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## Infection control for norovirus

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### Abstract

Norovirus infections are notoriously difficult to prevent and control, owing to their low infectious dose, high shedding titre, and environmental stability. The virus can spread through multiple transmission routes, of which person-to-person and foodborne are the most important. Recent advances in molecular diagnostics have helped to establish norovirus as the most common cause of sporadic gastroenteritis and the most common cause of outbreaks of acute gastroenteritis across all ages. In this article, we review the epidemiology and virology of noroviruses, and prevention and control guidelines, with a focus on the principles of disinfection and decontamination. Outbreak management relies on sound infection control principles, including hand hygiene, limiting exposure to infectious individuals, and thorough environmental decontamination. Ideally, all infection control recommendations would rely on empirical evidence, but a number of challenges, including the inability to culture noroviruses in the laboratory and the challenges of outbreak management in complex environments, has made it difficult to garner clear evidence of efficacy in certain areas of infection control. New experimental data on cultivable surrogates for human norovirus and on environmental survivability and relative resistance to commonly used disinfectants are providing new insights for further refining disinfection practices. Finally, clinical trials are underway to evaluate the efficacy of vaccines, which may shift the current infection control principles to more targeted interventions.

### Keywords

Disinfection; epidemiology; infection control; norovirus; nosocomial

### Introduction

Norovirus is a leading cause of acute gastroenteritis in people of all ages and settings. Approximately 19–21 million norovirus illnesses occur each year in the USA [1]. A high titre of shedding by infected persons, a low infectious dose and environmental stability are some of the attributes that facilitate effective norovirus transmission through a variety of modes (person-to-person, food, water, and environment) [2–5]. These attributes present an

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#### Transparency Declaration

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array of challenges for prevention and control, in particular in institutional settings [4,6]. Specialists involved with infection and environmental control use a range of strategies aimed at preventing and controlling norovirus outbreaks [7–9]. However, some of these measures, such as ward/unit closures in hospitals, can place a substantial burden on institutions and personnel; a UK study estimated a loss of *c.* \$1 million for every 1000 beds [10–12]. Ideally, outbreak management guidelines would be supported by high-quality empirical evidence. However, generating high-quality evidence for efficacy is difficult, as the evidence for outbreak management is largely empirical, and there are challenges associated with a non-cultivable virus. Here, we review the current knowledge of norovirus outbreak epidemiology and virology, and infection control guidelines, with a focus on disinfection and decontamination, and highlight areas for future research.

## Norovirus Outbreaks

### Settings

Outbreaks provide an opportunity to study norovirus epidemiology, including how these viruses spread and what control measures are effective. Outbreaks occur in the diverse range of settings where humans congregate. In the USA, outbreaks in restaurants and on cruise ships are frequently picked up by the media. However, one would have a skewed sense of the distribution of norovirus outbreak patterns from media reports alone. Data from broad-based surveillance in high-income countries show that the majority of outbreaks occur in healthcare facilities; however, the specific types of facility reporting outbreaks can differ between countries. In the USA, >60% of all norovirus outbreaks occur in long-term-care facilities [13,14]. This contrasts with the settings reported in Europe, Japan, and other high-income settings, where outbreaks in acute-care hospitals are common and roughly equal in number to outbreaks in long-term-care facilities (Fig. 1) [15]. In the USA, acute-care outbreaks are relatively uncommon, constituting *c.* 5% of norovirus outbreaks [13,16]. Whether the lower frequency of outbreaks reported from US hospitals represents a real difference in epidemiology or infection control, or an artefact of reporting bias, is not well understood.

### Modes of transmission

Although noroviruses have been detected in bovines, mice, and canines, these virus strains appear to be highly species-specific, and zoonotic transmission does not seem to be common. In humans, the virus typically spreads directly via person-to-person transmission (faecal–oral and vomit–oral) or indirectly through foodborne, waterborne and environmental transmission. Direct person-to-person transmission is reported in >90% of the norovirus outbreaks in healthcare facilities [6,13,17]. Food-borne, waterborne and environmental transmission have some features in common, in the sense that a food product, water source or fomite may become contaminated by an infected person, and another individual then ingests virus after coming into contact with that object. In the USA, norovirus is estimated to be the most common aetiological cause of foodborne illness, which accounts for 7–24% of norovirus outbreaks worldwide [13,14,18–20]. Although food may become contaminated at any point in the ‘farm to fork continuum’, the majority of foodborne norovirus illness is a result of contamination by infected food-handlers during preparation [21]. Ready-to-eat

foods (such as leafy greens) and foods handled after cooking are the most frequently identified products associated with outbreaks [21]. Each of these transmission modes presents specific challenges in terms of infection prevention and control, as discussed below.

