Viral Foodborne Disease Agents of Concern

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ABSTRACT

Viruses transmitted to humans via foods generally emanate from the human intestines. In the United States, Norwalk virus ranked #5, hepatitis A virus #6, and “other viruses” (principally rotavirus) #10 among the top 10 causes of foodborne disease during 1983-1987. Molluscs are the most frequently reported vehicles, but any food handled by humans may transmit human enteric viruses. Some fruit and vegetable vehicles may have been contaminated in the field before or during harvesting. Viruses in foods may be inactivated before the food is eaten, and thus, not cause infection. Increasingly sensitive detection methods, largely based on “molecular” techniques, are becoming available for these viruses but are not applicable to monitoring foods on a routine basis.

FOODBORNE VIRUSES ARE HUMAN ENTERIC VIRUSES

From early in the Twentieth Century until World War II, the only known foodborne viral disease was poliomyelitis (5). Then--shortly after hepatitis A was recognized as foodborne and before the poliomyelitis vaccines were licensed--food-associated outbreaks of poliomyelitis ceased to be reported in the United States. In areas of the world where paralytic poliomyelitis still occurs, the virus may be transmitted via food from time to time. However, the United States has recorded just two outbreaks of foodborne echovirus (close relatives of the polioviruses) illness, and none of poliomyelitis, since 1949.

Like the polioviruses, viruses transmitted to humans via foods in North America and Western Europe generally come from the human intestines (9). The hepatitis A virus is produced in the liver and drains into the intestinal lumen via the common bile duct, whereas the other foodborne enteric viruses are apparently synthesized in the intestinal mucosa and shed directly into the lumen. All are shed exclusively in feces, except for the Norwalk virus (and perhaps other “Norwalk-like” viruses), which is also shed in vomitus. These viruses are highly host adapted and infect no species other than humans. Like all enteric viruses, they are transmissible directly from person to person and via drinking water, as well as by foods. Only about 1% of reported cases of hepatitis A (the only “reportable” foodborne virus) have been associated with recognized foodborne or waterborne disease outbreaks (7), though more recent studies have placed the figure as high as 3%-8% (4); many others may be sporadic (individual) foodborne illnesses.

The hepatitis A virus (a picornavirus) causes acute hepatitis by infecting liver parenchyma cells that are eventually destroyed by the host's immune response to the infection. The incubation period averages 28-30 d (range of 15-50 d), during the last half of which virus is often shed at high levels in feces (2). The illness includes fever, malaise, and often jaundice; long-term debility or permanent sequelae may occur, but death is rare. Infection produces durable immunity. Of four other known types of human hepatitis viruses, types B, C, and D are transmitted only parenterally. Type E (a calicivirus, apparently not yet endemic in North America) is transmitted by a fecal-oral cycle through person-to-person contact and via water but has not yet been reported to be foodborne.

Gastroenteritis caused by the Norwalk virus (a calicivirus) and several “Norwalk-like” or small round structured viruses -- “SRSV” (some of which are astroviruses)--has a rapid onset (24-48 h) and a short duration (24-48 h) (2). Vomiting, diarrhea, or both are common. The virus is shed principally during illness, though an occasional foodborne disease outbreak has occurred when a food handler who had recovered from the illness continued to shed the virus for a few days more. Immunity, at least in response to infection with the Norwalk virus proper, is apparently ephemeral.

Rotavirus are more often causes of gastroenteritis spread person-to-person among children, but are occasionally foodborne (2). Incubation and duration are slightly longer than those of the Norwalk-like viruses, and immunity to the specific serotype that caused the illness is apparently durable.

Tick-borne encephalitis virus, which has occasionally been transmitted to humans via raw milk and milk products from tick-bitten dairy animals in Central Europe, does not occur in North America (11). Many viruses of animal disease occur in foods from time to time but do not represent a threat to human health (6).

VIRUSES AMONG FOODBORNE DISEASE AGENTS IN THE UNITED STATES

In the United States, Norwalk virus ranked #5, hepatitis A virus #6, and “other viruses” (principally rotavirus)
#10 among the top 10 causes of foodborne disease during 1983-1987 (1). It is difficult to take these rankings very seriously, given that the compilations are never issued timely, a great deal of foodborne disease is still of undetermined etiology, and viruses may or may not be less likely than bacteria to be implicated when they have in fact caused an outbreak (8).

Diagnosis of hepatitis A is well advanced; commercial kits are available to measure antibody of the immunoglobulin M class against the hepatitis A virus, which is an indicator of recent infection. Kits are also available for the diagnosis of rotavirus gastroenteritis. Until now, however, diagnosis of Norwalk virus gastroenteritis is far from routine, so that only a few laboratories have the capability; and the means to detect gastroenteritis caused by “Norwalk-like” viruses is rarer still. Detection of the viruses themselves in foods has been reported but is seldom undertaken because of the difficulty of the detection methods, problems with extracting the viruses from foods (10), and the frequent unavailability of suitable samples after an outbreak—especially given the average 4-week incubation period of hepatitis A. Hepatitis A is a reportable disease, but clearly is not always reported when it is diagnosed; whereas, the viral gastroenteritides are seldom confirmed by laboratory diagnosis and even then need not be reported. Because other countries’ systems for reporting foodborne diseases differ so greatly from that of the United States, it is impossible to compare incidences of foodborne viral disease internationally.

