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Risk profile

Virus in food and drinking water in Sweden

– Norovirus and Hepatitis A virus

by Flemming Lund and Roland Lindqvist

LIVSMEDELS VERKET
NATIONAL FOOD ADMINISTRATION, Sweden

Sammanfattning

Syftet med riskprofilen är att identifiera virus som kan spridas via livsmedel (inklusive dricksvatten) och att sammanfatta kunskapsläget ur ett svenskt perspektiv. Utifrån rapporten ges rekommendationer för hur Livsmedelsverket kan minska antalet fall (”matförgiftningar”) av sjukdom orsakade av livsmedel och virus.

De viktigaste virusen som kan spridas via livsmedel är norovirus och Hepatit A (HAV). Norovirus är den mikroorganism som orsakar flest fall av matförgiftningar i Sverige, uppskattningsvis mellan 135 000 till 220 000 fall per år. Endast 122 fall av HAV rapporterades under 2003 i Sverige, men HAV-smittad mat utgör ändå en potentiell fara som kan leda till stora utbrott med förhållandevis allvarliga konsekvenser. Symptomen orsakade av norovirus går normalt över på några dagar medan sjukdom orsakad av HAV ofta varar flera veckor och i sällsynta fall kan leda till döden.

Smittvägarna för norovirus och HAV och deras egenskaper är likartade. Båda sprids från människor via en fekal-oral väg, antingen via livsmedel (inklusive vatten) eller via person till person smitta genom kroppskontakt eller droppsmitta. Norovirus och HAV är båda mycket smittsamma. Infektionsdosen kan vara så låg som 10 – 100 viruspartiklar. Avföring från smittade personer innehåller 1 miljon till 100 miljarder viruspartiklar per gram medan en uppkastning innehåller omkring 10 miljoner viruspartiklar. Vidare utsöndrar infekterade personer ofta virus redan under inkubationsperioden och fortsätter en viss tid efter symptomen har försvunnit. Mat som produceras av infekterade personer kan därför lätt blir kontaminerad med virus. Särskild risk utgör mat som kräver mycket manuell hantering och som inte upphettas före konsumtion som exempelvis smörgåstårta, bakelser, och buffémat. Kontamination av mat genom avloppsvatten är en annan spridningsväg. Mat som kontamineras på detta sätt är exempelvis dricksvatten, frukt (t ex frusna hallon) och grönsaker (t ex ruccolasallad) samt ostron.

Norovirus och and HAV kan inte tillväxa utanför den mänskliga kroppen men överlever förhållandevis länge i miljön utanför kroppen. Frysning är ett utmärkt sätt att bevara virus på. Flera PCR-metoder har tagits fram för att påvisa norovirus och HAV i livsmedel men det finns ingen validerad standardmetod. Organismer som används som indikatorer för förekomst av virus såsom *Escherichia coli* bakterier och bakteriofager har alla sina begränsningar.

För att reducera antalet matförgiftningar orsakade av virus som spridits via smittade personer och kontaminerad mat föreslås informationsinsatser riktade till dels konsumenter och dels till personer som arbetar med livsmedel.

För att kunna fylla viktiga kunskapsluckor förslås stöd till framtagandet av standardmetoder för att påvisa norovirus (viktigast) samt HAV, och om det bedöms nödvändigt indikatororganismer för virus i mat. Standardiserade metoder är en förutsättning för tillförlitliga resultat vid utbrottsundersökningar, övervakning och livsmedelskontroll.

Contents

Abstract

The aims of this report are to identify the most important foodborne (includes drinking water) viruses and to present existing knowledge of these viruses from a Swedish perspective. Based on the report, recommendations for further actions to reduce the number of viral foodborne illnesses are given.

In Sweden the most important viruses in food are norovirus and Hepatitis A virus (HAV). Norovirus is the most common single foodborne agent in Sweden and causes 135 000–220 000 estimated cases of foodborne illnesses annually. In 2003, a total of 122 cases of HAV were reported in Sweden. However, there exists a potential hazard of HAV contaminated food in Sweden, which may lead to large outbreaks with severe health consequences. Norovirus normally causes illness lasting for few days whereas HAV causes illness for several weeks and can even cause death.

In general, the transmission routes for norovirus and HAV are the same and they have similar properties. Both viruses are transmitted by the human faecal-oral route either via food or person to person spread through body contact or release of aerosols.

Norovirus and HAV are both highly contagious. The infected dose can be as low as 10–100 virus particles. Infected stools contain 10^6–10^{11} virus particles per gram and infected vomit contains approximately 10^7 particles per vomiting incident. Infected persons, e.g. food handlers, may shed virus particles during the incubation period and some time after symptoms have disappeared. Foods produced by infected food handlers may therefore easily be contaminated. Examples include foods that require much handling and which are not heated before consumption such as layer cake, cakes, pastry and buffets. Another way to contaminate food is by human sewage water. Foods at risk for contamination by sewage includes drinking water, fruits (e.g. frozen raspberries) oysters and vegetables (e.g. rocket salad).

Norovirus and HAV do not grow in food or outside the human body, but are quite stable in the environment and e.g. freezing is an excellent way to preserve the viruses.

Several PCR methods have been reported to detect norovirus and HAV in food but no standard method exists. Virus indicator organisms such as *Escherichia coli* and male specific bacteriophages have limitations.

To reduce virus transmission from infected persons and contaminated food several recommendations to direct relevant information to consumers and food handlers are suggested.

Recommendations are also given to fill important knowledge gaps by supporting efforts to develop standardised methods for detection of norovirus (primarily), HAV (secondly), and, if necessary, indicator organisms in food. Standard methods are crucial for reliable results from outbreak investigations, surveillance, and control.

Introduction

Viruses are infectious micro-organisms, much smaller than bacteria, characterised by their inability to reproduce outside of a living host cell. Viruses cause a wide range of diseases in plants, animals and humans by taking over the host cells normal functions to change the behaviour of the host in a manner determined by the virus. Each group of virus is host specific with a typical range and preference of cells they can infect. Transmission of viruses to humans can occur in many different ways. For example, by droplets generated when an infected person coughs, by contamination with stool samples from a person infected with an intestinal virus, by sexual intercourse, by contact with blood from persons infected with blood-borne viruses, by contact with infected animals with zoonotic viruses, by vectors, such as mosquitoes or ticks, for arthropod-borne viruses or by food and water. Viruses that infect the cells lining the intestinal tract and disperse by shed-ding into the stool or through vomit are of greatest concern in terms of foodborne transmission.