The high levels of virus shed in faeces and vomit [2], the low infectious dose [3] and the environmental stability of the virus [4] all contribute to the ability of noroviruses to utilize various modes of transmission (Table 1). Furthermore, transmission has been reported to occur before the onset of symptoms [22], in the post-symptomatic period, and during subclinical infections [23]. However, the currently available evidence suggests that individuals are less infectious when they are asymptomatic, and that vomiting [23] is strongly associated with transmission [24].

### **Importance of genotyping noroviruses for understanding transmission**

Noroviruses are a group of genetically diverse single-stranded RNA viruses. There are six known genogroups (G), two of which (I and II) commonly cause human disease, and can be further subdivided into nine and 22 genotypes, respectively [5]. Immunity appears to be largely restricted to homotypic genogroups or genotypes [25]. This genetic diversity has public health relevance, in that certain genotypes are associated with different modes of transmission and, perhaps, severity of disease outcomes. Genogroup I viruses are more often associated with food and waterborne outbreaks (Fig. 2). For example, the recently emerged GI.6 virus is more often associated with foodborne disease [26]. Conversely, GII.4 viruses are strongly associated with person-to-person transmission and healthcare settings [27]. Factors that may promote GII.4 transmission in closed settings include a possibly longer duration of shedding [25], more frequent vomiting [28], and different environmental survival and disinfection resistance profiles [29,30]. Moreover, GII.4 infections are likely to be of greater severity and result in more hospitalizations and deaths than those caused by other GII or GI viruses, even after accounting for the different case mix of the populations affected by the different viruses (that is, GII.4 viruses primarily cause outbreaks among the elderly in institutionalized healthcare settings) [31].

## **Current Guidelines and the Evidence Base for their Efficacy**

### **Outbreak management**

Healthcare institutions provide services to vulnerable populations, and are the most common settings for norovirus outbreaks. For these reasons, these settings will constitute the focus of our discussions on infection control issues, but most of these principles also apply to other settings. Outbreak management is a multistage process: preparedness, identification, response, and evaluation [32]. An institutional structure conducive to organizing and timing the actions to prevent and control infection facilitates the containment of outbreaks [33]. The ability to identify a norovirus outbreak as early as possible is a key aspect in initiating infection control measures [8]. Although outbreak control measures are based on sound infection control principles [8,34], there are scant data to demonstrate that implementing specific infection control measures decrease the magnitude or duration of norovirus outbreaks [35]. Evaluating the effectiveness of infection control measures for norovirus

outbreaks is an important part of the process for developing evidence-based guidelines [7,35,36], but there are ethical and scientific challenges to conducting such studies [37].

Guidelines for managing norovirus outbreaks have been issued by public health agencies in several countries, including Australia, Ireland, the UK, and the USA [7,8,36,38,39]. Some guidelines, such as those from the UK and the USA, used systematic literature reviews followed by grading the strength of recommendations. Guidelines from other countries based their recommendations on a more expert opinion-driven approach to assessing evidence. Regardless of the methods used, recent guidelines are generally consistent in the measures that they recommend. The main approaches to preventing and containing norovirus outbreaks that are common across several guidelines include implementing policies concerning hand hygiene, patient isolation (separation of symptomatic patients) and cohorting (grouping of patients based on symptoms), staff exclusion from work, visitor restrictions, enhanced environmental cleaning and disinfection, and ward closures (Table 2) [7,8,33,36,38,39].

## Hygiene

A diverse set of recommendations for the prevention and control of norovirus outbreaks are needed, given the various transmission modes by which norovirus spreads and the lack of a 'magic bullet' to curtail transmission. In general, hand hygiene adherence should be actively promoted among healthcare personnel, patients and visitors in patient-care areas affected by outbreaks of norovirus gastroenteritis. During outbreaks, hands should be washed with soap and running water for a minimum of 20 s after providing care for patients with suspected or confirmed infection [7,8]. Data from several studies suggest that this method of hand hygiene is an effective intervention for reducing norovirus risk [7,40–42]. Despite widespread use, there is inconclusive evidence for the effectiveness of alcohol-based hand sanitizers for norovirus [29,43–45]. Therefore, during outbreaks, they should be used as an adjunct to hand-washing [8]. Aerosolization of noroviruses and close, direct contact with an infected individual contribute to the high risk of transmission [46]. Therefore, the use of appropriate personal protective equipment, i.e. gloves and masks, especially when cleaning up vomit, is another measure for limiting the further spread of norovirus infection to staff in healthcare facilities [7]. During an outbreak, personal protective equipment should be disposable and single-use [7,39].

