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Foodborne viral illness – status in Australia

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Abstract

Norwalk-like virus contamination of oysters and orange juice, and hepatitis A virus contamination of oysters have been responsible for large outbreaks of foodborne viral disease in Australia. Rotavirus, adenovirus, astrovirus, parvovirus and other enteroviruses also contribute to the incidence of gastroenteritis in this country but the role of foods and waters in transmitting these viruses is unclear. Protocols for the investigation, surveillance and reporting of foodborne viral illness require further development to enable a more accurate description of the problem. Few laboratories have the capability to analyse foods for viruses and specific training in this technology is needed. Management of food safety in Australia largely relies on the implementation of HACCP principles, but these need to be adapted to address the specific risks from viruses.

Keywords: Food; Virus; Norwalk virus; Hepatitis A; Gastroenteritis; Australia

1. Introduction

The food industry is one of the largest manufacturing and service sectors in Australia. It employs about 750 000 individuals and has an annual turnover value of approximately $US 50 billion (Anonymous, 1999a). Because it has a strong focus on export, it is intimately linked with international trade. The success of the industry depends on production, managerial and regulatory practices that ensure high quality, safe products. Microbiological safety commands high priority by both the industry and the government regulatory sectors. To achieve this safety, both sectors closely follow recommendations of the Codex Alimentarius Commission by implementing appropriate quality assurance, HACCP, and risk management programs, and ensuring that products conform to appropriate microbiological specifications and standards (Fleet, 1996; Jouve, 1998; Peters, 1998, 1999; Hathaway, 1999).

As a nation, Australia is comprised of six States and two Territories. Food safety is regulated by legislation that is administered through each State and Territory, using a common policy that has been developed at the national level by The Australia New Zealand Food Authority (ANZFA) (Anonymous, 1999a). ANZFA is presently developing national food safety standards based on HACCP principles

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that will also be administered at the State and Territory level. Foods for export are regulated separately by the Australian Quarantine Inspection Service (AQIS) which is an agency of the national Government but, in essence, follows the safety policies of ANZFA. Imported foods are also monitored by AQIS which administers the Imported Food Inspection Program based on policies developed by ANZFA (Fleet, 1996; Oram-Miles, 1999).

In addition to the general requirements for due diligence and good manufacturing practice, the microbiological safety of foods in Australia is regulated through a series of microbiological standards that are published in the Australian Food Standards Code (Anonymous, 1997a). Currently, these standards are based on bacteriological and fungal criteria. Virological criteria are not part of these standards, mainly because little information is known about the occurrence of viruses in local foods and because of the technical difficulties associated with analysis of viruses in foods. Nevertheless, as will be discussed in the following sections, Australia has a significant history of food and waterborne viral diseases. The incidence of these outbreaks appear to be on the increase which, combined with newer methods that enable the analysis of foods for viruses, are increasing demands for greater quality assurance and regulation of this problem. As a basis for developing future programs for prevention and management, this article examines the past and current status of food and waterborne viral diseases in Australia.

2. Surveillance systems and epidemiological statistics

Each State and Territory within Australia has public health legislation that requires official notification of communicable diseases. Notification simply means that clinical diagnosis of the disease or isolation of the causative organism must be reported to the appropriate Department of Health or equivalent. Communicable foodborne diseases that are listed for notification are campylobacteriosis, salmonellosis, shigellosis, yersiniosis, typhoid, listeriosis and hepatitis A. Annual reports of these notifications are published by each State and Territory and have been accessed for the purposes of this contribution. Over the years, there have been various attempts to collate the data of these reports at the national level, but these initiatives have been ad hoc and irregular (Murrell, 1986a,b; Crerar et al., 1996). In 1991, the Australian Government established the National Notifiable Diseases Surveillance System (NNDSS) to compile national statistics on communicable diseases including foodborne diseases. It receives data from the State and Territory notification programs, as well as other voluntary reporting schemes such as the recently established National Enteric Pathogen Surveillance Scheme (NEPSS), the Australian Paediatric Surveillance Unit (APSU), the *Escherichia coli* Reference Laboratory and the Virology and Serology Laboratory Reporting Scheme (LabVISE). The NEPSS collates reports on the isolation of pathogenic organisms from foods and other environmental sources and the APSU monitors disease outbreaks in children. LabVISE collates data on virus diseases as diagnosed in 21 sentinel laboratories operating throughout Australia. One objective of the NNDSS is to provide information that will assist in developing a uniform, national policy towards management of food safety (Anonymous, 1997b, 1999a). Reports of the NNDSS are published in the *Communicable Diseases Intelligence*, through the Department of Health and Family Services of the Australian Government.

