FRI FOOD SAFETY REVIEWS

White Paper on Effectiveness of Existing Interventions on Virus Inactivation in Meat and Poultry Products

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INTRODUCTION

Viruses are responsible for an estimated 9,282,170 cases of foodborne illness in the U.S. each year, which accounts for approximately 67% of all cases of foodborne illness. Usually viral infections are relatively mild (in developed countries), accounting for only about 34% of hospitalizations and 7% of deaths related to foodborne illness (*169*). Nevertheless, because of the large number of people sickened by foodborne viruses, there is a significant economic impact associated with viral foodborne infections (*151*). In less developed countries, enteric viruses present in water and food are an important cause of mortality in infants and young children (*15*).

Foodborne disease that was likely caused by viruses was first described in 1914 and involved 4 cases of paralytic illness among children consuming milk from a common source. During the next 35 years another 9 outbreaks of foodborne poliomyelitis were described in the U.S. and the UK. Raw milk was the most frequently reported vehicle, with lemonade and cream-filled pastries implicated in other outbreaks. Foodborne hepatitis was first associated with raw milk in 1943 and with shellfish in 1955. As diagnostic methods improved in recent years, viruses have been identified in increasing numbers of food- and waterborne outbreaks (*42*).

Noroviruses and hepatitis A virus are the most common viruses transmitted in food, and their presence in shellfish, fresh produce and prepared foods is the greatest public health priority according to a microbial risk assessment by the World Health Organization (WHO) (*283*). Data from CDC indicated that 321 of 329 foodborne viral outbreaks in the U.S. in 2007 were caused by norovirus and 4 outbreaks were due to hepatitis A [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx]. Data from the European Union for the same year demonstrated that caliciviruses (primarily norovirus) were responsible for 507 of 668 foodborne viral outbreaks, while hepatitis A caused another 46 outbreaks. Rotavirus was implicated in 122 outbreaks (*64*). A number of other human enteric viruses may be present in food (*58;84;129*). Infected food handlers have been responsible for numerous outbreaks of foodborne illness by contaminating foods with bacteria and viruses (*85*). Foods may also be contaminated with enteric viruses in the field through contact with feces (used as fertilizer) or fecally contaminated water.

Some animal viruses are known to infect humans but foodborne transmission appears to be rare. Viruses that infect food-producing animals may be present in skeletal muscles, milk, and other tissues that are commonly consumed. Some of these viruses have caused illness in cats and other carnivores eating raw meat but, in most cases, transmission to humans has not been demonstrated. Certain animal viruses of potential human concern are also present in animal fecal material and may contaminate meat during slaughter and processing. A recent review presented information on 13 animal viruses of possible relevance to the food industry. Characteristics of the viruses, their animal hosts, and intervention strategies were discussed (*107*).

Since viruses are not living cells like bacteria, controlling viral contamination of foods presents unique challenges, as discussed in several review articles (*9;137;198;215;270*). In addition to issues related to controlling conditions in domestic food production, processing, and preparation facilities, there are also concerns about importing viral zoonotic diseases through wildlife trade (*201*) and the potential presence of infectious viruses, such as avian influenza, foot and mouth disease, African swine fever, classical swine fever, and swine vesicular disease, in smuggled meat and meat products (*94;286*). Wild boars and perhaps wild ruminants may be reservoirs for some viral diseases that have largely been eliminated in domesticated animals (*93;112*).

CHARACTERISTICS OF VIRUSES

Structure and Life Cycle

Viruses have a single (ss)- or double (ds)-stranded piece of RNA or DNA as genetic material, a protein coat, and, in some cases, a lipid envelope around the protein coat. It is generally true that RNA viruses can mutate and evolve more rapidly than DNA viruses and that non-enveloped viruses are more resistant to environmental stresses (*103*).

The first step in the infection of a cell is an interaction between viral proteins and cell surface molecules that allows attachment of the virus and injection of viral DNA or RNA into the cell. Certain viral proteins interact with specific cell surface receptors and thus viruses may infect only cells of certain species of animals or plants or certain types of cells within these organisms. Once inside the cell, the virus takes over the metabolic activities of the cell and directs them to produce more viral proteins and nucleic acids. Viruses can only replicate inside cells of living organisms by utilizing cellular enzymes and structures necessary for protein synthesis and replication of genetic material. Therefore, viruses do not replicate in foods under any conditions of temperature or water activity but may survive for extended periods under conditions that would inactivate most bacterial cells. Their simple

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structure, and the fact that they are not physiologically active in foods or on environmental surfaces, protects viruses from many interventions that are used to inactivate more complex and metabolically active foodborne bacteria and parasites.

General Features of Foodborne Viruses

Viruses that are efficiently transmitted through food are generally small, lack a lipid envelope, and can reproduce in human, or at least mammalian, cells. These viruses must be able to survive for some time in foods and are often stable during extended periods of storage. Frequent mutations in the RNA genome produce new variants during each replication cycle. Some of these mutations may permit more efficient replication in new host animals or cell types and may increase resistance of the viruses to environmental stresses (*58*).

Foodborne viruses include **human enteric viruses** that primarily infect humans and may be transmitted from person to person directly or may contaminate food through exposure to human feces. Even though many enteric viruses have been detected mainly in shellfish, a food handler excreting any enteric viruses could potentially contaminate a wide variety of foods. **Animal viruses** cause infections primarily in animals and may be present in various tissues of animals, including milk, meat, and eggs. Very few animal viruses are known to cause human infections. It is not clear whether most animal viruses are unable to survive in foods and cause human infections or whether there is massive underreporting because of inadequate analytical methods and/or the occurrence of mild, subclinical infections. **Table 1** lists human enteric and animal viruses that have been reported to be present in foods. This table was compiled mainly from three recent references that listed viruses that have the potential to be transmitted by food (*58;84;107*). Foodborne transmission has not been documented for most of these viruses, and some of the animal viruses listed here are currently not frequently detected because of control and eradication programs. These viruses will be discussed further in later sections.

Environmental Resistance

For a virus to cause foodborne illness, it must be able to persist and remain infectious outside of living cells for some period of time. Factors impacting survival of viruses in the environment include: temperature, pH, moisture, sunlight or ultraviolet light, certain inorganic ions, organic matter, and presence of microbes that consume viruses. Different viruses, even within the same family, vary in their sensitivity to these factors. Resistance to environmental stress is related to the type of nucleic acids in a virus, the structure of the proteins forming the capsid coat, and the lipid envelope, if present. For example, double-stranded DNA viruses are more resistant to UV inactivation than single-stranded RNA viruses. Viruses with lipid envelopes are generally less resistant to adverse conditions.

Most enteric viruses are probably capable of surviving for weeks or months in the environment at ambient temperatures and perhaps for years at low temperatures (*21;31;223*). Some viruses readily attach to materials used to manufacture food contact materials, including stainless steel, copper, polythene, and polyvinyl chloride (*131*). Many viruses, including hepatitis A, remain infectious in frozen foods for over a year as demonstrated by the 1997 strawberry outbreak in the U.S. (*106*). Norovirus survives for at least 10 days on refrigerated foods, including turkey and lettuce (*136*), and hepatitis A virus persisted for 6 weeks on refrigerated spinach (*235*). Survival in water, soil, aerosols, and on surfaces is usually longer at lower temperatures. Survival of avian influenza viruses in landfills was estimated to range from 30 days to >600 days depending on temperature and pH of the surroundings (*83*). Swine vesicular disease virus was found on and in worms in soil where infected pigs were buried. Viruses in soil may be washed into surface waters or percolate into groundwater.

Viruses are less tolerant of heat and lose infectivity more rapidly at warm ambient temperatures on surfaces or in soil or water. Tolerance to acidity is an advantage for a foodborne virus as this enables it to survive in the gastrointestinal tract of its hosts. Presence of organic material around viruses in any environment increases tolerance to heat and pH extremes (*223*).

Data on survival of viruses should be interpreted carefully. Some viruses, including human norovirus which is responsible for a large percentage of foodborne viral infections, cannot yet be cultivated in vitro. Therefore, detection of these viruses is usually accomplished by a method such as PCR to identify viral DNA or RNA in a sample. This may demonstrate that a virus has been present in a sample but it does not prove that the virus is still infectious. Surrogate viruses, such as feline calicivirus and murine norovirus, are used to predict survival of noroviruses under different conditions. However, these viruses may be more or less sensitive to particular stresses than human noroviruses.

Furthermore, experiments on viral persistence are usually done in a laboratory where one or a few conditions are tested. Results from these tests may not accurately predict what will happen in a complex environment, such as a food or soil. Processing aids that affect survival, such as high pressure, ozone, irra-

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diation, and sanitizers/disinfectants, will be discussed below.

Transmission Pathways

Human enteric viruses are present at high concentrations in feces of infected persons and may be shed before a person has a symptomatic infection and also for an extended period following recovery. Volunteers infected with norovirus shed a median peak level of 95×10^9 genomic copies/g feces. Although symptomatic illness lasted 1–2 days, some volunteers shed virus for up to 56 days (*6*). These viruses are transmitted from person to person by some variation of the fecal–oral route.

Infected food handlers are suspected of causing a large percentage of foodborne enteric viral outbreaks. A recent quantitative exposure model included input data or estimates on fecal shedding, hand hygiene behaviors, inactivation of viruses on surfaces, and efficiency of transfer of virus between surfaces. Dynamics of foodborne transmission and effectiveness of different interventions can be tested with these models (*176*). Pathogens can spread through food processing and preparation environments in complex and unexpected ways. Transmission and survival of foodborne bacteria, viruses, and parasites in these environments were recently reviewed. Viruses are more resistant to most adverse environmental conditions than bacteria (*260*).

Foods may also be exposed to viruses during production and harvesting:

- Vegetables and fruits can be contaminated in the field by water, containing human sewage, that is used for irrigation, pesticide applications, or washing after harvest. Fecal material from animals and humans that is used as fertilizer, including biosolids, may contain viruses that attach to lettuce and other plants (*77;276*). High temperatures achieved during proper composting eliminates infectivity of many viruses (*89*).
- Meat from slaughter animals may contain some animal viruses even when animals appear healthy. Feces from infected animals are recognized as a source of bacterial contamination of meat during slaughter and may also be a source of viruses.
- Shellfish are filter feeders that ingest and pass out large quantities of water in order to screen out and consume tiny food particles. As part of this process, they can accumulate pathogenic bacteria and viruses if the water has been contaminated with human or animal sewage.

Transmission of viruses from one host to another follows many other routes. Some are carried by insects while others are dispersed in aerosols. Contagious viruses are often spread by direct or close contact between animals or humans. Enteric viruses may be present in drinking water contaminated with sewage and have also caused numerous outbreaks in people exposed to contaminated recreational water (*238*).

Table 2 indicates the transmission pathways that have been reported for the viruses considered here. Foodborne transmission to humans has been demonstrated for some viruses. Animals have been reported to acquire some viruses from contaminated feed.

HUMAN VIRUSES

Hepatitis A

Hepatitis A virus (HAV) is a non-enveloped single strand RNA virus with an estimated infectious dose of 10–100 viral particles. Illness is usually mild, with symptoms of fever and abdominal discomfort followed by jaundice, and recovery is usually complete within 1–2 weeks with no lingering chronic illness (as occurs with hepatitis B). Very few cases in the U.S. are fatal. According to data from CDC, an average of 28,000 cases of HA occurred yearly between 1987 and 1997. A vaccine became available in 1995. In subsequent years, CDC recommended that people in at-risk groups and children living in states that historically had a high incidence of HA be vaccinated. As a result, fewer than 3000 cases of HA were reported in 2007, and incidence of this disease declined from 12 cases/100,000 population in 1995 to 1 case/100,000 in 2007 [http://www.cdc.gov/hepatitis/HAV/StatisticsHAV.htm].

Hepatitis A is rarely a problem in underdeveloped countries. Most children are exposed to the virus by age 6 and have a mild illness or are asymptomatic. This exposure induces life-long immunity. In more-developed countries with better drinking water and sewage systems, children are not typically exposed to HAV and a high percentage of the population lacks immunity. When these people travel to underdeveloped countries or consume foods harvested in those countries, they may be exposed to HAV and become ill (*202*).

HAV is excreted in feces and infection occurs by some variation of the fecal–oral route. Outbreaks are often a result of waterborne transmission but have also been traced to foods contaminated by a food handler and to foods contaminated before distribution. The incubation period for HAV averages 28 days, making it difficult to trace the origin of many cases and outbreaks. HAV is relatively heat stable and survives over a month in the environment and over a year in the freezer (*84*).

CDC lists 74 foodborne outbreaks occurring from 1998 to 2007 [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx].

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Table 3 presents data on several recent HA outbreaks to illustrate different modes of transmission.