FOOD VEHICLES OF VIRUSES

Any food that is subject to contamination with human feces may harbor viruses (9). Because most feces travel via water-carriage toilets to sewage systems, it is important that sewage be properly treated before discharge, to limit dissemination of viruses. Discharge of raw sewage, due to storms or other disasters, can lead to disease outbreaks (13). Coastal communities, especially of the eastern and southern United States, have lagged in sewage treatment, with the result that many estuaries in which shellfish (bivalve molluscs) grow are contaminated with human enteric viruses (6). The shellfish feed by filtration, collecting viruses from their environmental waters and parting with these viruses only reluctantly. Sewage has also been known to contaminate other foods on an accidental, occasional basis.

Alternately, any food handled by an infected human may become contaminated (9). As was mentioned above, an apparently healthy person may shed hepatitis A virus and contaminate food during the last 10-14 d of the incubation period. Given that the foodborne viruses are shed exclusively in feces (with the possible exception of Norwalk virus in vomitus), contamination of food in handling indicates a failure of the source to wash his or her hands properly after defecation. Implicated food handlers have usually had contact with food during final preparation and serving. However, some fruit and vegetable vehicles may have been contaminated in the field before or during harvesting.

INACTIVATION OF VIRUSES IN FOODS

Viruses cannot, under any circumstances, multiply in foods. They may, however, be inactivated before the food is eaten and thus not cause infection (9). Heat is the most generally applicable means of inactivating viruses inside a food—the heat resistance of these viruses is generally comparable to that of the more heat-resistant foodborne vegetative bacterial pathogens. Foodborne viruses are approximately as resistant to drying as, and more acid stable than, the Enterobacteriaceae. Viruses on surfaces or in water can be inactivated by strong oxidizing agents (e.g., chlorine) or by ultraviolet light. Ionizing radiation will inactivate viruses inside of food, but the viruses offer very small targets, so large doses of irradiation are required to cause significant decreases in titer. Although the enteric viruses are generally more durable than viruses transmitted by other routes, recorded outbreaks have seldom depended on the ability of the virus to withstand cooking or other rigorous treatment.

DETECTION AND MONITORING

The infectivity of foodborne viruses was long expected to provide the basis for their detection: cultured primate cells inoculated with food extracts would become demonstrably infected, and this would show that the food had been contaminated with virus (10). However, the hepatitis A virus, in its principal “wild-type” form, does not infect cell cultures or—if it does—does not cause cytopathic effects (changes due to infection, visible with a microscope). The rotaviruses have presented similar problems; and the Norwalk virus (as well as the other SRSV) seemingly do not replicate at all in cultured cells. This essentially negates infectivity as a basis for detecting foodborne viruses.

The foodborne viruses comprise RNA (a single, + strand for hepatitis A virus and the Norwalk-like agents but a segmented, double-stranded form for the rotaviruses) coated with protein. Methods based on the interaction of antibody with the viral coat protein (radioimmunoassay, enzyme immunoassay, etc.) have been much used in diagnostic tests, but often are based on reaction of the host’s antibody with a laboratory reagent viral antigen, rather than on detection of the viral protein as antigen. In contrast to infectivity tests, in which the virus amplifies itself, serologic tests require other means of signal amplification and have generally not offered adequate potential sensitivity to be adapted to detection of viruses in foods. Serologic procedures have been combined with electron microscopy or with nucleic acid-detection techniques in some instances, either to allow detection of previously unknown viruses or to enhance the sensitivity and specificity of the detection method.

Increasingly sensitive detection methods, largely based on “molecular” techniques, are becoming available for these viruses (12). A complementary strand of nucleic acid, specific for a portion of the viral genome, reacts in demonstrable fashion with the viral RNA. The degree of signal amplification depends to some extent on the stringency of the reaction conditions but generally quite high sensitivities.
are being reported. In that a given test will detect only a single type of virus, these are most likely to be applied when an outbreak has occurred and diagnostic tests have already identified the causative agent. They are not generally applicable to monitoring foods on a routine basis because they are extremely specific, relatively laborious, and incapable of distinguishing an infectious virus from one that has been inactivated.

The ideal indicator, for use in routine monitoring of foods for viral contamination, is still being sought. Fecal coliform bacteria that have formed the basis of standards for shellfish are poorly correlated with the presence of viruses (3). Bacterial viruses, such as “F-RNA coliphages” and bacteriophages of Bacteroides fragilis, are under consideration. These offer more rapid results, and lower cost per test, than most tests directed to human enteric viruses. However, it remains to be seen whether any food other than shellfish will be perceived to present a high enough risk to merit routine monitoring even for a bacteriophage indicator. It seems certain that no before-the-fact test will ensure against virus transmission via a food contaminated during final preparation or serving.

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