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MINIREVIEW

Significance of Fomites in the Spread of Respiratory and Enteric Viral Disease

Stephanie A. Boone* and Charles P. Gerba

Department of Soil, Water and Environmental Science, University of Arizona, Tucson, Arizona 85721

Worldwide annually there are 1.7 million deaths from diarrheal diseases and 1.5 million deaths from respiratory infections (56). Viruses cause an estimated 60% of human infections, and most common illnesses are produced by respiratory and enteric viruses (7, 49). Unlike bacterial disease, viral illness cannot be resolved with the use of antibiotics. Prevention and management of viral disease heavily relies upon vaccines and antiviral medications (49). Both vaccines and antiviral medications are only 60% effective (39, 49). Additionally, to date there are no vaccines or antiviral drugs for most common enteric and respiratory viruses with the exception of influenza virus and hepatitis A virus (HAV). Consequently, viral disease spread is most effectively deterred by preclusion of viral infection.

Increases in population growth and mobility have enhanced pathogen transmission and intensified the difficulty of interrupting disease spread (14). Control of viral disease spread requires a clear understanding of how viruses are transmitted in the environment (27). For centuries it was assumed that infectious diseases were spread primarily by the airborne route or through direct patient contact, and the surrounding environment played little or no role in disease transmission (19, 27). Up until 1987 the Centers for Disease Control and the American Hospital Association focused on patient diagnosis due to the belief that nosocomial infections were not related to microbial contamination of surfaces (19). Over the years studies have changed the perspective on viral transmission to include a more complex multifactorial model of disease spread (27). There is now growing evidence that contaminated fomites or surfaces play a key role in the spread of viral infections (3, 7, 38, 71).

Viral transmission is dependent on interaction with the host as well as interaction with the environment (60). Viruses are probably the most common cause of infectious disease acquired indoors (7, 71). The rapid spread of viral disease in crowded indoor establishments, including schools, day care facilities, nursing homes, business offices, and hospitals, consistently facilitates disease morbidity and mortality (71). Yet, fundamental knowledge concerning the role of surfaces and objects in viral disease transmission is lacking, and further investigation is needed (52, 60, 61). The goal of this article was to use existing published literature to assess the significance of fomites in the transmission of viral disease by clarifying the role of fomites in the spread of common pathogenic respiratory and enteric viruses.

ROLE OF FOMITES IN VIRAL DISEASE TRANSMISSION

Fomites consist of both porous and nonporous surfaces or objects that can become contaminated with pathogenic microorganisms and serve as vehicles in transmission (Table 1) (24, 31, 58, 63, 66). During and after illness, viruses are shed in large numbers in body secretions, including blood, feces, urine, saliva, and nasal fluid (10, 33, 34, 39, 48, 58). Fomites become contaminated with virus by direct contact with body secretions or fluids, contact with soiled hands, contact with aerosolized virus (large droplet spread) generated via talking, sneezing, coughing, or vomiting, or contact with airborne virus that settles after disturbance of a contaminated fomite (i.e., shaking a contaminated blanket) (22, 24, 27, 58, 66). Once a fomite is contaminated, the transfer of infectious virus may readily occur between inanimate and animate objects, or vice versa, and between two separate fomites (if brought together) (27, 66). The Panic study (52) recovered 3 to 1,800 PFU of rhinovirus from fingertips of volunteers who handled contaminated doorknobs or faucets. Using coliphage PRD-1 as a model, Rusin et al. (60) demonstrated that 65% of virus could be transferred to uncontaminated hands and 34% to the mouth. The nature and frequency of contact with contaminated surfaces vary for each person depending on age, personal habits, type of activities, personal mobility, and the level of cleanliness in the surroundings (66). Viral transfer and disease transmission is further complicated by variations in virus survival on surfaces and the release of viruses from fomites upon casual contact (24, 66). Virus survival on fomites is influenced by intrinsic factors which include fomite properties or virus characteristics and extrinsic factors, including environmental temperature, humidity, etc. (Fig. 1) (24, 66). If viruses remain viable on surfaces long enough to come in contact with a host, the virus may only need to be present in small numbers to infect the host (10, 58, 66, 71). After contact with the host is achieved, viruses can gain entry into the host systems through portals of entry or contact with the mouth, nasopharynx, and eyes (10, 24, 58, 66). Host susceptibility to viruses is influenced by previous contact with the virus and the condition of the host immune system at the time of infection (27).
There are many complex variables that influence virus survival on fomites, viral transfer from fomites, and viral infection of the host (7, 10, 24, 66). As a result, direct experimental evidence of viral transmission via fomite has been very difficult to generate due to a variety of uncontrollable variables and the unpredictability of human infection (7, 66). An example of the difficulty in producing illness in the host after exposure was demonstrated that viruses can remain infective on surfaces for different time periods (1, 2, 9, 13, 33, 48, 64, 68). The length of time a virus remains viable depends on a number of complex variables (Fig. 2). In general, UV exposure and pH have minimal effects on viral survival in indoor environments. Viral survival may increase or decrease with the number of microbes present on a surface. Increasing amounts of microbes can protect viruses from desiccation and disinfection, but deleterious effects may also result from microbial proteases and fungal enzymes (67, 69). Typically, viral presence on fomites may decrease with surface cleanliness and increase with surface usage (66). However, some cleaning products or disinfectants are ineffective against viruses and can result in viral spread or cross-contamination of surfaces (8). Easily measured and predictable factors that influence viral survival on surfaces include fomite properties, initial viral titer, virus strain, temperature, humidity, and suspending medium (66, 69).

