Viruses have “emerged” as causes of foodborne disease, according to data compiled by the Centers for Disease Control and Prevention. During 1983–87 in the United States, Norwalk virus was the fifth leading cause of foodborne disease among outbreak-associated illnesses; hepatitis A virus was the sixth; and other viruses (principally rotaviruses) were tenth (Bean et al., 1990). By 1988–1992, the most recent period for which data have been issued, Hepatitis A virus had risen to the fourth leading cause and Norwalk-like viruses first appeared on the top 10 list as the ninth leading cause (Bean et al., 1996).

The numbers of reported foodborne illnesses are fewer than actually occur because the CDC’s passive data collection system records only illnesses occurring as outbreaks, rather than those occurring sporadically. Hepatitis A, which is notoriously under reported in the United States (Cliver, 1985), is the only foodborne viral disease in which official reporting is mandatory for all diagnosed cases. Thus, records of the incidence of the other viral diseases are certain to be less accurate. And, just as with Norwalk-like viruses, other new viral agents are likely to appear among the top 10 causes of foodborne disease in future compilations.

Special Features of Viruses Among Foodborne Disease Agents

Particle as the Transmissible Form. Viruses pass from host to host in the form of inert particles. The particles are roughly spherical, with diameters of 25 to 35 nm (picornavirus and calicivirus; for example, hepatitis A and Norwalk-like viruses, respectively) or as large as 75 nm (rotaviruses; Table 1). The smaller foodborne viruses contain single-stranded RNA, whereas the rotaviruses contain double-stranded RNA. Human enteric viruses containing DNA are known, but none have been proven to be transmitted via food or water. The outer surface of the particle is a highly specific protein coat that protects the RNA, interacts with a susceptible host cell to initiate infection, and acts as the antigen against which the host’s immune responses are mounted. Because these particles are totally inert, they cannot multiply in foods or anywhere outside the host. Neither can they carry out any metabolic activity, nor respond to stresses encountered in the environment.

Viral Infection. The virus particle will enter only a suitable host cell. Specificity depends on the interaction of the coat protein with receptors on the host cell. Only certain cells in the bodies of certain species can be infected; essentially all viruses transmitted to humans via foods are specific for humans and perhaps a few other primates. In practical terms, zoonotic viruses are not transmitted via foods.

When the viral coat protein reacts with homologous receptors on the cell membrane, the host cell engulfs and uncoats the viral RNA (Cliver, 1990). The RNA is translated to various virus-specific proteins and replicated (with the help of virus-specific, RNA-dependent RNA polymerase) into additional copies of viral RNA. The replicative cycle takes place in the host cell’s cytoplasm, without participation of DNA. (Reverse transcription of the viral genome does not occur.) As coat protein and viral RNA accumulate in the host cell, progeny particles assemble themselves and eventually leave the cell via leakage or in blebs that pinch off the cell’s surface membrane.

Viral disease may occur when progeny virus spreads and infects enough host cells to interfere with some normal bodily function. The viral replication may kill or subvert the host cells. In the case of hepatitis A, however, the host’s infected liver cells apparently are little affected until the body mounts an immune response and destroys the infected cells by means of “killer” (cytotoxic) T-cells. Whether the liver or the lining of the small intestine is the site of viral infection, the hepatitis or gastroenteritis that results is seldom fatal.

Epidemiology of Foodborne Viruses

Enteric (fecal-oral) Transmission. Essentially all foodborne viruses are transmitted enterically; they are shed with feces and infect by being ingested (Cliver, 1990). Like many other infectious agents that are enterically transmitted, the majority of infections are probably contracted by person-to-person contact, most probably via fecally soiled hand to mouth. If vomiting is part of the illness, viral particles may be shed with vomitus. Indirect transmission...
of enteric agents may occur via vectors such as flies, fomites such as soiled diapers, but most importantly via the vehicles of food and water. In the United States, more foodborne than waterborne viral illnesses are recorded.