Numerous viruses can be found in the human gut but only a few are commonly recognised as important foodborne pathogens (Table 1). According to the type of illness they produce foodborne viruses can be classified into three main groups, of which the two first groups appear to be most common: 1) Viruses that cause gastro-enteritis. 2) Enterically transmitted hepatitis viruses and, 3) viruses that replicate in the human intestine but cause illness after they migrate to other organs such as the central nervous system (e.g. poliovirus). So far most enteric viruses appears to be quite host specific and humans are the main reservoir. Host range variants do exist but zoonotic transmission has not been proven which implies that this risk may be low. However, given the similarity between some animal and human strains and the genetic flexibility of some viruses, the possibility of zoonotic transmission should not be completely ruled out.

Table 1. Likelihood of food- or water-borne transmission of enterically transmitable viruses according to the type of illness associated with the infection (Koopmans and Duizer 2004).

	Illness		
Likelihood of transmission	Gastroenteritis	Hepatitis	Other
Common	Noroviruses	Hepatitis A virus	
Occasional	Enteric adenoviruses (types 40/41) Rotavirus (groups A-C) Sapovirus Astrovirus Coronavirus Aichivirus	Hepatitis E virus	Enterovirus*

*Enteroviruses (e.g. polioviruses) are associated with a range of symptoms, including neurological symptoms.

Since humans are the main source of foodborne viruses, transmission occurs by the human faecal-oral route either via the ingestion of contaminated food and water or via person-to-person through body contact or release of aerosols. Food can be contaminated by infected food handlers or by contact with sewage sludge or polluted water.

There are several similarities between foodborne viruses in terms of their properties and, consequently, risk management options may be similar for many of these hazards. In general, only a few particles of foodborne viruses are needed to produce illness whereas high numbers of viral particles are shed in the stools or vomit of infected persons. These viruses are generally quite stable and resistant outside the host but cannot multiply in food or water. There is also a general lack of methods for detection of foodborne viruses in food and water.

Although several groups of viruses have been implicated in foodborne outbreaks (Table 1) this risk profile focus on norovirus and Hepatitis A viruses (HAV). Recent studies have shown that norovirus and HAV are the most common cause of illness due to foodborne viral transmission. Furthermore these groups of viruses represent in Sweden the extremes in terms of occurrence and, to some extent, health impact. Noroviruses are common and usually cause only a mild illness whereas foodborne HAV viruses are rare but in some cases can cause severe illness and even death.

Objectives

The objectives of this report are:

- To identify and describe the most important foodborne viruses from a Swedish perspective
- To summarise existing knowledge and data of importance for understanding the health impacts and the most important vehicles of foodborne viral infection
- To identify important knowledge gaps
- To propose further actions for the National Food Administration to reduce and to minimise foodborne viral infections.

In accordance with Swedish legislation viral infections acquired via ingestion of drinking water is also considered as foodborne transmission.

Much of the information in this report was extracted from the following review papers: Greening et al. 2003, Seymour and Appleton 2001, Koopmans and Duiser 2002, Kruse et al. 2002, Hutson et al. 2004, Fiore 2004, SMI 2004, CDC 2001. General statements in the report that are not followed by a literature quotation are taken from one or more of these papers and further information can be found in them. The omission of numerous citations of these papers was done to increase the readability of the report.

Norovirus

Taxonomy

In October 1968 an outbreak of gastroenteritis occurred in an elementary school in Norwalk, Ohio in USA. During a 2-day period, 50 % of the students and teachers (116/232) developed a gastrointestinal illness. The incubation period was approximately 48 hours, and the illness, which lasted approximately 24 hours, was described as "winter vomiting disease". Indeed the Norwalk outbreak caused vomiting and nausea in most patients although some patients developed diarrhoea.

Laboratory studies did not reveal an etiologic agent. However, a rectal swab specimen, prepared as a 2 % bacteria free filtrate, could cause gastroenteritis when the filtrate was administered orally to volunteers. Virus was suspected as the etiologic agent. Attempts to detect the virus associated with gastroenteritis by novel organ culture techniques failed and even today it is not possible to cultivate this virus outside the human body. In 1972 the virus was detected by Albert Z. Kapikian and named the Norwalk virus (Kapikian 2000).

Kapikian discovered a 27 nm virus-like particle by use of immune electron microscopy in an infectious stool from the Norwalk outbreak and showed by experiments that this particle was the etiological agent of Norwalk gastroenteritis.

The Norwalk virus was initially described as a picornavirus or parvovirus based on its morphological appearance using electron microscopy. Later, in 1981, this virus was reported as more consistent with the family *Caliciviridae*. A breakthrough came in 1990 when RNA sequence relatedness between Norwalk virus and other calicivirus was demonstrated and led to the recognition of Norwalk virus and other related "small round structured viruses" (SRSVs) as members of the family *Caliciviridae*. The name SRSVs was based on their morphological appearance as seen by electron microscopy.

In the sixth report of the Int. Committee on Taxonomy of Viruses (ICTV) (Cubitt et al. 1995) all viruses in *Caliciviridae* were grouped into one genus, *Calicivirus*. Later ICTV (Green et al. 2000) divided *Caliciviridae* into 4 genera. However the two genera associated with human gastroenteritis were assigned by provisional names: "Norwalk-like viruses" and "Sapporo-like viruses". Recently these names were changed to norovirus and sapovirus by ICTV (Mayo 2002) and should be used in the future. In Table 2 former names for norovirus are listed.

Table 2. Former names for norovirus and some examples of genogroup I and II strains (Gallimore et al. 2004).

Family	Genus / previously used names	Genogroups/strains
Caliciviridae	*Norovirus*/	GI-1 / Norwalk virus
	"Norwalk-like viruses"	GI-2 /Southampton
	Caliciviruses	GI-3/ Desert Shield virus
	Small round structured viruses (= SRSVs)	GI-4/Valetta virus
		GII-1/Hawaii virus
		GII-2/Melksham virus
		GII-3/Mexico virus
		GII-4/Grimsby virus

Division of norovirus and sapovirus into additional species has not been described yet. But norovirus has been divided into genogroups. The prototype strain of norovirus is the original Norwalk virus, a genogroup I strain. The genomic diversity of human norovirus includes two genogroups (I and II) and a number of genotypes, which have yet to be formally agreed on (Gallimore et al. 2004). Some strains belonging to the proposed genotypes in the two genogroups are listed in Table 2.

