread primarily though respiratory droplets, could be spread through the fecal-oral route or via contaminated food. In this article, we present a critical review of the literature exploring the potential foodborne transmission of several respiratory viruses including human coronaviruses, avian influenza virus (AVI), parainfluenza viruses, human respiratory syncytial virus, adenoviruses, rhinoviruses, and Nipah virus. Multiple lines of evidence, including documented expression of receptor proteins on gastrointestinal epithelial cells, *in vivo* viral replication in gastrointestinal epithelial cell lines, extended fecal shedding of respiratory viruses, and the ability to remain infectious in food environments for extended periods of time raises the theoretical ability of some human respiratory viruses, particularly human coronaviruses and AVI, to spread via food. However, to date, neither epidemiological data nor case reports of clear foodborne transmission of either viruses exist. Thus, foodborne transmission of human respiratory viruses remains only a theoretical possibility.

1. Introduction

Respiratory infections are the major causes of morbidity and mortality around the world (Ferkol and Schraufnagel, 2014; Forum of International Respiratory Societies, 2017; Troeger et al., 2018) and viruses are responsible for the majority of respiratory infections with the potential to cause pandemics (WHO, 2020a). The estimated annual economic burden of the common cold in the United States is over $40 billion (Fendrick et al., 2003) and for influenza is over $11.2 billion (Putri et al., 2018). To date, at least nine distinct viral families have been identified as common causative agents for respiratory tract infections in humans including, *Orthomyxoviridae*, *Paramyxoviridae, Coronaviridae*, *Pneumoviridae*, *Picornaviridae*, *Adenoviridae*, *Parvoviridae*, and *Circoviridae* (Moriyama et al., 2020). The main transmission route for respiratory viruses is by contaminated respiratory droplets (>10 μm) that people sneeze, cough, or exhale during conversation (Dhand and Li, 2020; Jones and Brosseau, 2015; Stadnytskyi et al., 2020). These droplets travel short distances (1–2 m) before settlings on surfaces, where viruses can remain infectious for hours to days, depending on the virion structure, as well as environmental factors such as temperature, humidity, pH, and exposure to ultraviolet light (La Rosa et al., 2013). Generally, it has been shown that enveloped viruses are less stable on inanimate surfaces and more sensitive to heat and drying than non-enveloped viruses (Firquet et al., 2015). The human nose can effectively filter large particles, however, the oropharynx is not as effective a filter as the nose, and thus mouth breathing increases the dose of respiratory particles to the lung compared with nose breathing (Dhand and Li, 2020).

On the other hand, multiple human viruses are easily spread via the consumption of contaminated food including noroviruses, hepatitis A virus, hepatitis E virus, rotaviruses, poliovirus, sapovirus, and astroviruses (Bosch et al., 2018). Viruses are unable to multiply outside of a living host; therefore, foodborne viruses must have the innate ability to maintain viability despite the stresses associated with the food environment. Stresses in food systems vary widely, but can include solar irradiation, desiccation, freezing, cooking, enzymes or unfavorable chemicals such as acids or surfactants (Li et al., 2021). It is worth noting that common foodborne viruses are non-enveloped, which are acknowledged to have a better ability to retain viability in the environment than enveloped viruses (Firquet et al., 2015). Foodborne viruses also commonly have fecal-oral transmission pathways, with the primary infection occurring in the human gastrointestinal (GI) tract. While much is unknown regarding the specific receptors for enteric

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viruses, interactions with carbohydrate ligands have been shown to be critical for their entry (Farkas et al., 2019). For example, depending on the human norovirus strain, histo-blood group antigen (HGBA), heparin sulfate or sialic acid have been identified in viral binding to the intestinal epithelial cells (Bartnicki et al., 2017). Moreover, a wealth of evidence indicate that these viruses interact with polysaccharides of commensal bacteria to facilitate infection (Karst, 2016).

Interestingly, the receptors for cell entry of respiratory viruses can also be expressed by the epithelial cells of the GI tract. For example, angiotensin-converting enzyme 2 (ACE2), which is the main receptor for SARS-CoV-1 and SARS-CoV-2, is abundantly expressed in the lung and upper respiratory epithelia, as well as in the duodenum and small intestine, with lower levels in stomach and large intestine (Hikmet et al., 2020). Although the mere expression of viral receptors in the GI system does not mean these cells are permissive to respiratory virus infection.

There is growing evidence that specific enveloped respiratory viruses can endure low pH, enzymes, and bile in the upper GI tract and replicate in the intestinal epithelium (Bertran and Swayne, 2014; de Wit et al., 2014; Zhou et al., 2017). The potential deleterious effect of low pH and digestive enzymes on viruses can also be mitigated if viruses are swallowed with water or food (Han et al., 2019). Food and water can also decrease the activity of pepsin, which could otherwise degrade virus particles (Witkowski et al., 2017). In this case, food contaminated with these respiratory viruses, either by zoonotic viruses from animal sources, or by contamination with respiratory droplets from infected food-handlers, has the potential to be a vehicle for viral transmission. The former mechanism could pose a health risk to individuals who encounter susceptible and infected animals in settings such as farms or slaughterhouses as these individuals are at higher exposure risk to contaminated saliva, feces, and respiratory droplets (Munnink et al., 2020). Both mechanisms could be of particular concern for foods that are consumed raw or undercooked such as fruits and vegetables. In epidemic and pandemic situations, a proportion of food-handlers could be carriers of infectious viruses regardless of being asymptomatic. Thus, assumptions could be made that foods that are prepared by infected food-handlers could become contaminated as well. Data suggests that some respiratory viruses, such as human coronaviruses and influenza viruses can persist in foods for days and weeks (Li et al., 2021). Herein, we will briefly discuss viral families (Table 1), which are known to cause respiratory illness in humans, but also have the potential to be transmitted through contaminated food, and we present a critical review of the evidence to date on possible foodborne transmission of these respiratory viruses.