Intrinsic factors, like fomite properties, virus strain, and viral inoculation titer, consistently impact the total virus survival end point (hours, days). The majority of viruses remain viable longer on nonporous surfaces (Tables 2 and 3); however, there are exceptions (1, 27). Astrovirus survives for 90 days on porous paper but only 60 days on nonporous aluminum (2). Initial inoculation titer can prolong viral survival on environmental surfaces (66). Brady et al. (13) found that the viral survival decay rate increased with inoculum titer: a 10⁴ virus
inoculation could be detected up to 6 h longer than a $10^3$ virus inoculation. Virus survival on fomites can also vary significantly within viral type and strain. Typically, nonenveloped enteric viruses remain viable longer on surfaces than enveloped respiratory viruses. The enteric viruses HAV, astrovirus, and rotavirus can all remain infective on surfaces for 2 months or longer (Table 3). In contrast, respiratory viruses usually remain viable for several hours to several days (Table 2). Virus inactivation rates can be expressed as the log decay of virus titer divided by the total time of viral survival. For comparative purposes, we calculated inactivation coefficients ($K_i$) using the following calculation after all viral titers were normalized to the 50% tissue culture infective dose (TCID$_{50}$) per ml of virus: 

$$\frac{\log_{10} \text{reduction (initial viral titer} - \text{final viral titer)/ml}}{\text{total hours of viral viability}}$$

Inactivation coefficients are linear functions and were not used to calculate $T_{90}$ or $T_{99}$ values, these values were calculated using the viral survival curve, which is typically not linear. Therefore, $T_{90}$ and $T_{99}$ values underestimate viral survival compared to inactivation coefficients ($K_i$) values. On nonporous surfaces, the enteric viruses reviewed typically exhibited inactivation rates at least 2 logs lower than respiratory viruses, with the exception of adenovirus and influenza virus (Fig. 2 and 3; Tables 2 and 3). Four out of five enteric viruses examined in this review produced inactivation coefficients between 0.0021 and 0.0059 log$_{10}$/h, whereas four out of five respiratory viruses produced inactivation rates between 0.167 and 0.625 log$_{10}$/h. The higher inactivation coefficient found among the respiratory viruses indicates a faster decay rate or decreased survival on surfaces (Fig. 2 and 3). Variations in virus survival may also occur within a viral family or strain (66, 71), as seen between the 12-h survival of coronavirus 229E and the 3-h survival of coronavirus OC43 (Table 2). Consequently, variations in fomite composition, initial viral inoculation, and virus type can dramatically influence the amount of time the virus survives on a surface.

