Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
What makes a foodborne virus: comparing coronaviruses with human noroviruses
Dan Li, Mitchie Y Zhao and Turk Hsern Malcolm Tan

In order to answer the question whether coronaviruses (CoVs) can be transmitted via foods, this review made a comparison between CoVs with the most recognized foodborne virus, human noroviruses (NoVs). As a result, although CoVs indeed have shown the possibilities to remain infectious on foods and/or food packaging materials long enough (from several days to several weeks) to potentially cause transmission, they seem to be less persistent than NoVs towards common disinfection practices with alcohols, chlorine and ultraviolet (UV). More importantly, the chance of foodborne transmission of CoVs is considered low as CoVs mainly spread through the respiratory tract and there is no clear evidence showing CoVs can follow fecal-oral routes like human NoVs and other foodborne viruses.

Address
Department of Food Science & Technology, Faculty of Science, National University of Singapore, Singapore

Corresponding author: Li, Dan (fstlda@nus.edu.sg)

Introduction
Human noroviruses (NoVs) are the most frequently linked virus with foodborne outbreaks, and as such are identified as the foodborne virus with the highest priority worldwide. In 2015, the World Health Organization (WHO) listed human NoVs as the ‘Number 1’ cause of foodborne illnesses [1]. Next to NoVs, commonly recognized foodborne viruses also include hepatitis A virus, hepatitis E virus, rotaviruses, astroviruses, and so on [2].

Because of the current pandemic of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), numerous concerns have been raised over whether SARS-CoVs-2 can be transmitted via foods and/or food packaging materials. Indeed, the possibility of foodborne transmission cannot be ruled out for any virus. However, these possibilities should be understood in-depth as supported by scientific data and analysis so that the virus spread could be controlled in a more focused and efficient way. In this review, we intend to make a comparison between coronaviruses (CoVs) with the most recognized foodborne virus, human NoVs, in order to supply evidence to evaluate the possibilities of foodborne transmission of CoVs.

The comparisons were performed from four different perspectives including the epidemiological evidence, their presence in foods, their persistence in food systems, and their relevant clinical manifestations.

Foodborne illnesses are linked clearly with the consumption of contaminated foods
In 2015, WHO estimated 684 million diarrheal disease cases caused by human NoVs annually, amongst which 212 000 deaths were caused [2]. The link between many of the illnesses and the human NoVs-food contaminated-food consumption has been clearly demonstrated (Table 1) thanks to the comprehensive investigations among all the components of a food control system. Typically, a successful foodborne outbreak investigation will need collaborative efforts from food law and regulations, food control management, inspection services, epidemiological and food monitoring (laboratory services) and consumers’ education and communication. Verhoef et al. [3] estimated the proportion of foodborne infections caused by human NoVs on a global scale to be as high as ~14%. Meanwhile, it should be well noted that large-scale outbreaks are often the result of a combination of several transmission routes. For example, the virus can first infect a sensitive population by food, water or an asymptomatic shedder, and a more efficient viral spread in a large group of population could be followed by direct person-to-person contact or via a contaminated environment.

In comparison, to the best of our knowledge, despite the long history and wide spread of CoVs in human communities, there is no epidemiological evidence showing that any of the illnesses was due to food consumption.

Foodborne viruses are detected frequently from foods
The discovery of human NoVs from food systems is not rare. Numerous reports have been published for human NoVs screening from food and environmental samples (Table 2). The most common categories of food linked to outbreaks are shellfish, which can bio-accumulate viral particles from a large volume of water and is often consumed uncooked; and fresh produce, especially soft
berry fruits and leafy green vegetables, which can be contaminated during the primary production, and are generally consumed without effective treatment to get rid of the contaminated viruses. However, one must realize that any food can be implicated in outbreaks, especially when the contamination is due to infected food handlers [1].