Differences between norovirus and sapovirus can be detected by electron microscopy or multiplex PCR technique. Furthermore there are suggestions that the epidemiology of these viruses differs in that sapovirus cause infections of young children whereas norovirus cause infections of people of all age groups.

Clinical presentation

The incubation period for norovirus-associated gastroenteritis in humans is usually between 12 and 48 hours. Norovirus infection usually presents by one or more of the following symptoms: nausea, diarrhoea, vomiting, and stomach pain. Additio-nal symptoms are present in some patients: low-grade fever, chills, headache, and myalgia.

The most common symptoms are acute-onset vomiting, watery non-bloody diarrhoea with abdominal cramps and nausea. Vomiting is more common in children whereas diarrhoea is more common in adults (in contrast to that norovirus illness popularly is called "winter vomiting disease") (Carrique-Mas et al. 2003).

Symptoms usually last 24 to 60 hours and recovery is normally complete. Dehydration is the most common complication and sometimes hospitalisation with parenteral fluid therapy is necessary.

Virus particles are shed via stools and vomits, starting during the incubation period and lasting up to 3 weeks. It is important to know that asymptomatic infections are not uncommon. In an outbreak in the Netherlands 75 % of people with gastroenteritis were found to shed norovirus compared to 20 % of healthy contacts (Vinjé et al. 1997).

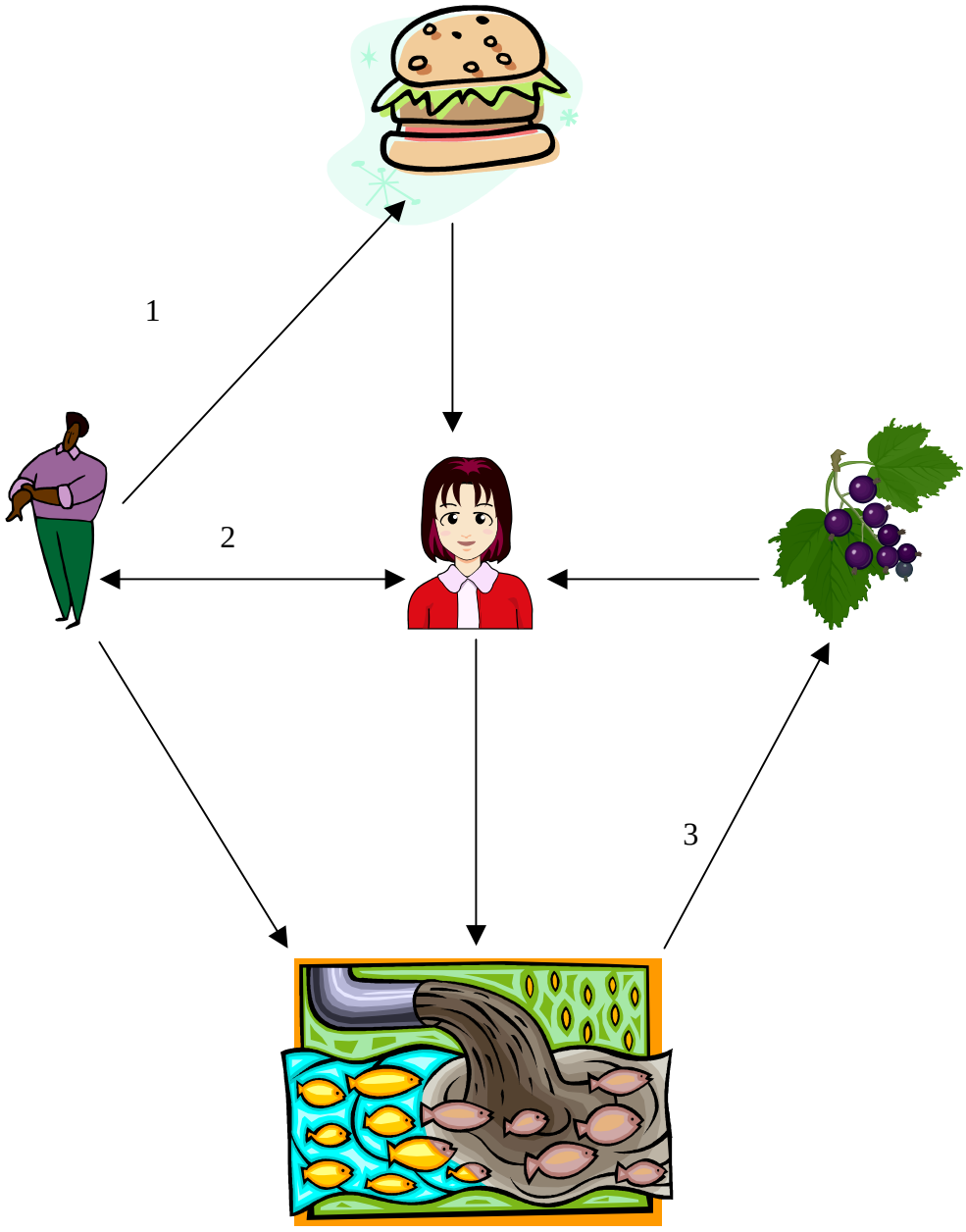

Figure 1. Routes of transmission of norovirus and HAV. 1. Food contaminated by food handler. 2. Person to person spread. 3) Food contaminated via sewage (e.g. berries, drinking water and shellfish).

Norovirus transmission

Noroviruses are transmitted primarily through the faecal-oral route, either by consumption of faecally contaminated food or water, or by direct person to person spread (Figure 1). Several outbreaks start by foodborne transmission e.g. in a restaurant followed by secondary person to person transmission in household contacts. In other words person to person spread is common and often occurring as a secondary transmission route after primary infection via food or drinking water.

Noroviruses are highly contagious. The infection dose can be as low as 10–100 virus particles. Infected stool samples contain 10^6–10^{11} virus particles per gram and infected vomiting samples contain approximately 10^7 virus particles per vomiting incident. Obviously toilets are places where infected persons can spread and infect other persons.