- Shellfish are filter feeders that can concentrate viruses and bacteria from contaminated water. Two outbreaks in Spain (1999, 2008) were traced to frozen cockles imported from Peru. These clams had been grilled but apparently at too low a temperature or for too short a time to inactivate the virus. Contaminated shellfish also originate in more developed countries, as was the case with the French raw oyster outbreak in 2007. Oysters had been stored in submersible tanks near a storm sewer outlet. Sewage overflows were the suspected source of the virus. A huge outbreak attributed to contaminated clams occurred in China in 1988.
- Fresh produce may also harbor viruses. Green onions that caused a large outbreak at a Chi-Chi's restaurant were traced back to two farms in Mexico. HAV may have attached to the onions when they were exposed to contaminated water during irrigation, washing, or cooling. Contaminated frozen strawberries caused an outbreak at schools in Michigan and other states. It was believed that these berries were contaminated during harvest when each berry was handled to remove the stem. A 2009 outbreak in Australia has been linked to imported frozen semi-dried tomatoes. A similar strain of hepatitis A virus, also associated with semi-dried tomatoes, sickened 13 people in The Netherlands (*204*).
- Ingestion of fecally contaminated water has also caused several outbreaks associated with bottled water in China (said to be caused by contamination of the water source with runoff from melting of heavy snowfall), with ice snacks in China made from contaminated well water, and from water in a spa pool in Australia that was contaminated by an ill child.
- Poor hygiene during processing was the apparent cause of an orange-juice-associated outbreak in Egypt. Fruit or machinery may have been in contact with contaminated water or an ill worker may have spread HAV to the oranges.
- Infected food handlers with poor hygiene were the probable sources for a variety of outbreaks, including some with unexpected food vehicles: an outbreak attributed to coleslaw at a catered youth camp gathering in Australia, an outbreak at a McDonald's restaurant near Chicago in 2009, an outbreak in Austria associated with deli food purchased at a supermarket, an outbreak linked to raw beef contaminated by a food handler in Belgium at a meat distribution plant, and an outbreak traced to glaze on donuts made by an infected baker.
- Direct person-to-person transmission has occurred among men having sex with men in Spain (and in other countries). A prolonged community-wide hepatitis outbreak occurred in a socio-economically depressed area in Australia where housing was crowded, families had many children, and there was an unemployment problem. Some cases were injected-drug users.

Norovirus

Noroviruses are small, non-enveloped, enteric RNA viruses that cause an estimated 23 million cases of acute gastroenteritis in the U.S. annually. Approximately 55–60% of outbreaks are a result of contamination of food by food handlers, by sewage contaminating shellfish in coastal waters, or by contaminated water or surfaces used in food production, processing or preparation. Other vehicles of infection are contaminated drinking and recreational water and direct contact with an infected person [http://www.cdc.gov/ncidod/dvrd/revb/gastro/norovirus.htm] Five principal norovirus genogroups have been described: GI, GII, GIII, GIV, and GV. Human isolates belong to groups GI, GII, and GIV. Members of the GII genogroup are the most common strains and evolve into new variants over time, causing new waves of pandemic outbreaks about every 2–4 years (*80;237*).

Noroviruses generally cause a relatively mild case of gastroenteritis, with vomiting and diarrhea that lasts 1–3 days. However, there have been reports of serious consequences of norovirus infection, including benign seizures in infants (*36*) and chronic debilitating diarrhea in immunocompromised patients (*218*). Incubation period following exposure is usually between 24 and 48 hours (in contrast to HAV where incubation period may be as long as 28 days). Infectious dose for noroviruses is very low (median dose estimated at 18 viral particles) while feces of infected individuals may contain 10^6 or more virus particles per gram (*253*). Even if only 5% of fecal material on a contaminated surface were transferred to a ready-to-eat food, this would be greater than the infectious dose (*43*). Virus shedding precedes clinical illness in 30% of exposed people and may continue for weeks following recovery. In an experiment, volunteers infected with norovirus shed a median peak level of 95×10^9 genomic copies/g feces. Although symptomatic illness lasted 1–2 days, some volunteers shed virus for up to 56 days (*6*). A large number of food handlers associated with norovirus outbreaks in Japan were asymptomatic, but analyses of their fecal samples revealed similar concentrations of noroviruses as those detected in symptomatic individuals (*196*).

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Table 3. Selected outbreaks of hepatitis A.

Food / Vehicle	Year	Location	# cases	Reference
Tomatoes, semi-dried	2009	Australia	>70	(*52*)
Restaurant food	2009	Illinois	34	(*54*)
Sexual contact	2008–2009	Spain	150	(*263*)
Water, bottled	2008	China	269	(*5*)
Deli food (supermarket)	2008	Austria	21	(*229*)
Cockles (clams)	2008	Spain	100	(*205*)
Oysters	2007	France	111	(*90*)
Ice snacks	2006	China	116	(*295*)
Orange juice	2004	Egypt	351	(*74*)
Beef, raw	2004	Belgium	269	(*217*)
Coleslaw	2003	Australia	21	(*181*)
Onions, green	2003	Pennsylvania	601	(*281*)
Cockles (clams)	1999	Spain	184	(*22*; *205*)
Strawberries	1997	U.S.: Multistate	262	(*106*)
Water, spa pool	1997	Australia	6	(*248*)
Person to person	1996	Australia	58	(*97*)
Glazed donuts	1994	New York	79	(*279*)
Clams, raw	1988	China	292,301	(*91*)

One region of the noroviral capsid protein recognizes the histo-blood-group antigens. Persons with blood type B and nonsecretors are much less likely to develop symptoms when exposed to common norovirus genotypes because the virus does not bind to the B antigens expressed on mucosal cells and in saliva. However, a norovirus strain responsible for a Swedish foodborne outbreak in 2007 caused symptoms in people regardless of secretor status or ABO blood group (*186*). A mutation in the capsid protein apparently expanded the host range of this variant.

Norovirus strains also infect animals: GIII genotypes infect cattle and GV genotypes have been isolated from mice. GII genotypes, distinct from human strains, infect swine. Zoonotic transfer of noroviruses from livestock to humans has not been demonstrated. However, analyses of feces from swine and cattle in Canada detected human-like GII.4 strains in a small number of samples. In addition, one sample of raw pork from a supermarket had a human norovirus strain. Although the pork could have been contaminated by an infected food handler, the discovery of human noroviruses in some swine suggests the possibility that meat could be contaminated with noroviruses in animal feces during slaughter and processing (*162*).

Epidemiological data indicate that infected food handlers are often involved in transmission of viruses, if they practice poor personal hygiene (*258;259*). They may contaminate foods directly or contaminate food preparation surfaces with fecal material. Survival of noroviruses and some related caliciviruses on food preparation surfaces and the probability of their transfer to ready-to-eat foods were examined in a series of experiments. Ceramic, formica, and stainless steel coupons were contaminated with norovirus, norovirus RNA, or feline calicivirus and stored for 7 days at 22ºC. Purified RNA was not detectable on the coupons after 24 hours but norovirus particles persisted on all coupons throughout the experiment. After an initial drop of 2–3 logs during the first hour of storage, virus titers decreased slowly during the week but were still present on all materials after a week. Virus was transferred to lettuce that was pressed on the inoculated coupons, more efficiently if the lettuce was wet (*43*).

Noroviruses cannot be cultured, which makes it difficult to assess the effectiveness of processing methods and interventions to prevent contamination. PCR methods can be used to determine presence of viral RNA but this does not provide reliable estimates of the presence of infectious viral particles. Surrogate viruses, feline calicivirus (a respiratory pathogen), canine calicivirus (a gastrointestinal pathogen), and murine norovirus have been used to predict behavior of human noroviruses under different conditions. However, some experiments suggest that human noroviruses are more resistant to heat than murine noroviruses and feline calicivirus (*100;136*).

Norovirus is a frequent cause of outbreaks of foodborne illness. In 2006, in Minnesota, norovirus was the confirmed or suspected agent in 54 of 81 foodborne outbreaks of gastroenteritis. Foods associated with outbreaks included ham, sandwiches, salads,

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egg rolls, lemon curd, and iced tea. In many outbreaks, a specific food was not identified as the source (*175*). Data for 2007, presented on the CDC website, showed that of 329 outbreaks with a viral etiology, 321 were caused by norovirus with only 4 outbreaks attributed to hepatitis A virus [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx]. Caliciviruses (primarily norovirus) caused 507 of 668 foodborne viral outbreaks in the European Union in 2007 (*64*). Noroviruses have also been notorious for causing large outbreaks on cruise ships. Of the 102 diarrheal outbreaks on cruise ships investigated by CDC in 2005–2009, a causative agent was identified in 85 and norovirus was responsible for 80 of the outbreaks [http://www.cdc.gov/nceh/vsp/surv/GIlist.htm].

Table 4 presents information on some representative norovirus outbreaks.

- Outbreaks often occur in long-term-care institutions, and difficulties involved in effectively disinfecting the premises may lead to recurrent outbreaks as observed in three Oregon outbreaks at the same facility in 2007. Recurring outbreaks have also occurred on cruise ships. One memorable outbreak occurred on an airplane in 2008. Although some cases, who were part of a tour group, apparently were infected prior to boarding the plane, other passengers were infected during the flight as numerous ill passengers suffered bouts of diarrhea and vomiting.
- Packaged deli meats were identified as the source of infection for river rafters in the Grand Canyon in 2005. Traceback investigations revealed that an employee at the packaging plant, responsible for slicing the meat, had recently recovered from gastroenteritis.
- Shellfish, consumed raw or lightly cooked, are a common vehicle for norovirus infections, as in the 2009 Fat Duck restaurant outbreak in the UK. Traceback to the source of the implicated shellfish found more contaminated shellfish still in the water. Cross-contamination and person-to-person transfer also occurred in the restaurant.
- Produce, such as raspberries and raw vegetables, is another frequent vehicle as demonstrated in the recent Finnish outbreaks.
- An unusual vehicle was pastry served at several Japanese schools. The source of contamination was not identified but apparently occurred at the central kitchen preparing school lunches.
- Another school-related outbreak, in Washington DC, was traced to contamination on computer mice and keyboards, presumably deposited by an infected individual who hadn't practiced good hygiene.
- Some waterborne outbreaks occur when there is contamination of drinking water distribution systems, as in the capitol city of Montenegro or where there are lapses of sanitation in swimming pool water.
- Three outbreaks, occurring at different college campuses in 2008, were never epidemiologically associated with particular sources. This is often the case with viral foodborne outbreaks.

Table 4. Selected recent norovirus outbreaks.

Food	Year	Location	No. cases	Reference
Raspberries	2009	Finland	200	(*163*)
Shellfish	2009	UK	>240	(*96*)
Water, drinking	2008	Montenegro	1699	(*280*)
Unknown	2008	CA, MI, WI	1085	(*216*)
Aerosolization	2008	Airplane; U.S.	22	(*124*)
Keyboards; computer mice	2007	Washington, DC	266	(*53*)
Meal at restaurant	2007	Sweden	33	(*186*)
Aerosolization (vomiting)	2007	Austria	176	(*134*)
Pastry	2007	Japan	26	(*194*)
Person to person	2007	Oregon	145	(*34*)
Vegetables, raw	2006	Finland	>400	(*154*)
Meat, packaged deli	2005	Arizona	137	(*155*)
Water, pool	2004	Vermont	53	(*33*)

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Other Human Enteric Viruses

Adenoviruses have DNA as their genetic material and are most often associated with respiratory infections. They have been detected in shellfish, wastewater, and surface waters in many locations, and certain serotypes, types 40 and 41, cause gastroenteritis and have been suspected in foodborne infections (*84*). According to data from CDC, adenovirus was a suspected cause of two restaurant-associated outbreaks attributed to sausage and ice in 2003 and 2007 [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx].

Aichi virus is a non-enveloped single-strand RNA virus that was recently recognized as a cause of oyster-associated gastroenteritis (*289*). It has since been detected in shellfish and diarrheic stool samples from China, Japan, France, Germany and other countries (*138;271;290*). It is suspected that there are other vehicles of foodborne infection besides shellfish, but they have not been identified as yet.

Astroviruses are non-enveloped RNA viruses that cause enteric disease particularly in children, the elderly, and immunocompromised persons. According to data from CDC on foodborne disease outbreaks, astrovirus was a confirmed cause of a restaurant outbreak but no specific food was identified [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx]. A large outbreak of diarrhea affecting >4700 people at 14 schools in Japan was caused by astrovirus. Food provided to the schools by a common supplier was believed to be the vehicle of infection (*193*). Astroviruses also cause an important disease in turkeys but there is no evidence that this virus is zoonotic (*126*).

Parvoviruses are very small, unenveloped DNA viruses that have been associated with human respiratory and foodborne illness (traced to contaminated shellfish) (*84*). **Bocavirus**, first isolated in 2005, is a related virus usually associated with respiratory disease but has also been detected in the digestive tract. These viruses are spread by the fecal–oral and respiratory routes and are very resistant to heat and many disinfectants (*37;63*).

Poliovirus caused at least 10 foodborne outbreaks of disease in the U.S. and the UK from 1914 to 1949. Raw milk was the most frequently reported vehicle, with lemonade and cream-filled pastries implicated in other outbreaks (*42*). Pasteurization of milk became widespread in the 1950s and decreased transmission of polio by this route. With mass immunization campaigns, few human cases of polio now occur except in a few countries where routine vaccination of children has not been achieved. Poliovirus was detected in market samples of raw meat in the U.S. in the 1960s–1970s (*41*). Research has shown that poliovirus can survive in the middle of hamburgers cooked to 60ºC (rare) (*242*) and in dry and semi-dry fermented sausages (*114*).