2. Human coronaviruses

Human coronaviruses are the second most prevalent cause of the common cold in humans (Kingsley, 2016). They belong to the *Coronaviridae* family, which is further divided into four genera; alpha, beta, gamma, and delta. To date, only alpha and beta coronaviruses are found to infect humans. Their genome consists of a single-stranded RNA with positive polarity and is about 30 kb in size, which is the largest known RNA genome (Brian and Baric, 2005). The genome is packaged inside a helical capsid formed by the nucleocapsid protein (N), which is further surrounded by an envelope. Coronaviruses have at least three structural proteins: The membrane protein (M) and the envelope protein (E),

Table 1
Characteristics of human respiratory viruses.

Virus Name	Genus	Strains/ Species	Genome Characteristics	Virion Structure	Alternative Hosts	References
Human Coronaviruses	Alphacoronavirus Betacoronavirus	HCoV-229E, HCoV-NL63, SARS-CoV-1, SARS-CoV-2, HCoV-OC43, MERS-CoV	+ssRNA ~26–32 kb	Enveloped Helical symmetry 120–160 nm diameter	Bats, cattle, pigs, civets, dromedary camels	Lim et al. (2016) Cui et al. (2019) (ICTV), 2020)
Highly Pathogenic Asian Avian Influenza (HPAI)	Alphainfluenzavirus	H5N1 H9N2	Segmented -ssRNA ~13.5 kb	Enveloped Helical symmetry 80–120 nm diameter	Poultry, wild birds	Peiris et al. (2007) CDC (2015) Sangsiriwut et al. (2018) (ICTV), 2020)
Human Parainfluenza Virus (HPIV)	Respirovirus Rubulavirus	HPIV-1, HPIV-3 HPIV-2, HPIV-4	-ssRNA ~15 kb	Enveloped Helical symmetry 150–300 nm diameter	Hamsters, guinea pigs, Ferrets, non-human primates	GC (2011) Henrickson (2003) ICTV (2020)
Human Respiratory Syncytial Virus (HRSV)	Orthopneumovirus	RSV	-ssRNA ~15 kb	Enveloped Helical symmetry 80-140 diameter, Filamentous form 70–190 diameter, up to 2 μm in length	Chimpanzees	Lee et al. (2012) Walsh and Hall (2015) ICTV (2020)
Human Adenoviruses (HAdVs)	Mastadenovirus	HAdV A-G	dsDNA ~36 kb	Non-enveloped Icosahedral symmetry 70-90 diameter	Non-human primates	Bots and Hoeben (2020) Saha et al. (2014) ICTV (2020)
Human Rhinoviruses (HRVs)	Enterovirus	HRV A-C	+ssRNA ~7.2 kb	Non-enveloped Icosahedral symmetry 27 nm	None	Jacobs et al. (2013) ICTV (2020)
Nipah Virus (NiV)	Henipavirus	NiV-B, NiV-M	-ssRNA ~18.2 Kb	Enveloped Pleiomorphic 40-600 nn diameter	Pigs, bats	Harcourt et al. (2000) CDC (2014) Rockx et al. (2012)

which are involved in virus assembly, whereas the spike protein (S) mediates virus entry into host cells and is the main antigenic viral protein (Li, 2016).

Severe acute respiratory syndrome (SARS), which was first reported in 2002, was caused by a coronavirus, SARS-CoV-1. In 2019, SARS-CoV-2 emerged, which is currently causing a global pandemic involving millions of people. The receptor for both viruses is angiotensin-converting enzyme 2 (ACE2) (Walls et al., 2020; Wan et al., 2020), which is highly expressed in alveolar epithelial type II cells and ciliated cells of human lungs, as well as in intestinal enterocytes (Lamers et al., 2020). Although the most common symptoms of infection for both SARS-CoV-1 and SARS-CoV-2 include fever, cough, and shortness of breath (Huang et al., 2020), about 13–50% of patients report gastrointestinal symptoms such as nausea, vomiting, and diarrhea (Cheung et al., 2020; Zhou et al., 2020). Viral RNA can be detected in patients' respiratory and stool specimens (Cha et al., 2020; Huang et al., 2020). Moreover, persistent fecal viral shedding is prominent in pediatric patients (Xu et al., 2020) and it has been shown that the virus excreted in feces is infectious in tissue culture (Xiao et al., 2020). Active SARS-CoV-2 replication in human enteroids and enterocytes has been reported (Lamers et al., 2020; Zhou et al., 2020). Furthermore, autopsy results have revealed intestinal tissue damage as a result of direct viral replication and inflammation (Ye et al., 2020). The presence of the virus in stool samples has made SARS-CoV-2 a candidate for Wastewater-Based Epidemiology (WBE), as numerous researchers around the globe have begun quantifying the SARS-CoV-2 RNA in untreated wastewater (Daughton, 2020; Venugopal et al., 2020). Moreover, intra-gastric inoculation of a mouse model expressing human ACE2 with SARS-CoV-2 has led to productive infection in upper respiratory tract and lungs (Sun et al., 2020). Collectively, these observations suggest that the gastrointestinal tract can serve as an alternative infection route for SARS viruses. Importantly, it was demonstrated that orally inoculated golden Syrian hamsters, develop infection in both respiratory and intestinal tract (Chak-Yiu Lee et al., 2020). This is an important piece of evidence demonstrating that SARS-CoV-2 is able to survive the gastrointestinal fluids and enzymes, and establish a productive infection in the intestine. Thus, this finding further supports the notion that food and waterborne transmission of SARS-CoV-2 is plausible.