## Cleaning and disinfection

Enhanced cleaning and disinfection protocols may control and prevent the spread of norovirus [47–49]. This includes increasing the frequency of cleaning and paying closer attention to high-traffic areas and frequently touched surfaces, including, for example, door handles and telephones [4,7,8]. For disinfection, a bleach solution at a minimum concentration of 1000 p.p.m. sodium hypochlorite prepared fresh daily is recommended [8]. The results from several studies have demonstrated that bleach effectively disinfects norovirus better than other products, i.e. quaternary ammonium-based products [50–53]. In areas where bleach is not available or is corrosive to materials, EPA-registered products, in particular List G, are available that can be effective against norovirus surrogates [54]. Cleaning and disinfection should proceed from unaffected areas to affected areas, with care

being taken to clean from low-contamination areas to high-contamination areas [36]. Steam cleaning can be considered for soft furnishings, i.e. rugs, carpets, chairs, and other fabrics, that are adversely affected by bleach [7,36].

### Isolation and cohorting

Isolation, cohorting (grouping of patients on the basis of symptoms) and exclusion of symptomatic staff, patients and visitors constitute another class of recommended strategies for infection control [7,8,33,36,38,39]. These strategies can prevent the amount of secondary transmission, and decrease the outbreak duration [55–59]. Although most guidelines recommend cohorting patients into groups on the basis of symptomatic, exposed asymptomatic and unexposed asymptomatic status [7,8,33,36,38,39], at a minimum, symptomatic patients should be isolated in a single ward or care unit in order to minimize secondary transmission [39]. Several guidelines stress that symptomatic patients should not be transferred to other wards/units within the facility or between facilities until at least 48 h after symptoms have been resolved, in order to reduce the spread of infection to unaffected areas or facilities [7,8,33,36,38]. To minimize the spread of norovirus between patient cohorts, healthcare institution staff should care for one patient cohort at a time, and movement of staff between patient cohorts should be limited. In particular, staff assigned to symptomatic patients should strictly adhere to all enhanced infection control policies [7]. Exclusion of staff members from work during illness and for at least 48 h after resolution of symptoms can reduce transmission to patients during the symptomatic and post-symptomatic phases of infection [60]. Sick pay and sick leave policies in healthcare institutions that do not penalize ill workers may help to prevent staff from working while infectious [8] but these measures may also lead to unintended consequences, such as staff shortages [11,61]. Minimizing access of visitors and non-essential personnel to affected areas and the exclusion of symptomatic visitors is strongly recommended. As visitors may not be knowledgeable about norovirus, facilities can provide educational material describing the risks of norovirus transmission and measures to prevent infection [7,33,39]. Finally, and perhaps most controversially, some guidelines recommend closing units, or parts thereof, to new admissions or transfers [7,8,33,36,38,39]. Most data suggest that ward closure is effective in terms of reducing the number of cases and the duration of outbreaks [12,46,62].

### Organizational structure and response

A common theme in several national guidelines is the value of an organizational structure within a healthcare institution that is capable of providing timely response to outbreaks [36,38,39]. One department that is accountable for identifying and implementing recommendations can streamline the initiation of protocols and infection control measures [63]. The reporting of norovirus outbreaks from healthcare institutions to appropriate public health authorities may assist in outbreak control and, ultimately, through collection of surveillance data, provide evidence supporting specific actions [7,8,33,36,38,39].