According to data compiled by the NNDSS, notification of foodborne microbial diseases in Australia increased from approximately 17 000 cases in 1991 to 23 000 cases in 1999. Hepatitis A, the only notifiable virus disease, represented about 10–13% of the cases over this period. However, it is generally accepted that these statistics are a substantial underestimation of the real incidence of foodborne disease, and highlight major limitations of the surveillance system (Crerar et al., 1996; Hellard and Findlay, 1997; Anonymous, 1999a). Many cases of foodborne illness remain unreported or not diagnosed and, consequently, are not notified. Also, the notification system does not include illness caused by many foodborne pathogens, such as *Staphylococcus aureus*, *Bacillus cereus*, *Clostridium perfringens* or viruses other than hepatitis A. For example, orange juice contaminated with Norwalk virus was responsible for over 3000 cases of gastroenteritis in Australia in 1991, but these data are not included in the official notifiable statistics. Based on the data available and the limitations inherent in the existing

surveillance system, several groups have independently estimated that the true incidence of foodborne microbial illness in Australia is about 4.0–5.0 million cases per year. About 30–40% of these cases are probably attributable to viruses (Anonymous, 1997b, 1999a). This proportion of foodborne viral illness, relative to the total, is similar to that reported for the USA (Anonymous, 1999b).

As in other countries, significant outbreaks of poliomyelitis occurred in Australia during the early part of the twentieth century, and foods and waters were frequently suspected as being involved in transmitting the disease (Eyles, 1978). However, genuine interest in food or waterborne viral diseases did not develop in Australia until 1978 when oysters were found to be responsible for a very large outbreak of gastroenteritis caused by Norwalk-like agents. Table 1 shows the range of viral diseases that have the potential to be food or waterborne and are now routinely monitored throughout the country by LabVISE. The values in Table 1 do not represent the true incidences of each of the viral diseases since they are compilations of reports from only 21 sentinel laboratories. However, based on comparisons with NNDSS data for hepatitis A virus, a fully notifiable viral disease, the values in Table 1 probably represent an approximate five-fold underestimation of the real incidence. Nevertheless, the data of Table 1 are significant in showing annual trends and the relative frequency of the different viruses in causing disease. Eyles (1978, 1989) and Grohmann (1997) have made passing references to outbreaks of foodborne viral disease in Australia as part of more general discussions of viruses in foods. The following sections provide a more detailed discussion of viruses associated with or thought to be associated with outbreaks of foodborne disease in Australia.

3. Rotavirus

Rotaviruses were first discovered in Australia in 1973, and were found in the faeces and duodenal mucosal epithelial cells of children who had been hospitalised with acute, non-bacterial gastroenteritis (Bishop et al., 1974). These viruses are now recognised as a major cause of gastroenteritis in children throughout the world. Rotaviruses belong to the family of *Reoviridae*, and infect the epithelial cells of the small intestine (Christensen, 1989; Desselberger, 1998). Illness becomes evident after 1–2 days incubation and is characterised by the sudden onset of acute, watery diarrhoea and vomiting, often accompanied by fever. The symptoms persist for several days or longer, leading to dehydration which, in severe cases, can cause death. High levels of infectious virus particles are excreted with the faeces. Transmission of the disease from person to person occurs mostly via the faecal–oral route, because the virus is highly infectious. However,

Table 1
Australian statistics on the incidence of illness caused by viruses that are potentially foodborne[a]

Virus	1991	1992	1993	1994	1995	1996	1997	1998	1999
Rotavirus	2643	2134	1989	2274	1616	1640	1585	1325	1507
Adenovirus group	1513	1716	1861	1542	1182	1611	1258	1033	992
Adenovirus type 40	4	6	9	–	–	34	16	15	59
Adenovirus type 41	–	–	–	–	–	1	3	–	–
Enterovirus	687	804	944	1101	923	880	642	502	638
Parvovirus	28	178	86	109	102	258	366	248	342
Hepatitis A virus	444	371	452	373	430	415	723	380	296
Echovirus	151	1	490	443	201	79	47	60	277
Norwalk agent	21	6	21	11	47	43	104	38	60
Coxsackievirus	213	214	277	159	43	60	67	34	23
Astrovirus	19	14	4	1	6	–	8	10	4
Calicivirus	37	19	12	6	1	6	–	1	1
Coronavirus	36	26	11	2	1	–	–	–	–
Small virus (like) particle	59	64	33	33	16	16	3	2	–
Total	5855	5553	6189	6054	4568	5043	4822	3648	4199

[a] Data are compiled from LabVISE reports published in *Communicable Disease Intelligence*; –: No data reported.

contaminated foods and waters can be significant in its transmission since the organism has a strong survival rate in the environment (Christensen, 1989; Hedberg and Osterholm, 1993; Bishop, 1994).