Middle East respiratory syndrome coronavirus (MERS-CoV) has caused human respiratory infections with a high case fatality rate since 2012 (Assiri et al., 2013; Zaki et al., 2012). Evidence suggests that, similar to SARS coronaviruses, bats may have been the original source of MERS-CoV, and dromedary camels are the main reservoirs of the virus (Haagmans et al., 2014). Although common symptoms of MERS are fever, cough, and shortness of breath, about one third of MERS patients report gastrointestinal symptoms such as abdominal pain, nausea, vomiting, and diarrhea (Zhou et al., 2017). Importantly, MERS-CoV has been shown to be able to survive in gastrointestinal fluids, and productively replicate in primary human intestinal epithelial cells (Chafekar and Fielding, 2018; Zhou et al., 2017). The viral receptor for MERS-CoV is a transmembrane glycoprotein called dipeptidyl peptidase-4 (DPP-4), which has a wide tissue distribution in humans, including on alveolar cells of the lower respiratory tract and in the small intestine (Mackay and Arden, 2017). Although fecal-oral transmission has not been confirmed in camels, low infectious viral titre was found in camel saliva (Adney et al., 2014) and rectal swabs (Reusken et al., 2016). Moreover, MERS-CoV RNA has been detected in camel milk and it was demonstrated that the virus is stable in camel milk (Reusken et al., 2014; van Doremalen et al., 2014). Thus, it has been suggested that fecal-oral and foodborne transmission of MERS-CoV is possible.

Human coronavirus (HCoV)-229E and HCoV-OC43 have been known for more than 50 years, while HCoV-NL63 and HCoV-HKU1 were first characterised in 2004 and 2005, respectively (Mackay and Arden, 2017). These viruses cause mild upper respiratory diseases in immunocompetent hosts, although some of them can lead to severe infections in infants, young children, and elderly individuals, and are generally responsible for 10–30% of common colds (Cui et al., 2019). There is evidence that these viruses have animal origins as well. HCoV-NL63 and HCoV-229E are considered to have originated in bats, while HCoV-OC43 and HCoV-HKU1 likely originated from rodents (Forni et al., 2017). Interestingly, HCoV-NL63, which is frequently associated with croup, also uses ACE2 for entry, although it binds to a different part of the protein than SARS-coronaviruses (Perlman and Netland, 2009). Even though not common, HCoV-229E, HCoV-OC43, HCoV-NL63, and HCoV-HKU1 have been occasionally associated with symptoms of gastroenteritis, including vomiting and diarrhea in pediatric patients, which typically occur along with respiratory symptoms (Risku et al., 2010; Xiong et al., 2020). HCoV-HKU1 RNA has also been identified in stool samples of less than 2% of the patients with acute viral gastroenteritis (Esper et al., 2010).

The HCoV presence in foods is drastically understudied and the authors could not find any record on the presence of seasonal and endemic HCoVs in foods in the literature. In addition, there is no food surveillance system to investigate the presence of HCoVs in foodstuff. Recently, the finding of SARS-CoV-2 in imported frozen food commodities was reported (Klein, 2020; Roxanne Liu, August 13, 2020; Yusha, 2020), and genetic evidence was provided that would link COVID-19 resurgence in Beijing to cold-chain food contamination (Pang et al., 2020). It was also demonstrated that this virus is stable for weeks in cold storage (−80 °C to +4 °C) on artificially contaminated pork, chicken, and salmon (Fisher et al., 2020). While the infectivity of HCoV-229 on fresh produce at ambient temperature is reduced below the limit of detection 1–4 days post-inoculation (Blondin-Brosseau et al., 2020).

In addition, many of the mammal- and avian-associated coronaviruses cause severe gastroenteritis in their host species including agricultural and domestic animals such as poultry, swine, bovine, equine, canine, and feline hosts (Cimolai, 2020). Fecal-oral and foodborne modes have been shown to be the primary transmission route for porcine epidemic diarrhea virus (PEDV), and swine acute diarrhea syndrome coronavirus (SADS-CoV), which have caused fatal enteric disease outbreaks in pigs (Wang et al., 2019), as well as for equine coronavirus (ECoV) responsible for severe gastroenteritis in horses (Pusterla et al., 2018). With the accumulation of data on the gastrointestinal implications of HCoV, especially SARS-CoV-2, as well as the reported finding of trace amounts of the virus in food and environment, it is expected that there will be an increased understanding of the potential risk of foodborne transmission of these viruses in the near future.