Again, no record over the presence of CoVs in foods could be found from the literature. One may argue that the CoV presence could have been understudied and in the future, especially with the use of metagenomics technologies, CoVs might be able to be found within the viromes of foods. However, care should be taken when interpreting the results of virus detection from foods with the use of molecular methods. In-depth understanding over the virus quantities in relation to a dose-dependent effect and the virus viability (as the molecular methods detecting the presence of nucleic acids are not able to differentiate between infectious and non-infectious viruses) are of crucial importance in order to determine the relevant public health influence.

**Table 1**

| Foods involved in the outbreaks | Period and origin | Epidemiological description | Laboratory investigation | Reference |
|---------------------------------|-------------------|----------------------------|--------------------------|-----------|
| Oyster                          | Jan. 2020, Denmark and Sweden | At least 180 people in Denmark and 70 people in Sweden were sick with vomiting and diarrhoea. | Symptomatic individuals and oyster samples were positive for NoV. | [20] |
| Turkey                          | Mar., 2018, Spain | The acute gastroenteritis outbreak affected 137 out of 361 people of a nursing home. | Ten of the 28 stool samples were positive for NoVs (two G1, six GII and two GII/GII). Turkey was suggested to be the initial source of the outbreak and was subsequently spreading via person-to-person transmission. | [21] |
| Mussels                         | 2017, Spain       | Thirty-nine people were sick after consuming mussels contaminated with NoV | Three stool samples from symptomatic individuals were positive for NoV. Mussel samples from the affected batch were positive for NoV G1 and GII. | [22] |
| Oyster                          | Jan., 2017, New Zealand | Eleven people became ill after consuming oyster harvested from Mahurangi Harbour | NoVs identified from symptomatic individuals and oysters were the same. | [22] |
| Chipotle chili                  | Oct. and Nov., 2016, United Kingdom | A total of 1112 customers and staff reported with gastroenteritis after eating at all branches of a restaurant group | Thirty out of 48 samples from staff were positive for NoV strain GII.6. New chipotle chili imported from outside the European Union was most likely to be the vehicle of the transmission | [23] |
| Coleslaw                        | 2015, Sweden      | A two-episode outbreak; the first outbreak affected 542 out of 1109 employees in a large office based location in Stockholm. Three weeks later (second outbreak), 54 employees and a restaurant personnel fell ill with gastrointestinal-symptoms. | First outbreak: 8 faecal samples from symptomatic individuals and coleslaw samples were positive for NoV GII. Nucleotide sequencing of the faecal samples reveals that the outbreak strain belongs to GII.6 genotype. Second outbreak: 3 employees and 2 out of 10 restaurant personnel were positive for NoV GII. The close connection between two outbreaks suggests the possible spread of the same NoV genotype (GII.6), which could be attributed to a mixture of foodborne and person-to-person transmission. | [24] |

**Foodborne viruses show high stability and resistance towards environmental stress in food systems**

Figure 1 illustrates the foodborne transmission routes of human NoVs. Since viruses cannot multiply themselves without a host, after being shed to the environment, the viruses must be able to resist the possible environmental stress in the food systems, such as solar irradiation, desiccation, high or low temperature, unfavourable chemicals, and so on, and remain infectious for durations long enough until being ingested again. In fact, human NoVs are known as the ‘super survivor’ in the food systems as shown by numerous studies (Table 3).

Table 3 intends to compare the stability between NoVs and CoVs under different possible conditions in the food systems. Since molecular methods underestimate largely the infectivity decrease of viruses, we only included data generated with the use of cell culture based methods or from human volunteer studies. Although there have been recent breakthroughs reported in human NoV tissue culture models [4*], it is not yet commonly used for
routine food and environmental testing due to the presence of residual food matrix components as well as the cost and labour implications. Surrogates including feline calicivirus (FCV), murine norovirus (MNV), and coliphage MS2 that share pathological and/or biological features with human NoVs have been widely used to study the stability of human NoVs. For CoVs, although the most-of-interest strains are SARS-CoVs and other severe syndrome strains such as Middle East respiratory syndrome coronavirus (MERS-CoV), working with BSL-3 laboratory containment places can cause significant practical challenges, and thus researchers have employed surrogates such as transmissible gastroenteritis virus (TGEV), a diarrheal pathogen of swine, and mouse hepatitis virus (MHV), a respiratory and enteric pathogen of laboratory mice, to study the survival and persistence of CoVs. Consequently, large variabilities were observed even within the NoVs or CoVs used in different studies (Table 3).