In Australia, gastroenteritis caused by rotaviruses is responsible for the annual hospitalisation of about 12 000 children under the age of 5 years. This represents about 50% of all children hospitalised with gastroenteritis and is estimated to cost the nation about $US 10 million annually (Bishop and Barnes, 1996; Carlin et al., 1998; Masendycz et al., 1999). Outbreaks of the disease are mostly associated with child care centres, pre-schools and other situations where there are large numbers of children (Hanna, 1992; Hanna and Brookes, 1993; Ferson, 1995). However, outbreaks in adults, especially at nursing homes for the elderly, can occur (Bishop, 1994). While person to person contact is considered to be the principal mode for spread of the virus in these situations, the involvement of faecally contaminated water and foods is often suspected, but requires more specific investigation. Crerar et al. (1996) refer to one outbreak, involving 55 individuals, where salad vegetables were considered to have transferred the virus. Diagnosis of the disease generally relies on the symptoms it causes and detection of the virus in faecal specimens by serological and electron microscopic techniques.

The epidemiology of the illness has been extensively monitored over the last 25 years by serotyping and electrophoretic differentiation of the viral RNA (Rodger et al., 1981; Barnes et al., 1998; Diwarkala and Palombo, 1999). The incidence of outbreaks follows a seasonal pattern, predominating in mid to late winter months, but minor variations in this pattern occur across the country. In tropical regions, peak times of infection may occur in summer months (Bishop and Barnes, 1996). Different states within Australia also have different rotavirus notification rates, ranging from 9.2 per 1000 to 49.8 per 1000 children under the age of 5 years (Carlin et al., 1998). Similar serotype patterns are observed in different cities, emphasising the endemic nature of rotavirus infection in the population. The majority of the outbreaks are caused by Group A rotavirus, serotype G1 (about 80% of cases), but epidemics caused by serotypes G2, G3, G4, G6 and G8 have occurred (Palombo and Bishop, 1995). Substantial heterogeneity occurs in the RNA electrophoretic profiles of the serotypes, and suggests the continual emergence of new strains of the virus (Palombo et al., 1996). A National Rotavirus Reference Centre has recently been established to improve surveillance and management of the disease (Masendycz et al., 1999).

4. Norwalk or Norwalk-like viruses

The Norwalk or Norwalk-like viruses belong to a group generally called the small round structured viruses (SRSVs) because of their morphology. Their genome consists of single stranded RNA, the sequence of which corresponds with their classification in the family *Caliciviridae* (Appleton, 1994; Desselberger, 1998; Anonymous, 1999a). They have been a major cause of food or waterborne gastroenteritis in Australia (Eyles, 1989; Grohmann, 1997; Marshall and Wright, 1999). Characteristic symptoms of nausea, vomiting and diarrhoea generally appear after 24–48 h of incubation and last about 48–72 h.

The first evidence of a problem with these viruses occurred in 1977, when oysters harvested from Georges River, Sydney, caused outbreaks of gastroenteritis in the UK. The oysters had been opened and frozen in the half shells before export. The frozen oysters conformed to *Escherichia coli* standards (<2.3 *E. coli*/g) as tested by exporting authorities in Australia and importing authorities in the UK. No bacterial pathogens were detected in the oysters at levels that would cause gastroenteritis. Subsequent investigations revealed that the implicated oysters had been harvested from Georges River at a time during rainfall and that unfrozen batches of the oysters had exhibited counts exceeding the *E. coli* standard. Evidently, freezing of the oysters killed the *E. coli* but not the suspected virus. The same batch of frozen oysters caused gastroenteritis more than 6 months after storage (Fleet, unpublished data). Although oysters and specimens from victims of the outbreaks were not examined for viruses, the symptoms of gastroenteritis were characteristic of Norwalk or Norwalk-like viruses. Moreover, oysters harvested from the same location several months later in 1978 caused outbreaks of similar gastroenteritis in Australia affecting over 2000 consumers. These outbreaks have been extensively studied, and Norwalk virus was detected by electron microscopy, immunoelectron microscopy and reverse transcrip-