Foods as Vehicles for Viruses. A few enteric viruses of humans have been reported to be indirectly transmitted via vehicles other than foods; whereas others are foodborne with some frequency. Because hepatitis A virus is the only reportable virus in the United States, it is the only virus for which transmission via food or water can be compared by using total recorded incidence figures (Cliver, 1985). The proportion of reported hepatitis A cases attributed to food- and waterborne transmission, collectively, in the United States is only 3 to 9% (CDC, 1994). However, it must be recognized that the numerator (e.g., foodborne illnesses) and the denominator (total reported illnesses) are compiled in very different ways. That is, the foodborne illnesses are only those occurring in outbreaks that happen to have been investigated. Many are not. Thus, illnesses in unrecorded outbreaks and those occurring sporadically, though also foodborne, would not be included. It is also certain that not all diagnosed cases of hepatitis A are reported through official channels, but the proportion missed in this way is probably less than for the foodborne category. In any case, it seems likely that if all of the recorded foodborne illnesses had been prevented, no statistically significant change in the total rate of recorded hepatitis A in the United States would be detected. Data from other countries are very difficult to obtain and are generally not compiled in ways that permit comparison with those from the United States.

Viruses among Foodborne Disease Agents. Various groups of viruses occupied three of the top 10 spots as causes of reported foodborne disease in the U.S. during 1983–1987 (Bean et al., 1990). This was a period when the Norwalk virus was regarded as one agent, rather than a group of variably related viruses. Therefore, one would have expected that the number of reported food-associated cases would increase as the diagnostic “net” broadened. However, Norwalk-like viruses fell to ninth position (two outbreaks comprising 292 cases) during 1988–1992 (Bean et al., 1996), which suggests that the many improved diagnostic methods that have appeared in the literature are being applied very sparingly. Among reported outbreaks of foodborne disease in the United States in 1988–1992, etiologies were determined in 1,001 (41%), comprising 36,890 (48%) of the cases; 4% of these outbreaks and 6% of the cases were attributed to viruses. Until methods for detecting viruses in foods are improved and widely applied, knowledge of foodborne virus transmission will result from successful diagnoses of human illnesses. Here, too, it should be noted that much foodborne illness is endured without consulting a physician and that physicians are unlikely to order diagnostic tests for virus disease because, if the viral diagnosis is confirmed, the physician has no means of treating the illness. The hepatitis A virus now appears to be causing more foodborne illnesses than many of the better-known bacterial pathogens. Among the 1,422 recorded foodborne outbreaks during 1988–1992 that were of undetermined etiology (presumably gastroenteritis in most instances), it was surmised on the basis of incubation periods longer than 15 h that 35% might have been caused by viruses (Bean et al., 1996).

Shellfish as Special Vehicles for Viruses. Bivalve molluscs, such as clams, cockles, mussels, and oysters, are especially prone to transmit viruses. The waters in which they grow are increasingly subject to human fecal contamination, sometimes from sewage discharges and sometimes from infected shellfish harvesters. The shellfish collect viruses in the course of their filter feeding activity. Human viruses do not infect these species, but they are harbored for days or weeks in the shellfish digestive tract and are apparently more difficult to remove than bacteria during processes intended to cleanse the shellfish (e.g., depuration; Grohman et al., 1981; Power and Collins, 1989).

Unlike many other seafoods, shellfish are usually eaten with their digestive tracts in place. They are often eaten raw or lightly cooked. Shellfish, unlike other foods, may also protect viruses from thermal inactivation during cooking (DiGiarlo et al., 1970).

The first recorded outbreak of shellfish-associated viral disease resulted from storing clean oysters in a illegally contaminated harbor while awaiting sale (Gard, 1957). Over 600 cases of hepatitis A resulted. More recently, outbreaks of viral gastroenteritis and hepatitis A have been associated with eating usually uncooked shellfish. A clam-associated outbreak of hepatitis A in Shanghai may have been the largest recorded outbreak of foodborne disease in history (Halliday et al., 1991). Sporadic viral illnesses associated with shellfish have also been demonstrated (Koff et al., 1967); it is difficult to avoid bias entirely in such studies because, at least in coastal states, a diagnosis of hepatitis A regularly leads to asking the patient about shellfish consumption, to the exclusion of other foods.

Shellfish-growing waters are typically monitored for fecal contamination by testing for bacteria of the fecal coliform group or for Escherichia coli. The presence of these bacteria, however, has been shown to be a poor predictor of the presence of human enteric viruses (Wait et al., 1983). Unfortunately, no more accurate index of the presence of viruses in shellfish or their growing waters has yet been identified. Because it has no other way to guarantee the safety of raw cockles, the U.K. government allows their sale only if cooked by an approved method.