3. Avian influenza viruses

Avian influenza viruses (AIV) are a sub-group of influenza A viruses that are readily spread between wild-birds, occasionally result in high mortality in outbreaks in domestic poultry, and can cause sporadic human infections that are severe and fatal in up to 48–60% of cases (Freidl et al., 2014; Van Kerkhove et al., 2011). Influenza A viruses (IAVs) are enveloped viruses that contain eight segments of single-stranded negative-sense RNA. IAVs, in general, spread easily from human-to-human through respiratory droplets and aerosols (Lindsley et al., 2012). AIVs, however, have little ability to spread from human-to-human (Harder et al., 2016), but can spread through zoonosis from infected birds after prolonged close contact. The exact mechanisms of bird-to-human transmission are not completely understood. AIVs which cause mild disease in susceptible birds are classified as low pathogenic AIVs (LPAIV), AIVs that cause serious disease which can result in 100% mortality in under 48 h are classified as highly pathogenic AIVs (HPAIV) (Gonzales et al., 2018). To date, 16 hemagglutinin (HA) (H1 to H16), and 9 neuraminidase (NA) (N1 to N9) subtypes of AIV have been identified. Subtypes H5 and H7 LPAIVs are of particular note because of their propensity for mutation to HPAIVs (Gonzales et al., 2018). In avian populations, fecal-oral transmission of AIV is the most important route of transmission since AIVs are excreted in feces of infected birds which is then ingested by susceptible birds via

contaminated surface water and feedings grounds (Rohani et al., 2009). In the appropriate conditions, AIV can remain infective for up to 6 months in surface waters (Keeler et al., 2014). AIV are rapidly inactivated by low pH and bile acids (Hirose et al., 2016; Scholtissek, 1985); although, evidence has shown that mucus can provide protection from inactivation in the gastrointestinal environment (Hirose et al., 2016), and the possibility of water being a diluent to lower the pH of the stomach and allow AVI to pass has also been suggested (Han et al., 2019).

The H5N1 AIV virus emerged in Asia in 1997 and spread rapidly in avian populations. Since 2003, H5N1 has been the primary cause of human AIV infections, although H9N2 is rising in prevalence (WHO, 2018). These viruses recognize receptors with saccharides terminating in sialic acid-α2,3-galactose (SAα2,3Gal), which are highly expressed in human lower respiratory tract (Paulson and de Vries, 2013). The H5N1 AIV is highly pathogenic and causes systemic infections. Infected birds can have high viral titres in various internal organs as well as in muscle tissue (Rohani et al., 2009). As a result, H5N1 viruses have caused fatal infections in domestic dogs (Songserm et al., 2006) and cats (Kuiken et al., 2004), ferrets (Bertran and Swayne, 2014), leopards (Keawcharoen et al., 2004), tigers (Hu et al., 2016; Keawcharoen et al., 2004), and lions (Chen et al., 2016) when these mammals are fed with infected poultry carcases. While felids are not affected by most IAVs, intratracheal inoculation of H5N1 AVIs can cause a respiratory infection in cats (Kuiken et al., 2004). Several well-controlled laboratory experiments have since demonstrated systemic infections, including fecal shedding of live virus, in felids after feeding on infected poultry carcasses (Kuiken et al., 2004; Rimmelzwaan et al., 2006). However, these do not rule out the generation and respiratory inoculation of viruses in feeding experiments during chewing. Subsequently, direct intragastric inoculation of H5N1 AIV in hamsters ($10^{7.1}$ to $10^{7.3}$ tissue culture infectious doses ($TCID_{50}$)), ferrets ($10^{8.5}$ to $10^{8.8}$ $TCID_{50}$), and domestic cats ($10^{7.8}$ $TCID_{50}$) resulted in fatal systemic infection including lymphoid organs and lungs (Reperant et al., 2012; Shinya et al., 2011). Spread of AVI from the intestine to other organs occurred via the vascular system, with high viral titres being found in highly vascularized organs such as the liver, kidney, and respiratory tract, which resulted in hemorrhages (Reperant et al., 2012). However, as with most studies of oral inoculation of respiratory viruses, 1 or 2 log higher viral doses are required for infection via the oral route, than for infection through respiratory exposure in permissive animals (Shinya et al., 2011); although, the exact infectious and fatal dosages also depend on viral strain. For example, a study comparing oral and intranasal inoculation of AVI in ferrets found a mean ferret lethal dose (FLD_{50}) of $>10^{9.2}$ mean egg infectious doses (EID_{50}) for the Mong/05 strain of AVI, and a FLD_{50} of $10^{8.9}$ EID_{50} for the VN/04 AVI strain (Bertran and Swayne, 2014). A dose of 10^{7} EID_{50} has been consistently shown to infect ferrets with AVI (Hinshaw et al., 1981; Zitzow et al., 2002). It is unknown to what extent this higher dosage represents an accurate, and biologically relevant, risk of oral transmission. The AIV H9N2 lineage is also able to spread via fecal-oral transmission in birds (Bertran and Swayne, 2014), and appears to also have the ability to replicate in the intestinal tract of some mammals. Additionally, H9N2 can infect mouse intestinal organoids (Huang et al., 2017), as well as human intestinal epithelial cells, including the HT-29 and Caco-2 lines, which are susceptible to infection with H9N2 AVI and show high levels of apoptosis soon after infection (Bertran and Swayne, 2014; Jahangir et al., 2010). Other non-avian IAVs have been shown to be shed in fecal material persistently, even after respiratory shedding stops (Hirose et al., 2016).