Rotaviruses are double-stranded RNA viruses that are responsible for a large percentage of cases of diarrhea in children <5 years of age. Rotaviruses have been the most common cause of hospitalization for diarrhea among children in the U.S. but a recently developed vaccine for children is expected to decrease morbidity (*15*). Rotavirus was recently detected in potato stew served during a sanatorium outbreak in Germany (*165*). Sandwiches, probably contaminated by a cafeteria worker, were associated with an outbreak in 2000 at a college in the U.S. (*72*). Rotavirus was also the confirmed or suspected cause of 7 other foodborne outbreaks in the U.S. between 1999 and 2005. Other vehicles included deli meat, tuna salad, conch, and multiple other foods [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx].

Sapoviruses have been detected along with other enteric viruses in stools of outbreak cases of oyster-associated gastroenteritis in Japan. Oyster samples were not available for analysis but a similar sapovirus was detected in clams (*184*). Sapovirus caused a gastroenteritis outbreak affecting 55 college students in Taiwan in 2007. No vehicle was identified but it was believed to be a foodborne illness (*287*). Sapoviruses were implicated in 5 outbreaks in the U.S. during 2001–2004. Vehicles included salad, water, bun, and chicken or potato [http://wwwn.cdc.gov/foodborneoutbreaks/Default.aspx]. High concentrations of sapovirus were detected in stool samples from asymptomatic food handlers associated with an outbreak in Japan (*294*). Sapoviruses can also infect animals. They were detected in 7.6% of 1050 swine fecal specimens (44% of farms) in Europe. However, genetic characterization of these viruses indicated that they are not closely related to known human sapoviruses (*213*). Sapoviruses, including two strains closely related to human isolates, were also detected in Japanese swine (*185*).

ANIMAL VIRUSES

Livestock are subject to numerous viral diseases but many of these are effectively controlled on the farm by vaccination and some rearing practices. Nevertheless, apparently healthy animals at slaughter may harbor viruses in internal tissues, and high concentrations of viruses may be present in fecal material. Relatively few viruses present in animals have been shown to cause human illness; these include high pathogenicity avian influenza (HPAI), foot and mouth disease (FMD), hepatitis E, Newcastle disease, bluetongue, rabies, Rift Valley fever, SARS, swine vesicular disease, and vesicular stomatitis. A recent

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review by ILSI Europe presented information on 13 animal viruses of potential concern to the food industry (*107*). A review of recent outbreaks of viral diseases in livestock in Europe (avian influenza, foot and mouth disease, classical swine fever, and bluetongue) emphasized the importance of national plans for the detection of exotic diseases, organized procedures for dealing with epidemics, and effective communication among all stakeholders (*241*).

Hepatitis E

Hepatitis E virus (HEV) is a non-enveloped single-stranded RNA virus that has been classified into four genotypes. Genotypes 1 and 2 exclusively infect humans, primarily in less developed countries, and have been associated with large waterborne outbreaks. Several huge epidemics of hepatitis E have occurred in Asia, Africa, and Mexico. These include an outbreak with more than 100,000 cases in China in 1986–88, 29,000 cases in India in 1955, and a prolonged outbreak in Uganda during 2007–2009 with more than 10,000 cases and at least 160 deaths. Genotypes 3 and 4 have been identified in both humans and other animals, particularly swine, and are usually associated with sporadic cases and small outbreaks. HEV does not appear to cause illness in pigs (*2*). HEV has been detected in chicken eggs (*82*) but this strain is not closely related to human and porcine isolates (*269*). Several variants have been described for each of these genotypes. Recently a new genotype was proposed for an HEV strain isolated from farmed rabbits in China (*296*).

Some recent outbreaks of HEV are listed in **Table 5**. Waterborne outbreaks tend to affect hundreds or thousands of people whereas foodborne outbreaks involve fewer cases. A recent outbreak, associated with consumption of shellfish, occurred on a cruise ship that had traveled around the world (*225*). Reports from Japan have implicated consumption of raw or lightly cooked meat from deer and wild boar as the cause of some human cases of hepatitis E (*249*).

Hepatitis E virus can cause subclinical infections and, in symptomatic cases, typically causes a self-limiting, moderately severe hepatitis. Case-fatality rates are typically 0.5–4.0% but much higher rates of 15–20% have been reported for pregnant women who may develop fulminant hepatitis (*269*). A virulent strain of HEV genotype 3 was detected in 8 human cases and several swine samples in Japan. Three of the patients suffering severe hepatitis reported consuming undercooked pork or wild boar meat (*247*). There has been one report in Europe of Guillain-Barre syndrome following acute hepatitis E infection (*147*).

In Japan, Europe, and North America, clinical hepatitis E cases are often associated with overseas travel to developing countries where water quality may be questionable. However, several countries have reported increasing numbers of sporadic infections apparently acquired domestically. Surveys indicate that 17–21% of people in the U.S. have been exposed to hepatitis E without experiencing clinical symptoms (*172*). Data from the UK, Denmark, and Okinawa indicate that prevalence of antibodies to HEV in the general population has decreased in the past 10–12 years (*38*).

Persons with occupational exposure to animals, particularly swine, have a higher prevalence of anti-HEV antibodies. This includes about 50% of Danish farmers tested in 1983 (*38*), 26% of veterinarians tested in the U.S. (*172*), about 19% of swine workers in Spain (compared to 4% of non-workers; *75*), 28% of swine workers in Thailand (compared to 17% of non-workers; *209*), and 25.3% of wild boar hunters on Okinawa (compared to 7.7% of non-hunters) (*264*). Contact with livestock, particularly swine, has been shown to increase the likelihood, by 1.5 to 5 times, that antibodies to HEV will be present in the blood of farm workers and veterinarians (*75;172*).

Table 5. Recent outbreaks of hepatitis E.

Vehicle	Year	Location	No. cases	Reference
Shellfish	2008	Cruise ship	33	(*225*)
Drinking water	2007–2009	Uganda	10,196	(*252*)
Drinking water	2005–2006	India	3170	(*11*)
Drinking water	2005	India	1611	(*226*)
Pork, barbecued	2004	Japan	8	(*160*)
River water	2004	India	538	(*243*)
Deer meat	2003	Japan	4	(*250*)
Wild boar meat	2003	Japan	8	(*249*)
Wild boar liver	2003	Japan	2	(*161*)

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Surveys and experimental studies have demonstrated that hepatitis E can infect a number of animal species, including pigs, cows, sheep, goats, deer, chickens, cats, rodents, monkeys, and other primates. Hepatitis E virus (HEV) strains vary somewhat geographically and in different animal species. Nevertheless, the strains are very similar, and cross-species transfer has been observed. This suggests the possibility that sporadic cases in developed countries may result from contact with infected animals or may be foodborne infections (*172;269*). Hepatitis E virus is shed in feces of infected animals and may therefore be present in the farm environment and could contaminate meat during slaughter. HEV appears to be common in swine (*166;231;293*) and is also prevalent in wild boar in Europe and Japan (*1;46;158;227;228*) and in some kinds of deer (*73;262*). Numerous surveys worldwide have documented the widespread presence of anti-HEV antibodies in swine, with reported prevalences ranging from 6% to 85% (*269*). Infections in pigs are spread by contact, and virus is excreted in feces and urine for several weeks after exposure. Most infections in pigs are subclinical, with small lesions observed in the liver (*24;139;141*). Infectious virus is present in fresh feces and in manure slurry in lagoons and pits. This is a potential source for contamination of surface waters and shallow wells (*115;116*).

Analyses of tissues of naturally and experimentally infected pigs demonstrated the presence of HEV RNA in bile, liver, lymph nodes, and intestines. HEV appears to replicate primarily in hepatocytes and is most frequently detected in liver of infected swine (*47;141*). At least 3 surveys, in India, The Netherlands, and the U.S., have detected HEV in pig liver in grocery stores, with prevalences of 0.83%, 6.5%, and 11% of pig livers tested in those countries, respectively, containing detectable hepatitis E. When infected raw liver was fed to other pigs, many developed hepatitis (*23;66;132*).

Proper cooking of porcine liver does destroy infectivity of hepatitis E virus. Boiling in water for 5 min and stir-frying for 5 min at 191ºC to an internal temperature of 71ºC inactivates this virus. However, incubation of infected liver for one hour at 56ºC was not an effective heat treatment (*67*). Since pork is usually cooked until well done in the U.S., it is not considered likely that hepatitis E is a common foodborne infection. But it is possible that some sporadic infections result from consuming undercooked meat and from cross-contamination during food preparation.

High Pathogenicity Avian Influenza (HPAI)

Avian influenza (AI) viruses primarily infect birds and occasionally cause illness in humans working closely with infected birds and in predators and scavengers consuming sick or dead birds. AI is caused by relatively large RNA viruses that are covered with a lipid envelope. AI viruses are notorious for mutating frequently and these mutations could affect host range, pathogenicity, and effectiveness of treatments to control the viruses. AI viruses are designated low pathogenicity (LP) or high pathogenicity (HP) depending on the severity of the illness they cause. Wild ducks, farm ducks, geese, gulls, and other birds are commonly infected with LPAI strains, and sporadic outbreaks of LPAI occur in domestic poultry in many countries.

LPAI (low pathogenicity avian influenza) viruses are usually detected only in the respiratory and gastrointestinal systems and feces of infected birds (*246*). There is one report of an LPAI strain, H9N2, present in meat of infected chickens (*62*). LPAI viruses have also been isolated from untreated lake water where large numbers of ducks and other water birds live, and contaminated lake water appears to be an efficient vehicle for spreading infection among flocks (*78*). A recent study on the survival of 12 LPAI viruses in water at different conditions of temperature, pH, and salinity found that they generally persisted longer at a slightly basic pH, temperatures <17ºC, and in fresh or slightly brackish water. Some LPAI viruses survived up to 150 days (*219*).

HPAI strains cause rapid and severe, sometimes fatal infections in chickens and turkeys. Three major outbreaks of HPAI were reported in U.S. poultry, in 1924, 1983–1984, and 2004. There was no apparent transmission to humans during these outbreaks (*266*). However, during a large HPAI H7N7 outbreak on commercial poultry farms in Europe in 2003, 453 people reported symptoms and H7N7 virus was detected in 89 people working with the sick birds. The primary symptom was conjunctivitis; 7 confirmed cases also had influenza-like illness. Most cases were working directly with infected birds but person to person transmission occurred in at least 3 cases (*130;268*).

The HPAI strain of current concern is the H5N1 virus, first detected in 1996 in domestic geese in southeast China and widely recognized in the next year as it spread through live-bird markets in Hong Kong infecting many birds and eighteen humans, killing six. This virus has spread to Europe and Africa, infecting poultry in 50 countries as of 28 December 2009 [http://www.oie.int/downld/AVIAN%20INFLUENZA/A_AI-Asia.htm]. H5N1 viruses spread rapidly among chickens and turkeys and typically cause death within 48 hours. Huge numbers of chickens have become ill or have been slaughtered to prevent spread of this illness. Efforts to control HPAI, including culling, stamping out, cleaning and disinfection, and vaccination, have

not been successful in eradicating H5N1 in Asia, but have been more effective in Europe (*291*).

According to surveillance information published by WHO, the cumulative number of confirmed human cases of H5N1 avian influenza from 2003 to 30 December 2009 is 467, with 282 deaths. In nearly all cases, it has been determined that humans affected by this disease have had close contact with poultry, and foodborne transmission was considered unlikely. Cases have been identified in 15 countries, with the top 5 countries in numbers of cases: Indonesia, Viet Nam, Egypt, China, and Thailand [http://www.who.int/csr/disease/avian_influenza/country/en/]. These numbers are likely an underestimate because surveillance programs are designed mainly to detect severe cases and vary in their efficiency in different countries. Testing of 674 villagers in Cambodia who lived near two fatal avian influenza cases found that 7 previously unidentified people had high antibody titers to this virus. Although these seropositive villagers comprise only a small fraction of the population (about 1%), it does indicate that the surveillance program detected only 2 of 9 infected people in this village (*272*).

HPAI viruses cause systemic infections in birds and have been detected in blood, bone, and breast and thigh meat of chickens (*246*). HPAI H5N1 viruses also infect ducks, but ducks often appear healthy even when virus is present in muscles and internal organs (*30*). Titers of HPAI H5N1 virus in chicken thigh and breast meat were reported to be $10^{6.8}$ to $10^{8.0}$ and $10^{5.5}$ to $10^{7.9}$ EID_{50} (median infectious dose)/g, respectively, while titers for an HPAI H5N2 strain in chicken thigh and breast meat were reported to be $10^{2.8}$ and $10^{2.3}$ EID_{50}/g, respectively. HPAI H5N1 titers in thigh meat of sick ducks were reported as $10^{4.0}$ to $10^{6.0}$ EID_{50}/g, and in infected but clinically normal ducks as $10^{2.0}$ to $10^{3.4}$ EID_{50}/g (*244;246;255*). H5N1 viruses were detected in frozen duck carcasses from German duck fattening farms in 2007. There were no obvious clinical signs of disease in the ducks (*92*). Avian influenza virus (H5N1) is also present at high levels in feathers of infected ducks (*288*).