Low temperature is favourable for both NoVs and CoVs to survive both on food-contact surfaces/solid foods and in water/liquid foods as shown consistently in multiple studies as summarized in Table 3. However, the influence of relative humidity (RH) is contradictory for different viruses. HAV survived better at higher RH [5], while MS2 and MERS-CoV survived better at lower RH [5, 6].

| Sample type                                      | Period and origin | Positive rate | Detection and analysis methods                             | Reference |
|--------------------------------------------------|-------------------|---------------|------------------------------------------------------------|-----------|
| Fresh produce (raspberries and lettuce) frozen   | Mar., 2015 to     | Human NoV was detected in 5.3% (30/568) of lettuce samples, 2.3% (7/310) of fresh raspberry samples and 3.6% (10/274) of frozen raspberry samples. | Real-time RT-PCR (Taqman) | [25] |
| produce (Raspberries)                            | Apr., 2017, the   |               |                                                            |           |
|                                                  | United Kingdom    |               |                                                            |           |
| Fresh/frozen berries (strawberries, blueberries,| Jan., 2016 to     | Human NoV was detected in 9% (81/900) of frozen and 12.1% (109/900) of fresh domestic retailed berry samples. | Real-time RT-PCR (Taqman) | [26] |
| raspberries, cranberries, blackberries and       | Dec., 2017, China |               |                                                            |           |
| blackcurrants)                                   |                   |               |                                                            |           |
| Fresh seafood (oysters, clams, shrimps and       | India             | NoV GII was detected in 41 out of 104 (41.3%) fresh seafood samples. The incidence of NoV was the highest in bivalves (52.7%–39/74), followed by finfish (16.7%–2/12) and lastly crustaceans (11%–2/18) | Reverse-transcription PCR (RT-PCR), nested PCR, Southern hybridization (for confirmation purpose) | [27] |
| finfish)                                         |                   |               |                                                            |           |
| Shellfish (oysters and clams)                    | Oct., 2015 to     | Human NoV was detected in 81.8% (99/121) of the analyzed samples. Multiple strains of NoV were identified (GI.2, GI.4, GI.5, GI.6, GII.3, GI.4, GIII.6, GIII.7, GIII.13, GIII.14, GIII.17, GIII.21 and GIII.18) | Real-time RT-PCR (Taqman) | [28] |
|                                                  | June, 2016, Vietnam |               |                                                            |           |
| Shellfish (oyster)                               | Sep., 2015 to     | Human NoV was detected in 20.7% (155/622) of the oyster samples. | Real-time RT-PCR (Taqman) | [29] |
|                                                  | Sep., 2016, China |               |                                                            |           |
| Shellfish (oyster)                               | Nov., 2014 to     | NoV GII was detected in 89% (48/54) of the composite oyster samples pooled from 162 individual oysters. Multiple genotypes of GII were identified (GII.3, GII.4, GIII.6, GIII.13, GIII.17) | Reverse transcription (RT) and quantitative real time PCR (qPCR), nested PCR (for unquantifiable but possibly positive sample), pyrosequencing (for genotyping and phylogenetic analysis). | [30] |
|                                                  | Mar., 2015, Japan |               |                                                            |           |

Similarly, no consensus could be reached for the influence of organic (food) matters. On one hand, MNV showed 6.2-log reduction on residue-free coupons and only 1.4-log reduction on coupons with lettuce, cabbage, or ground...
Table 3