Despite the multiple lines of evidence supporting the possibility of foodborne spread of IAV to humans, there are no well-documented cases of human IAV infection from food, where respiratory exposure could be completely ruled out. Though it should be noted that fatal cases of H5N1AIV where intestinal symptoms were present but respiratory symptoms were absent, have been documented (de Jong et al., 2005). In addition, proper cooking and industrial processing of poultry meat and egg products result in complete inactivation of IAVs, lessening the potential for foodborne spread (Chmielewski and Swayne, 2011).

4. Parainfluenza viruses

Human parainfluenza virus (HPIV) belongs to the *Paramyxoviridae* family and is most commonly known to cause respiratory tract infections in children, the elderly, and those who are immunocompromised (Henrickson, 2003). . HPIV is divided into four major serotypes, HPIV-1, HPIV-2, HPIV-3, and HPIV-4, which are categorized into two genera: Respirovirus (HPIV-1 and HPIV-3) and Rubulavirus (HPIV-2 and HPIV-4) (Schaap-Nutt et al., 2012). A study conducted between 1990 and 2004 demonstrated that HPIV-3 was the most prevalent strain (52%), followed by HPIV-1 (26%), HPIV-2 (12%), and HPIV-4 (2%) among 40,630 reported HPIV-positive test results (Fry et al., 2006). Following the discovery of HPIV within a group of children in the late 1950s, much research has been devoted to understanding their role in zoonotic diseases (Henrickson, 2003).

HPIVs are pleomorphic, medium-sized, enveloped viruses containing a single negative-sense strand of RNA (Henrickson, 2003). The HPIV genome encodes 6 proteins: a nucleocapsid protein (NP), a phosphoprotein (P), a matrix protein (M), a fusion glycoprotein (F), a hemagglutinin neuraminidase (HN) glycoprotein, and an RNA polymerase (L). HN and fusion proteins are responsible for attaching to sialic acid residues on host epithelial cells and fusing the viral envelope with the host membrane, respectively (Hu et al., 1992). Typically, HPIV replicates in ciliated epithelial cells of the respiratory tract, leading to either an upper respiratory tract infection (URTI) or lower respiratory tract infection (LRTI) (Branche and Falsey, 2016; Moscona, 2005).

LRTIs are among the top five causes of death in children less than 5 years of age (Mortality and Causes of Death, 2015). In comparison to other serotypes, HPIV-3 is more often associated with LRTIs, causing bronchiolitis and pneumonia in neonates and infants (Henrickson et al., 2004). Additionally, HPIV-3 and HPIV-1 are most commonly associated with pneumonia, accounting for 1–6% and 2–12% of HPIV-related hospitalizations, respectively (Branche and Falsey, 2016).

Approximately 40–60% of pediatric HPIV infections result in URTIs (Branche and Falsey, 2016). Croup - a common URTI - is often caused by HPIV-1 and HPIV-2. Illnesses related to HPIV-4 are often mild and subclinical, making the virus more difficult to detect (Billaud et al., 2005; Vachon et al., 2006).

Parainfluenza viruses may also cause respiratory infections in other animals. For example, bovine parainfluenza virus type 3 (BPIV-3) is one of the main causes of respiratory infections in cattle (Timurkan et al., 2019). Typically, the infection is subclinical, however, some bovine may develop bronchointerstitial pneumonia. Transmission in susceptible animals is typically caused by aerosol and fomites resulting from nasal discharge (Timurkan et al., 2019). HPIV-3 and BPIV-3 are very similar in genome organization, making BPIV-3 a good vaccine candidate to protect against HPIV-3 (Schmidt et al., 2000). In comparison to HPIV-3, BPIV-3 is both non-pathogenic and poorly transmitted in humans (2017) However, cross-species infections of BPIV-3 in humans has been reported (Ben-Ishai et al., 1980). Moreover, infection of humans with BPIV-3 results in 1- to 3-log lower viral titers recovered in comparison to HPIV-3. Additionally, viral replication of BPIV-3 occurs, and antibody responses specific to BPIV-3 are induced (Thibault et al., 2017). Thus, the species restriction for BPIV-3 and HPIV-3 is not absolute and transmission from bovine to humans is possible.