Since HPAI viruses can be detected in muscle tissue and internal organs of infected birds, they could potentially be present and infective in raw or lightly cooked meat. There is no definitive evidence of foodborne transmission of H5N1 to humans although consumption of fresh duck blood and undercooked poultry was reported to be associated with some human illness (*95*). Eagles, cats, tigers, dogs, and some laboratory animals have become ill after consuming raw meat from infected birds (*125;240;267*).

Foot and Mouth Disease Virus (FMDV)

Foot and Mouth Disease (FMD) is caused by a small, non-enveloped, single-stranded RNA virus that typically infects cattle, pigs, sheep, goats, buffalo and other hoofed animals. Seven main serotypes of FMD have been described. Infected animals contain high titers of virus in blood, skin, muscle, and internal organs and shed numerous virus particles in saliva, milk, semen, urine, and feces even before clinical symptoms are evident. Viruses persist in some animals for months and years after disease symptoms have cleared. FMD is highly contagious, spreading rapidly through contact and aerosols and, if outbreaks are not quickly contained, they can have devastating economic consequences not only on farmers and food processors but also on a variety of retail businesses and unrelated industries such as tourism (*179;222;239*).

A nationwide epidemic of FMD in the UK in 2001 initially infected pigs, likely through consumption of contaminated meat products. Apparently healthy swine that carried FMD were slaughtered, contaminating the abattoir, and personnel working there spread the virus to other farms. FMD also spread from the index pig farm by aerosol to sheep on nearby farms. Sheep often experience less severe symptoms of disease and some infected sheep were sent for processing, further spreading FMD around the country before the outbreak was recognized (*68*). Over 2000 cases of FMD were confirmed and six million animals were culled, with an estimated loss of £3.1 billion to agriculture and food processors [http://footandmouth.csl.gov.uk/ ; http://www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/fmd/2001/index.htm].
A localized outbreak occurred in the UK in 2007 and was apparently due to escape of infective virus from the drainage system of a vaccine production plant. FMD virus was most likely carried off-site by soil, water, or other material contaminated by effluent and deposited on the road leading to the first infected premises [http://www.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/fmd/2007/index.htm].There was no evidence of transmission of this virus to humans in either of these outbreaks.

Potential spread of FMD among cattle in California (*157*) and Texas (*274*) and among wild deer and feral pigs (*275*) in the U.S. has been studied and transmission patterns modeled.

Severe consequences of FMD outbreaks on livestock producers and processors have prompted extensive research, dating back to 1927, on survival of FMD virus in various animal products. Two recent reviews summarized this research and noted conditions and processes that inactivated this virus

(*199;222*). Following slaughter, lactic acid levels in muscle tissue increase causing a drop in pH to 5.6–5.7. FMD virus is inactivated in meat under these conditions but if meat is frozen soon after slaughter, the smaller decrease in pH may not be sufficient to inactivate the virus. Virus in lymph nodes, bone marrow, and blood clots is protected from acid and can survive for weeks or months during refrigerated or frozen storage. Virus also survives for extended periods in pork fat and may persist for weeks in refrigerated dairy products.

Heat inactivates FMD virus, with D values of 6.06–10.87 sec (70ºC) and 1.65–3.18 sec (90ºC) measured for virus suspended in saline solutions (*113*). Fats and proteins in foods protect viruses during heating so D values are greater during thermal processing of milk and meat. Not all viral particles are inactivated during some pasteurization protocols and data on thermal inactivation in meat are inconsistent. Results from thermal treatments reported in older papers may or may not be relevant to current processing methods. It should be noted that the oral infectious dose of FMD virus (for animals) is greater than the respiratory infectious dose. Even if some viral particles survive processing, the number of viruses remaining may be lower than the infectious dose (*222*).

FMD virus is detectable in some hams (Parma, Serrano, Iberian) during the first few months of curing but is not present when hams are fully cured (*168;170*). FMD virus has been reported to survive for 2–3 months in pork sausage and bacon (*222*). Salt treatment of intestinal casings enhances destruction of FMD virus during storage at 20ºC (*285*).

Several thermal processes were recently tested for inactivation of FMDV in milk. Only UHT (135ºC, 1 sec) and double set pasteurization at 72ºC, 30 sec reliably inactivate the virus (*4*). FMDV survived HTST pasteurization (72–95ºC for 18.6 or 36 sec) although up to a 4-log reduction occurred under some conditions (*261*). Inactivation of FMD during cheese production depends on the type of cheese, including the length of curing and curing temperature (*56;222*). FMD virus does not survive in yogurt because of the low pH of 4.4 (*4*).

Only a few proven cases of FMD have occurred in people who have been in close contact with infected animals. Illness has been mild, usually with tingling blisters on the hands. Fever, sore throat, and blisters on the feet have also been reported. However, the incidence of human infection is rare considering the large number of people exposed to infected animals worldwide, with about 40 human cases reported in the literature. Transmission of the virus to humans usually occurs via the respiratory system. In one case a veterinarian was apparently infected through a cut on his hand. Although the virus is present in milk of infected cows, the only reported human illness from consuming contaminated milk was said to have occurred in 1834 when 3 veterinarians deliberately drank milk from an infected cow (*13;55*).

Other Viruses Reported as Transmitted by Food

Nipah Virus is a relatively large single-stranded RNA virus (Paramyxoviridae) with a lipid envelope. Symptomatic infections in people resemble influenza-like illness which may be followed by encephalitis, coma, and death in 40–75% of patients. It is naturally found in fruit bats in Southeast Asia but does not cause illness in them. Bats secrete this virus in saliva and urine and may contaminate fruit that is then consumed by people, cats, pigs, horses, or other animals. An outbreak of febrile encephalitis was first described in more than 250 people in Malaysia and Singapore in 1999. Epidemiological investigations indicated that infection spread from pigs to people working on large pig farms and in abattoirs processing swine carcasses (*39*). An outbreak of encephalitis caused by Nipah virus in Bangladesh in 2004–2005 was traced to consumption of raw date palm sap. Bats drink this sap during the night from clay pots used to collect it. The sap, gathered by people the following day, contained infective virus. Twelve persons met the case definition, with eleven fatalities (*149*). During the past 10 years there have been recurrent outbreaks of Nipah virus in Bangladesh. About half of these cases are believed to have developed from person-to-person contact, and some of the other cases may have resulted from contamination of fruit or juices by fruit bats (*148*). A related bat-borne virus, **Hendra virus**, has infected a few people caring for sick animals and has also been transmitted orally to cats and horses (*277*).

Tick-borne Encephalitis Virus, another enveloped RNA virus (Flaviviridae)**,** is normally spread to humans and animals through tick bites (*156*). Six human cases of this disease in a mountainous area of Austria were recently traced to consumption of raw goat milk and cheese from a goat that had been infected with this virus (*104*). A 2007 outbreak in Hungary affecting 25 people was also attributed to raw goat milk (*12*). Other cases, associated with raw milk and products made with raw milk including cheese and yogurt, have been reported previously in Europe. Infected goats, sheep, and cows have been shown to excrete this virus into their milk for several days early in an infection. Heat treatment will readily inactivate this virus (*58*).

West Nile Virus (WNV), an enveloped RNA virus (Flaviviridae)**,** is usually spread to birds, horses, humans, and other animals by mosquitoes but there is one report of an infant acquiring this virus from his mother's milk (*192*). Other human infections have

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occurred in workers at a turkey farm and in persons receiving blood transfusions or organ transplants from infected people. Cats consuming infected mice (*7*) and alligators consuming infected raw horse meat (*109*) have both developed symptoms of WNV infection. Owls and corvids (crows and relatives), which are seriously affected by WNV, excrete >6 logs of WNV virus particles in their feces which could contaminate foods consumed by other animals. Chickens are not seriously affected by West Nile virus and excrete much lower titers of virus. WNV is detectable in blood and internal organs of chickens (*79;127;232*).

Other Viruses of Potential Concern

These animal viral diseases have been mentioned by some authors as viruses of concern because they cause disease in farm animals and may be present in meat or milk. Further information on these diseases is available at a number of websites, including Iowa State University [http://www.cfsph.iastate.edu/DiseaseInfo/] and OIE [http://www.oie.int/eng/maladies/en_technical_diseasecards.htm]. No human cases of many of these diseases (Aujeszky's Disease, African Swine Fever, Classical Swine Fever, Lumpy Skin Disease, Peste des Petits Ruminants, Porcine Circovirus 2, Porcine Reproductive and Respiratory Syndrome, Rinderpest, Sheep and Goat Pox) have been reported either in people handling the animals or in those consuming raw or lightly cooked animal products. Given the high mutation rate of viruses, of course, it is possible that one or more of these viruses could, in the future, acquire traits that would permit infection of humans.

Other viruses included here (Bluetongue, Newcastle Disease, Rabies, Rift Valley Fever, SARS, Swine Vesicular Disease, Vesicular Stomatitis) have caused human illness. Transmission has not been foodborne but has occurred through direct contact, inhalation of aerosolized viruses, or insect vectors. One human case of bluetongue was reported in a laboratory worker.

African Swine Fever is a serious disease of wild and domestic pigs, usually spread by ticks or contact with infected animals, that is currently confined to Africa and feral pigs in Sardinia. The virus is very resistant to environmental stress and survives for long periods in the environment, for months in refrigerated boned meat and salted hams, and for years in frozen meat (*107;170;203*). This virus is resistant to many common disinfectants, but sodium hypochlorite and some iodine, phenolic, and quaternary ammonium compounds inactivate the virus. No human illness caused by this virus has been reported.

Bluetongue is a disease of wild and domesticated ruminants that is usually spread by biting midges and not by contact. Sheep and deer generally develop more severe symptoms than cattle, goats, and elk. This disease has been detected in many parts of the world and has recently (since 1998) spread into Europe (*98*). The virus is susceptible to sodium hypochlorite and 3% sodium hydroxide. There has been one report of a human infection in a laboratory worker but this disease is not considered zoonotic (*107*).

Classical Swine Fever (hog cholera) is a very contagious disease found in much of Asia and Central and South America and some countries in Africa. Pigs, both domesticated and wild, are the only known reservoir of the disease and acquire infections from contact with other pigs or consuming meat from infected animals. There are no records of human infection with this virus. The virus can survive up to 300 days in some cured hams (*170*), up to 75 days in salami (*197*), for months in the refrigerator, and for years in the freezer. However, the virus is readily inactivated by sodium hypochlorite, phenolic compounds, detergents, quaternary ammonium compounds, formaldehyde, and pH >11 and <3. Heating at 71ºC for 1 min or 65.5ºC for >30 min inactivates the virus (*59;60;107*).

Lumpy Skin Disease Virus (LSDV) causes a pox-type illness in cattle in Africa and the Middle East. Transmission occurs primarily through biting insects; virus may also be spread by ingestion of contaminated milk. LSDV survives for months on dry surfaces in the environment but is susceptible to ether, chloroform, formalin, phenol, sodium hydroxide and quaternary ammonium compounds. Heating at 60ºC for 10 min or 56ºC for 30 min inactivates the virus in milk. There are no reports of human infection with this virus (*8;107*).

Newcastle Disease (ND) is a viral disease of birds that most severely affects chickens, although there have been virulent outbreaks among other domestic and wild birds. Low pathogenicity strains (lentogenic) are common worldwide, including the U.S., while higher pathogenicity strains (velogenic) have been eradicated from more developed countries. Some pets, such as parrots, and wild birds can harbor the virus and excrete it without exhibiting symptoms. Virus is acquired by birds by inhalation and ingestion since the virus is shed in both feces and respiratory secretions. Sodium hypochlorite, phenolic compounds and oxidizing agents can inactivate NDV. Laboratory workers and vaccination crews, exposed to high concentrations of virus, have developed conjunctivitis. There have also been reports of a mild, self-limiting influenza-like illness in exposed persons and one reported case of a lethal lung infection in an immunocompromised person, associated with a pigeon strain of NDV (*81*). NDV is inactivated in egg products using industry standard pasteurization protocols (*245*). D values for thermal destruction of NDV in meat

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homogenate ranged from 120 sec at 65ºC to 29 sec at 80ºC (*3*). NDV in naturally and in artificially contaminated chicken meat was inactivated in <1 sec at 70 or 73.9ºC. The FSIS time-temperature guidelines to achieve a 7-log reduction in *Salmonella* in chicken meat effectively inactivated both high and low pathogenicity NDV strains (*107;254*).

Peste des Petit Ruminants (PPR) and Rinderpest are caused by related enveloped, RNA viruses. PPR affects primarily sheep and goats with some cases reported in other wild and domesticated ruminants while rinderpest was a highly contagious, serious disease of cattle. Rinderpest (virulent strains) appears to have been eradicated from the world although there may be infectious virus remaining in remote areas of Africa. PPR appears to be spreading. Transmission is usually by close contact between animals. Both viruses are considered fragile and do not survive more than 4 days in the environment. Little information is available on inactivation of PPRV in the environment but data on rinderpest suggest that it would be inactivated by UV light, desiccation, pH <5.6 or >9.6, and temperatures >70ºC. PPRV may survive for some time in refrigerated and frozen meat but is inactivated by many disinfectants: sodium hypochlorite, sodium hydroxide, phenolic compounds, alcohols, iodophores. The virus may be present in milk but there are no reported human cases and these are not considered zoonotic diseases (*107;174*).