Costs and Special Features of Outbreaks. Hepatitis A is one of the more severe of foodborne diseases; a few weeks of debility are common, and permanent impairment of some liver functions occurs occasionally. In contrast, viral gastroenteritis typically lasts 1 or 2 days. In the absence of directly applicable data, costs per case of foodborne hepatitis A have been estimated at $5000, versus $887 for Norwalk gastroenteritis (CAST, 1994).

The span of onsets in an “explosive” outbreak — or one in which many people were infected on the same occasion — tends to equal the median incubation period of the disease. Therefore, an explosive outbreak of hepatitis A may show onsets of illness over a 28- or 30-day period (Cromeans et al., 1994). Under similar circumstances, outbreaks of Norwalk virus gastroenteritis would probably show onsets of primary illness over no more than 2 days (Appleton, 1994). Secondary cases may occur in both instances, with transmission of the infectious agent from those who ate
contaminated food, via contact, to others who did not. Norwalk gastroenteritis is often characterized by shedding of the virus in vomitus as well as in feces, so opportunities for secondary transmission are enhanced. Because immunity after Norwalk virus infection is only briefly protective, and perhaps because infectious doses are small, attack rates (number of persons ill divided by number of persons exposed) are often quite high (e.g., >50%; Appleton, 1994).

In outbreaks from food contaminated by a single infected person, or “diffuse” outbreaks, the long duration of shedding (10–14 days) may further spread the period of onsets of hepatitis A in situations where the infected person contaminates food on several days. Shedding of Norwalk-like viruses can continue for a week, so diffuse outbreaks of viral gastroenteritis are also possible. Either virus can persist in contaminated food and, thus, infect people who eat the food on different days, further “blurring” the outbreak. Given all these complications, it is remarkable that epidemiologists have been able to solve as many of the mysteries of hepatitis A transmission as they have. As yet, sporadic transmission resulting in single illnesses has defied characterization, except in the study concerning shellfish that was cited earlier (Koff et al., 1967).

**Risk Assessment.** The process of risk assessment has generally comprised hazard identification, dose-response assessment, exposure assessment, and risk characterization (CAST, 1994). Because foodborne infectious agents present some special features not seen with chemical hazards, the risk assessment process may need some modification in this application (Potter, 1996). Beyond this, viruses transmissible via foods offer some problems all their own. Hazard identification, based on the epidemiological record, focuses attention on hepatitis A virus and the Norwalk-like agents of gastroenteritis. Dose-response assessment for these viruses is complicated by the lack of laboratory hosts that they will infect; when human volunteer trials have been conducted, precise quantification of the doses administered was impossible (Dolin et al., 1972; Grohmann et al., 1981). Even when cell cultured enteric viruses have been administered orally, the number of viral particles comprising a cell culture infectious dose (e.g., a plaque-forming unit) was not determined. Assumptions based on various models for infectivity yield quite variable predictions. Exposure assessment is also difficult for foodborne viruses because there are no standardized methods for qualitative detection of viruses in foods, and even the best methods typically are not quantitative. Distribution of the few human viruses present in the food supply can be expected to be heterogeneous and nonrandom. The virus detection methods applicable to foods are unlikely to be used on a routine basis to determine which foods contain viruses because the cost of performing predominantly negative tests would be huge. For these reasons, precise risk characterization is not really an option. All the same, attempts have been made; results do not conform closely to present knowledge of the incidence of viral infections in the United States (Rose and Sobsey, 1993).

**Viruses Transmitted via Foods**

The hepatitis A and small gastroenteritis viruses are more often transmitted via foods than are other viruses. All known foodborne viruses except the agent of tick-borne encephalitis are human-specific and transmitted by a fecal-oral cycle. Perhaps incidentally, all of these “enteric” viruses contain RNA rather than DNA (parvovirus, if it is really foodborne, is an exception) and have no lipid envelope around their protein coats (Table 1).