Transmission of HPIV between humans has been reported to be predominantly through direct or indirect contact with infectious respiratory droplets, with minimal aerosol transmission. Therefore, contact with surfaces, including foodstuff, contaminated with infectious respiratory droplets may also lead to infection (Burke et al., 2013). There are no reports of the persistence of HPIV on food, however, HPIV-1, HPIV-2, and HPIV-3 have been investigated and determined to survive up to 10 h on non-absorptive surfaces when the sites remained moist, with up to

80% and 50% reduction in viral recovery after 10 h on stainless steel and laminated plastic, respectively. Moreover, HPIV-1, HPIV-2, and HPIV-3 could survive up to 4 h on absorptive surfaces, with 100% reduction in viral recovery on facial tissues after 4 h, 0% reduction in viral recovery from an unlaundered hospital gown and lab coat after 4 h, and 100% reduction in viral recovery from a washed hospital gown after only 30 min of incubation. Such results were dependent on the material being tested, the concentrations of viral inoculum which mimicked HPIV viral titers found in symptomatic patients, and the environmental conditions (Brady et al., 1990). HPIV is easily affected by temperature, humidity, and pH. Viral survival decreases above 37 °C, under low humidity, or when the pH is between 3.0 and 3.4. Viral survival increases when exposed to physiological pH (7.4–8.0) and HPIV is most stable at 4 °C or if frozen (−74 °C) (Henrickson, 2003). Additional research should be conducted to understand the viability of HPIV specifically on food surfaces and how contamination of food may lead to infection.

5. Orthopneumovirus

The genus Orthopneumovirus of the family *Pneumoviridae* contains various species capable of causing significant respiratory infections in mammals (Rima et al., 2017). The species *human orthopneumovirus*, includes the human respiratory syncytial virus (RSV) which was first discovered in 1955 in chimpanzees and was subsequently confirmed to be a human pathogen in 1956 (Blount et al., 1956). RSV is an enveloped virus containing a negative-stranded RNA genome of approximately 15.2 kb (Collins and Graham, 2008). RSV can spread easily in hospitals, nursing homes, and other close-contact settings, and is thus considered one of the most contagious human pathogens, primarily infecting children and the elderly (Collins and Graham, 2008). In fact, it is predicted that RSV infects 90% of children within the first two years of life and frequently re-infects older children and adults partially due to the lack of long-term immunity (Schweitzer and Justice, 2019).

RSV-G glycoprotein can attach to CX3XR1, a G-coupled transmembrane chemokine receptor (Chirkova et al., 2015), on airway epithelial cells (Jones et al., 2018) and RSV-F fusion glycoprotein can subsequently fuse the two membranes together (Schweitzer and Justice, 2019; Taleb et al., 2018). The majority of infected patients will develop an upper respiratory tract illness that usually presents as an airway obstruction, runny nose, shortness of breath, wheezing, and/or hypoxia (Schweitzer and Justice, 2019; Taleb et al., 2018). However, a significant minority of patients may develop a lower respiratory tract illness, namely pneumonia or bronchiolitis (Schweitzer and Justice, 2019).

Other common species found in the Orthopneumovirus genus include Bovine orthopneumovirus and Murine orthopneumovirus, which contain bovine respiratory syncytial virus (BRSV) and murine pneumonia virus (MPV), respectively (Collins and Graham, 2008). BRSV is frequently associated with bovine respiratory disease (BRD), which contributes to revenue losses of more than $1 billion USD annually (Brodersen, 2010; Leme et al., 2020). MPV causes natural infections in mice, rats, hamsters, other rodents, as well as rabbits (Whary et al., 2015). However, humans are not normally exposed to MPV nor is MPV cross-protective against RSV (Brock et al., 2018). There is no animal reservoir for RSV, however alternative animal versions of RSV suggest that interspecies transmission has occurred during viral evolution (Collins and Graham, 2008).

RSV has been demonstrated to spread person-to-person via respiratory droplets and may survive on contaminated surfaces, thus allowing for the transfer of infectious viral particles to humans (Hall et al., 1980; Schweitzer and Justice, 2019). RSV was recovered from Formica® countertops for up to 6h, rubber gloves for up to 1.5h, cloth gowns and paper tissues for 30–45 min, and skin for up to 20 min (Hall et al., 1980). As such, fomites, including food, may be potential modes of transmission, although, no studies have specifically investigated the viral persistence on food surfaces and there is no evidence to link any clinical case to foods or fomites.

6. Adenoviruses

Human adenoviruses (HAdVs) are non-enveloped, double-stranded DNA viruses in *Adenoviridae* family, genus Mastadenovirus. To date, over 100 types of HAdVs have been identified and divided across seven species, A-G (Human Adenovirus Working Group, 2019). The adenovirus virion consists of an icosahedral capsid with fiber proteins extending from the vertices. The fiber proteins are the main antigenic proteins that bind to the host cell-surface receptors through the terminal globular domain, and mediate cell entry (Berk, 2013). Adenoviruses use remarkably diverse attachment receptors including Coxsackie and Adenovirus Receptor (CAR), CD46, and heparin sulfate glycosaminoglycans, which are involved in viral binding, and the integrin molecules that facilitate entry (Stasiak and Stehle, 2020; Zhang and Bergelson, 2005). The genome of HAdVs encodes about 20 early genes, responsible for genome replication, and about 15 late genes that are involved in viral assembly and progeny release (Ison, 2013). HAdVs usually cause lytic infection in epithelial cells; however, they are capable of establishing latency in lymphoid cells (Kosulin et al., 2016).

HAdVs can cause an array of diseases including respiratory, eye, GI, and urinary tract infections (Ison, 2013). In most cases, these infections are mild, self-limiting, and occur in children under the age of 5 (Ghebremedhin, 2014). Of the seven HAdV species (A to G), species A, F, and G have been associated with GI infections (Hassan et al., 2019). Types 40/41 in species F are responsible for about 10% of pediatric gastroenteritis around the world (Lee et al., 2020). Species B and C are more common to the respiratory tract (Ghebremedhin, 2014), species D mainly causes conjunctivitis (Ismail et al., 2019), and species E is associated with respiratory and ocular infections (Ghebremedhin, 2014).