Porcine Circovirus 2 (PCV2) contains a short covalently closed, single strand of DNA and is associated with several diseases in pigs (*282*). The usual route of transmission appears to be fecal–oral but the virus is also present in urine and oral and respiratory secretions. PCV2 is present in lymphoid tissue, bone marrow, and skeletal muscle of infected pigs at concentrations sufficient to cause infection in naïve pigs by the oral route (*195*). Experiments have demonstrated that this virus is very resistant to dry heat up to 120ºC for 30 min and moist heat at 75ºC for 15 min (*188;278*). Sodium hydroxide and sodium hypochlorite effectively inactivate PCV2 and some quaternary ammonium compounds also reduce infectivity (*118*). There are no reports of human infection with this virus.

Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) was first identified in 1991 and has since spread worldwide to cause respiratory illness in growing pigs and a high rate of abortion in sows. During the first two weeks after infection, PRRSV reaches its highest levels in lungs and lymphoid tissue, with lower levels detected in serum and muscles. Pigs generally acquire the infection through the respiratory tract but PRRSV has been transmitted experimentally to pigs by feeding meat from infected pigs to healthy pigs. However, it appears that there is rarely enough infective virus in muscle tissue to transmit infection by this route (*177*) [http://www.efsa.europa.eu/en/efsajournal/scdoc/239.htm]. Houseflies can transport the virus between different pig populations (*206*) and PRRSV has been detected in wild boars in the U.S. and Europe (*212*). Several disinfectants, including phenolic compounds, a quaternary ammonium compound, sodium hypochlorite, and sodium hydroxide inactivate PRRSV (*118*). Commercial spray drying of animal plasma effectively destroys infectivity of PRRSV (*175*). No human cases of infection with this virus have been described.

Pseudorabies (Aujeszky's Disease) Virus (ADV) is an enveloped DNA-containing herpes virus that causes a highly contagious neurological disease of pigs. This disease has been eradicated in domestic swine in many countries but does persist in wild boars in Europe and the U.S. (*93;112;212*). The virus has been detected in a number of tissues and organs of infected animals, and pigs and carnivores such as dogs have become ill by consuming meat of infected pigs (*164*). Cattle, sheep and goats can be infected but are usually dead-end hosts (*32*). ADV can be inactivated by some disinfectants (orthophenyl phenols and quaternary ammonium compounds), sunlight, drying, and high temperatures. Commercial spray drying of animal plasma effectively destroyed infectivity of pseudorabies virus (*175*). No symptoms of Aujeszky's disease have been reported in humans but seroconversion does occur.

Rabies. There have been several incidents in recent years in the U.S. in which people consumed raw milk and shortly afterward learned that one of the cows supplying the milk was rabid (*167*). In 2005, 62 people in Oklahoma who were potentially exposed to rabies by this route received post-exposure prophylaxis [http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/MilkSafety/ConsumerInformationAboutMilkSafety/ucm183174.htm].Although there are no published data on the concentration of rabies virus in milk of rabid animals, the virus is known to be present throughout the bodies of sick animals, and consumption of raw milk and meat from rabid animals is not recommended. No human cases of rabies associated with oral exposure have been reported but experimental animals and bats have acquired rabies from oral exposure (*14;71;133*).

Rift Valley Fever (RVF) is a zoonotic disease, spread by mosquitoes, that has serious effects on domestic ruminants in Africa and the Middle East. Humans can also be infected by mosquitoes and by direct contact with tissues of infected animals during slaughter and meat preparation for cooking. Most human infections are self-limiting febrile illness but 1–2% of people develop serious complications. Foodborne transmission of RVF is considered possible

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because the virus is present in milk and blood/meat of animals, and humans are known to be susceptible to infection. The virus is inactivated by pH< 6.2 and would therefore be destroyed in meat that has matured and may be destroyed by stomach acid following ingestion. The virus can survive for months at refrigeration temperatures and for years in the freezer. Lipid solvents, detergents, and sodium hypochlorite are effective disinfectants (*19;107*).

SARS (Severe Acute Respiratory Syndrome) Virus emerged as a human pathogen in China in 2002 and rapidly spread to 25 countries within 3–4 months. Early in the outbreak, the virus was associated with animals and people at live animal markets selling a variety of wild-caught animals, but later evidence indicated that the SARS virus likely originated in bats. Although SARS is a respiratory illness primarily spread by aerosols, it also causes gastrointestinal symptoms in about 40% of cases and fecal shedding occurs. There have been no confirmed cases of foodborne transmission of SARS but it is possible that the virus could be transmitted by infected food handlers (as with human enteric viruses) or by raw or undercooked meat of infected animals (*58*). Coronaviruses can persist in water for nearly 3 weeks at 25ºC, suggesting that contaminated water is a potential vehicle of infection (*30*).

Sheep and Goat Pox Viruses cause a pox-type illness in sheep and goats in Africa, Asia, and the Middle East. Transmission occurs primarily through the respiratory route during close contact. Viruses may survive for months in shaded sheep pens and on wool or hair but are susceptible to ether, chloroform, formalin, phenol, sodium hydroxide and detergents containing lipid solvents. Heat sensitivity varies among strains. Generally, 65ºC for 30 min or 56ºC for 120 min inactivates the virus. There are no reliable reports of human infection with this virus (*8;107*).

Swine Vesicular Disease Virus (SVDV) causes moderate to severe disease in swine and has caused mild flu-like infections in laboratory workers. The virus appears to be related to a human pathogenic coxsackievirus. Farmers and veterinarians have not been infected by sick pigs. Pigs become infected through close contact with infected animals or contaminated materials and by consuming meat from infected animals. SVDV is present in all swine tissues, is resistant to fermentation and smoking, and is stable at a wide pH range of 2–12. It survives in hams for 180 days, dried sausages for >1 year, and processed intestinal casings for >2 years. SVDV is also relatively heat stable and resistant to most commonly used disinfectants. Sodium hydroxide combined with a detergent is one recommended disinfectant (*107;170;171*).

Vesicular Stomatitis Virus (VSV) is an important livestock disease in the Americas that affects mainly cattle, swine and horses. Serological evidence of infection has been detected in a wide range of domestic and wild animals including rabbits, rodents, turkeys, ducks, ruminants, carnivores, and raccoons. Humans are also susceptible to this virus. People handling affected animals and contaminated materials and working in laboratories may be infected by contact or by aerosols. Insects may transmit the virus to animals and possibly to humans. Cattle that ingest contaminated grasshoppers become ill but there are no reports of foodborne transmission to humans. VSV has been detected in raw milk but not in other edible tissues of livestock. It does not appear to survive manufacture of cheddar cheese (*40*). VSV does not survive long in the environment and is inactivated by UV light, lipid solvents, sodium hypochlorite, sodium hydroxide, iodophores, and sodium carbonate (*107;133*).

STRATEGIES FOR CONTROL OF VIRUSES

Interventions for control of microbes in foods include refrigeration and freezing, acidification, reduced water activity, thermal processing, high pressure, irradiation, ozone, ultraviolet light, modified atmospheres, and various disinfectants and sanitizers. Some interventions, such as refrigeration/freezing and modified atmospheres, have little or no effect on reducing infectivity of viruses and, in fact, may protect viruses from destruction. These processes will not be discussed further. There is some variation in sensitivity to other processes among different viral strains, and the food matrix can protect viruses from processing methods and sanitizers. Recent experiments with human norovirus in a variety of foods demonstrated that freezing, cooling and mild heat treatment were not effective in significantly reducing virus titers (*178*). Reviews have summarized information on the effects of food processing methods on viruses (*9;101;283*).

Little data is available for some viruses because they cannot be cultivated in tissue culture (e.g. norovirus) or high-level biosecurity facilities must be used for testing (e.g., SARS virus). For these viruses, surrogate viruses (feline calicivirus for norovirus and murine hepatitis virus for SARS) have been tested to estimate efficacy of some interventions. These surrogates are useful but do not always accurately predict responses of pathogens to stresses (*100*). Detection of non-cultivatable viruses may be accomplished by PCR which identifies viral DNA or RNA in a sample. This technique may demonstrate that a virus has been present in a sample but the virus may no longer be infectious because the protein coat has been denatured.

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Thermal Treatments

Heat disrupts viral proteins and can be an effective means of destroying the infectivity of viruses. Heating to 56–60ºC in laboratory media or buffer reduces concentrations of many viruses by 3 logs or more. Thermal inactivation curves for viruses are often biphasic, with some viruses, perhaps aggregated together or attached to protective food constituents, exhibiting greater thermal stability (*18*). Viruses can be extremely resistant to heat when dried on surfaces, particularly in the presence of organic material. For example, porcine circovirus survives 30 min at 120ºC and lumpy skin disease virus retains infectivity for at least 10 min at 100ºC. An enteric virus, adenovirus, can withstand 10 min at 80ºC when dried (*107;188;278*).

Cooking and commercial thermal processes do not always inactivate all the viruses present in a product. Noroviruses, hepatitis A virus, and other enteric viruses often remain infective in shellfish after steaming. Shellfish are usually heated just until the shell opens (about 70ºC, 47 sec) to avoid toughening the meat. However, this is not enough heat to destroy poliovirus or HAV (*102*). Immersion of shellfish in boiling water for 3 min is recommended for destroying HAV and norovirus (*99*).

Thermal inactivation studies of high pathogenicity avian influenza viruses in infected chicken meat have demonstrated that virus titers remained unchanged up to 50ºC and then start to decline at 60ºC. H5N1 virus was undetectable in all meat samples after 5 sec at 70ºC. Viruses were found to be consistently inactivated when meat changed from the pink-tan color of raw meat to the white, firm cooked product (*244*). Hepatitis E virus in meat is inactivated by 71ºC within 5 min (*67*). FMD virus survives some, but not all, pasteurization processes in milk (*222*).

Differences in viral proteins impact heat sensitivity, and food components such as fat exert a protective effect, permitting viruses to withstand higher temperatures (*101*). Other ingredients in foods also affect thermal stability. For example, 44% more norovirus survived heating for 1 min at 72–74ºC in spiced tomato sauce than in buffer solution (*180*). Therefore D10 values should be measured in foods of interest and not just in laboratory media.

Table 6 presents available recent data on effective thermal treatments that have been reported for viruses in media or buffer, meat, milk, or eggs. Experimental conditions used in generating these data varied, so the values are not exactly comparable for different viruses. Much more information is available for some viruses than for others.

Drying / Decreased Water Activity

Salt (sodium chloride) and drying have been used for thousands of years to decrease water activity in meat, fish, vegetables, eggs, and even some fruit and thereby limit bacterial growth in preserved foods. Salt may have some antiviral effects but reduced water activity does not rapidly inactivate animal viruses. Pig intestines used for natural sausage casings may contain porcine viruses. Storage of these casings at 20ºC for 30 days in a salt solution inactivated classical swine fever virus and foot and mouth disease virus (*284;285*). However, experiments with different varieties of cured hams demonstrated survival of FMD virus for 108–182 days, African swine fever virus for 140–300 days, classical swine fever virus for 140–252 days, and swine vesicular disease virus for 365–560 days (*168;170*). In dried pepperoni and salami, African swine fever virus was present for >15 days, classical swine fever virus for >30 days, FMD virus for 56 days, and swine vesicular disease virus for 400 days (*20*).

Salt has a protective effect on hepatitis A virus subjected to high pressure in media. In the presence of 6% sodium chloride, 1 min of 400 MPa, 50ºC reduced HAV titer by 0.4 log; in the absence of added salt, HAV titer was reduced by 3.9 log. A similar protective effect of sodium chloride was observed in high pressure processing of samples containing norovirus (*119;120*).

Viruses lose some infectivity when dried on surfaces but many viruses persist for a month or more dried on paper, cloth, plastic, aluminum, and ceramics. Length of survival is related to the type of surface, relative humidity, and the presence/absence or organic material, such as food or feces, as well as to the structure of the viral capsid proteins. Viruses with lipid envelopes, such as pseudorabies, do not persist as long on surfaces as those without a lipid envelope, for example hepatitis A and parvovirus (*128;251*). Drying often increases viral resistance to other stresses such as heat and sanitizers (*251;278*).

Commercial spray drying of animal plasma (to produce a feed additive) effectively destroyed infectivity of pseudorabies, porcine circovirus, swine vesicular disease virus, and porcine reproductive and respiratory virus. PCV2 DNA was detected in the spray dried plasma but was not infective when tested in young pigs (*208;210*).

Vacuum freeze-drying is used to manufacture high quality dehydrated products. This process inactivated HAV on berries and herbs by 0.29–1.24 logs (measured by PCR) or by 1.24–2.42 (measured by viral culture). Noroviruses appeared to be more sensitive to this process, with log reductions ranging from 0.63 to 3.52 logs (by PCR) (*27*). Freeze-drying is also used to produce plasma-derived medicinal products.

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Experiments testing viral survival during freeze-drying demonstrated that HAV and pseudorabies titers were reduced, generally by 2 to 5 logs. Previous studies have shown that poliovirus is also significantly inactivated by lyophilization but less than one log reduction was observed for parvoviruses (*265*).