The stability of NoVs and CoVs and their surrogates under different conditions as reported in the literature

| Conditions | Virus stability | NoVs and the surrogates | CoVs and the surrogates | Reference |
|------------|-----------------|-------------------------|-------------------------|-----------|
| On possible food-contact surfaces and solid foods | MNV, NoV surrogate at room temperature for 28 days: Rank order of reduction, from highest to lowest, was stainless steel (2.28-log reduction after 28 days), plastic, rubber, glass, ceramic, and wood (1.29-log after 28 days). | | SARS-CoV-2 at 21–23°C and 40% relative humidity (RH): more stable on plastic (3.1-log reduction after 3 days) and stainless steel (3.1-log reduction on plastic after 2 days) than on copper and cardboard, and viable virus was detected up to 3 days after application to these surfaces. | [31] |
| | On dried stainless steel surfaces for 7 days: MNV and FCV showed ~1-log reduction at 4°C; ~4-log reduction at room temperature after 7 days. | | SARS-CoV remained stable on plastic surface at room temperature with 40–50% RH for up to 4 weeks, yet lost its infectivity significantly at 38°C with >95% RH during 24 hours in air. | [33] |
| | On stainless steel coupons for 30 days at 25°C: MNV showed 6.2-log reduction on residue-free coupons and 1.4-log reduction on coupons with lettuce, cabbage, or ground pork residues Bacteriophage MS2 4°C: <1-log reduction for all produce types by day 7, <2-log reduction in cabbage and carrots by day 87; 8°C: <1-log reduction for all produce types by day 7, ~1-log reduction in tomato, cabbage, carrots and lettuce by day 39; 22°C: 1-log reduction on lettuce and <1-log reduction on tomato and parsley by day 7 Hepatitis A virus (HAV), MS2, MNV on oyster and peppers at 4°C, 15°C, 25°C, and 40°C: viruses survived best at 4°C and were inactivated most at 40°C. On oysters, a 1-log reduction of both HAV and MNV occurred at 4°C, even after 14 days. However, a 5-log reduction of MNV occurred on peppers at 4°C. MNV showed the shortest survival duration on peppers at all temperatures compared to other viruses. Viral survival was better on oysters than on peppers. At a given temperature, HAV survived better at higher RH, while MS2 survived better at lower RH. At 40°C, inactivation of HAV was 1 log at 50% RH but only 0.1-log at 70% RH on day-1 post inoculation. | | MERS-CoV survived on both plastic and steel surfaces after 48 hours at 20°C, 40% RH, while it remained viable only for 8 hours at 30°C, 80%RH and 24 hours at 30°C, 30% RH. At 20°C, MERS-CoV’s viability decreased 7% at 40% RH, and 89% at 70% RH respectively. | [36] |
| | In water and liquid foods | MNV showed infectivity reduction rate of 0.16-log PFU/day in surface water and 0.04-log PFU/day in groundwater at 25°C. Norwalk virus (NV, prototype of NoVs) remained infectious at least for 61 days in groundwater at room temperature in the dark as tested by human volunteer studies. | | Human coronavirus (hCoV) strain 229E survived on lettuce during 2 days of storage at 4°C, yet became non-infectious by day 4 (reduction > 1.31-log). No hCoV could be recovered from raspberries or strawberries after spiking. | [8] |
| | towards alcohols | Regardless of concentration or exposure time, alcohols slightly reduced, but did not completely inactivate, human norovirus (3 GII.4 strains tested by the enteroid culture model). | | HCoV (with 99.9% decrease of infectivity) for 10 days at 23°C, for >100 days at 4°C in tap water, yet for only 2–4 days in wastewater. At 25°C, transmissible gastroenteritis (TGEV) survived for 22 days, and mouse hepatitis virus (MHV) survived for 17 days in reagent-grade water, whereas in wastewater, TGEV survived for 9 days and MHV survived for 7 days (with 99% decrease of infectivity). At 4°C, both viruses survived longer than four weeks. | [38] |
| | towards chlorine | Complete inactivation of the 3 GII.4 viruses occurred at concentrations at 50 ppm of chlorine after incubating the solutions for 1 min at room temperature strains tested by the enteroid culture model. | | SARS-CoV residual infectivity was detected after fixation with 70% ethanol for 10 min or 100% ethanol for 5 min. Isopropanol 70% and 100% achieved >3.31-log reduction of SARS-CoV infectivity after 30 s. SARS-CoV could be completely inactivated with 10 ppm chlorine for 10 min or more, and with 20 ppm chlorine for 1 min or more. | [39] |
| | towards UV | The susceptibility of MHV was 7-10 times that of the MS2. | | [39] |
pork residues [7]. On the other hand, MNV showed infectivity reduction rate of 0.16-log PFU/day in surface water and 0.04-log PFU/day in groundwater at 25°C [8]. Similarly, human coronavirus (HCoV) survived for 10–100 days in tap water, yet for only 2–4 days in wastewater [9]. At 25°C, TGEV survived for 22 days, and MHV survived for 17 days in reagent-grade water, whereas in wastewater, TGEV survived for 9 days and MHV survived for 7 days [10]. Therefore, the matrices with different components tested with the viruses may play different roles, either in viral protection from the environmental stress, or as antiviral agents alone or together with the external stress.