Enteric HAdVs are transmitted through the fecal-oral route, however, all HAdV types, including respiratory types can be detected in stool specimens (Afrad et al., 2018; Kumthip et al., 2019; Lee et al., 2020). The duration of fecal shedding following primary HAdVs infection in children can be very prolonged, up to 3 months (Ye et al., 2017). Plus, it has been demonstrated that multiple HAdVs species are able to establish latency in lymphoid cells of the lamina propria in the GI tract (Kosulin et al., 2016). Thus, it has been suggested that fecal shedding of respiratory HAdVs plays a role in community spread of these viruses (Kim et al., 2017). Furthermore, the intestinal tract appears to be a common site for persistence of HAdVs in non-symptomatic and immunocompetent adults (Kosulin, 2019), and conditions of immunosuppression lead to reactivation of HAdVs in the GI tract, which could have severe consequences (Lion, 2014).

No contaminated food has been directly associated with HAdV infection but there is evidence for water-borne transmission in public water systems and in swimming pools (Rodríguez-Lázaro et al., 2012).

7. Rhinoviruses

Human Rhinoviruses (HRVs) are members of the *Picornaviridae* family and Enterovirus genus and are estimated to cause approximately 50% of common colds worldwide (Jacobs et al., 2013; Winther, 2008). Since the discovery of HRVs in the 1950s, more than 150 HRV strains have been classified as either species A, B, or C (Blaas and Fuchs, 2016; Jacobs et al., 2013). HRVs are non-enveloped viruses containing a positive-sense single-strand of RNA, encoding a single polyprotein which is cleaved into 11 proteins (Megremis et al., 2012). The viral capsid is composed of four viral proteins (VPs): VP1, VP2, VP3, and VP4. VP1 is known to mediate cell surface attachment by binding to various cell surface receptors (Stobart et al., 2017). Twelve HRV-A strains bind to members of the low-density lipoprotein (LDLR) family, while the remaining A and B types bind to intercellular adhesion molecule-1 (ICAM-1) (Hofer et al., 1994; Staunton et al., 1989); HRV-C has been shown to bind to human cadherin-related family member 3 (CDHR3) (Bochkov et al., 2015). HRV has been demonstrated to replicate in the

nasal epithelium and nasopharynx, perhaps due to the high expression of ICAM-1, LDLR, and/or CDHR3 (Arruda et al., 1995; Watters and Palmenberg, 2018).

HRV is normally thought to cause relatively benign upper respiratory tract illnesses and is thus considered the most common cause of upper respiratory tract infections (URTIs) (Jacobs et al., 2013). However, HRVs have now been linked to asthma development, exacerbations of chronic pulmonary disease, severe bronchiolitis in infants, and fatal pneumonia in the elderly and immunocompromised (Henquell et al., 2012; Kennedy et al., 2019; Mallia et al., 2011). Further evidence suggests that HRVs are associated with considerable economic burdens due to medical visits and absenteeism from work and/or school (Fendrick et al., 2003; Nichol et al., 2005). Unfortunately, there are no approved antiviral agents to prevent such infections and vaccine development efforts are hindered due to the large amount of serotypes with high sequence variability in antigenic sites (Jacobs et al., 2013; To et al., 2017).

Children are considered the main transmission vector for HRVs due to the high rate (12–32%) of asymptomatic infections in children less than 4 years old, and the high viral load relative to adults (Blaas and Fuchs, 2016; L'Huillier et al., 2015). HRVs are transmitted person-to-person by either direct or indirect contact, or by aerosol particles (Jacobs et al., 2013). The virus has been shown to regularly deposit onto the hands of infected individuals and into the environment. Under experimental conditions, HRV was transferred from surfaces to the fingertips of participants in 60% (18/30) of trials 1 h after contamination and 33% (10/30) of trials 18 h after contamination (Winther et al., 2007). Additionally, HRV can survive on undisturbed forskin for 2 h (Winther et al., 2007) and aerosols produced by coughing or sneezing contain a viral load 30 times lower than that of nasal secretions (L'Huillier et al., 2015). Thus, person-to-person transmission is most likely due to contamination of hands by nasal secretions, either directly from the hands of an infected person or an intermediary (L'Huillier et al., 2015). All HRV species have frequently been reported in stool samples of young children, as well as in sewage water, suggesting a possible fecal-oral transmission route (Blomqvist et al., 2009; Honkanen et al., 2013). There has been no reported literature regarding foodborne outbreaks of HRVs, as well as no studies investigating the viral survival in or on food products.

8. Nipah virus

Nipah virus (NiV) is one of the deadliest zoonotic emerging pathogens (Soman Pillai et al., 2020). It is an enveloped virus that belongs to the *Paramyxoviridae* family and its genome consists of a single strand of RNA with negative polarity, about 18.2 kb long (Harcourt et al., 2000). Following an incubation period of less than two weeks, although it might vary from 4 days to two months (Aditi and Shariff, 2019), patients develop fever, headache, vomiting, respiratory distress, and encephalitis, manifested as seizure and unconsciousness (Ang et al., 2018). The NiV mortality rate ranges from 68% to 91% (Soman Pillai et al., 2020).