Projectile vomiting is a common feature following a virus infection. Vomit contaminated with norovirus can be aerosolised and this is recognized as an important vehicle for transmission. This was illustrated by an outbreak of gastroenteritis following a meal in a large hotel during which one of the guests vomited. Later an inverse relationship was found between the attack rate per table and the distance from the person who became sick. The aerosolization of vomit presumably resulted in droplets contaminating surfaces of food, dish or cutlery, which then were ingested (Marks et al. 2000). No evidence suggests that infection occurs through the respiratory system.

Immunity to norovirus

More than 25 years ago, the initial norovirus challenge studies conducted in volunteers found that one group of individuals were repeatedly susceptible to norovirus, whereas a second group was repeatedly resistant to infection. Furthermore, based on estimates of the number of persons exposed to viruses in reported novrovirus outbreaks the attack rate is seldom 100 %. Attack rate range is normally from 30 to 80 % in food borne outbreaks. Thus, some persons appear to be resistant to norovirus.

Recently a mechanism that explains susceptibility or resistance to norovirus infection has been identified. Norovirus attach to potential host cells in the gut, only if the host cell expresses specific, genetically determined carbohydrates. This discovery was a breakthrough in understanding norovirus host-susceptibility factors .

Carbohydrate binding is a common mechanism by which many viruses attach to their host cells. Noroviruses bind to a group of carbohydrates called the H, Lewis and ABO histo-blood group antigens. Unique varieties of these carbohydrates are determined by the presence or absence of specific glycotransferase enzymes as a result of a person's genetics.

In volunteer challenge studies it has been shown that carbohydrate binding is essential for the original Norwalk virus strain. Individuals who are non-secretors

(Se$^-$), i.e. did not make a special H type 1, or Lewis type b antigen, did not become infected after challenge with this Norwalk strain. It should be noted that other strains of norovirus display different H, Lewis and ABO carbohydrate-binding profiles; i.e. individuals resistant to the Norwalk virus strain may be susceptible to other norovirus strains.

These data also help to explain why the absence of antibodies to a norovirus does not necessarily predict susceptibility to infection with that virus strain. Persons without norovirus specific antibodies could be completely protected to norovirus infection (i.e. did not become infected after challenge with norovirus) because of the lack of expression of carbohydrates necessary for virus attachment to host cells. Conversely, the presence of norovirus specific antibodies could indicate previous infection and therefore susceptibility at the virus receptor binding level. A single norovirus infection does not appear to provide long-term protection. Without re-exposure to a particular norovirus strain, protection wanes after six months. It has been reported that susceptible individuals have been re-infected two years after prior exposure.

In epidemiological studies with norovirus it is therefore very important to include the possibility of immunity in specific consumers when a suspected food is investigated. Furthermore, popular conclusions such as excluding a suspected food item when not all consumers developed illness – may indeed be wrong.

Epidemiology in Sweden

In the last 10 years the number of reported norovirus infected persons in Sweden and other developed countries has increased steeply. Factors influencing this trend are likely the development of better detection techniques such as the reverse transcriptase polymerase chain reaction (RT-PCR) and better surveillance systems. For example the Center for Diseases Control and Prevention (CDC) in the USA found that in 1993–1997, only 9 of 2 751 foodborne outbreaks were caused by norovirus. Now CDC estimates that up to 50 % of all food borne outbreaks of gastroenteritis may be attributed to noroviruses. Among 232 outbreaks of norovirus reported to CDC from July 1997 to June 2000, 57 % were food borne, 16 % were due to person to person spread, and 3 % were water borne; in 23 % of the outbreaks, the transmission was unknown (Widdowson 2004).

Oysters (and other bivalve molluscs) contaminated with norovirus often cause outbreaks of gastroenteritis. In Sweden four oysters outbreaks with 58 cases have been reported (Table 4). Oysters feed by filtering small particles such as algae from the surrounding water but occasionally the water in the growing areas is contaminated with human sewage and norovirus. Growing oysters in restricted areas far from sewage contamination seems to be a way to limit the problems. However, sewage effluents from recreational boats have been identified as the likely source of norovirus contamination in commercially farmed New Zealand oysters (Simmons et al. 2001).

Hedlund et al. (2000) investigated the epidemiology of norovirus infection in Sweden, 1994–1998, using electron microscopy. Of 5 800 faecal samples from persons with gastro-enteritis 3 700 were associated with outbreaks. A total of 676 outbreaks were analysed and norovirus were detected in 407 (60 %) of the outbreaks. Contaminated food or water was suspected in 15 % of all 676 outbreaks, and norovirus had the highest detection rate (72 %) in foodborne outbreaks (Table 3).

Lindqvist et al. (2000) also found that norovirus was the most reported agent both in terms of incidents and cases of food borne disease incidents in Sweden, 1992 to 1997. In this period 39 (8 %) norovirus outbreaks including 2 961 (27 %) cases were reported (Table 3). In a one-year (1998–99) study of food borne illnesses in Uppsala in Sweden, Lindqvist et al. (2001) again found that norovirus was the most common aetiological agent with 20 detected incidents including 41 cases (Table 3).

In a comparison of the number of reported cases in Sweden with the estimated number of cases based on a population-based interview study (Norling 1995) underreporting in Sweden was estimated to a factor of 270 (Lindqvist et al. 2000). Extrapolation of the yearly incidence of foodborne illnesses found by Norling (1995), i.e. 500 000 cases, gives an estimated incidence of 57 cases per 1 000 persons in Sweden. Lindqvist et al. (2000) reported that 27 % of foodborne disease cases were due to norovirus infection. In 2003, 25 % of foodborne disease outbreaks in Sweden were caused by norovirus and this virus was the aetiological agent in 44 % of the reported foodborne disease cases (National Food Administration, unpublished data). Thus an estimated number of 15 to 24 cases per 1 000 persons, or 135 000–220 000, occurred annually due to foodborne norovirus infection based on data from 1992 to 1997 and 2003 (Table 3).

Table 3. Number of foodborne outbreaks or cases caused by norovirus compared with the total number in Sweden.