Irradiation

Irradiation may be useful in controlling bacteria, parasites, and insects in foods but the high irradiation doses required to significantly reduce viral titers may adversely affect organoleptic properties of foods. The small size and low moisture content of viruses ensures that they are more resistant to irradiation than bacteria, including spore-formers. Data on some foodborne viruses (hepatitis, polio, rotavirus) tested in fish, shellfish, or beef indicate that D values (dose required to reduce virus titers by one log) range from 2 to 10 kGy (*200*). Tests with Newcastle Disease virus in "egg fluid" yielded a D value of 2 kGy (*257*). A low pathogenicity avian influenza strain exposed to high energy electron beam irradiation was inactivated with D10 values of 1.6 kGy in egg white and 2.6 kGy in ground turkey (*25*). Poliovirus, hepatitis A virus, and rotavirus in oysters were inactivated with a D10 values of 2.94, 2.0 and 2.4 kGy, respectively (*9;111*). Therefore, at approved radiation doses (**Table 7**), titers of these viruses would be reduced by only 2–3 logs in meat and shellfish. A dose of 3 kGy was required to achieve 1-log reduction of hepatitis A virus on lettuce or strawberries (*17*).

Table 7. Approved maximum limits for irradiating foods in the U.S. (21CFR179.26, January, 2010).

Shellfish, fresh or frozen	5.5 kGy
Meat, uncooked, refrigerated	4.5 kGy
Meat, uncooked, frozen	7.0 kGy
Meat, frozen, packaged for NASA only	44.0 kGy
Poultry, uncooked, fresh or frozen	3.0 kGy
Eggs, shell, fresh	3.0 kGy
Spinach and lettuce, fresh	4.0 kGy
Spices and herbs, dried	10.0 kGy
Seeds for sprouting	8.0 kGy

Table 8 presents more reported data on inactivation of viruses by irradiation. Although relatively low doses of irradiation may inactivate viruses suspended in buffer, proteins and other organic compounds in foods quench the radicals generated by irradiation and reduce its antiviral effects (*48*). Very high doses of gamma rays are needed in some cases. Porcine circovirus, the smallest animal virus, is extremely resistant to gamma irradiation: 45 kGy causes only a 1-log reduction (*207*). Irradiation at 40 kGy was recommended for destroying infectivity of FMD virus dried on a glass surface (*49*).

High Pressure

High hydrostatic pressure effectively inactivates some viruses under certain conditions (*9;101*). Exposure of HAV and rotavirus to 450 MPa at 22ºC for 5 min resulted in a 7–8-log reduction in titer (*87*). However, a small fraction of rotaviruses suspended in buffer appeared to be much more resistant to high pressure (*117*). Large capsid proteins may protect some viruses, such as poliovirus and aichi virus which experience <1-log reduction in titer following 60 min at 600 MPa at 20ºC (*69*).

As with other processing methods, viruses are more readily inactivated by high pressure in buffer solutions and simple media than in foods where sugars, salts and other components protect viral integrity. Higher salt and sucrose concentrations in buffer solutions protected HAV and feline calicivirus from the effects of high pressure (*86;88;120*). Treatment at low temperatures (refrigeration or freezing) increases destruction of some viruses but HAV is more sensitive to pressure at high temperatures (*122*). Feline calicivirus is most resistant to pressure at 20ºC and becomes more sensitive at temperatures above and below this (*35*). Decreasing pH of suspending solution enhanced pressure inactivation of HAV but decreased inactivation of feline calicvirus (*119;120*).

High pressure destruction of viruses in foods has also been tested. Feline calicivirus and HAV were inactivated by 2.89–3.23 logs in sausage by 5 min of 500 MPa at 4ºC (*234*). In tests with an HPAI H7N7 virus suspended in chicken meat, 25 seconds at 15ºC and 500 MPa induced a 5-log reduction in virus titer (*108*). Other viral inactivation studies have been done with oysters (*29;123;143*), strawberries, and green onions (*121*). Viruses in oysters are less sensitive to pressure than in buffer solutions. Relatively mild pressure treatments (250 MPa), commonly used in seafood processing, caused little inactivation of viruses in oysters (*182*). Two models for control of viruses by high pressure processing have been published: HAV (*88*) and feline calicivirus (norovirus surrogate; *26*). Other available data on effects of high pressure processing are presented in **Table 8**.

Ultraviolet Light

UV light primarily damages RNA and DNA of viruses, although at very high doses, it can damage proteins as well. Thymine or uracil dimers are produced in the nucleic acids, which results in mutations. Double-stranded DNA and RNA viruses are more resistant to UV light because only one strand is damaged and the other strand can serve as a template for repair (*61;101*). Adenoviruses are more resistant to UV than other enteric viruses. A medium pressure UV system proved to be a more effective disinfectant than

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low pressure systems, causing >4-log reduction in titer at 100 mJ/cm^2 (*145*).

UV light is a clean technology leaving no residues but has limited applications in the food industry because it does not penetrate into foods, and turbidity in liquids decreases its effectiveness. Rotavirus was inactivated by 2.5 logs in some juices (*189*). UV light is used for disinfection of water and for inactivating microbes on environmental and food surfaces. In laboratory media, D values for inactivation of feline calicivirus (FCV), hepatitis A virus, and poliovirus were 47.85, 36.5, and 24.1 mJ/cm^2, respectively (*187*). Inactivation of HAV, FCV, and aichi virus on lettuce by UV was in the range of 4.5–4.6 logs whereas on strawberries, only 1.9–2.6 logs were inactivated. The uneven topography of the strawberry surface probably shielded some virus particles from the light (*70*). See **Table 8** for more data on ultraviolet light.

Ozone

Ozone gas is a strong oxidizing agent that can effectively kill microbes. Non-enveloped viruses, with their protein coats exposed, are subject to protein peroxidation while lipid oxides are formed in enveloped viruses (*183*). Ozone gas is used to disinfect surfaces (*105*) and may be bubbled through liquids (such as water and juices), and ozonated water has been used to rinse produce and chicken carcasses. Ozone can also be used in commercial laundry systems to inactivate microbes on cloths and mops (*214*). However, there are limitations to its use in foods. Organic compounds in foods react with and consume ozone, thereby deceasing the amount available to inactivate microbes. When ozone reacts with food components, it may alter flavors and colors. As temperature increases, ozone reacts more rapidly with organic material but it also decomposes more quickly. Ozone is most stable in solution at pH 5 and starts to decompose as pH increases (*101*). However, some viruses, including poliovirus, are more resistant to ozone under more acidic conditions (*220*). **Table 8** includes data on ozone concentrations used to inactivate viruses.

Acid or Alkali Treatment

Many enteric viruses tolerate pH values as low as 3 or 4 and as high as 9 or 10 (*283*). For example, HAV remained infectious after 90 min at pH 1 and norovirus in a stool filtrate remained infectious after 3 hours at pH 2.7 (*9*). Tolerance to acid is an adaptive feature since foodborne viruses must survive passage through the acidic stomach. Little or no reduction in norovirus titers occurred during storage in foods such as ketchup and salads at pH 4.5–5.5 (*180*). Several animal viruses, such as FMD, are sensitive to pH <6 and lose infectivity as animal carcasses are aged (*211*). One strain of HPAI H5N1 was unaffected by exposure to media of pH 3 or 12 for 10 min (*273*) but other strains are sensitive to pH <5 and >9 (*45;233*). Many viruses are sensitive to alkali and are inactivated by sodium hydroxide. Porcine circovirus lost about 2 logs of infectivity when exposed to 0.8% sodium hydroxide (*159*). See **Table 8** for more data on pH tolerances.

Sanitizers and Disinfectants

Sanitizers and disinfectants can inactivate viruses as well as bacteria but their effectiveness depends on temperature, viral concentration and aggregation, viral structure and size, and the presence of organic matter or dirt. Viruses with lipid envelopes are generally more susceptible to all disinfectants than non-enveloped viruses. Many animal viruses are surrounded by a lipid layer, but human enteric viruses and some animal viruses, including bluetongue, hepatitis E, FMD, parvovirus, circovirus, and swine vesicular disease, do not have a lipid envelope and can be more difficult to control (*45;101*). Some viruses, including porcine parvovirus, are very resistant to disinfectants that are generally effective against non-enveloped viruses (*63*).

Disinfectants are used in food preparation, processing, and production to sanitize water and to clean work surfaces, the surfaces of fresh produce, and employees' hands. Although disinfectants are usually tested initially in in vitro systems, viruses that have dried on surfaces are more resistant to disinfectants than viruses in liquid suspension (*63;251*). Furthermore, the topography of fruits and vegetables may include crevices that protect viruses from the effects of sanitizers (*9*).

Hand sanitation requires compounds that are effective and also mild enough to use on skin. Enveloped viruses, including influenza viruses, are sensitive to alcohol-based compounds but several studies demonstrated that these products have little capacity to reduce hepatitis A, norovirus or feline calicivirus levels on fingers. Antibacterial liquid soaps, including soaps containing triclosan, were not significantly more effective than washing hands with tap water (*16;135;144;146*). An evaluation of handwashing by volunteers whose hands were contaminated with feline calicivirus (FCV) in artificial feces noted that the greatest reduction in viral titer occurred after washing with regular liquid soap and using a nail brush. It is difficult to clean microbes from under the fingernails; keeping fingernails short will reduce the possibility of microbial contaminants on hands (*144*). A solution of 10% povidone-iodine reduced levels of FCV on fingers by 2.67 logs but is unlikely to be a popular choice for general use (*135*). An alcohol-based sanitizer that contained an organic acid and a polyquaternium polymer reduced murine norovirus, poliovirus, adeno-

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virus, and rotavirus on hands by 2.48, 2.98, 3.16, and 4.32 logs, respectively (*152*).

Fresh produce. Washing produce with water removes some dirt and about 1 log of inoculated viruses. Approved antimicrobials for washing produce include chlorine, chlorite, acidified sodium chlorite, organic acids, ozone, and peroxy acids. Chlorine, sodium hypochlorite, hydrogen peroxide, and peroxyacetic acid have also been used in several experiments to remove hepatitis A virus and surrogates for norovirus from lettuce, spinach, and strawberries. These sanitizers are less effective against viruses than bacteria and generally inactivated up to an additional 1 to 2 logs of virus particles. The presence of other organic compounds on the surface of produce (both natural compounds and "dirt") diminishes the effect of the sanitizers on viral infectivity (*9;10;101*). In addition, viruses may become internalized into leaves or other plant parts and are then likely protected from sanitizers (*276*).

Surfaces of equipment and food preparation areas are routinely cleaned with sanitizers and disinfectants to remove food residues and inactivate pathogens. Chlorine, as a gas or as calcium or sodium hypochlorite, is probably the most commonly used sanitizing agent. It has been reported to cause 2–5-log reductions in titers of several enteric viruses in buffer solutions. However, in kitchens and processing plants, its efficacy is often reduced by suboptimal pH or temperature conditions or the presence of organic material that reacts with chlorine (*9*). Other sanitizers include: oxidizing agents, such as peroxyacetic acid and hydrogen peroxide; quaternary ammonium compounds; phenolic compounds; and aldehydes, including glutaraldehyde and formaldehyde. Ethanol is more effective against enveloped viruses, such as avian influenza, than against non-enveloped viruses such as caliciviruses (*45;57;63*). Data from the literature on inactivation of viruses with these sanitizers is included in **Table 9**.

PERSPECTIVES

GMPs and SSOPs related to employee training, preventing carcass contamination with fecal material, ensuring cleanliness of water used for processing, and adequate thermal processing of foods are particularly important for meat processors to prevent viral contamination of products. Careless workers with poor personal hygiene and fecal material from animals or humans (potentially present in inadequately treated water) are likely to be the main sources of viruses contaminating meat products. Heat does kill viruses but temperature or time of exposure may need to be greater than that used to kill bacteria. This should be tested with viruses of concern in real foods. Although viruses, unlike bacteria, will not multiply on surfaces or in crevices containing food residues, cleaning and sanitation of equipment and surfaces is important to reduce the possibility of food contamination.

Human enteric viruses cause the overwhelming majority of cases of viral foodborne illness. These viruses are difficult to eradicate because they lack a lipid envelope, making them relatively insensitive to many sanitizers, acids, and some other interventions. Therefore, preventing their introduction into a food processing and preparation environment is certainly preferable. Education of workers with an emphasis on hygiene and providing facilities for maintaining cleanliness and the use of treated water in production and processing will be major deterrents to contamination of food with these viruses. A point to keep in mind is that there are enteric viruses in livestock that are similar to human enteric viruses. No zoonotic transmission of these viruses has been demonstrated. However, the high mutation rate of these RNA viruses suggests the possibility that the host range of some of these viruses could expand to include humans. Interventions to avoid fecal contamination of carcasses to prevent transmission of bacteria may also aid in preventing transmission of viruses. However, many viruses are acid tolerant and would probably not be affected by some procedures such as acid washes.

Few animal viruses have been transmitted by food to humans. These include hepatitis E, tickborne encephalitis, and Nipah virus. Hepatitis E viruses lack an envelope whereas the others are surrounded by a lipid envelope. In all human cases, these viruses were transmitted by unheated or lightly cooked foods.