Extrinsic environmental factors, such as temperature, humidity, and surrounding viral medium, have a varying effect on viral decay rate, depending on the viral strain. In the Abad et al. study (1), media changes had no noticeable effect on enteric virus survival (HAV and rotavirus); however, medium changes adversely affected the survival of adenovirus. Changes in viral suspension medium from tryptose phosphate broth to nasal discharge decreased rhinovirus survival in research by Sattar et al. (63) (Table 2). Additionally, Abad et al. (1) demonstrated that temperature and humidity variations had no effect on the survival (60 days) of HAV and rotavirus (Table 3). However, temperature variations from 4°C to 20°C decreased the survival of astrovirus ($T_{90}$ change from 8 days to <24 h) and feline calicivirus ($T_{90}$ change from 10 days to <24 h) (2, 21). Humidity influences the viral desiccation rate. Humidity in the United States can range from 14 to 94% in outdoor environments (76). Indoor humidity varies depending on outdoor humidity, temperature, and varying indoor factors (76). Abad et al. (1) found that decreases in humidity could negatively impact HAV, rotavirus, and adenovirus survival (Table 3). Humidity variations in the Abad et al. study (1) caused a significant decline in HAV survival ($T_{90}$ change from 35 days at 85% humidity to 11 days at 45% humidity). The majority of studies investigating the effects of humidity on respiratory viruses are aerosol studies. However, Sattar et al. (64) was one of the few studies that investigated respiratory virus survival on surfaces in which humidity was used as a variable. The study found that rhinovirus exhibits optimum survival at 50% humidity (Table 2) (64).

### LABORATORY EVIDENCE OF RESPIRATORY VIRUS TRANSMISSION VIA FOMITES

Several different viruses cause respiratory infections, including respiratory syncytial virus (RSV), human parainfluenza virus (1 thru 4) (HPIV), influenza virus (A and B), human coronavirus (SARS, OC43, and 229E), rhinovirus, and adenovirus (serotypes 4 and 7) (18). It is generally accepted that respiratory viruses are spread person to person via aerosol transmission (7, 27). Nevertheless, current scientific evidence also suggests that fomites are an important vehicle in the spread of respiratory viruses (7). By using an aerosolized source, HPIV1 was found to infect only 2 of 40 children at a
distance of 60 cm (37). Therefore, HPIV transmission by aerosol was considered improbable; however, transmission may have taken place by surface contamination or close contact (37). Respiratory viruses cause sneezing and coughing, which expel an estimated $10^7$ infectious virions per ml of nasal fluid (18). Nasal secretions can travel at a velocity of over 20 m per second and a distance greater than 3 m (about 10 feet) to contaminate surrounding fomites (42, 57, 78).

Viruses have been isolated on fomites in day care centers and homes (influenza A virus) (12), offices (parainfluenza virus) (S. A. Boone and C. P. Gerba, submitted for publication), and hospitals (coronavirus, parainfluenza virus, and RSV) (23) using PCR. A hospital in Taiwan used reverse transcriptase PCR to detect coronavirus on hospital phones, doorknobs, computer mouses, and toilet handles during an outbreak of severe acute respiratory syndrome (SARS) (23). Studies have proven that RSV, HPIV, influenza virus, coronavirus, and rhinovirus can remain viable on fomites for several hours to several days (Tables 1 and 3) (5, 7, 9, 51). Avian influenza virus was detected on several surfaces for over 6 days (73).

