VIRUSES AND PRIONS TRANSMITTED VIA FOOD AND WATER

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Introduction
Viruses transmitted via food and water are, with one exception, human enteric viruses. Only a few primate species other than humans are infected by these viruses. Enteric viruses infect perorally and are shed in feces.
Classification: see Table 1
Replication occurs only in susceptible cells; no DNA is involved in the replicative cycle. Prions are a newly recognized class of infectious agents that cause fatal neurological illnesses.

Hepatitis A virus
CDC (U.S., ’93–’97): 23 outbreaks, 729 cases, 0 deaths (Table 2); est. ~11,000 cases/yr
CAST: 4,800–35,000 cases/year, ≤14 deaths, $5030/case; no FoodNet coverage
Picornavirus: ca. 28 nm diameter, single (+) strand RNA; coat protein comprises 60 copies of each of four structural polypeptides; virus is relatively resistant to heat and to drying
History — Viral hepatitis recognized ca. time of World War II; fecal-oral transmission of “infectious hepatitis” (now hepatitis A) recognized much later; 5–6 hepatitis viruses now known, but only hepatitis A is known to be transmitted via food and water in North America
The disease
Virus in feces of infected person
Entry via intestines
Liver colonized; infected cells destroyed by host’s immune response; incubation 15–50 days (average 28–30); virus shed in feces 1–2 weeks before onset
Illness: fever, malaise, anorexia, nausea, abdominal discomfort—jaundice (?); usually complete recovery after a few weeks, with permanent immunity
Transmission/Control
Routes — person-to-person, or via water (drinking, irrigation?), “undercooked” shellfish, or food handled by an infected person
Prevention — sanitation, cooking, vaccination (U.S., 1995)
Food as a vehicle

Norwalk-like gastroenteritis viruses “Noroviruses”
CDC (U.S., ’93–’97): 9 outbreaks, 1233 illnesses, 0 deaths; est. 9.2 million foodborne/yr
CAST: 181,000 cases/year, 0 deaths, $890/case
History and naming
Norwalk, Ohio, gastroenteritis outbreak, 1972
Small round structured viruses (SRSV), “Norwalk-like” Calicivirus group — small (~30 nm), single-stranded RNA, protein coat has
“dimples”
The disease — vomiting and diarrhea; less common than rotavirus in infants
  Virus from ill or convalescent person via feces or vomitus
Colonization of intestines — incubation: 1–2 days
Severe diarrhea & vomiting — 12–60 hr (usually 24–48), virus shedding up to 7
days
Antibody is not protective

Transmission/Control
  Routes — person-to-person, or via water, “undercooked” shellfish, or food
  handled by an infected person
  Prevention — sanitation, cooking

Diagnosis
  Virus in feces: ELISA for antigen; RT-PCR for viral nucleic acid; serogroups
  Antibody production

Other gastroenteritis viruses
  Astroviruses — occasionally foodborne, some replicate in cell culture
  Rotaviruses — more often infant diarrhea than foodborne disease
  Adenoviruses — serotypes 40 & 41, not known to be foodborne
  Coronavirus — questionable cause of human diarrhea, foodborne once?

Other viruses and food
  Human enteroviruses (polioviruses, coxsackieviruses, echoviruses)
  Hepatitis E virus — water, food?
  Tick-borne encephalitis virus — milk & milk products, Slovakia
  Industry alarms
  “Non-problems” — hepatitis B, C, & D; herpes, HIV, hantavirus

Detection & monitoring: cytopathic effects in cell culture, plaques
  Diagnosis
  Detection of viruses in food
    Sample processing — liquefaction, clarification, concentration
    Test methods — probes, PCR, antigen capture
  Indicators — bacteria, viruses, phages

Prevention
  Food vehicles
    Shellfish (bivalve mollusks)
    Other food vehicles
  Sanitation (handwashing)
  Depuration of shellfish — not very successful
  Cooking, inactivation
Prions

Transmissible spongiform encephalopathies (TSEs)

Accumulation of abnormal prions in brain leads to spongiform degeneration
All are fatal
Some are “contagious”

Prions are
Low MW peptides found in CNS & some other organs
Normal folding (“PrP°C”) depends on amino acid sequence.
Abnormal (various causes, “PrPSc”) produces a protease-resistant molecule