In addition, different experimental set-ups were used in different studies, including the tested environmental parameters, the virus spike levels, the test durations, the virus recovery methods, and the data interpretation methods (log-reductions versus durations until the viruses became non-infectious), and so on. Therefore, it is not possible to make direct comparisons between NoVs and CoVs over their stabilities on foods (both solid and liquid foods) or possible food-contact surfaces. Nevertheless, it seems that both NoVs and CoVs were able to remain infectious on foods and/or food packaging materials long enough (from several days to several weeks) to potentially cause transmission especially at low temperatures.

Fortunately, when it comes to the disinfection studies, more straightforward comparisons become possible thanks to the availability of studies evaluating human NoV viability with the use of tissue culture model [11] and studies directly comparing NoV and CoV surrogates [12]. According to the results demonstrated in Table 3, NoVs were clearly much more resistant than CoVs towards alcohols, chlorine and ultraviolet (UV) disinfection.

**Foodborne viruses are transmitted via fecal-oral routes**

So far, all of the well-recognized foodborne viruses are transmitted via fecal-oral routes. For human NoVs, although our understanding on the cellular pathways that control infection and the exact pathogenesis remains limited [13,14], the recent breakthrough of human NoV in vitro cultivation systems with mucosa-derived intestinal epithelial organoids [4] reveals clearly that human NoVs infection occurs primarily in the human digestive tracts. Besides, the clinical manifestations of human NoVs also have the following features being believed to contribute to its ‘achievement as a successful foodborne virus’. First, human NoVs are extremely contagious. The infectious dose of human NoVs was estimated to be as low as 10 particles [15]. Second, once infected, human NoV particles can be shed from the stool and vomit of the patients in large quantities (e.g. up to \( >10^{10} \) genomic copies per gram of feces [16]). Moreover, it has been discovered that asymptomatic infections with long-term fecal shedding (up to three weeks) of human NoVs can have a high prevalence, especially in the group of children [17].

CoVs, including the newly emerged SARS-CoV-2, mainly spread through the respiratory tract. There are indeed reports showing the presence of CoVs in human fecal samples. For instance, in a recent investigation, 41 (55%) out of 74 SARS-CoV-2 infected patients were tested positive for SARS-CoV-2 RNA in their fecal samples and the fecal shedding remained for a mean of 27.9 days after the first symptom onset [18]. However, there is so far no clear evidence showing SARS-CoV-2 can cause infection in human digestive tracts. Considering the high probability of infection of SARS-CoV-2 (basic reproduction number estimated to be above 2.0 by WHO [19]), assumptions could be made that the viruses may migrate from the oral ingestion to the respiratory tracts via, for instance, the throat. However, this assumption will need sound experimental and/or clinical supports not only in a qualitative way (to show whether it is possible for the virus to migrate from oral ingestion to the respiratory tract), but also in a quantitative way. Since if large quantities of viruses must be ingested in order to cause the migration, the chance of such occurrence could be very low in reality.