### Table 2. Experimental conditions for studies assaying survival of respiratory viruses on fomites

<table>
<thead>
<tr>
<th>Virus (reference)</th>
<th>Suspension medium</th>
<th>Temp (°C)</th>
<th>% Humidity</th>
<th>Fomite</th>
<th>Survival (h)</th>
<th>$K_r$</th>
<th>$T_{90}$ (h)</th>
<th>$T_{99}$ (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Influenza A virus (9)</td>
<td>Dulbecco’s PBS$^b$</td>
<td>27.8–28.3</td>
<td>35–40</td>
<td>Stainless steel</td>
<td>72</td>
<td>0.0278</td>
<td>30</td>
<td>47</td>
</tr>
<tr>
<td>Influenza B virus (9)</td>
<td>Dulbecco’s PBS</td>
<td>26.7–28.9</td>
<td>55–56</td>
<td>Stainless steel</td>
<td>72</td>
<td>0.0417</td>
<td>12</td>
<td>37</td>
</tr>
<tr>
<td>Avian influenza virus (73)</td>
<td>EMEM$^d$ with Earle’s salts</td>
<td>Room temp</td>
<td>Not specified</td>
<td>Stainless steel</td>
<td>144</td>
<td>0.0138</td>
<td>0</td>
<td>48</td>
</tr>
<tr>
<td>Coronavirus 229E (64)</td>
<td>Dulbecco’s PBS</td>
<td>21</td>
<td>55–70</td>
<td>Aluminum</td>
<td>12</td>
<td>0.167</td>
<td>5.25</td>
<td>9</td>
</tr>
<tr>
<td>Coronavirus OC43 (64)</td>
<td>Dulbecco’s PBS</td>
<td>21</td>
<td>55–70</td>
<td>Aluminum</td>
<td>3</td>
<td>0.25</td>
<td>1.5</td>
<td>3</td>
</tr>
<tr>
<td>Parainfluenza virus 2 (12)</td>
<td>MEM$^d$</td>
<td>22</td>
<td>Not specified</td>
<td>Stainless steel</td>
<td>10</td>
<td>0.5</td>
<td>3.75</td>
<td>6</td>
</tr>
<tr>
<td>Respiratory syncytial virus (33)</td>
<td>MEM$^d$ with pooled nasal secretions</td>
<td>22.25–25.25</td>
<td>35–50</td>
<td>Formica countertop</td>
<td>8</td>
<td>0.625</td>
<td>2.8</td>
<td>3.3</td>
</tr>
<tr>
<td>Rhinovirus 14 (61)</td>
<td>TPB$^e$ or nasal secretions</td>
<td>22</td>
<td>15–25</td>
<td>Steel disc w/ TPB</td>
<td>≥25</td>
<td>&lt;0.2</td>
<td>25</td>
<td>&gt;25</td>
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<td></td>
<td></td>
<td>45–55</td>
<td>Steel w/ nasal discharge</td>
<td>≥25</td>
<td>1.25</td>
<td>2.5</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>75–85</td>
<td>Steel w/ TPB</td>
<td>≥25</td>
<td>&lt;0.2</td>
<td>25</td>
<td>&gt;25</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Steel w/ nasal discharge</td>
<td>≥25</td>
<td>0.625</td>
<td>6</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Steel w/ TPB</td>
<td>≥25</td>
<td>&lt;0.2</td>
<td>25</td>
<td>&gt;25</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Steel w/ nasal discharge</td>
<td>≥25</td>
<td>0.625</td>
<td>4</td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

$^a$ Inactivation coefficient ($K_r$) = $\log_{10}$ reduction in virus titer per ml = (initial viral titer – final viral titer)/survival (in hours) (64).

$^b$ PBS, phosphate buffer solution.

$^c$ EMEM, Eagle’s minimal essential medium.

$^d$ MEM, minimal essential medium.

$^e$ TPB, tryptose phosphate broth.

$^f$ ND, not done due to lack of data.

have demonstrated that RSV, influenza virus, parainfluenza virus, and rhinovirus can survive on hands for significant periods of time and that these viruses can be transferred from hands and fingers to fomites and back again (Tables 1 and 2) (5, 7, 33, 51). After a 10-second exposure, 70% of rhinovirus was transferred from donor to recipient hands in the 1978 study by Gwaltney et al. (30). Also, Gwaltney et al. demonstrated that subjects with cold symptoms had rhinovirus on their hands, and the virus was recovered from 43% of the plastic tiles they touched (30). Contaminated hands frequently come into contact with portals of entry, and so the potential for viral infection from contaminated fomites and hands exists. A study by Hendley et al. (36) found that 1 in 2.7 hospital grand round attendees rubbed their eyes and 33% picked their nose within a 1-hour observation period (36). Indirect evidence from clinical and laboratory studies clearly supports the involvement of fomites in respiratory virus infection. However, direct evidence supporting respiratory virus transmission or infection is still scarce. A study by Gwaltney et al. (29) observed that 50% of subjects developed infections after han-
duling a coffee cup contaminated with rhinovirus. The study also demonstrated that rhinovirus self-inoculation can result from rubbing the nasal mucosa with contaminated fingers and could lead to infection (29).