### Food-handling

Foodborne outbreaks arise from a variety of contamination points, i.e. during production, processing, preparation, or service. Infected food-handlers contaminating ready-to-eat food is the most common source of foodborne norovirus outbreaks [21]. Leafy vegetables, fruits,

and shellfish, all of which are commonly consumed raw or undercooked, are the food commodities most commonly reported as the cause of food-borne norovirus outbreaks [21,64]. Determining whether food is the cause of the outbreak as early as possible can facilitate the withdrawal of implicated food or the exclusion of infected food-handlers, hence limiting both primary food exposures and the secondary spread of norovirus infection [21,64]. Contaminated food and exposed utensils should be removed and appropriately disinfected, as should contaminated common areas such as dining halls [8,9,39]. Like healthcare workers, food-handlers should remain off work for at least 48 h after symptom resolution [65,66]. Ensuring that staff involved in food preparation, storage and serving adhere to the US Food and Drug Administration Food Code is important in preventing foodborne norovirus outbreaks [8,9,34]. Two key infection control measures specific to food-service settings include eliminating bare-handed contact with ready-to-eat foods and the presence of certified kitchen managers with food safety training [9].

### Implications of Environmental Stability of Human Norovirus

The infectiousness of norovirus outside the human host is influenced by intrinsic characteristics of the virus, such as physiochemical properties (thermal and desiccation resistance) and extrinsic characteristics (surface types). Norwalk virus seeded into ground water for at least 61 days was still able to infect human volunteers [67]. Although human noroviruses cannot yet be cultured *in vitro* [68], cultivable viruses, i.e. coliphage MS2 (MS2), murine norovirus (MNV), and feline calicivirus (FCV), have been used widely to assess the use of physiochemical abilities to predict the infectivity of human norovirus [69–71]. Such surrogate-based studies have estimated that human norovirus could stay potentially infectious on frozen foods (less than or equal to  $-20^{\circ}\text{C}$ ), refrigerated foods ( $10^{\circ}\text{C}$ ) and fomites for up to 6 months [72,73], up to 7 days [74,75], and 7 days [76], respectively. Robust stability ( $<1 \log_{10}$  of infectivity loss for 1 h of contact) of virus on hands was also demonstrated in an *in vivo* study with FCV and MNV [77]. Additionally, norovirus can be easily transferred between hands and surfaces through casual contact, which probably contributes to the spread of norovirus in the community [3,78,79].

### Further considerations on norovirus interventions

The basis for recommending washing of hands with soap is that soap, in several *in vivo* experiments, has been demonstrated to be more effective in removing viruses from hands than topical agents (e.g. alcohol-based hand sanitizers) [41,45]. However, few data are available on the level and frequency of contamination on hands from infected individuals, and it therefore remains uncertain whether hand-washing alone is sufficient to reduce the risk [80]. Also, hand-washing compliance is a general issue, with implications for a range of healthcare-associated pathogens, and not only norovirus. Alcohol-based hand sanitizers may be used as an adjunct but not as a substitute for hand-washing during norovirus outbreaks [7,81]. A number of studies have supported the virucidal activity of alcohol-based hand sanitizers against human norovirus and multiple surrogates [29,43,52,77]; other active ingredients (e.g. benzalkonium chlorite (Quat), triclosan, or chlorhexidine) were ineffective [29]. However, the clinical value (i.e. effectiveness) of alcohol-based sanitizers is a function of both: (i) their ability to inactivate viruses (i.e. efficacy), which depends both on the

formulation and on the way they are tested *in vitro* or *in vivo* [29,41,77,82,83]; and (ii) compliance, which includes both the frequency of use and proper application [34]. Overall, the lack of data on real-world effectiveness makes it difficult to generalize claims simply based on *in vitro* or *in vivo* experiments on a particular formulation. More comprehensive studies are warranted.

There is little information on the bio-burden of norovirus on hard surfaces, but recent data have shown that the surfaces of a few high-contact objects (i.e. doorknobs, toilet seats, and faucets) can be contaminated with up to  $10^4$  virus particles per object (unpublished data), which strongly suggests that reductions in levels of 3–4  $\log_{10}$  are required to eliminate norovirus contamination on high-contact surfaces [7,8]. The use of sodium hypochlorite solution (1000 p.p.m.) remains reliable for achieving a higher than 3  $\log_{10}$  reduction of human norovirus on surfaces, but pre-cleaning before its application is strongly recommended, to reduce the faecal organic load [7,8,48]. EPA-registered products claimed to have efficacy against human norovirus (e.g. List G) can be considered as alternative options. However, care should be taken, as FCV, which is officially used for claims of efficacy against human norovirus in EPA-registered products [54], is not the most resistant surrogate virus for predicting inactivation of human norovirus [29,71,86,87]. However, some recent EPA-registered products, which claimed norovirus antiviral activity, provided additional efficacy information against other norovirus surrogate viruses, such as MNV. The utilization of multiple norovirus surrogates demonstrating efficacy against norovirus can allow for a more conservative selection of appropriate disinfectants. In addition, the EPA test protocol allows for a longer duration of contact between disinfectant and inoculum (usually 5 min), whereas a shorter exposure time (1–3 min or shorter) is the practice more likely to be used in the field, potentially reducing the efficacy of these disinfectants [84,85]. Thus, strict compliance with the manufacturer's instructions is strongly advised to achieve the claimed efficacy.