**Conclusions**

On the basis of our understanding, four important features are shared by foodborne viruses. I) Clear epidemiological evidence showing the link between relevant illnesses and the consumption of virus-contaminated foods; II) Records of virus presence in foods by the monitoring or surveillance studies, which in reality facilitate due diligence in the food supply chains or initiate recalls; III) High stability and resistance towards environmental stress in the food systems; IV) Fecal-oral transmission routes with infection occurring primarily in the human digestive tracts.

In correspondence, the chance of foodborne transmission of CoVs is considered low and thus CoVs should not be recognized as foodborne viruses. CoV infection has never been found to link with food consumption, and so far CoVs have never been detected from foods either. Although CoVs indeed showed the possibilities to remain infectious on foods and/or food packaging materials long enough (from several days to several weeks) to potentially cause transmission, they were found to be less resistant to chemical and physical disinfections than NoVs. More importantly, CoVs mainly spread through the respiratory tract and there is no clear evidence showing CoVs can follow fecal-oral routes and cause infection in the human digestive tracts.

In the future, the possibility of CoV infection via oral ingestion should be monitored closely, as many facts of
these viruses still remain unrevealed and the viruses may evolve rapidly. In addition, care should be taken when interpreting results obtained with the emerging molecular technologies. As the trace of virus genetic materials may neither necessarily represent the presence of viable viruses thus nor the public health threats. Lastly, when multiple transmission routes are identifiable, comprehensive consideration is necessary to set up the priorities and to control the virus spread efficiently.

Conflict of interest statement
Nothing declared.

Acknowledgement
This study was supported by a Ministry of Education (MOE) academic research fund (AcRF) TIER I project ‘Study of important foodborne viruses from relevant foods in Singapore’ (Jan 2019 to Dec 2021).

References and recommended reading
Papers of particular interest, published within the period of review, have been highlighted as:
• of special interest

1. World Health Organization: WHO Estimates of the Global Burden of Foodborne Diseases: Foodborne Disease Burden Epidemiology Reference Group 2007-2015. World Health Organization; 2015:255.

2. Bosch A, Gikogka E, Le Guyader FS, Loisy-Hamon F, Lee A, van Karantik L, Tengie VR, Neil FH, Blunt SE, Zheng X-L, Qu L et al.: Foodborne viruses: detection, risk assessment, and control options in food processing. Int J Food Microbiol 2018; 285:110-128.

3. Verhoef L, Hewitt J, Barclay L, Ahmed S, Lake R, Hall AJ, Lopman BA, Kroneman A, Venenema H, Vinje J et al.: Norovirus genotype profiles associated with foodborne transmission, 1999–2012. Emerg Infect Dis (Open Access) 2015; 21:592-599.

4. Ettayebi K, Crawford SE, Murakami K, Broughman JR, • Karandikar UJ, Tengie VR, Neil FH, Blunt SE, Zheng X-L, Qu L et al.: Replication of human noroviruses in stem cell-derived human enteroids. Science (New York, N.Y.) 2016; 353:1387-1393.

This is the only currently well-accepted human norovirus tissue culture model.

5. Lee SJ, Si J, Yun HS, Ko G: Effect of temperature and relative humidity on the survival of foodborne viruses during food storage. Appl Environ Microbiol 2015; 81:2075-2081.

6. van Dorremen N, Bushmaker T, Munster VJ: Stability of Middle East respiratory syndrome coronavirus (MERS-CoV) under different environmental conditions. Euro Surveill 2013; 18:20509.

7. Takahashi H, Ohuchi A, Miyas I, Izawa Y, Kimura B: Effect of food residues on norovirus survival on stainless steel surfaces. PLoS One 2011; 6:e21951.

8. Bae J, Schwab KJ: Evaluation of murine norovirus, feline calicivirus, poliovirus, and MS2 as surrogates for human norovirus in a model of viral persistence in surface water and groundwater. Appl Environ Microbiol 2008; 74:477-484.

9. Gundy PM, Gundy PM, Gerba CP, Gerba CP, Pepper IL, Pepper IL: Survival of coronaviruses in water and wastewater. Food Environ Virol 2009; 1:10-14.