**LABORATORY EVIDENCE OF ENTERIC VIRUS TRANSMISSION VIA FOMITES**

Enteric viruses which cause gastrointestinal symptoms include rotavirus, adenovirus (serotypes 40 and 41), astrovirus, calicivirus (norovirus and sapoviruses), and HAV (40, 41). However, gastrointestinal symptoms like nausea and vomiting are found at a lower frequency in hepatitis A virus infections (74). Enteric viruses spread by the fecal-oral route. In many disease outbreaks viral transmission occurs via contaminated surfaces (1, 2). It has been estimated that one single vomiting incident may produce an estimated 30 million viral particles (7, 39, 61). In addition, at the peak of an enteric virus infection, more than $10^{11}$ virions per gram may be excreted in the stool (2, 6, 7, 59, 61, 77). Contamination of fomites from enteric viruses can originate from aerosolized vomit or the transfer of vomit and fecal matter from hands to surfaces (7, 59, 61). Viruses aerosolized from flushing the toilet can remain airborne long enough to contaminate surfaces throughout the bathroom (27). Enteric viruses have been detected in carpets, curtains, and lockers, which can serve as viral reservoirs (39). Surfaces contaminated (e.g., knives or sinks) by virus-infected individuals during food preparation have been documented to be the source of several food-borne outbreaks (53).

Studies on virus survival have indicated that enteric viruses are viable for at least 45 days on nonporous fomites (Table 3). A study by Fischer et al. found that rotavirus stored in feces remained infective for 2.5 months at 30°C and 32 months at 10°C (25). In addition, norovirus, adenovirus, and rotavirus have all been isolated from naturally contaminated fomites. Norovirus has been detected on fomites in hotels, hospital wards, and cruise ships during outbreaks of gastroenteritis (7, 61). GII norovirus and HAV RNA were detected on nonporous surfaces for over 21 days using real-time PCR (J. H. Park, D. H. D. Souza, P. Lui, C. L. Moe, and L. A. Jaykus, unpublished data). Adenovirus has been isolated on drinking glasses from bars and coffee shops, and rotavirus was detected on 16 to 30% of fomites in day care centers (7, 15, 24). Very small amounts of enteric virus (e.g., norovirus, estimated at 10 to 100 virions) can cause infection, with many viral infections being largely asymptomatic or subclinical in healthy adults (7, 59, 61). As a result, viral shedding onto surfaces or the spreading of virions into the environment by infected individuals can go on undetected (6–8, 39).

**TABLE 3. Experimental conditions for studies assaying survival of enteric viruses on fomites**

<table>
<thead>
<tr>
<th>Virus (reference(s))</th>
<th>Suspension medium</th>
<th>Fomite</th>
<th>Temp (°C)</th>
<th>% Humidity</th>
<th>PBS or BEM*</th>
<th>FS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis A virus (1, 48)</td>
<td>PBS or FS</td>
<td>Alum.</td>
<td>4</td>
<td>85–90</td>
<td>&gt;60</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alum.</td>
<td>20</td>
<td>85–90</td>
<td>&gt;60</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paper</td>
<td>20</td>
<td>45–55</td>
<td>&gt;60</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paper</td>
<td>4</td>
<td>85–90</td>
<td>&gt;60</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paper</td>
<td>20</td>
<td>85–90</td>
<td>&gt;60</td>
<td>&lt;1</td>
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<td>20</td>
<td>45–55</td>
<td>&gt;60</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Adenovirus 40 (1)</td>
<td>PBS or FS</td>
<td>Alum.</td>
<td>4</td>
<td>85–90</td>
<td>15</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alum.</td>
<td>20</td>
<td>85–90</td>
<td>15</td>
<td>&lt;1</td>
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<tr>
<td></td>
<td></td>
<td>Alum.</td>
<td>20</td>
<td>45–55</td>
<td>15</td>
<td>&lt;1</td>
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<td></td>
<td>Paper</td>
<td>4</td>
<td>85–90</td>
<td>&gt;30</td>
<td>&lt;1</td>
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<td>Paper</td>
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<td>85–90</td>
<td>&gt;30</td>
<td>&lt;1</td>
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<tr>
<td></td>
<td></td>
<td>Paper</td>
<td>20</td>
<td>45–55</td>
<td>&gt;30</td>
<td>&lt;1</td>
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<tr>
<td>Rotavirus p13 (1)</td>
<td>PBS or FS</td>
<td>Alum.</td>
<td>4</td>
<td>85–90</td>
<td>&gt;60</td>
<td>&lt;1</td>
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<tr>
<td></td>
<td></td>
<td>Alum.</td>
<td>20</td>
<td>85–90</td>
<td>&gt;60</td>
<td>&lt;1</td>
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<tr>
<td></td>
<td></td>
<td>Alum.</td>
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<td>45–55</td>
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<td>45–55</td>
<td>&gt;60</td>
<td>&lt;1</td>
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<tr>
<td>Astrovirus (type 4) (2)</td>
<td>PBS or FS</td>
<td>China</td>
<td>4</td>
<td>85–95</td>
<td>60</td>
<td>&lt;1</td>
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<td></td>
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<td>60</td>
<td>&lt;1</td>
<td>&lt;1</td>
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<tr>
<td>Feline calicivirus F9 (22)</td>
<td>BEM</td>
<td>Glass coverslip</td>
<td>4</td>
<td>ND*</td>
<td>57</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Glass</td>
<td>20</td>
<td>ND</td>
<td>35</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Glass</td>
<td>37</td>
<td>ND</td>
<td>7</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