**Hepatitis A Virus.** When hepatitis transmission via food was first recorded, it was not known that multiple types of viral hepatitis existed (Cliver, 1966). Now, at least five serological types of hepatitis viruses are recognized, belonging to various taxonomic groups; but only hepatitis A is documented as being foodborne (Cromeans et al., 1994; Table 2). Hepatitis E virus (a calicivirus) is apparently not present in North America; it has been implicated in water-associated, but as yet not food-associated, outbreaks (Cromeans et al., 1994). Hepatitis A is one of the more severe of foodborne diseases, especially among those caused by viruses (Table 3). Both hepatitis A and E viruses are listed as Severe Hazards in Appendix V of the 1995 FDA Food Code (USPHS, 1995). Virus produced in the liver is shed via the common bile duct and occurs at levels above 10^9 particles per gram of feces for days or weeks before the onset of illness. Food becomes contaminated via fecally soiled hands of infected persons or via fecally contaminated water, as is usual with shellfish. The virus is more heat resistant than most enteric viruses and is quite resistant to drying. Now that a formalin-killed vaccine has been licensed for use in the United States and Europe, food workers could be immunized to preclude the possibility that they might contaminate food with the virus (WHO, 1995). The vaccine is produced from a mutant strain of the virus that replicates in cell culture; wild type virus either does not replicate in cell culture or replicates very slowly, often without cytopathic effects (Cromeans et al., 1994).

**Small Round Structured Viruses Causing Gastroenteritis.** The Norwalk virus was the first gastroenteritis virus reported to be foodborne (Greenberg et al., 1979). Later, other serologically and genetically related viruses belonging to the calicivirus group were recognized (Table 4). The term “small round structured viruses” was applied to the agents when they were detected by electron microscopy or immune electron microscopy (Appleton, 1994). Astroviruses, which also have visible surface structure, are variably included in this group; they are discussed separately below. Gastroenteritis caused by the Norwalk-like viruses often includes vomiting, and virus shed in vomitus may contaminate food. Because

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### Table 1 — Major groups of nonenveloped human enteric viruses

<table>
<thead>
<tr>
<th>Diameter of nucleic acid strands</th>
<th>Nucleic acid type</th>
<th>Virus group</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-35 nm (single)</td>
<td>RNA</td>
<td>Caliciviruses, Picornaviruses</td>
</tr>
<tr>
<td>70-85 nm (double)</td>
<td>DNA</td>
<td>Adenoviruses, Rotaviruses</td>
</tr>
</tbody>
</table>

1. An envelope on a virus is a lipid-containing outer wrap that derives from the plasma membrane of the host cell in which the virus was produced.
immunity is transient, persons who have been infected and ill with the Norwalk virus are subject to reinfection with it, as well as with other serotypes, after perhaps a year (Parrino et al., 1977). Susceptibility is common, and attack rates in outbreaks are often quite high. Outbreaks have been traced both to ill persons (Kuritsky et al., 1984) and to food workers who had recovered from illness days earlier (White et al., 1986). The virus evidently is shed in large quantities, and small (not yet measured) quantities are infectious perorally. The individual viral particles, however, do not appear to be exceptionally resistant to inactivation by heat or chlorine (Appleton, 1994). Members of this group have not been shown to replicate in cell cultures.

Other Viruses Occasionally Transmitted via Foods. Many other groups of human enteric viruses are known, yet these are reported to be transmitted via foods only infrequently or not at all (Table 5). Factors such as duration and level of fecal shedding of the virus, efficiency of peroral infection, or stability of the virus in the food vehicle may play a role. Given that some of these viruses are able to replicate in laboratory cell cultures and cause cytopathic effects, they may be better characterized than the more important foodborne viruses discussed above.

• Astroviruses comprise a distinct group of small round gastroenteritis viruses that have surface projections in patterns often resembling five- or six-pointed stars (Appleton, 1994). The non-enveloped protein coat surrounds single-stranded RNA (Table 1). Usual features of the disease vary slightly from those of the Norwalk-like viruses; the incubation period is somewhat longer, vomiting is less common, and very young (<1 yr) children are more often affected. Epidemiologic evidence of transmission via foods is limited.

• Rotaviruses contain segmented, double-stranded RNA (Table 1) surrounded by a double protein coat (Sattar et al., 1994). They, too, infect young children most often and are occasionally associated with food and water.