Contaminated hands and surfaces may both contribute to norovirus transmission via regular interactions between hands and their surroundings, and hand and surface interventions should therefore complement each other. It is important to note the limitations of these traditional hygiene interventions. In particular, if sufficient decontamination is achieved, surfaces in areas of high contamination risk (e.g. toilets) are susceptible to recontamination by contact with affected or asymptomatic carriers. However, the effects of surface disinfection or hand-washing are transient, because commercial chemical disinfectants do not have any residual antimicrobial activity [4]. In addition to having proven effectiveness against norovirus, chemical disinfectants must also satisfy other requirements, such as low toxicity for personnel, and a low risk of damaging contaminated surface materials [88]. Novel disinfection methods are being considered as alternatives or complements to traditional hygiene interventions, but further research is needed (Table 3) [89–94].

## Future Research

Advances in disinfection technology for environmental and food safety use may direct updated guidelines for infection control practices [95,96]. A successful technology, such as high hydrostatic pressure, may have the potential for use in food safety [97–100]. Short of

developing a norovirus cell culture system, norovirus surrogates such as Tulane virus, porcine enteric calicivirus, MNV and FCV may help in better assessment of the efficacy of cleaning and disinfection practices [98,100]. Future studies should be directed towards quantitative assessment of norovirus contamination at each stage of the infection transmission cycle. Carefully designed observational studies or, preferably, intervention trials may help to answer the question of whether cohorting and/or unit closures alone or in conjunction with other strategies, i.e. cleaning and disinfection, are effective at controlling norovirus outbreaks. Progress is also being made in the development of a norovirus vaccine [101–105]. Accordingly, there are a number of possible strategies (e.g. vaccinating healthcare workers or nursing home residents) that will require careful evaluation. None of these developments in infection prevention will happen in isolation, so the costs and benefits of both individual interventions and combinations should be assessed [10,106].

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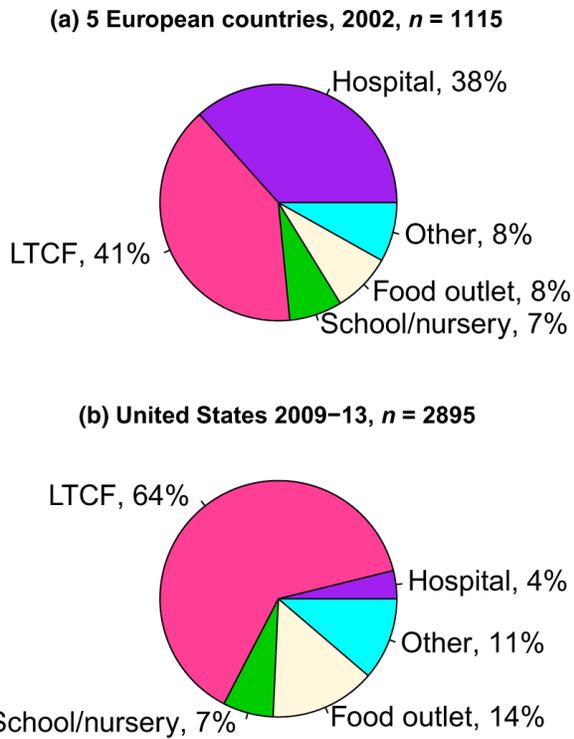
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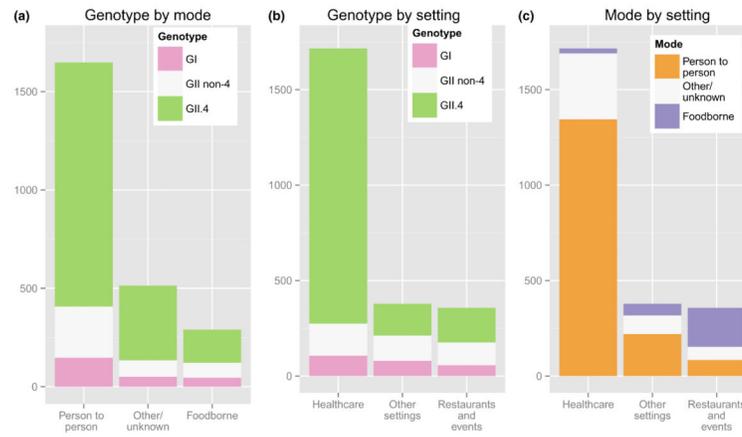
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**FIG. 1.** Setting of (a) norovirus outbreaks reported in five European countries with broad-based surveillance, 2002,  $n = 1115$ , and (b) the USA, 2009–2013,  $n = 2895$ . Long Term Care Facility (LTCF). Adapted from Lopman *et al.* [15] and Vega *et al.* [13].