Some other animal viruses (avian influenza, bluetongue, foot and mouth disease, Newcastle disease, rabies, Rift Valley fever, SARS, swine vesicular disease, vesicular stomatitis, West Nile virus) are known to cause human illness. Transmission has not been foodborne but has occurred through direct contact, inhalation of aerosolized viruses, laboratory accidents, or insect vectors. (There is a report of

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West Nile transmitted by breast milk and some suggestions that a few cases of avian influenza may have resulted from foodborne transmission.) No human cases of other animal viruses (Aujeszky's disease, African swine fever, classical swine fever, lumpy skin disease, peste des petits ruminants, porcine circovirus 2, porcine reproductive and respiratory syndrome, rinderpest, sheep and goat pox) have been reported either in people handling the animals or in those consuming raw or lightly cooked animal products. We should remain alert to the possibility that a virus could develop the capability to infect humans in the future (given the high mutation rate of viruses). It is also true that, although humans have apparently not become ill from consuming food containing these viruses, several of these animal viruses can be transmitted to other species of animals through food.

Little data is available for some viruses because they cannot be cultivated in tissue culture (e.g. norovirus) or because high level biosecurity facilities must be used for testing (e.g., SARS virus). For these viruses, surrogate viruses (feline calicivirus for norovirus and murine hepatitis virus for SARS) have been tested to estimate efficacy of some interventions. These surrogates are useful but do not always accurately predict responses of pathogens to stresses. Development of cell culture methods for important viruses, particularly human noroviruses, should be a priority.

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Table 1. Human enteric and animal viruses potentially present in foods.
(Only the [*] viruses have been directly associated with foodborne illness; others have been suggested as potential foodborne pathogens.)

Virus	Family	Genetic material	Envelope	Foods / tissues detected in
Human Viruses				
Adenovirus*	Adenoviridae	ds DNA	No	Shellfish
Aichi Virus*	Picornaviridae	ss RNA	No	Shellfish
Astrovirus*	Astroviridae	ss RNA	No	Shellfish
Hepatitis A*	Picornaviridae	ss RNA	No	Shellfish, strawberries
Norovirus*	Caliciviridae	ss RNA	No	Shellfish, raspberries, deli meats, sandwiches
Parvovirus* / Bocavirus	Parvoviridae	ss DNA	No	Shellfish
Poliovirus*	Picornaviridae	ss RNA	No	Milk
Rotavirus*	Reoviridae	ds RNA	No	Sandwiches, shellfish, stew
Sapovirus*	Caliciviridae	ss RNA	No	Shellfish
Animal Viruses				
African Swine Fever	Asfaviridae	ds DNA	Yes	Meat, blood
Avian Influenza	Orthomyxoviridae	ss RNA	Yes	Lungs, meat, intestines
Bluetongue	Reoviridae	ds RNA	No	Blood, lymph nodes, milk?
Classical Swine Fever	Flaviviridae	ss RNA	Yes	Pork
Foot and Mouth Disease	Picornaviridae	ss RNA	No	Milk, meat, blood
Hepatitis E*	Hepeviridae	ss RNA	No	Liver and meat: deer & pigs
Lumpy Skin Disease	Poxviridae	ds DNA	Yes	Milk, skin, lungs
Newcastle Disease	Paramyxoviridae	ss RNA	Yes	Eggs, poultry products
Nipah virus*	Paramyxoviridae	ss RNA	Yes	Date palm sap, fruit
Peste des Petits Ruminants	Paramyxoviridae	ss RNA	Yes	Lungs, lymph nodes, milk?
Porcine Circovirus 2	Circoviridae	ss DNA	No	Swine tissues
Porcine Reproductive and Respiratory Virus	Arteriviridae	ss RNA	Yes	Meat from swine
Pseudorabies (Aujeszky's Disease)	Herpesviridae	ds DNA	Yes	Swine tissues
Rabies	Rhabdoviridae	ss RNA	Yes	Milk?
Rift Valley Fever	Bunyaviridae	ss RNA	Yes	Milk, meat
SARS Virus	Coronaviridae	ss RNA	Yes	Meat: raccoons, bats, civet
Sheep and Goat Pox	Poxviridae	ds DNA	Yes	Blood, skin, digestive tract, milk?, meat?
Swine Vesicular Disease	Picornaviridae	ss RNA	No	Meat and tissues
Tickborne Encephalitis*	Flaviviridae	ss RNA	Yes	Milk, cheese
Vesicular Stomatitis	Rhabdoviridae	ss RNA	Yes	Milk, skin
West Nile Virus	Flaviviridae	ss RNA	Yes	Milk, horse meat, birds

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Table 2. Reported transmission pathways for viruses.
(Only the [X] pathways have been reported; [--] indicates no report.)

	Foodborne to humans	Foodborne to animals	Ticks / Insects	Aerosols / Respiratory	Direct contact
Adenovirus	X	--	--	X	X
African Swine Fever	--	--	X	--	X
Aichi Virus	X	--	--	--	X
Astrovirus	X	X	--	--	X
Avian Influenza	--	X	--	X	X
Bluetongue	--	--	X	--	--
Classical Swine Fever	--	X	--	X	X
Foot and Mouth Disease	--	X	--	X	X
Hepatitis A	X	--	--	--	X
Hepatitis E	X	--	--	--	X
Lumpy Skin Disease	--	X	X	--	X
Newcastle Disease	--	X	--	X	X
Nipah Virus	X	X	--	--	X
Norovirus	X	--	--	--	X
Parvovirus & Bocavirus	--	--	--	X	X
Peste des Petits Ruminants	--	--	--	X	X
Poliovirus	X	--	--	X	--
Porcine Circovirus 2	--	X	--	X	X
Porcine Reproductive and Respiratory Virus	--	X	X	X	X
Pseudorabies (Aujeszky's Disease)	--	X	--	X	X
Rabies	--	X	--	--	X
Rift Valley Fever	--	--	X	X	X
Rotavirus	X	--	--	--	X
Sapovirus	X	--	--	--	X
SARS Virus	--	--	--	X	--
Sheep and Goat Pox	--	--	--	X	X
Swine Vesicular Disease	-	X	--	--	X
Tickborne Encephalitis	X	--	X	--	--
Vesicular Stomatitis	--	X	X	X	X
West Nile Virus	X	X	X	--	--

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Table 6. Effective thermal treatments for destruction of viruses in media or buffer, meat or shellfish, and milk or eggs.
($D = D_{10}$ value for inactivation. Log = log reduction. [--] indicates no data available.)

Virus / Disease	Media / Buffer	Milk / Egg / Other	Meat / Shellfish	References
Adenovirus	56°C, 1 min, 3 log; Dried on surface: dry heat, 80°C, 10 min, 3.8 log; moist heat, 70°C, 10 min >4 log	--	--	(*63*; *101*)
African Swine Fever	56°C, 70 min; 60°C, 20 min	Serum: 60°C, 30 min	70°C, 30 min	(*191*)
Aichi Virus	--	--	--	
Astrovirus	60°C, 10 min: no effect	--	--	(*230*)
Avian Influenza (HP)	45°C: D = 330 min; 55°C: D < 28 min	Dried egg: 60°C, D = 192.2 min; Whole egg: 60°C, D = 188 sec	Chicken: 70°C, 5 sec	(89; *244*; *256*)
Bluetongue	60°C, 15 min; 50°C, 180 min	--	--	(*191*)
Classical Swine Fever	--	Blood: 66°C, 60 min	65°C, 30 min; 71°C, 1 min	(*107*)
Foot and Mouth Disease	80°C, 3.75 min	70°C, 30 min	70°C, 30 min	(*113*; *191*)
Hepatitis A	70°C, 4 min, >6 log	80°C, 0.68 min, 5 log	60°C, 10 min, 2 log	(*9*; *101*)
Hepatitis E	--	--	71°C, 5 min	(*67*)
Lumpy Skin Disease	Dried: 100°C, survives 10 min	Milk: 30 min, 56°C; 10 min, 60°C	65°C, 30 min	(*107*; *191*)
Newcastle Disease	60°C, 30 min; 56°, 180 min	Milk: 65°C, 20-30 sec	Chicken: <1 sec, 70°C	(*190*; *191*; *254*)
Nipah Virus	60°C, 60 min	--	--	(*191*)
Norovirus *surrogate	60°C, 30 min, still infectious; 71.3°C, 1 min effective*; 75°C, 2 min*	--	Boiling, 30 min; Roasting at 200°C, 30 min	(*26*; *57*; *101*; *180*)
Peste des Petits Ruminants	50°C, 60 min	--	60°C, 60 min	(*107*; *191*)
Parvovirus / Bocavirus	Dried on surface: dry heat, 80°C, 10 min, 0.5 log; moist heat, 70°C, 10 min, 0.7 log	Biowaste: 70°C, 30 min, 3 log	--	(*63*; *224*)
Poliovirus	Dried on surface: dry heat, 80°C, 10 min, >4.6 log; moist heat, 70°C, 10 min, 3.8 log	72°C, 0.5 min, >5 log	Steaming, 30 min, 2 log	(*63*)
Porcine Circovirus 2	Dried on surface: 120°C, 30 min, not effective; Media: 80°C, 15 min	Plasma: 80°C, 10 hr, >3.2 log; 60°C, 10 hr, 1.6 log	--	(*188*; *278*)
Porcine Reproductive and Respiratory Virus	--	--	70°C, 11 min	(*188*)
Pseudorabies (Aujeszky's Disease)	--	--	--	
Rabies	--	65°C, 20-30 sec	--	(*190*)
Rift Valley Fever	--	Serum: 56°C, >120 min	--	(*191*)
Rotavirus	50°C, 30 min, 2 log; 60°C, 10 min, 7 log	--	--	(*101*)
Sapovirus	--	--	--	
SARS Virus	75°C, 45 min, complete inactivation; 56°C, 20 min, most inactivated	--	--	(*44*)
Sheep and Goat Pox	56°C,120 min; 65°C, 30 min	--	--	(*191*)
Swine Vesicular Disease	56°C, 60 min	Biowaste: 70°C, 30 min	--	(*191*; *224*)
Tickborne Encephalitis	--	--	--	
Vesicular Stomatitis	58°C, 30 min	--	--	(*191*)
West Nile Virus	56°C, 30 min	--	--	(*65*)

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Table 8. Effective interventions to inactivate viruses.
(D = D_{10} value for inactivation. Log = log reduction. [--] indicates no data available.)

Virus / Disease	Irradiation	Ozone	pH	Pressure	UV light	References
Adenovirus	--	Buffer: 0.30 mg/L, 30 sec, 3.04 log; Buffer: 1200 ppmv, 60 min, 4 log	pH <6 or>9.5	Media: 400 MPa, 15 min, 20°C	Buffer: 226 mJ/cm^2, 4 log	(*31*; *101*; *183*)
African Swine Fever	--	--	pH <3.9 or >11.5	--	--	(*191*)
Aichi Virus	--	--	--	Media: 600 MPa, 5 min, 21°C	Media: 50 mJ/cm^2, 4 log; Lettuce: 40 mJ/cm^2, 4 log; Strawberries: 40 mJ/cm^2, 1.5 log	(*9*; *101*)
Astrovirus	--	--	pH <3	--	--	(*230*)
Avian Influenza (HP)	Turkey: D = 2.6 kGy	--	pH 5-9 stable; some strains stable at pH 3-12	500 MPa, 25 sec, 15°C	Water: 10 mJ/cm^2, 3.6 log	(*25*; *108*; *150*; *191*; *233*; *273*)
Bluetongue	--	--	pH <6 or >8	--	--	(*107*; *191*)
Classical Swine Fever	--	--	pH <3 or >11	--	--	(*191*)
Foot and Mouth Disease	Dried: 40 kGy	--	pH <6 or >9	--	--	(*49*; *191*)
Hepatitis A	--	Buffer: 0.25 mg/L, 1.2 sec, 2.7 log	pH <2	Buffer: 450 MPa, 5 min, 22°C; Oysters: 400 MPa, 1 min, 9°C, 3 log; Sausage: 500 MPa, 5 min, 4°C, 3.23 log; Strawberries: 375 MPa, 5 min, 21°C, 4.3 log	Media: 75 mJ/cm^2, 4 log; D = 36.5 mJ/cm^2	(9; 31;*86*; *88*; *101;* *183*; *187*)
Lumpy Skin Disease	--	--	pH <6 or >8.6	--	--	(*191*)
Newcastle Disease	D = 2 kGy	--	pH ≤ 2	--	--	(*191*; *257*)
Nipah Virus	--	--	pH <4 or >10	--	--	(*191*)
Norovirus *surrogate virus	--	Buffer: 0.37 mg/L, 10 sec, >3 log	pH <3 or >8*	Media: 450 MPa, 15°C, 7 log*; Media: 300 MPa, 3 min *; Oysters: 400 MPa, 5 min, 5°C, 4 log	25 mJ/cm^2*; 34 mJ/cm^2*; 12 mJ/cm^2*; D = 47.85 mJ/cm^2	(*26*; *48*; *57*; *86*; *101*; *123*; *140*; *183*; 187)
Peste des Petits Ruminants	--	--	pH <4 or >11	--	--	(*191*)
Parvovirus / Bocavirus	30 kGy, 5.8 log	--	--	--	--	(*173*)
Poliovirus	Buffer: D = 0.46 kGy; Oyster: D = 2.84 kGy; Frozen oyster: D = 8.2 kGy	Buffer: 0.37 mg/L, 10 sec, 3 log	Stable at pH 3	Media: 600 MPa, 60 min, 20°C, <1 log	Media: 21.7 mJ/cm^2, 4 log; D = 24.1 mJ/cm^2	(*31*; *86*; *101*; *111*; *183*; *187*)
Porcine Circovirus 2	45 kGy: 1 log	--	0.8% NaOH, 2 log	--	--	(*159*; *207*)
Porcine Reproductive and Respiratory Virus	0.25 kGy	--	pH ≤2 or ≥12	--	100 mJ/cm^2	(*51*)
Rift Valley Fever	--	--	pH < 6	--	--	(*107*; *191*)

Table 8, *continued next page*

Table 8, *continued*.