• Picornaviruses other than HAV have single (+) stranded RNA (Table 1) in a simple protein coat that is featureless as seen by electron microscopy. The polioviruses were the first viruses shown to be foodborne, but virulent strains are now rare, and the vaccine strains are potential indicators of the possible presence of other, virulent viruses in food and water (Alhajjar et al., 1988). Although the coxsackieviruses have sometimes been reported in association with food and water, only one outbreak of foodborne coxsackievirus illness has been reported (Osherovich and Chasovnikova, 1967). Two foodborne outbreaks of echovirus illness have been recorded (NYDH, 1989; USDHEW, 1979).

• Tick-borne encephalitis virus is the only known foodborne virus that is not transmitted by a fecal-oral route (Greššková, 1994). The agent infects dairy animals in central Europe (principally Slovakia) via the bites of vector ticks, Ixodes persulcatus and I. ricinus. Infected animals shed the virus in their milk, which, if ingested without pasteurization, infects humans. Products made from the unpasteurized milk may also be vehicles. The virus belongs to the genus Flavivirus, meaning that the particle contains single (+) stranded RNA like most foodborne viruses, but has a lipid envelope around the protein coat. This is the only enveloped virus known to be foodborne. It is highly specific to its vectors and has a limited range, so that outbreaks are now rare. Seven people in Slovakia were affected during a 1993 incident (WHO, 1994).

• Hepatitis E virus belongs to the calicivirus group, so it has single (+) stranded RNA coated with protein that shows characteristic cup-like depressions (Cromeans et al., 1994). It occurs widely in Asia, Africa, and Latin America, but rarely elsewhere and apparently not at all in the United States and Canada (except for rare imported cases). It is the non-A, non-B hepatitis virus that was known to be transmitted by the fecal-oral route. Waterborne outbreaks are common; but for some reason, foodborne outbreaks have not yet been documented.

• Paroviruses are proposed as causes of human gastroenteritis. Although documented food-associated outbreaks are evidently quite rare, Appleton (1994) describes an outbreak associated with cockle consumption in England that involved at least 800 persons.

DETECTING FOODBORNE VIRUSES

Detection of viruses in food is most likely to be undertaken when an outbreak has occurred. It would be helpful, however, if some routine testing method were available to apply to foods, such as shellfish, that often serve as vehicles for viruses.

Outbreak investigation and routine monitoring present very different sets of priorities. When people have already been made ill, the relatively high costs of attempting to detect viruses in food samples may be acceptable, especially if litigation is anticipated. However, as was mentioned earlier, an outbreak of hepatitis A affecting several people is likely to be recognized only 4 weeks or more after the contaminated food was consumed, so pertinent food samples most likely will be unavailable for testing. With other viral diseases that have shorter incubation periods, clinical histories and diagnostic samples of feces and blood serum may afford some indication of which virus is to be sought in the food, which could prove very helpful. In contrast, testing foods for virus contamination in the hope of preventing human illness presents special problems. Costs are likely to outweigh demonstrable benefits. In addition, since almost all foodborne viruses are transmitted by fecal-oral cycles, one virus is as likely as another to occur in food, subject to fecal contamination. As ingenious as the methods are that have been devised for detecting foodborne viruses, none could be considered routine.

A fundamental problem is that the viruses of greatest concern, hepatitis A viruses and the Norwalk-like gastroenteritis viruses, replicate slowly and

<table>
<thead>
<tr>
<th>Type</th>
<th>Former name</th>
<th>Transmission Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Infectious hepatitis</td>
<td>Fecal-oral</td>
</tr>
<tr>
<td>B</td>
<td>Serum hepatitis</td>
<td>Parenteral</td>
</tr>
<tr>
<td>C</td>
<td>Non-A, non-B</td>
<td>Parenteral</td>
</tr>
<tr>
<td>D</td>
<td>Delta agent</td>
<td>Parenteral</td>
</tr>
<tr>
<td>E</td>
<td>Non-A, non-B</td>
<td>Fecal-oral</td>
</tr>
</tbody>
</table>


**Table 3 — Hepatitis A virus and disease**

| Picornavirus: particles, featureless spheres, 28 nm in diameter, single-stranded RNA coated with protein |
| Infection via intestine to liver; incubation period 15-50 days (mean 28 days) |
| Illness from immune destruction of infected liver cells: fever, malaise, anorexia, nausea, abdominal discomfort, often followed by jaundice; severity tends to increase with age and ranges from inapparent infection to weeks of debility, occasionally with permanent sequelae |
| Shedding: peaks during the second half of the incubation period (10-14 days); usually ends by 7 days after onset of jaundice |
| Diagnosis: based on detection of IgM-class antibody against hepatitis A virus in the patient’s blood serum (kits available) |
| Immunity: durable (possibly lifelong) after infection; active immunity can be produced with a killed-virus vaccine; passive immunity is imparted by injection of pooled human immune serum globulin |

Adapted from Cromeans et al., 1994.