tion (RT)-PCR in faeces of many of the affected consumers (Murphy et al., 1979; Grohmann et al., 1980; Linco and Grohmann, 1980; Moe et al., 1994). Unfortunately, Norwalk virus could not be detected in oysters implicated in these outbreaks, although echovirus type 8 and reovirus were isolated, but not considered responsible for the gastroenteritis (Eyles et al., 1981). These outbreaks resulted in major changes to the regulation of oyster production in the state of New South Wales (NSW) of Australia and, subsequently, it became mandatory for all oysters to be subjected to a process of depuration before sale (Souness and Fleet, 1991; Ayres, 1991). While depuration combined with quality assurance programs have managed to prevent further major outbreaks of oyster associated Norwalk virus, some authors have questioned the efficacy of depuration in eliminating the virus (Eyles, 1980, 1989; Grohmann et al., 1981; Grohmann, 1997). Oysters harvested from an estuary in northern NSW and supposedly depurated were suspected of causing an outbreak of 97 cases of Norwalk virus gastroenteritis in 1996. The virus could not be detected in faecal specimens but was detected in one sample of oysters by RT-PCR (Stafford et al., 1997). Further research is being conducted at the University of New South Wales to determine the kinetics of virus depuration from local shellfish, and to evaluate the use of bacteriophages as potential indicators of estuary and shellfish contamination with human viruses such as the Norwalk or Norwalk-like viruses.

The largest outbreak of Norwalk virus associated gastroenteritis involved over 3000 individuals who had consumed orange juice that was largely served to passengers on domestic airline flights during August 1991. Extensive surveying of passengers as to what they had consumed, enabled identification of the causative food, in this case orange juice which was traced to one manufacturer. Inspection of the production facilities identified several areas where contamination of the juice could have occurred. The virus was not detected in the juice but the outbreak terminated when the juice was withdrawn from the market. Faecal specimens from affected individuals showed the presence of characteristic Norwalk-like particles (Lester et al., 1991).

Sewage contaminated drinking water was suspected to be the cause of an outbreak of Norwalk-like gastroenteritis in several hundred guests at a caravan park in NSW (McAnulty et al., 1993). Analysis of faecal specimens from affected individuals by direct- and immuno-electron microscopy did not give conclusive evidence of virus presence, but ELISA testing of sera as well as epidemiological data suggested the occurrence of Norwalk-like agents. Private hospitals, hostels, parenting centres, and nursing homes are often involved in outbreaks of Norwalk-like gastroenteritis (Oliver et al., 1985; Selden et al., 1993; Taylor and Murphy, 1994; Wilby and Ferriera, 1995; Marshall et al., 1997; Marshall and Wright, 1999). Transmission of the disease by mechanisms other than food or water seems to have occurred in these outbreaks but, generally, the source of the virus and its mode of transfer were not thoroughly investigated.

Wright et al. (1998) have completed a major retrospective study of the incidence of Norwalk-like gastroenteritis in Australia, principally the state of Victoria, by examining for the virus in faecal specimens that had been submitted for suspected viral gastroenteritis over the period 1980–1996. Faecal samples were screened for presence of the virus by electron microscopy. Positive samples were further examined by RT-PCR followed by partial sequencing of the amplified DNA. Of the 6226 samples examined, about 3.5% were positive for presence of Norwalk-like virus with the majority (82%) representing submissions from adults or elderly persons. Of the positive samples, 36% were submissions from hospitals, 19% were from nursing homes, 13% were associated with outbreaks related to a restaurant or reception, 5% from camp, chalet or child-care settings, and 26% were from family or uncertain origins. Outbreaks were more common in late winter and early summer months. Based on DNA sequences, the viruses were classified into genogroups 1 and 2, with most cases of gastroenteritis being associated with genogroup 2. While the source of the virus was not reported in this study, epidemiological data suggested a significant involvement of foods or waters. A recent report has suggested that Norwalk and Norwalk-like viruses probably account for the greatest incidence of foodborne disease in Australia (Anonymous, 1999a).

5. Hepatitis A

Hepatitis A virus (HAV) is classified in the family *Picornaviridae* which also includes the poliovirus

and enterovirus groups. Their genome consists of single stranded RNA which, in the case of HAV, is highly conserved, giving only four genotypes and one serotype (White and Fenner, 1994). Spread of the virus occurs by the faecal–oral route, where contaminated foods and waters can be significant vectors. After ingestion, the virus multiplies within the intestinal epithelium, passes into the blood stream and then attacks liver cells. Onset of the disease occurs after an incubation period of 2–6 weeks and is evidenced by symptoms of malaise, anorexia, nausea, lethargy, jaundice, pale faeces, dark urine and liver pain. Illness may last 4–6 weeks, after which full recovery is usual. However, shedding of the virus in the faeces can extend for 3–6 months and some individuals experience relapses of the disease. Death due to liver failure may occur, but is rare (White and Fenner, 1994; Anonymous, 1999b).