10. Casanova L, Rutala WA, Weber DJ, Sobsey MD: Survival of surrogate coronaviruses in water. Water Res 2009; 43:1893-1898.

11. Costantini V, Morantz EK, Browne W, Ettayebi K, Zeng X-L, • Atmar RL, Estes MK, Vinje J: Human norovirus replication in human intestinal enteroids as model to evaluate virus inactivation. Emerg Infect Dis 2018; 24:1453-1464.

This is the only article we could find evaluating the human norovirus viability with the use of tissue culture model.

12. Walker CM, Ko Q: Effect of ultraviolet germicidal irradiation on • viral aerosols. Environ Sci Technol 2007; 41:5460-5466.

This is the only article we could find making direct comparison between coronavirus and norovirus surrogate (MS2) towards disinfection treatment.

13. Hosmillo M, Chaudhry Y, Nayak K, Sorgeloos F, Koo B-K, Merenda A, Lillestol R, Drumright L, Zibauer M, Goodfellow I: Norovirus replication in human intestinal epithelial cells is restricted by the interferon-induced JAK/STAT signaling pathway and RNA polymerase II-mediated transcriptional responses. mBio 2020; 11.

14. Zhang D, Tan M, Zhong W, Xia M, Huang P, Jiang X: Human intestinal organoids express histo-blood group antigens, bind norovirus VLPs, and support limited norovirus replication. Sci Rep 2017; 7:12621.

15. Teunis PFM, Moe CL, Liu P, Miller SE, Lindesmith L, Baric RS, Le Pendu J, Calderon RL: Norwalk virus: how infectious is it? J Med Virol 2008; 80:1468-1476.

16. Atmar RL, Estes MK: The epidemiologic and clinical importance of norovirus infection. Gastroenterol Clin North Am 2006; 35:275-290.

17. Ayukkongb J, Lindh M, Nenonen N, Tah F, Nkou-Akenji T, Bergström T, Institute of Biomedicine DöM, Sahlgrenska a, Institutionen för biomedicin af, Göteborgs u et al.: Enteric viruses in healthy children in cameron: viral load and genotyping of norovirus strains. J Med Virol 2011; 83:2135-2142.

18. Wu Y, Guo C, Tang L, Hong Z, Zhou J, Dong X, Yin H, Xiao Q, Tang Y, Qu X et al.: Prolonged presence of SARS-CoV-2 viral RNA in faecal samples. Lancet Gastroenterol Hepatol 2020; 5:434-435.

19. World Health Organization: Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19). World Health Organization: 2020;40.

20. Whitworth J: More than 1,000 Sick in France from Contaminated Raw Shellfish. Denver: Neweast: 2020 https://www.foodsafetynews.com/2020/01/ more-than-1000-sick-in-france-from-contaminated-raw-shellfish/.

21. Parrón I, Álvarez J, Jané M, Cornejo Sánchez T, Razoquin E, Guix S, Camps G, Pérez C, Domínguez A: A foodborne norovirus outbreak in a nursing home and spread to staff and their household contacts. Epidemiol Infect 2019; 147:e225.

22. Whitworth JJ: Oysters behind Norovirus Outbreak in New Zealand. Edited by 2017 https://www.foodnavigator-asia.com/Article/ 2017/02/07/Norovirus-outbreak-linked-to-oysters.

23. Morgan M, Watts V, Allen D, Curtis D, Kirollos A, Macdonald N, Maslen E, Morgan D, Saeli A, Sedgwick J et al.: Challenges of investigating a large food-borne norovirus outbreak across all branches of a restaurant group in the United Kingdom, October 2016. Eur Surveill 2019; 24:190051.

24. Sharma S, Hagbom M, Carlsson B, Nederby Öh d J, Insulander M, Eriksson R, Simonsson M, Widström M, Nordgren J: Secretor status is associated with susceptibility to disease in a large GI.6 norovirus foodborne outbreak. Food Environ Virol 2020; 12:28-34.