**Table 4 — Norwalk-like, small round structured viruses of gastroenteritis**

| Calicivirus: particles, spheres 25-35 nm in diameter, single-stranded RNA coated with protein that has characteristic cupped surface depressions; Norwalk-like viruses include Cockle, Ditchling, Hawaii, Oklahoma, Parramatta, Snow Mountain, and Taunton agents |
| Infection of intestinal lining, incubation period usually 24-48 hours |
| Illness: nausea, vomiting, diarrhea, etc., generally lasting 24-48 hours |
| Shedding: during illness in vomitus and feces, possibly 7 days after onset |
| Diagnosis: detection of virus in stool by ELISA or PCR or of antibody against the virus in patient’s blood serum; no standard methods, reagents not readily available for most agents |
| Immunity: apparently transient |

Adapted from Appleton, 1994.

Inapparently or not at all in laboratory cell cultures. If foodborne viruses cannot be detected on the basis of their infectivity, alternate bases include their morphology (as seen by electron microscopy), their antigenic specificity (as demonstrated by reactions with homologous antibody), their genetic specificity (as demonstrated with complementary probes or polymerase chain reaction [PCR] primers), or combinations of these. These methods may be less sensitive than tests based on infectivity, and by their nature all carry some risk of yielding a positive result with viruses that has been inactivated (no longer infectious). The final detection method to be used must be considered when the food sample is being processed for testing. An additional problem encountered with the hepatitis A virus is that the incubation period of the illness is so long (average 4 weeks) that pertinent food samples are unlikely to be available once the disease has been recognized. These problems will be addressed briefly here, as they have recently been reviewed fairly extensively elsewhere (Cliver, 1995; Cliver et al., 1992). The remaining detection methods are based on the morphology, antigenic specificity, or genetic specificity of the viral particle.

**Methods for Detecting Viruses Extracted from Foods.** As was mentioned above, infectivity tests are not applicable to the viruses that are most often foodborne. Nevertheless, infectivity testing may be appropriate when astroviruses, rotaviruses, and perhaps a few others are sought (Chung et al., 1996; Wait et al., 1983). The remaining detection methods are the same as those used to detect other viruses. For example, one method is to use nucleic acid probes to detect specific sequences of nucleic acid from the virus. Another method is to use specific antibodies to detect the presence of the virus in the sample. These methods are not applicable to the viruses that are most often foodborne. Nevertheless, infectivity testing may be appropriate when astroviruses, rotaviruses, and perhaps a few others are sought (Chung et al., 1996; Wait et al., 1983). The remaining detection methods are based on the morphology, antigenic specificity, or genetic specificity of the viral particle.
Table 5 — Diseases caused by less commonly recognized foodborne viruses

<table>
<thead>
<tr>
<th>Virus group</th>
<th>Illness (major signs)</th>
<th>Incubation</th>
<th>Duration</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Astroviruses</td>
<td>Diarrhea</td>
<td>3-4 days</td>
<td>2-3 days</td>
<td>Some replicate in cell culture</td>
</tr>
<tr>
<td>Picornaviruses&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Meningitis, myalgia, etc.</td>
<td>3-5 days&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Varies</td>
<td>Food vehicle rare</td>
</tr>
<tr>
<td>Rotaviruses&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Fever, vomiting, watery diarrhea, in children, even occasionally severe dehydration</td>
<td>1-3 days</td>
<td>4-6 days</td>
<td>Often severe death</td>
</tr>
<tr>
<td>Tick-borne encephalitis (Flavivirus)</td>
<td>Fever, headache, nausea, vomiting, encephalitis or aseptic meningitis</td>
<td>7-14 days</td>
<td>Varies</td>
<td>Virus shed in milk of tick-bitten animals</td>
</tr>
</tbody>
</table>

<sup>a</sup> Sometimes longer  
<sup>b</sup> Other than hepatitis A virus  
<sup>c</sup> Variable

are available. If the convalescent phase serum reacts with the virus and the acute phase serum does not, this is likely to have been the agent that caused the illness. Unfortunately, the method will not detect the smaller numbers of viral particles that are capable of causing disease.