* PBS, phosphate buffer solution (for all viruses except feline calicivirus F9); BEM, basal Eagle’s medium (used only for feline calicivirus F9).

b FS, 20% fecal suspension.

c Inactivation coefficient ($K_i = \log_{10}$ reduction in virus titer per ml = (initial viral titer – final viral titer)/survival (in hours) (64).

d Alum., aluminum surface.

e ND, not determined.
The spread of HAV, rotavirus, and astrovirus from hands to fomites and vice versa has been well documented in several studies (Table 1). Artificially contaminated finger pads transferred 9.2% of HAV to lettuce (11). Gloved hands transferred feline calicivirus to spatulas, lettuce, forks, doorknobs, and cutting boards (54). A study by Barker et al. demonstrated that norovirus could be transferred from contaminated surfaces to clean hands and then contaminated hands could transfer virus to a secondary surface, such as a phone or door handle (8). It was also found that norovirus-contaminated hands could cross-contaminate a series of seven clean surfaces without additional recontamination of hands (8). Viruses can be easily spread to the mouth when fomites and hands become contaminated (58, 60). A small child puts fingers in his mouth once every 3 minutes, and children up to 6 years average a hand-to-mouth frequency of 9.5 contacts per hour (31, 75).

Like respiratory viruses, laboratory studies documenting direct evidence of enteric virus transmission via surfaces are limited. The Ward study (77) found that all the volunteers who licked a rotavirus-contaminated dinner plate became infected. In the same study, only half of the volunteers who touched the contaminated dinner plate and subsequently licked contaminated fingers became infected (77). Overall, laboratory evidence supporting viral transmission via fomites is considered indirect and circumstantial, but it represents an important component in understanding potential virus transmission (6, 7).

**EPIDEMIOLOGICAL EVIDENCE OF VIRUS TRANSMISSION VIA FOMITES**

The involvement of fomites in viral disease transmission was first recognized long before the identification of pathogenic organisms, when smallpox outbreaks were traced to imported cotton in 1908 (24). Initially, epidemiology studies on viral disease transmission lacked the scientific methods to detect and distinguish between a variety of bacterial and viral illnesses. Consequently, most epidemiology studies did not identify the microbial cause of a disease, and outbreaks were characterized by disease symptoms only. For example, in 1929 an epidemic of nonbacterial gastroenteritis was described as the winter vomiting disease by epidemiologists (41). Molecular methods are now being used by epidemiologists to link enteric and respiratory viruses to disease outbreaks by identifying the viral pathogens in the host and the environment.