Year	Type of data	Number of norovirus	Total number	Reference
1992-97	Outbreaks with known agent	39	180	Lindqvist et al. 2000
1992-97	Cases with known agent	2 961	7 121	Lindqvist et al. 2000
1992-97	Estimated cases per year	135 000	500 000	Lindqvist et al. 2000
1994-98	Outbreaks of gastroenteritis	73	101	Hedlund et al. 2000
1998-99	Incidents* with known agent	20	55	Lindqvist et al. 2001
1998-99	Cases with known agent	41	181	Lindqvist et al. 2001
2003	Estimated cases per year	220 000	500 000	National Food Administration, unpublished data

*Incidents included both outbreaks and sporadic cases

Hjertqvist et al. (2004) summarised all reported outbreaks and cases of norovirus in food and drinking water in Sweden between 1999 and 2004. Although the surveillance system is not mandatory their summary indicate that there has been an increase in the number of norovirus outbreaks (Table 5). The detection technique was based on electron microscopy but, in addition, samples from all outbreaks were analysed by RT-PCR and characterised by sequencing. From outbreaks in 2002, sequencing results showed a wide diversity of norovirus strains. During 2002, genogroup II strains were the most prevalent (52 %). In 2003 all but two outbreaks were caused by genogroup II strains.

Infection of norovirus causes gastroenteritis popularly called ”winter vomiting disease” and Hjertqvist et al. (2004) also found that the total number of outbreaks had the highest prevalence in January to March. However, the food- and water-borne outbreaks were evenly distributed throughout the year.

In approximately 55 % of the outbreaks a suspected food vehicle was incriminated. In general, it was still difficult to detect norovirus in food items. However, in Sweden norovirus has been detected in water and raspberries, and in these cases the corresponding genotype was detected among the patients (Hjertqvist et al. 2004).

In a Swedish investigation of outbreaks between 1994–98 Hedlund et al. (2000) using epidemiology also found frozen raspberries and drinking water associated with norovirus outbreaks and in addition they found contaminated bakery products and oysters as vehicles of norovirus transmission.

In Table 4 several food items suspected to be associated with norovirus outbreaks in Sweden 2000–2004 are listed. The results indicate that drinking water is the single known food vehicle causing most of the reported cases. Possibly, this is a reflection of the extent of consumption of drinking water in comparison with most other foods. In addition many other food items include a heating process and may inactivate possible norovirus. Therefore, it is important to focus on water borne outbreaks and two outbreaks are described here in detail.

Table 4. Suspected food associated with norovirus outbreaks in Sweden 2000–2004 (Hjertqvist et al. 2004.)

Food	Outbreaks	Cases
Drinking water	7	920
Raspberries	13	686
Cake/pastries	6	376
Meat products	2	358
Vegetables	4	165
Several dishes	9	157
Buffet	3	111
Oysters	4	58
Several layer cake	3	48
Baguette, sandwich	2	38
Christmas table	6	>18
Pizza	1	7
Unknown	28	> 910

One outbreak affected approximately 500 people in a Swedish ski resort in 2002. Drinking un-boiled water originating from communal water systems was identified as a significant risk factor for gastroenteritis and the risk increased with increasing amount of water consumed. Stool samples from patients were tested positive for norovirus. However, the drinking water was tested negative for *E. coli* and other faecal indicator bacteria, the presence of which could have suggested a possible crack in a sewage pipe. Despite this, a crack in a sewage pipe was discovered 10 m from the well. This outbreak highlights a common problem when an outbreak investigation implicates the vehicle of infection but the microbiological analysis fail to detect the pathogen or indicator organism in the suspected vehicle. In other words, despite the absence of indicator bacteria for sewage contamination, the samples may still contain norovirus from sewage contamination (Carrique-Mas et al. 2003).

Similarly, another outbreak in Swedish drinking water occurred in 2001 and affected at least 200 persons. No pathogenic bacteria were detected in water or stool specimens, but norovirus was detected in stool specimens and water samples. All norovirus strains were identical (a genogroup II strain) and in this way strongly indicated that this strain was the principal causative agent of this outbreak (Nygård et al. 2003).

After drinking water, raspberries caused the most number of norovirus cases between 2000-2004 in Sweden (Table 4). In an outbreak in a school in Sweden approximately 140 persons had gastroenteritis after eating food from the school kitchen. It was found that raspberry sauce made from frozen raspberries imported from Poland was contaminated with norovirus. Normally the kitchen staff boiled the sauce but in this case they had served the sauce un-boiled (Larsson, 2002).

Table 5. Reported norovirus outbreaks in Sweden 1999-2004 (Hjertqvist et al. 2004).

Year	Outbreaks	Cases
1999	7	452
2000	12	331
2001	16	759
2002	28	> 1212
2003	18	881
2004 (until summer)	11	566
Total	**92**	**> 4201**

In addition to drinking water and raspberries a variety of food items were associated with norovirus outbreaks (Table 4) and most outbreaks had taken place after consumption of food in restaurants (Table 6). This indicates that the foods likely could have been contaminated by food handlers. (A food handler is here defined as any person who works in an area where food is being prepared, produced, served or packed). Foodhandlers must strive for a good (personal) hygiene to prevent transmission of norovirus. Furthermore food handlers with suspected gastroenteritis must be sent home immediately.

Table 6. Reported places for norovirus outbreaks in Sweden 2000–2004 (Hjertqvist et al. 2004).

Place	Outbreaks
Restaurant	28
At home	6
Several places	6
Day care centre/school	5
Hotel	5
At work	4
Home for old people	3
Other	18
Unknown	11

The validity of this recommendation is illustrated by an outbreak in a hotel in Sweden. The cook in the hotel restaurant started vomiting and had diarrhoea while he prepared breakfast and other meals for the guest. He left the hotel after working for 1.5 hour. The following day five additional employees working in the hotel kitchen fell ill with gastroenteritis while preparing lunch for the hotel guests. Following this sequence of events an outbreak of gastroenteritis occurred which affected 158 of 219 guests and employees at the hotel. In stool specimens norovirus was detected by electron microscopy. Genotyping and sequence analyses revealed that all samples had identical sequence and clustered in genogroup I (Johansson et al. 2002).

Detection methods

Norovirus can not be cultivated in the laboratory and until recently detection relied solely on the use of electron microscopy. This technique is fairly insensitive and requires a minimum of 10^6 virus particles per ml of sample. It has been used widely for detection of norovirus in faecal samples and has worked well in outbreak investigations but can not be used for food, water and environmental samples.