- Antigenic specificity of the viral coat protein can serve directly as a means of detection by enzyme immunoassay or by radioimmunoassay. These methods have been of considerable use in diagnostic virology, where they are applied to fecal samples containing levels of virus often exceeding a million particles per gram. Because contaminated foods are likely to contain only imperceptible levels of feces, the quantities of virus present in food samples are likely to be much smaller, and immunoassay tests are seldom adequately sensitive. Therefore, serologic reactions between virus and antibody are most often applied in combination with electron microscopy or with nucleic acid-based tests.

- Nucleic acid tests are generally based on the specific interactions of portions of the viral genome with complementary probes, PCR primers, or both. PCR and other genetic amplification methods afford highly sensitive means of detecting small quantities of virus. Because almost all known foodborne viruses are RNA agents, the viral nucleic acid must be extracted and reverse-transcribed to complementary DNA before amplification by PCR can begin. Short complementary probes bracketing a selected region of the viral genome are introduced with a polymerase that functions at high temperature, and many successive cycles of replication, denaturation, and annealing are carried out by a programmed thermal cycler. The result is millions of short copies of the selected region of the viral genome. The specificity of these copies is demonstrated on the basis of their appropriate length (in terms of number of nucleotide base pairs), their reaction with a complementary probe that is directed at a portion of the amplified segment, or both. Probes used for this purpose are labeled with a radionuclide, an enzyme, or some other means of expressing their presence in association with the PCR-amplified product.

In addition to the challenges of performing the demanding PCR and probe-specificity tests themselves, testing food samples has been found to present some special problems. Certain food components interfere with reverse transcription or with PCR amplification. Specialized means of surmounting these problems are being reported (Gouvea et al., 1994). The genetic diversity of the Norwalk-like viruses also causes problems (Ando et al., 1995; Wang et al., 1994). Although such nucleic acid-based test methods are exquisitely specific, it is possible to amplify or probe for a segment of the viral genome that is common to more than one type, to afford a broader test when needed (Jaykus et al., 1995).

- Combined methods consist typically of a serologic method followed by another detection procedure, as with the immune electron microscopy approach described above. Virus has also been captured from sample extracts with homologous antibody for later detection by PCR (Deng et al., 1994). This method seems to obviate some of the problems with food inhibitors of reverse transcription and PCR, and it allows RNA to be released from the viral particle simply by heating. It seems that detection methods combining nucleic acid tests and morphologic tests have not been devised.

**Indicators as Alternatives for Monitoring Foods.** Fecal coliform bacteria and *Escherichia coli* have long been used for monitoring shellfish and their growing waters, but bacteria are essentially irrelevant to the presence of viruses in shellfish and in other foods (Berg, 1978; Wait et al., 1983). Human enteric viruses that are capable of replicating in laboratory cell cultures and producing cytopathic effects have been proposed as alternate indicators of virus contamination. At least in instances of contamination via community sewage, there may be enough of such viruses, particularly vaccine polioviruses, to afford an indication of viral contamination. Infectivity tests are inherently quite sensitive, but are expensive and may take several days to a few weeks to produce firm results. Other candidate indicators of viral contamination of food have been bacteriophages that infect intestinal bacteria, such as *E. coli* or *Bacteroides fragilis*. These bacteriophages are relatively easy to detect, with readout times of as few as 6 h, but they do not seem more reliably present than are cytopathic human viruses. In general, any of these indicators might be of value in identifying foodborne viruses transmitted via sewage contamination but not via feces-soiled hands.

**PREVENTING VIRUS TRANSMISSION VIA FOODS**

Clearly, the reason for compiling information regarding virus transmission via foods is to develop strategies for preventing viral foodborne diseases. Vi-
ruses present some unique features among foodborne disease agents. Since they cannot multiply in foods, one might reason that control would be relatively easy, but this has not proven to be the case.