Several epidemiological studies have supported laboratory studies by indicating environmental contamination as a potential vehicle for virus transmission. During an outbreak in a Honolulu nursing home, it was determined that staff hands or fomites (e.g., towels, medical cart items, etc.) spread influenza virus (51). An outbreak of coronavirus (SARS) in a Hong Kong apartment complex may have resulted from fecal-oral transmission combined with environmental contamination (62). Studies in day care centers have detected rotavirus on various surfaces, including toys, phones, toilet handles, sinks, and water fountains (40). The transmission of HAV by contaminated drinking glasses was associated with an outbreak of hepatitis in a public house when an ill barman with HAV served drinks (72). Nursing volunteers who touched infected infants or surrounding fomites developed RSV infection, while nurses with no infant or fomite contact did not develop RSV symptoms (27, 34).

Epidemiological studies also provide additional information by using statistical tools, such as risk assessments and attack rates, to illuminate viral transmission routes. The potential for norovirus transmission via fomites was demonstrated during a wedding reception where the guests suffered a 50% attack rate of gastroenteritis after a kitchen assistant vomited in the sink which was subsequently used for salad preparation (7). When natural rhinovirus colds were studied, rhinovirus was found on 39% of symptomatic individuals’ hands (35). Additionally, volunteers touching contaminated objects and/or the fingers of symptomatic individuals had a higher attack rate of colds if they inoculated their own eyes or nose (35). Risk exposure analysis completed after an outbreak of gastroenteritis on a hospital elderly care ward showed that areas where patients vomited were the most significant factor in the spread of norovirus (7). Another hospital ward study demonstrated that
Rotavirus remained infective for 32 days in feces, and 16% viral transfer from contaminated fingertips to steel disc after 20 min (4).

Influenza virus
- Survival at lab temp of 28°C and 40% humidity for 48 h on dry surface; 72 h for avian influenza virus on dry surface (73); 72 h for influenza A virus on wet surface (9).
- Virus transferred from contaminated surface to hands for up to 24 h after inoculation (9).
- Transferred to lettuce (11).
- Transferred to recipients' fingers (30).
- Not found; estimated at 10–100 TCID50 (7, 55).
- Estimated to be as few as 10–100 particles (7, 8, 17, 39).
- Transferred to lower arm or water tap handle (8).
- Estimated at 10–100 TCID50 (7, 55).
- Proven (7, 22).
- Not proven, indirect evidence supports (3, 22).
- Not proven but suspected (3, 38, 58).
- Not proven but suspected (3, 38, 58).
- Not proven but suspected (3, 38, 58).
- Proven (food and fecally contaminated surfaces) (1, 41).
- Not found; estimated at 10–100 TCID50 (7, 55).
- Proven (7, 22).
- Accepted (food and fecally contaminated surfaces) (1, 41).
- Widely accepted, contaminated surfaces (1).
- May play an important role in secondary transmission (2, 61).

**DISINFECTION AND HYGIENE INTERVENTION STUDIES**

Like epidemiological studies, many disinfection and hygiene intervention studies lack microbial specificity and identify diseases by symptoms (gastrointestinal, respiratory, or cold symptoms). For example, research by Krilov et al. demonstrated that when environmental surfaces (school bus, toys, etc.) were regularly cleaned or disinfected there was a reduction in gastrointestinal and respiratory illness among children attending the day care center (7). A study in 1980 by Carter et al. found that families using an iodine-based hand wash had lower rates of respiratory disease (16). In addition, a review article by Barker et al. cited over 15 research studies that indicated a decrease in viral contamination and viral infection when hand washing was used regularly as an intervention (7). Subsequently, disinfection and hygiene intervention studies, which have cited a reduction in nonspecific illnesses, only support interruption of disease transmission.

Recently, molecular methods and immunoassays have been used to detect and identify viral presence in the environment before and after disinfection or cleaning. In 2002 norovirus caused consecutive outbreaks of gastroenteritis on various cruise ships (38). Three out of five of the cruise ships required disinfection and hygiene intervention studies, which have cited a reduction in nonspecific illnesses, only support interruption of disease transmission.