Sequencing of the norovirus genome has led to the development of PCR assays with greatly enhanced sensibility. However, there exists a great genomic diversity among the norovirus strains and one set of PCR primers will not detect all strains. Workers in the field have proposed a variety of primers and probe considerations but no international consensus has yet emerged.

Only few methods have been developed for detection of norovirus in foods. Norovirus has been detected in samples of raspberries, water and shellfish (oysters). But several factors such as extraction efficiency, controls, contamination during the PCR process and PCR conditions have to be taken into account to achieve reliable and sensitive results. A standard monitoring method has not been designed yet and commercial test kits are not available. However, development of methods goes on. Recently, a real time RT-PCR method for quantification of norovirus in inactivation experiments has been reported (Duizer et al. 2004).

The present Swedish analytical capability for detection of norovirus in human samples according to K.O. Hedlund (pers. comm.) consists of one laboratory using electron microscopy and nine laboratories using RT-PCR technique. In addition, one laboratory in Sweden has the capability to analyse norovirus in shellfish, water and drinks (Lopman et al., 2002). Some work has been done to detect norovirus based on antigen methods and three laboratories in Sweden use ELISA technique (K.O. Hedlund, pers. comm.). However, immune responses are predominantly type specific and these assays are narrow in their applicability.

In most countries the detection of viral contaminants rely on the concept of faecal indicator organism to assess microbiological hazards. However, the faecal coliforms have been shown to inadequately reflect the presence of viral contaminants. This has been illustrated in outbreak reports by failure of bacterial indicators to indicate norovirus in contaminated shellfish and water.

A better indicator could be bacteriophages. Male-specific RNA (FRNA) bacteriophages was shown to be a better indicator of virus contamination than *E. coli* in oysters. In the UK, 10 outbreaks of norovirus were negative for *E. coli* whereas all samples were positive for FRNA bacteriophage. However, in more "clean" shellfish from cleaner harvesting areas an investigation showed that 13 shellfish samples were positive for norovirus but only 6 were also contaminated by FRNA. In such situations, direct monitoring of norovirus may be the only option (Le Guyader, unpublished data).

Important norovirus properties

Table 7. Some important properties of norovirus.

Property
Survives* freezing.
Survives* 60 °C in 30 min.
Survives* in carpets, lockers, toilet seats.
Survives* exposure to acidity levels below pH 3.
Survives* in a solution up to 3 000 ppm hypoclorite in 10 min.
Inactivates by boiling at 100 °C.
Can not be cultivated or proliferated outside the human body.
Is found in stools from asymptomatic persons.
Infects human by the oral route (contaminated food, water or by person to person transmission) see Figure 1.

*norovirus is not completely inactivated and may infect humans.

Consumption of even small numbers of norovirus particles can result in disease. The dose capable of causing infection has been estimated as approximately 10–100 viral particles (Caul 1994) but consumption of only 1 virus particle in drinking water has also been reported to cause infection (Moe et al. 1999).

Infected individuals produce large numbers of viruses in faeces and vomit. The levels of viral particles shed in faeces are 10^6–10^{11}/g and approximately 10^7 potentially infectious doses can also be generated per vomiting incident. Other important norovirus properties are listed in Table 7.

HAV (Hepatitis A virus)

Taxonomy

HAV particles are 22–30 nm and are classified as a picornavirus. There exist only one HAV serotype and immunity after infection is life long. However, using PCR methods subtyping of HAV strains is possible and can be used to trace cases infected by a common source and thus to detect possible outbreaks among unlinked cases.

Clinical presentation

The median incubation period is 28 days (range, 15–50 days). HAV begins with symptoms such as fever, anorexia, nausea, vomiting, diarrhoea, myalgia, and malaise. Jaundice, dark-coloured urine, and/or light coloured stools might be present at onset or within a few days. Physical findings can include abdominal tenderness, hepatomegaly, or splenomegaly. For most persons HAV lasts for several weeks. The overall case-fatality rate is 0.3 %, but it is 1.8 % among persons aged > 50 years. Persons with underlying chronic liver disease have increased risk of death.

Except symptom treatment of e.g. dehydration, no specific treatment for HAV has been shown to be effective.

HAV transmission

Several features of HAV transmission are the same as for norovirus transmission. HAV is transmitted by the faecal-oral route by ingestion of contaminated food/ drinking water or by person to person spread and is also infectious at very low doses (Figure 1). Large numbers of HAV particles can be excreted in faeces from an infected person. Levels of the order of 10^6–10^{11} infective units per gram have been estimated during the incubation period. A specific HAV dose response model has been described (Haas et al. 1999).

However unlike norovirus, HAV transmission may also occur after exposure to HAV contaminated blood or blood products.

Immunity to HAV

In 1995 a HAV vaccine was licensed for persons aged more than 2 years. More than 90 % of adults and children are protected against HAV by 4 weeks after receipt of a single dose of vaccine. The efficacy of the vaccine is 94–100 %, and protection is likely to last for more than 20 years after vaccination.

Injection of immunoglobulin provides short-term (1–2 month) protection from HAV. Immunoglobulin is a sterile preparation of concentrated antibodies (immunoglobulins) made from pooled human plasma processed in a way that inactivates HAV. Immunoglobolins are often used in persons who require immediate protection, e.g. persons who plan to travel within 2–4 weeks to HAV risk areas or in families who have members with HAV.

In developing countries, HAV transmission often is unrecognised, because most residents acquire HAV infection during early childhood without any obvious or very mild symptoms. In these countries foodborne outbreaks are uncommon because of high levels of immunity in the resident population, but foodborne transmission to non-immune travellers might be an important source of travel-associated HAV.

Epidemiology in Sweden

In Sweden HAV infection is a communicable disease and it is mandatory to report cases of HAV to the country medical officer responsible for communicable diseases. Between 1997 and 2003, 27 to 189 domestic cases have been reported per year. In 1997, 189 domestic cases were reported of which 176 cases were associated with a HAV outbreak in Stockholm among injection drug users. Contaminated amfetamin was suspected as the origin of outbreak followed up by person to person transmission.