**Preventing Contamination.** With the sole exception of the tick-borne encephalitis virus, foodborne viruses come from human feces and can be kept out of foods by preventing human fecal contamination. Obviously, this has proven a difficult task, even in the matter of pre-harvest monitoring of shellfish and their growing waters. Conditions that had been judged acceptable can change very rapidly, with highly unforeseen results (Halliday et al., 1991). Contamination via the hands of infected food handlers is quite another matter. Infections that are completely inapparent (Eisenstein et al., 1963) are known, but incubating infections, in the case of hepatitis A (USDHEW, 1973), and briefly persistent infections in persons convalescing from viral gastroenteritis (White et al., 1986) have been greater problems. With viruses transmitted by the fecal-oral route, there is a need for good personal hygiene practices and high standards of food protection and sanitation procedures. In these instances, proper hand washing, done frequently and efficiently (using friction action and a nail brush), is the general preventive (except for a very few instances in which contamination by vomitus has been suspected). Feces on the hands of infected persons may contaminate food, whether the person is a field worker, a kitchen worker, or a server. Gloves may be of some value in preventing hand contact, but availability of hand washing facilities and management’s emphasis on proper hand washing techniques are the most important precautions to prevent direct human contamination of foods with viruses (Ansari et al., 1989; Cliver and Kostenbader, 1984).

Vaccination of food workers against hepatitis A is also of value in locations where natural immunity from infection is not regularly attained early in life (WHO, 1995).

**Inactivation of Viruses in Foods.** Assuming that contamination has not been prevented, viruses in food cannot multiply, but may be inactivated before someone eats the food. Storage at room temperature may encourage inactivation of viruses in food, but may lead to bacterial hazards. Thermal processing is generally effective, although some further attention to the adequacy of milk pasteurization to inactivate hepatitis A virus is needed (Parry and Mortimer, 1984). The British Government recommends heating milk to 85–90°C for at least 90 seconds to destroy viruses (IFST, 1996). Alternatively, shellfish may be depurated (i.e., held in facilities supplied with clean saline water) or delayed from where they grew into clean water to purge themselves of contaminants. Although these practices have served well in removing pathogenic bacteria from shellfish, success with viruses is not guaranteed. Longer periods of treatment are required for removal of viruses than bacteria (Grohman et al., 1981; Power and Collins, 1989), and individual shellfish may not cleanse themselves. Other food processes that are not based on heat are less effective: viruses present a small target for ionizing radiation; enteric viruses are generally acid-resistant, and the hepatitis A virus, at least, is quite resistant to drying. Viruses in water or on surfaces can be inactivated by strong oxidizing agents, such as chlorine or ozone, and by ultraviolet light.

**HACCP.** Food safety, at least in the developed world, is increasingly predicated on the hazard analysis and critical control point (HACCP) system, along with appropriate general precautions. Foods subject to fecal contamination via wastewater or contact by the feces-soiled hands of infected persons are at risk of contamination with viruses; this is the essence of hazard analysis in this case. Critical control points are points at which contamination can be prevented (as in disinfecting potentially contaminated water so that virus is not carried to food) or undone (such as by cooking an “at-risk” food thoroughly after it is last handled). Thorough hand washing, or prevention of hand contact with foods that will not be cooked thereafter before being served, are also very important measures. Although depuration or relaying of potentially contaminated shellfish may have some preventive value, these probably should not be considered critical control points in preventing virus transmission via shellfish eaten raw.

**SUMMARY**

Viruses are transmitted to humans via foods as a result of direct or indirect contamination of the foods with human feces. Viruses transmitted by the fecal-oral route are not strongly dependent on foods as vehicles of transmission, but viruses are important among agents of foodborne disease. The viruses most often foodborne are the hepatitis A virus and the Norwalk-like gastroenteritis viruses. Detection methods for these viruses in foods are very difficult and costly; the methods are not routine. Indicators that would rapidly and reliably suggest the presence of viral contamination of foods are still being sought. Contamination can be prevented by keeping feces out of food or by treating vehicles such as water to inactivate viruses that might be carried to a food. Viruses cannot multiply in food, but can usually be inactivated by adequate heating. Other methods of inactivating viruses within a food are relatively unreliable, but viruses in water and on exposed surfaces can be inactivated with ultraviolet light or with strong oxidizing agents.

**REFERENCES**


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