In a study by Barker et al., surfaces cleaned with a detergent solution spread norovirus to uncontaminated surfaces (8). As a result, the contaminated surface, the cleaning cloth, and the cross-contaminated surface all tested positive for norovirus (8). However, cleaning with a 5,000 ppm chlorine solution was effective in preventing cross-contamination and eliminating norovirus from environmental
surfaces (8). In Taiwan a hospital reported that following an outbreak environmental samples which tested positive for coronavirus were negative after resampling the cleaned emergency department and isolating the infected patients (38). A study by Ward et al. demonstrated that spraying rotavirus-contaminated surfaces with disinfectant prevented infection (77). Infection occurred in 63% to 100% of volunteers who touched rotavirus-contaminated surfaces and then licked fingers, and no volunteers became infected after licking contaminated surfaces that had been disinfected (77). Overall, when a disinfection intervention study specifies the microbial cause of disease and details on environmental decontamination, the study relays more practical information about interruption of the specific virus spread.

DISCUSSION

In 2006 the World Health Organization reported that diarrhea and respiratory infections were two of four major diseases influenced by environmental conditions (56). To limit or prevent the spread of viral infections, pathogen transmission needs to be fully understood (27). Both respiratory and enteric viruses have more than one route of transmission (30). Respiratory viruses are known to be spread by person-to-person contact, the airborne route, and contaminated surfaces or fomites (7, 27). Enteric viruses are spread by the fecal-oral route via environmental and person-to-person contact (7, 61, 77). Respiratory viruses appear to be more efficient in disease spread (via the airborne route) than enteric viruses. Respiratory viruses spread faster (from a sneeze, airborne virus travels 3 m at 20 m/s) (78), have short incubation times (1 to 8 days), and greater infectivity (a lower dosage causes infection) (39). On the other hand, enteric viruses spread more slowly (water or food), have longer incubation times (1 to 60 days), and require a higher viral dosage (lower infectivity) (59). These facts suggest that enteric and respiratory viral disease transmissions have nothing in common. However, person-to-person contact and environmental contamination are common routes of transmission for both types of viruses. Viruses spread by person-to-person contact can be interrupted with isolation of the viral carrier. Yet, isolation may prove to be impractical or difficult if there are many people or if the source of infection is unknown (69). Consequently, interrupting disease spread via indoor fomites is one of the more practical methods for limiting or preventing enteric and respiratory viral infections.

A majority of respiratory viruses are enveloped (parainfluenza virus, influenza virus, RSV, and coronavirus) and survive on surfaces from hours to days. In contrast, most enteric viruses are nonenveloped and survive on fomites from weeks to months. Studies have demonstrated that viral transfer from hands to surrounding surfaces is possible in 7 out of 10 viruses reviewed (Table 4). Epidemiological studies have verified naturally occurring outbreaks for 8 out of 10 viruses (HAV, RSV, norovirus, rotavirus, influenza virus, coronavirus, astroviruses, and adenoviruses). Investigations of disease outbreaks and disinfection intervention studies have documented indoor surfaces as reservoirs for pathogenic viruses with potential spread of infectious disease (19). Epidemiological studies have also identified fomites as a potential vehicle for disease transmission. Hygiene and disinfection intervention studies have demonstrated two concepts that support transmission of viral infection via fomites. First, proper cleaning of hands decreases respiratory and gastrointestinal illness. Second, disinfection of fomites can decrease surface contamination and may interrupt disease spread (norovirus, coronavirus, and rotavirus). In addition, laboratory evidence from studies by Ward et al. (rotavirus) (77) and Hendley et al. (rhinoviruses) (34) support viral transmission via fomites. Disease transmission via contaminated fomites has been proven or is suspected for all 10 enteric and respiratory viruses reviewed (Table 3). Generally, research evidence suggests that a large portion of enteric and respiratory illnesses can be prevented through improved environmental hygiene, with an emphasis on better hand and surface cleaning practices (39).

Additional studies investigating the infectious dose of enteric and respiratory viruses would improve and/or validate current water, air, and other environmental exposure guidelines. There is also a need for better quantitative data in the form of viral inactivation rates and transfer rates on/from fomites. Viral research that further investigates survival on fomites and hand-to-surface transfer would be useful in understanding the ecology of fomites in virus transmission. Studies targeting the distribution of viruses on fomites within the home, work, and public places could aid the targeting of cleaning and disinfection procedures. Generally, new data could be used in risk assessment models that associate viral infection with fomite contact or to improve viral transmission models. The potential success of risk assessment interventions would benefit both public health and the medical community.

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REFERENCES