25. Cook N, Williams L, D’Agostino M: Prevalence of norovirus in produce sold at retail in the United Kingdom. Food Microbiol 2019; 79:85-89.

26. Gao X, Wang Z, Wang Y, Liu Z, Guan X, Ma Y, Zhou H, Jiang Y, Cui W, Wang L et al.: Surveillance of norovirus contamination in commercial fresh/frozen berries from Heilongjiang Province, China, using a TaqMan real-time RT-PCR assay. Food Microbiol 2019; 82:119-126.

27. Das O, Lekshmi M, Kumar S, Nayak BB: Incidence of norovirus in tropical seafood harbouring faecal indicator bacteria. Mar Pollut Bull 2020; 150:110777.

28. Suffredini E, Le OH, Di Pasquale S, Pham TD, Vicenza T, Losardo M, To KA, De Medici D: Occurrence and molecular
characterization of enteric viruses in bivalve shellfish marketed in Vietnam. Food Control 2020, 108:105828.

29. Tao J, Chunhui H, Fanning S, Nan L, Jiahui W, Hongyuan Z, Jing Z, Fengqin L: Norovirus contamination in retail oysters from Beijing and Qingdao, China. Food Control 2018, 86:415-419.

30. Pu J, Miura T, Kazama S, Konta Y, Azraini ND, Ito E, Ito H, Omura T, Watanabe T: Weekly variations in norovirus genogroup II genotypes in Japanese oysters. Int J Food Microbiol 2018, 294:48-55.

31. Kim A-N, Park SY, Bae S-C, Oh M-H, Ha S-D: Survival of norovirus surrogate on various food-contact surfaces. Food Environ Virol 2014, 6:182-188.

32. van Doremalen N, Bushmaker T, Morris DH, Holbrook MG, Gamble A, Williamson BN, Tamin A, Harcourt JL, Thornburg NJ, Gerber SI et al.: Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. N Engl J Med 2020.

This is a very recent report on the environmental stability of the pandemic SARS-CoV-2.

33. Cannon JL, Papafragkou E, Park GW, Osborne J, Jaykus L-A, Vinje J: Surrogates for the study of norovirus stability and inactivation in the environment: a comparison of murine norovirus and feline calicivirus. J Food Prot 2006, 69:2781-2785.

34. Chan KH, Peiris JSM, Lam SY, Poon LLM, Yuen KY, Seto WH: The effects of temperature and relative humidity on the viability of the SARS coronavirus. Adv Virol 2011, 2011:734690-734697.

35. Rabenau HF, Cinatl J, Morgenstem B, Bauer G, Preiser W, Doerr HW: Stability and inactivation of SARS coronavirus. Med Microbiol Immunol 2005, 194:1-6.

36. Dawson DJ, Paish A, Staffell LM, Seymour U, Appleton H: Survival of viruses on fresh produce, using MS2 as a surrogate for norovirus. J Appl Microbiol 2005, 98:203-209.

37. Yépez-Gómez MS, Gerba CP, Bright KR: Survival of respiratory viruses on fresh produce. Food Environ Virol 2013, 5:150-156.

38. Seitz SR, Leon JS, Schwab KJ, Lyon GM, Dowd M, McDaniels M, Abdulhafid G, Fernandez ML, Lindesmith LC, Baric RS et al.: Norovirus infectivity in humans and persistence in water. Appl Environ Microbiol 2011, 77:6884-6888.

39. Horm KM, D’Souza DH: Survival of human norovirus surrogates in milk, orange, and pomegranate juice, and juice blends at refrigeration (4 °C). Food Microbiol 2011, 28:1054-1061.

40. van Doremalen N, Bushmaker T, Karesh WB, Munster VJ: Stability of Middle East respiratory syndrome coronavirus in milk. Emerg Infect Dis 2014, 20:1263-1264.

41. Wang XW, Li JS, Jin M, Zhen B, Kong QX, Song N, Xiao WJ, Yin J, Wei W, Wang GJ et al.: Study on the resistance of severe acute respiratory syndrome-associated coronavirus. J Virol Methods 2005, 126:171-177.