Virus / Disease	Irradiation	Ozone	pH	Pressure	UV Light	References
Rotavirus	Oysters: 2.4 kGy, 1 log	Buffer: 0.1 mg/L, 6 sec, 3 log	pH <3 or>9	450 MPa, 5 min, 22°C; 300 MPa, 2 min, 25°C, 8 log	56 mJ/cm^2, 4 log	(*31*; *87*; *101*; *142*)
Sapovirus	--	--	pH <3.5	--	--	(*31*)
SARS Virus	0.03-0.15 kGy not effective	--	pH <5 or >9	--	UV-C effective at 3 cm, 15 min	(*44*)
Sheep and Goat Pox	--	--	2% acid, 15 min	--	--	(*191*)
Swine Vesicular Disease	--	--	pH <2.5 or>12	--	--	(*107*; *191*)
Vesicular Stomatitis	--	Buffer: 1200 ppmv, 10 min, 6 log; Serum: 60 min, 4 log	pH <4 or >10	--	--	(*183*; *191*)

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Table 9. Effective sanitizer / disinfectant concentrations to inactivate viruses.
(Log = log reduction. [--] indicates no data available.)

Virus / Disease	Hypochlorite	Peracetic acid	Quaternary NH_4	Glutaraldehyde	Phenols	References
Adenovirus	Dried: 2500 ppm, 1 min, >4.1 log	0.2%, 10 min, >4.1 log	0.05%, 10 min, 1.2 log	2%, 10 min, >4.1 log	--	(*63*)
African Swine Fever	2.3%, 30 min	--	Effective	--	3%, 30 min	(*191*; *236)*
Aichi Virus	--	--	--	--	--	
Astrovirus	Not effective	Effective	Not effective	--	Not effective	(*230*)
Avian Influenza (HP)	5.25% effective; 0.05%, 10 min not effective	--	0.02%, 10 min not effective	0.1%, 10 min not effective	0.1%, 10 min	(*273*)
Bluetongue	--	--	--	--	Effective	(*191*)
Classical Swine Fever	Effective	--	Effective	Effective	--	(*191*)
Foot and Mouth Disease	3%	Effective	Not effective	Effective	Not effective	(*191*; *211*)
Hepatitis A	Strawberries: 200 ppm, 0.5 min, 1 log	--	--	--	--	(*28*)
Hepatitis E	--	--	--	--	--	
Lumpy Skin Disease	2-3%	--	0.5%	--	2%	(*191*)
Newcastle Disease	1-6%	1%, 1 min	1%, 1 min	--	Effective	(*76*; *191*)
Nipah Virus	Effective	--	--	--	--	(*191*)
Norovirus *surrogate	≥160 ppm; 3000 ppm*	1000 ppm, 5 min*	Effective*	2500 ppm, 5 min*	Effective at 2-4X recommended levels*	(*57*; *86*; *110*; *146*; *153*)
Peste des Petits Ruminants	Effective	--	--	--	Effective	(*191*)
Parvovirus / Bocavirus	Dried: 2500 ppm, 10 min, 1 log	0.2%, 10 min, >6 log	0.05%, 10 min, 0.4 log	2%, 10 min, 3.6 log	--	(*63*)
Poliovirus	Dry: 2500 ppm, 10 min, 4.5 log	0.2%, 10 min, >4.8 log	0.05%, 10 min, 0.8 log	2%, 10 min, >4.6 log	--	(*63*)
Porcine Circovirus 2	0.3%, 1.41 log	1%, 2.2 log	Effective	--	Not effective	(*159*; *221*; *292*)
Porcine Reproductive and Respiratory Virus	--	--	--	--	--	
Pseudorabies (Aujeszky's Disease)	--	--	Effective	--	Effective	
Rabies	--	--	--	--	--	
Rift Valley Fever	Cl >5000 ppm	--	--	--	Not effective	(*191*)
Rotavirus	Strawberries: 200 ppm, 0.5 min, >1.5 log	--	--	--	--	(*28*)
Sapovirus	--	--	--	--	--	
SARS Virus (murine hepatitis virus)	0.21%	--	0.1% with ethanol	Effective	--	(*44*; *50*)
Sheep and Goat Pox	2-3%	--	0.5%	--	2%, 15 min	(*191*)
Swine Vesicular Disease	0.03% not effective	Effective	Effective with NaOH	--	--	(*191*; *236)*
Tickborne Encephalitis	--	--	--	--	--	
Vesicular Stomatitis	1-3%	--	Effective	2%	--	(*107*; *191; 236)*
West Nile Virus	500-5000 ppm	--	--	2%	--	

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FRI Briefings

Hepatitis A

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We know how the hepatitis A virus (HAV) is spread, but foodborne enteric viruses remain a difficult problem to control. An outbreak affecting 153 schoolchildren and staff in Michigan this spring emphasizes the need for continued vigilance. The frozen strawberries implicated in this outbreak were grown in Mexico and then packaged in a California plant before being distributed to schools (*1*). The origin of HAV in this outbreak has not been established. Viral contamination may have occurred during picking in the strawberry fields of Mexico and/or during processing in the California plant. Although HAV is spread most commonly by direct person-to-person contact, there are many documented outbreaks of hepatitis A in the USA and other industrialized countries which have been traced to an infected food handler or some local source of contamination. Postings to the FSNet listserve (*2*) and Promed's web site on monitoring infectious diseases (*3*) since the beginning of the year include descriptions of food- and waterborne hepatitis A outbreaks in Los Angeles, Pennsylvania, Iowa and nearby midwestern states, Sweden, Australia, Russia, and Mexico, in addition to the outbreak in Michigan. These are, undoubtedly, only a fraction of the cases which actually occurred. It has been estimated that about one-third of the U.S. population has been exposed to HAV (*4*).

According to the Centers for Disease Control and Prevention (CDC), 31,582 cases of hepatitis A were reported in the USA in 1995 but the real incidence of this disease is estimated to be 138,000 cases/year (*5*). Not all of these were food- or waterborne. In fact, the majority of hepatitis A cases probably result from fecal–oral transfer during close contact with an infected person. Nevertheless, in its report on cases of foodborne disease outbreaks in the USA during 1988–1992, the CDC lists HAV as the fourth leading cause of foodborne disease (*6*). Numbers of hepatitis A cases in the USA wax and wane in approximately 10-year cycles. The last peak was in 1989, with 35,821 cases reported (*7*). Outbreaks are most often associated with consumption of salads, fresh fruits or vegetables, or shellfish, such as oysters.

Multiple types of hepatitis viruses are known to exist but only HAV is commonly associated with foodborne illness. Hepatitis E virus has been associated with waterborne illness whereas hepatitis B, C, and D are spread only through body fluids. HAV is an RNA virus which is quite resistant to drying and is more heat resistant than many other enteric viruses. Foods become contaminated with HAV by exposure to fecal material from infected persons. (Feces from an infected person can contain one million virus particles per gram.) Once the contaminated food or water is ingested, the virus makes its way to the liver but it may be 15–50 days before symptoms appear. The disease typically has an abrupt onset with fever, malaise, anorexia, nausea, dark urine, and jaundice. Usually hepatitis A is less severe in younger children and is only rarely fatal, although symptoms may persist as long as two months. There is no treatment for the disease other than to relieve symptoms. It is very important to realize that peak shedding of viruses in the feces begins about 2 weeks before obvious signs of illness and continues for about a week after onset of symptoms. Therefore, an infected food handler with poor personal hygiene habits may be a source of infection even before he or she is aware of being sick.

Contamination of food may occur in one of several ways: An infected food handler with dirty hands working in the field picking fruits or vegetables, in a food-processing plant sorting or packing, or at a restaurant making salads or fresh fruit plates may pass on this disease to others. Irrigation or processing water contaminated with sewage may introduce viruses onto fresh fruits and vegetables. Shellfish living in relatively shallow coastal waters may be exposed to viruses from inadequately treated sewage discharged from treatment plants on shore or from dumping of wastes from ships. In fact, current water treatment practices are unable to completely inactivate all enteric viruses.

An outbreak of hepatitis A associated with frozen strawberries also occurred in 1990 and was responsible for 28 confirmed cases in Montana and Georgia. An epidemiological investigation revealed that the containers of strawberries associated with illness in the two states were processed on the same night in the same plant in California (*8*). The berries were grown in California and could have been contaminated either in the field or in the processing plant. None of the plant or field workers were diagnosed with hepatitis A within two months before or after that processing date. However, significant underreporting of HAV infection is known to occur and investigators considered it likely that a worker was infected.

An infected baker was demonstrated to be the most likely source of 79 cases of hepatitis A in a community outbreak in New York in 1994 (*9*). The baker apparently contaminated doughnuts while applying a sugar glaze. Investigation revealed that the source of HAV was clearly someone who had handled the doughnuts after they had been cooked. This outbreak illustrates the point that any food can harbor HAV if it has been handled by an infected person just before serving. An interesting sidelight to this outbreak is its similarity to a 1968 community outbreak of hepatitis A in Michigan that was also traced to an infected baker applying glaze to doughnuts. This incident had been widely used as an epidemiology teaching exercise. Apparently its lessons were forgotten (or never learned) by a later generation of bakery workers.

Oysters, clams, and mussels are filter feeders that process relatively large volumes of water and can concentrate food particles (and viruses) from the surrounding water. Since oysters are frequently consumed raw and mussels and clams are often steamed lightly, HAV is a significant problem in these foods. Recent experiments demonstrated that mussels immersed in virally contaminated water readily adsorbed 4–56% of the virus particles present (*10*). One approach to cleansing shellfish of viruses, depuration, involves placing them in tanks of virus-free water for a period of time to allow them to "wash out" contaminants. However, commercial depuration procedures did not completely remove HAV from contaminated mussels even after 96 hours' immersion in a continuous flow of ozonated marine water (*10*). Cooking was also not completely effective in eliminating HAV. Viruses were still detectable in steamed mussels five minutes after the opening of the shells (*10*).

Detection of viruses in foods has been, for the most part, difficult and relatively unsuccessful. Recently developed techniques using polymerase chain reaction (PCR) assays to amplify the nucleic acids of a pathogen have been modified for detection of HAV in oysters. Using total RNA extracted from oyster meat, contaminated with HAV by adsorption, bioaccumulation, or injection, and reverse transcription-PCR, Cromeans et al. were able to detect the equivalent of as few as 8 plaque-forming-units (pfu) of HAV/gram of oyster meat (*11*). One drawback of such assays is that PCR detects the presence of nucleic acids even if the virus particle is no longer infectious because its protein coat has been disrupted by disinfectants or heat. Using a magnetic immunoseparation step which immobilizes only undamaged viruses followed by PCR, Lopez-Sabater et al. detected as few as 10 pfu of infectious HAV in artificially contaminated oyster meat (*12*). Further development of these methods should aid in the detection of contaminated foods.

Outbreaks of foodborne disease may involve substantial costs to society and to food facilities as well as to the affected individuals. An investigation of a foodborne outbreak of hepatitis A involving 43 persons apparently infected by a food handler working for a catering company in Colorado concluded that the costs from a societal perspective were $809,706 (*13*). Medical costs incurred by the cases totaled $46,064 while disease control costs included $450,397 for 16,293 immune globulin injections and $105,699 for 2777 hours of health department personnel time. These estimated costs were considered conservative and did not include all of the preventive medical costs and the insurance compensation for pain and suffering for persons who developed the disease. This latter cost was expected to be several hundred thousand dollars.

Strategies for preventing outbreaks of hepatitis A include preventing contamination by: (a) strict attention to personal hygiene, especially hand washing, by all food handlers; (b) use of clean, not fecally contaminated, water for irrigating, washing and processing foods; and (c) prevention of the contamination of shellfish beds by sewage (*14*). If contamination of food has occurred, thermal processing is generally effective in destroying infectivity. However, HAV is relatively heat-resistant as well as resistant to drying and acid. Virus particles in water and on surfaces can be inactivated by UV light and by strong oxidizing agents, such as chlorine or ozone.

The recent introduction of a vaccine for hepatitis A offers the possibility for prevention of some outbreaks of this disease by vaccination of food handlers. Based on limited data, two doses of the vaccine are estimated to confer immunity for at least 6–7 years and maybe as long as 20 years (*4*). (Prior infection with HAV confers lifelong immunity.) This vaccine will be much more useful than the immunoglobulin shots which afford protection for only 3–6 months. Economic analyses will be needed to determine whether it is cost-effective and desirable that some or all food handlers be vaccinated (*15*). For example, in the Colorado outbreak, it would have cost about $9600 to vaccinate the 100 food handlers who were employed at the catering facility (*12*). Preventive efforts should be increased as we approach the time for next peak in the cycle of hepatitis A cases.

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