Some foodborne HAV outbreaks in Sweden have been reported. Around Christmas in 1955 several hundred persons became infected by HAV after consumption of HAV contaminated oysters. In 1976 a dish washer in a restaurant in Stockholm was infected by HAV. During his incubation period he had helped to produce a raw salad. Twenty-eight consumers of the salad became HAV infected and two of the consumers died (SMI 2004).

An increased incidence of HAV occurred in Sweden in 2001 and a case-control study was initiated. Sixteen cases and 31 controls were interviewed. Matched analysis showed that consumption of imported rocket (rucola) salad was associated with HAV infection. Sixty-seven % of the patients recalled having eaten rocket salad in two months before disease onset, compared with 32 % of the controls (Nygård et al. 2001).

In 2003, a total of 122 cases of HAV were reported in Sweden, i.e. 1.3 cases per 100 000 inhabitants. Sixty-two (51 %) persons were infected in Sweden. Of persons infected in foreign countries most persons were infected in Turkey (9),

Iraq (7) and Morocco (5). Most HAV cases were unrelated but two HAV outbreaks were reported. During a wedding HAV were spread to other guests from persons who had visited Turkey. At the other outbreak in a university in Stockholm several pupils were HAV infected during a party.

Detection methods

Unlike norovirus, HAV can be diagnosed by serologic analyses. Serologic testing is necessary to distinguish HAV from other forms of viral hepatitis. The serologic marker of acute HAV infection, IgM antibody to HAV (IgM anti-HAV), is detectable 5–10 days before onset of symptoms and usually decreases to undetectable concentrations within 6 months after recovery. The sensitivity and specificity of commercially available IgM anti-HAV test is more than 95 %.

The HAV can be cultured in the laboratory, but this is a long and unreliable procedure.

Due to the long incubation period, food items are not usually available for testing. However, some developed PCR methods to detect HAV in fruits, vegetables, shellfish and water have been reported (Seymour and Appleton 2001). As for norovirus, HAV has been detected in water and shellfish with acceptable counts of coliform bacteria.

HAV properties

In general HAV is transmitted the same way as norovirus and has the same properties as norovirus. Thus preventive actions described for norovius also include HAV with some important exceptions. HAV is also transmitted by blood. Therefore increased risk for HAV infection is associated to men who have sex with men and injection drug users.

Conclusions

- Norovirus is the most common single foodborne agent in Sweden causing an estimated 135 000–220 000 cases of foodborne illnesses annually

- HAV contamination of food or drinking water is a potential hazard in Sweden, which may lead to large outbreaks with severe health consequences

Following conclusions include both norovirus and HAV:

- Humans are the main source

- Transmission is by the faecal-oral route either via contaminated food/water or person to person spread through body contact or release of aerosols

- Food is contaminated either by infected food handlers or through contact with human sewage sludge or polluted water

- Food at risk for contamination by food handlers includes foods that requires much manual handling and which are not heated before consumption such as layer cake, cakes, pastry, and buffets

- Food at risk for contamination by sewage includes drinking water, fruits (e.g. frozen raspberries), oysters, and vegetables (e.g. rocket salad)

- Infected persons with or without symptoms may shed viruses also during the incubation period and some time after symptoms have disappeared

- A few virus particles are sufficient to cause disease and high numbers are shed in faeces or vomit

- Viruses do not grow in food, drinking water or in the environment

- Viruses are quite stable in the environment but data on survival is lacking partly due to a lack of methods, especially for norovirus, for cultivating the virus outside the human body

- There is a lack of standardized detection methods in food and drinking water

- The indicator organisms in use, *Escherichia coli* and male specific bacteriophages (F-RNA phages), have limitations

Recommendations

Information

To reduce virus transmission from infected persons and contaminated food NFA should:

- Update the information on foodborne viruses on its website
- Direct information to food handlers to improve their awareness of foodborne viruses as a potential hazard in water and raw materials
- Direct information to food importers, caterers and food handlers at institutional kitchens about the risk of foodborne viruses in frozen raspberries
- Direct information to food handlers to improve their awareness of the importance of the personal hygiene and health of their employees in terms of transmission of foodborne illness
- Direct information to consumers to improve their awareness of foodborne viruses and the importance of their personal hygiene in terms of transmission of foodborne illness
- Take actions so the problems of foodborne viruses are included as a subject in relevant courses and education programs at all levels in Sweden
- Develop detailed instructions and recommendations for food handlers with gastroenteritis

Knowledge gaps

To obtain validated and standardized methods for detection of viruses in food and drinking water to be used for outbreak investigations, for surveillance and control, and to generate data for the assessment of risk NFA should:

- In cooperation with other authorities in Sweden, work for the prompt development of standardized and validated methods for detection of norovirus (most importantly), HAV (secondly), and if necessary, indicator organisms in food and drinking water
- Decide, based on our responsibilities, where in Sweden this analytical competence is necessary and how the methods should be implemented
- Support efforts to improve our knowledge of the epidemiology of foodborne viruses.

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Appendix A 1

Food processes, virus inactivation factors, and resulting risk of the product if viruses are present before processing
(Koopmans and Duizer, 2004 with modifications and additions)

Process	**Example of food product**	**Virus inactivation (log_{10})**	**Risk of infection of consumer if viruses are present before processing[b]**	**Remarks**
Thermal treatments				
Boiling at 100 °C	Any liquid food (e.g. milk) or solid food boiled in water	HAV and PV>4 (Hollinger and Ticehurst, 1996)	Negligible	Likelihood of presence depending on food; kinetic data lacking. Inactivation in solid foods lower than in liquids, dependent on fat and protein content.
71,3 °C, 1 min	water	FeCV 3, CaCV 3 (Duizer et al., 2004)	Medium	
60 °C, 30 min	liquids or solid foods	HAV<2 (Hollinger and Ticehurst, 1996) or HAV>4 (Croci at al., 1999; Millard et al., 1987) PV<2 (Nissen et al., 1996) NoV: incomplete inactivation (Dolin et al., 1972)	Medium	
Pasteurisation of solid foods (70 °C, 2 min)	Paté or other cooked meats	HAV<2 (Millard et al., 1987) FeCV>3 (Doultree et al., 1999)	Medium	Inactivation dependent on fat and protein content
Pasteurisation of liquids and immediate packing (e.g. HTST 71,7 °C for 15 sec)	Milk, ice cream	HAV<2 (Bidawid et al., 2000a)	Medium	Inactivation dependent on fat and protein content
UHT and aseptic filling (>120 °C)	Long-life milk, other dairy products		Negligble	