**FIG. 2.** Distribution of norovirus genotype (GI, GII.4, GII non-4) by mode of transmission (a) and by outbreak setting (b), as well as mode of transmission by outbreak setting (c), from 2895 norovirus outbreaks reported to CaliciNet, 2009–2013. Adapted from Vega *et al.* [13].

TABLE 1

## Characteristics that facilitate norovirus transmission

Characteristics	Description
Low infectious dose	Estimates of the infectious dose ranges from 18 to 10 <sup>3</sup> virus particles [3]
High shedding titre	Peak shedding ranges from 10 <sup>5</sup> to 10 <sup>9</sup> particles/g of stool [2]
Prolonged shedding	Virus can be detected up to 8 weeks after symptom onset, with a median of 4 weeks; even longer durations of shedding may be detected in immunocompromised individuals [2,107]
Genetic diversity	Over 30 genotypes (nine GI and 22 GII) infect humans [5]. No long-lasting immunity [25,108]. Different genotypes can infect humans over their lifetime [25]
Environmental stability	Norovirus particles may be infectious for 2 weeks on environmental surfaces and for >2 months in water [67,109]
Resistant to common disinfectants	Surrogates used to determine the efficacy of EPA-registered disinfectant products have different physiochemical properties; therefore, different disinfection profiles exist, and overestimate the efficacy of disinfectant products [29,87]
Vomiting	Vomiting appears to be a particularly effective route of norovirus spread. Vomiting events may occur and lead to direct transmission (when in public) as well as environmental contamination from vomit droplets [59,110]
Transmission through multiple routes	Noroviruses are transmitted via the faecal–oral route and vomit–oral route, and through a number of specific modes, including foodborne, waterborne, environmental and direct person-to-person spread [6,13,21,57,64]

Adapted from [4].

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TABLE 2

Summary of infection control guidelines for the prevention and management of norovirus outbreaks in healthcare settings

Infection control category	Infection control strategy
Outbreak identification	<p>Define start of outbreak: enables the initiation of enhanced norovirus infection control measures</p> <p>Two or more associated patients with gastroenteritis onsets within 24–48 h of each other</p> <p>Use Kaplan's clinical and epidemiological criteria to identify norovirus outbreak, if clinical laboratory testing is not available</p> <p>Stool negative for bacteria</p> <p>Mean duration of illness of 12–60 h</p> <p>Vomiting in &gt;50% of cases</p> <p>Incubation period of 24–48 h</p>
Hygiene	<p>Wash hands with soap and warm running water for a minimum of 20 s before and after contact with patients, after using the lavatory, and/or before and after eating</p> <p>Wear appropriate personal protective equipment (PPE)</p> <p>Gloves: if directly contacting symptomatic patients</p> <p>Masks: if a potential risk of aerosolization, e.g. vomit, exists</p> <p>Gowns: if a potential risk of splashing exists</p> <p>Goggles/face shields: if a potential risk of splashing exists</p> <p>Change PPE frequently</p>
Cleaning and disinfection	<p>Increase the frequency of cleaning and disinfection of high-traffic areas and implicated areas</p> <p>Clean and disinfect from unaffected to affected areas</p> <p>Clean areas of any organic material</p> <p>Disinfect all surfaces with freshly prepared 0.1% (1000 p.p.m.) sodium hypochlorite (bleach)</p> <p>Clean carpets with detergent and warm water, and follow this with steam cleaning</p> <p>Steam-clean all soft furnishings that may be damaged by bleach</p> <p>Discard all disposable cloths in biohazard bags</p> <p>Launder all non-disposable cloths, i.e. linens, blankets, towels, and clothing</p>
Patient isolation/cohorting and transfer	<p>Separate patients on the basis of symptomatic, exposed asymptomatic, or unexposed asymptomatic</p> <p>Limit movement and transfer of symptomatic patients</p>
Staff exclusion and cohorting	<p>Exclude ill staff for at least 48 h after symptom resolution</p> <p>Assign staff to one patient cohort</p>
Visitors	<p>Limit visits to implicated wards</p> <p>Limit symptomatic visitors until 48 h after symptom resolution</p> <p>Provide educational material that describes the risks of norovirus transmission and measures to prevent infection</p>
Ward closures	<p>Consider closing the unit or ward to new admissions and transfers</p>
Outbreak reporting	<p>Notify appropriate local or state health departments, as per local and state public health regulations</p>
Food safety	<p>Discard exposed food</p> <p>Exclude ill staff for at least 48 h after symptom resolution</p> <p>Close communal dining areas</p> <p>Ensure proper food preparation, storage, and</p>