“Old” TSEs
Scrapie in sheep
Creutzfeldt-Jakob disease (CJD), sporadic, etc., in humans
Transmissible mink encephalopathy
Chronic wasting disease (deer, elk)

“New” TSEs
Bovine spongiform encephalopathy (BSE) — “mad cow disease”
Feline spongiform encephalopathy
New variant CJD (vCJD) in humans

BSE in cattle, UK
April 1985 to December 2004, 184,131 confirmed cases of BSE (3–5-yr incubation)
Control by not feeding rendered bovine meat-and-bone meal (MBM) to cattle — slow enforcement
Slaughter of affected herds
Enormous research effort
No BSE prions found in red meat (voluntary muscle) or milk
Vertical transmission (cow-to-calf) “unlikely”
Carcass disposal precautions

BSE in cattle elsewhere
Some cattle, much beef, and a lot of MBM exported from UK to other countries
Now ca. 24 countries have BSE (few thousand cases), all in Europe except Japan, Israel (occupied West Bank territories), Canada, and US, so far

Inter-species transmission
Ca. 1994, TSE in cats (UK), including zoo species
In 1995, something resembling CJD began occurring in young people in the UK — “new variant CJD” or “vCJD” (>10-yr incubation?)
vCJD differs in more than age distribution of victims
Impact of vCJD

-~153 people in UK, ~23 in the rest of the world affected by 12/05 (almost all deceased)
- Even in UK, <CJD rate (28 vs 49 in peak year, 2000)
- Far less than deaths from other foodborne diseases
- HUGE public reaction
- Specified bovine offals banned from the food chain, most BSE countries
- Cattle >30 months old not eaten in UK, carcasses incinerated
- Slaughter cattle >30 (24 in some countries) months old tested in other BSE countries
- Genetic susceptibility — all vCJD patients tested have been homozygous for methionine at codon 129 of their prion gene (40% of population)
- Restrictions on blood donation and use

Sporadic CJD and vCJD in the UK, 1990–2005

BSE in Canada, spring 2003
- One cow, no trace
- Exports to U.S. (& other countries) cut off

BSE in US (Washington), December 2003
- One cow, possible “downer”
- Huge interstate recall of meat
- Media orgy
- Embargoes on US beef
US control measures
- Ban on feeding mammalian meat and bone meal (MBM, product of rendering) to food-source ruminants
- Restrictions on blood donation
- Scrutiny of biologicals
- Slaughter of downer cattle prohibited
- Risk materials, from animals >30 months old, prohibited from human food supply
- Other prohibitions pending
- More testing of downers, dead-on-farms, and suspects at slaughter
- Major over-reaction to minimal threat to human health

Drama in North America — chronic wasting disease
- Deer & elk, Colorado, Wyoming
- Other states (Wisconsin), Canadian provinces, farmed and wild animals
- Environmental transmission (feces?)
- Transmissible to humans??
- Processing carcasses — food safety?
- Now upstaged by BSE

Summary
- Human enteric viruses, fecal contamination
- Cooking or other means of inactivation (depuration)
- Detection vs. indicator systems for monitoring
- Prion diseases are here in North America.
- Threat to human health is minimal.
- Measures being imposed may well lessen overall food safety.

Bibliography


http://www.fda.gov/cber/bse/bse.htm (has many links)

Table 1. Major groups of human enteric viruses.

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<th>Size (nm)</th>
<th>NA strands</th>
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<td></td>
<td></td>
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<td>picorna-</td>
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<td>70–85</td>
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<td>reo-</td>
<td>adeno-</td>
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Table 2. Causes of foodborne outbreaks, U.S., ’93–’97

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<th>Rank</th>
<th>Causative agent</th>
<th>Cases</th>
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<td><em>Salmonella</em> spp.</td>
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<td><em>Escherichia coli</em></td>
<td>3,260</td>
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<td>3</td>
<td><em>Clostridium perfringens</em></td>
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<td>5</td>
<td>Other viral</td>
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<td>7</td>
<td>Norwalk-like viruses</td>
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<tr>
<td>9</td>
<td>Hepatitis A virus</td>
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