Other physical/chemical/biological processes				
Drying (spray and freeze drying)	Dried milk, instant dried soups, dessert mixes, chocolate	HAV,FeCV<1 (Doultry et al., 1999; Mbirhi et al, 1991)	High	No information on commercial drying
Freezing	Ice-cream, frozen desserts mixes, chocolate	HAV, PV, FeCV<1 (Hollinger and Ticehurst, 1996)	High	
Fermentation	Cheese, yoghurt	No information		Microbial inactivation of viruses is found for sludge (Ward, 1982)
Acidification	Fruit juices, still fruit drinks	NoV: pH 2.7, 3h incomplete (Dolin et al., 1972) HAV: pH 1, 5h incomplete (Hollinger and Ticehurst, 1996)	Medium	No quantitative data on inactivation
Acid/base	Water	pH 2: NoV =0; pH<5 or pH>10: CaCV>5; pH<2 or pH>10: FeCV>5; pH 6: FeCV 2, CaCV 4 (Duizer et al., 2004)	Medium	
Homogenisation		Incomplete	High	Likelihood of presence depending on type of product
Depuration of oysters and mussels		NoV incomplete (Grohmann et al., 1981)	High	
High hydrostatic pressure (600 MPa, 1h)		PV<1 (Wilkinson et al., 2001)	High	Likelihood of presence depending on type of product
Virus inactivation in water				
70 % ethanol		8 min:FeCV, CaCV=1 30 min: FeCV, CaCV=3 (Duizer et al., 2004)		

Chlorination (0.5 mg free chlorine/l, 1 min)		HAV>3, HAV<2, HRV<2, PV>3 (Abad et al., 1994; Sobsey, 1989)	Variable	Risk is low for PV but medium for HRV and HAV
Sodium hypochlorite solution (10 min): 30 ppm free chlorine		FeCV, CaCV<1		
300 ppm free chlorine		FeCV=1, CACV=3		
3000 ppm free chlorine		FeCV, CaCV>5 (Duizer et al., 2004)		
UV radiation		20 mJ/cm^2: PV=3 or less (Sommer et al., 1989); HRV<3 (Sobsey, 1989); 21 mJ/cm^2: CaCV=3 22 mJ/cm^2:FeCV=3 (Duizer et al., 2004)		
Ozone treatment (0.2 mg/l, 10 min)		HAV>3, PV=2 or less, HRV<1 (Kim et al., 1999; Sobsey, 1989)	Variable	Risk is low for HAV but medium/high for PV and HRV
Cleaning of equipment and surfaces				
Rinsing with (lots of) water		HAV<2 (Bidawid et al., 2000b)	Medium/low	
Ethanol (70 %, 10 min)		HAV<2, HRV<3 (Abad et al., 1997)	Medium	
Chlorhexidine (0.05 %, 10 min)		HAV<1, HRV<1 (Abad et al., 1997)	High	
Sodium hypochlorite (0.125 %, 10 min)		HAV<3, HRV<3 (Abad et al., 1997; Kawana et al., 1997)	Low	
Sodium chlorite (30 %, 10 min)		HAV>3, HRV>5 (Abad et al., 1997)	Negligible	

Catering				
Washing, rinsing (where water > 1% of food) and the food is eaten without additional cooking	Washed salads, Fruits (strawberries)	No substantial removal or inactivation	High	Any removal of viruses will be by mechanical action only; very difficult to remove any microorganisms from foods by washing alone (Mariam and Cliver, 2000)
Catering				
Freezing of drinking water to prepare ice	Ice for drinks or for cold foods	No inactivation	High	Freezing is an excellent way to preserve viruses; therefore best to assume there will be no inactivation after one freeze/thaw cycle
Chilling of drinking water or use of water from tap without any treatment		No inactivation	High	Chilling will slow down the inactivation rate of viruses

[a]Viruses for which data were used to assemble this table are the (common) foodborne hepatitis A virus (HAV), Norovisuses (NoV) [and the animal model feline calicivirus (FeCV) and canine calicivirus (CaCV)], human rotavirus (HRV), rhesus rotavirus (RV), and poliovirus (PV). Note: estimates included in this table are based on extrapolation of data from scientific studies and should be regarded as indicative only. Data in this table cannot be used to calculate risks. For precise process calculations or predictions on food manufacturing processes, additional experimental information is needed.

[b]Treatment results in at least 4$\log_{10}$ inactivation of common foodborne viruses. Low risk = product unlikely to contain infectious viruses in numbers likely to cause disease in healthy individuals; treatment results in approximately 3$\log_{10}$ inactivation of common foodborne viruses. Medium risk = product may contain infectious viruses in numbers that may cause disease; treatment results in approximately 2$\log_{10}$ inactivation of common foodborne viruses. High risk = products in which the level of viruses is likely to be high enough to cause disease in healthy individuals; treatment results in less than 1$\log_{10}$ inactivation of common foodborne viruses. Variable risk = treatment results in significant differences in inactivation of several common foodborne viruses.

[c]Before spray drying in dried milk processes, a substantial heat step destroys viruses.

Appendix A 2

Preventing actions - norovirus and HAV (information for the public)

- proper hand washing with soap and warm water and dry the hands in clean towels, especially before eating or preparing food, after using the toilet and after changing diapers.
- make sure that all food preparation areas are clean before use.
- wash all fruits and vegetables.
- cook your food completely.
- (frozen imported) raspberries (used for sauce) should be boiled for 5 min.
- in an area that might have contaminated water: drink bottled water and beverages without ice.
- after episodes of vomiting or diarrhoea, clean contaminated surfaces immediately with a bleach-based household clean.
- foodhandlers (workers of food related business) who have gastroenteritis (norovirus) must stay out of work for at least 72 hours after their symptoms have stopped.
- foodhandlers should know that they shed norovirus up to 3 weeks after onset of symptoms (in that time foodhandler might not work with food production and only use a separated toilet).

Especially for HAV

- when you travel to an area with increased or high risk of HAV consider vaccination or injection immunoglobulins against HAV.
- vaccination is recommended to men who have sex with men.
- vaccination is recommended to illicit drug users (regardless of whether they inject the drugs or not).
- vaccination is recommended to families who have members with HAV.

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