Infection control category	Infection control strategy
	serving Eliminate bare-handed contact with ready-to-eat foods

Adapted from [7], [8], [36], and [39].

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TABLE 3

List of available alternative surface disinfection technologies for human noroviruses

Disinfectant or disinfection process	Proposed application	Efficacy
Fluorinated titanium dioxide film [95]	Self-sanitizing surface	Antimicrobial activity of fluorinated titanium dioxide (TiO <sub>2</sub> )-coated coupons are activated by fluorescent light. After 60 min of exposure to fluorescent light (10 μW/cm <sup>2</sup> ), fluorinated TiO <sub>2</sub> -coated coupons reduced the infectivity of MS2 <sup>a</sup> , FCV <sup>b</sup> and MNV-1 <sup>c</sup> by 1.7, 2.6 and 2.6 log <sub>10</sub> , respectively
Gaseous ozone [92]	Decontamination of larger surface areas	Gaseous ozone at 20–25 p.p.m. inactivated FCV by 5 log <sub>10</sub> after 20 min of exposure. The efficacy of gaseous ozone was not influenced by the test room size (34–47.6 m <sup>3</sup> ), location of viral contamination, or surface type
Hydrogen peroxide gas [91]	Decontamination of larger surface areas	Hydrogen peroxide gas (12% hydrogen peroxide) inactivated MNV-1 by >3 log <sub>10</sub> in a test room (7 × 5 × 2.7 m <sup>3</sup> ). The surface disinfection efficiency was not influenced by location of viral contamination or surface type
Super-oxidized water (hypochlorous acid) [90]	Food contact sanitizer	Super-oxidized water (hypochlorous acid solution: 188 p.p.m. Cl <sub>2</sub> , pH between 5.5 and 6.2), generated electrolytically from a dilute NaCl solution, inactivated MS2 dried on coupons (stainless steel and ceramic tile) by 3 log <sub>10</sub> after 1 min of contact time
Saturated steam vapour [93]	Food contact surfaces	Saturated steam vapour by VaporJet 2400 (Advanced Vapor Technologies, Seattle, WA, USA) inactivated MS2 dried on clay coupons by >3 log <sub>10</sub> after 2 s of exposure
Steam–ultrasound [94]	Food contact surfaces	Steam (130°C) in combination with ultrasound (30–40 kHz) applied with the SonoSteam <sup>®</sup> technique inactivated FCV and MS2 by >4 log <sub>10</sub> after 1 s, and MNV-1 by 3.7 log <sub>10</sub> after 3 s

<sup>a</sup>Coliphage MS2, a non-enveloped, (+) single-stranded RNA virus, classified in family *Leviviridae*, genus *Levivirus*; a model strain for human enteric viruses [69].

<sup>b</sup>Feline calicivirus, a non-enveloped, (+) single stranded RNA virus, classified in family *Caliciviridae*, genus *Vesivirus*; a surrogate for human norovirus [71].

<sup>c</sup>Murine norovirus, a non-enveloped, (+) single stranded RNA virus, classified in family *Caliciviridae*, genus *Norovirus*; a surrogate for human norovirus [70].