Fruit bats of the genus Pteropus (flying foxes) are the natural reservoirs for NiV (Halpin et al., 2011). NiV outbreak was first reported in 1998 in Sungai Nipah, a village in Malaysia, where humans contracted NiV from pigs, which in turn contracted the virus due to the consumption of fruits contaminated with saliva and excretes of fruit bats (Goh et al., 2000). Recurring NiV outbreaks have then been reported in different parts of South Asia, including Bangladesh, where infections occurred due to the consumption of raw date palm sap contaminated with saliva and feces of the fruit bats (Soman Pillai et al., 2020). Foodborne transmission of NiV has also been demonstrated in laboratory animals (Kingsley, 2016). Interestingly, the orally administered virus in hamsters was detected in respiratory tissues rather than in the intestinal tract (Kingsley, 2016). Based on genetic diversity, two strains of NiV have been identified: NiV-B (Bangladesh) and NiV-M (Malaysia). NiV-B and NiV-M share 91.8% genetic similarity; however, NiV-B has higher fatality rates and is more prevalent (Mire et al., 2016).

The attachment (G) and fusion (F) envelope glycoproteins are both required for viral entry into cells (Bradel-Tretheway et al., 2019). NiV uses evolutionary conserved ephrinB2 (B2) and ephrinB3 (B3) as its receptor for cell entry (Liu et al., 2015). Ephrin B2 and B3 are highly expressed in endothelial and neuronal cells and demonstrate over 95% amino acid sequence homology between different species of mammals (Pernet et al., 2012). NiV is highly pathogenic to a broad range of mammals and has pandemic potential due to its zoonotic as well as person-to-person transmission. The main reservoir of infection, Pteropus bat are endemic to tropical and subtropical regions of Asia, East Africa, Australian continents and some oceanic islands, thus where they reside has the potential to be the location of new spillover events in the future (Aditi and Shariff, 2019).

9. Conclusion and future remarks

Currently, there is no epidemiological evidence for foodborne transmission of respiratory viruses, particularly AIV and SARS-CoV-2. There is consensus among the World Health Organization (WHO), the Canadian Food Inspection Agency (CFIA), the United States Department of Agriculture, and the European Food Safety Authority that the risk of SARS-CoV-2 foodborne transmission is low (CFIA, 2020; EFSA, 2020; USDA, 2020; WHO, 2020b). The International Commission on Microbiological Specifications for Foods (ICMSF) recently released a statement that there is no documented evidence that food is a significant source or vehicle for transmission of COVID-19 (ICMSF, 2020). Additionally, the CFIA has conducted susceptibility analysis on livestock including domestic turkeys, chickens and pigs, and have demonstrated that these animals do not spread SARS-CoV-2 to humans, animals or the environment. Furthermore, the studies demonstrated that SARS-CoV-2 does not replicate in domestic turkeys and chicken and replicates poorly in domestic swine under laboratory conditions. In these animals, there was also no indication of the presence of SARS-CoV-2 in tissues destined for human consumption (CFIA, 2020).

However, it is unclear that the traditional epidemiological foodborne investigational approach is employed with respect to COVID-19 patients. For example, it is unlikely that infected people are asked to recall foods that they may have consumed during the period when they became infected. Without this information, any association between SARS-CoV-2 and foods are unlikely to be realized. Thus, the theoretical risk of transmission via foods cannot be ruled out based on a number of factors, including the expression of viral receptors on enterocytes, replication in enteric cell lines, and extended survival in the environment. The possibility of respiratory exposure to the virus via food could also be possible, since aerosols are generated during the process of chewing foods (Gavião and Bilt, 2004). It is notable that there are specific risk mitigation measures, such as respiratory etiquette, and frequent hand and surface hygiene (Health Canada, 2020) that could considerably reduce the risk of contamination of food with respiratory viruses.

Future studies should be conducted to understand the ability of various respiratory viruses to survive in various foods including meats, dairy, seafood, and fresh produce. Studies should also evaluate the heat and pH resistance in these foods. While enveloped viruses are not resistant to heat or low pH, the ability of high fat or protein foods to protect the virus as it transits the stomach should be assessed. Additional studies should be conducted to assess the ability of respiratory viruses to survive on food-contact surfaces, and surfaces in retail establishments, as well as disinfectant efficacy. Such studies should take into account the genetic variation that is often observed among respiratory viruses, as such difference may lead to differential survival in foods and the environment. Also, such applied studies are often conducted using high-titres of cell-culture adapted viruses that might not necessarily represent natural contamination.

The use of metagenomics technologies could aid in the detection of

respiratory viruses within the virome of foods (Ronholm et al., 2016). However, care should be taken when interpreting the results of virus detection from foods with the use of molecular methods, as these methods cannot discriminate between infectious viral particles and uninfectious viral nucleic acids (Nasheri et al., 2019; Suresh et al., 2019). Finally, given the high infectivity of respiratory viruses and the fact that many research institutions do not have the BSL-3/CL-3 biosafety capacity to work with them, additional work on viral surrogates should be conducted to identify appropriate surrogates for infectious viruses that can be tested in BSL-2/CL-2 labs in modelling and experimental studies.

Declaration of competing interest

The authors declare no conflict of interest.

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