Statistics on HAV infection in Australia have been reviewed and discussed by several authors (Scott and Sheridan, 1994; Selden et al., 1994; Ferson et al., 1998; Merritt et al., 1999; Amin et al., 1999). As noted already, HAV infection is a notifiable disease in Australia. Data from the NNDSS indicate 1500–3000 cases per year over the period 1991–1999. Notification statistics vary between States and Territories, and range from 1.9 to 51.7 cases/100 000 individuals. Significant outbreaks of HAV infection have been reported in homosexual men (Ferson et al., 1998), individuals within institutions (Hanna and Brookes, 1994; Bell et al., 1994), child care centres (Ferson et al., 1994; Tallis et al., 1996), in lower socioeconomic communities or families (Dick et al., 1994) and after consumption of foods (Dienstag et al., 1976; Anonymous, 1997c,d). Apart from the well described foodborne outbreaks, person to person contact is considered to be the main mode for spread of the virus. However, poor hygiene and faecal contamination of foods and waters may be responsible for spread of the virus in institutional and child care settings.

The first documented foodborne outbreak of HAV in Australia was attributed to the consumption of incompletely cooked mussels that had been harvested from faecally contaminated waters in the state of Victoria. Seven out of the 10 individuals who consumed the mussels developed symptoms of hepatitis A. Antibodies to HAV were subsequently detected in the sera of affected individuals (Dienstag et al., 1976; Locarnini and Gust, 1978). A dredge, discharging untreated sewage into the water near where the mussels were growing, was thought to be the source of the virus. The largest outbreak of HAV in Australia occurred over several months during 1996–1997 after consumption of oysters harvested from the Wallis Lakes region in northern NSW. Almost 500 individuals were affected and one person died as a result of the disease. Analysis of samples of oysters collected from the region during the time of the outbreak were positive for the presence of HAV as determined by a PCR method. The oysters also tested positive for the presence of adenoviruses and enteroviruses but not Norwalk viruses. However, they conformed to bacteriological standards (less than 2.3 *E. coli*/g) and, supposedly, had been subject to depuration according to legislative requirements of the state government. A subsequent coronial enquiry into the outbreak revealed numerous sources by which untreated sewage could contaminate the lake and oyster cultivation areas (e.g. no reticulated sewage systems in nearby villages, leaking public toilets, caravan parks, and recreation water craft) (Anonymous, 1997c; Grohmann, 1997; Wilcox, 1999). More stringent quality assurance programs for oyster cultivation and processing have been implemented to avoid future problems of this nature (Wilcox, 1999). However, the question as to whether HAV is effectively eliminated from oysters after commercial depuration processes remains unresolved and requires research. Another concern arising from this outbreak was the failure of *E. coli* standards to reliably indicate oyster contamination by viruses.

Sporadic outbreaks of foodborne HAV occur throughout the country and generally involve foods served at restaurants. One such outbreak involved 23 cases and was traced to the consumption of imported frozen, fresh water prawns. PCR testing of leftover prawns failed to detect HAV and blood samples taken from restaurant staff all tested negative for recent HAV infection (Anonymous, 1997d).

6. Astroviruses

Astroviruses contain single-stranded RNA within a capsid that has a characteristic star-shaped appearance when visualised by electron microscopy. They

cause gastroenteritis, especially in young children, giving symptoms similar to those caused by rotaviruses, but generally, are less severe. Transmission of the virus occurs by the faecal–oral route (Lew et al., 1991; Appleton, 1994). Very little has been reported about the occurrence of this virus in Australia. Using a specific DNA probe, astrovirus was detected in 4.2% of faecal specimens from 378 children who were hospitalised with gastroenteritis in the state of Victoria during 1995. Incidence of the virus was greatest during winter months and in infants of 6–12 months of age (Palombo and Bishop, 1996). The epidemiological significance of these findings was not reported. Clearly, astrovirus-associated gastroenteritis does occur in Australia (Table 1) but the role of food or water in its transmission needs further investigation. The specific role of shellfish in its transmission would be worthy of more thorough investigation, because of their previous association with outbreaks of HAV and Norwalk-like viruses in this country and overseas reports of shellfish-associated outbreaks of astrovirus gastroenteritis (Appleton, 1994).

7. Adenovirus

Adenoviruses contain double stranded DNA within an icosahedral capsid. Many of the 47 or more known adenovirus serotypes can multiply in the small intestine, but only types 40 and 41 have been associated with gastroenteritis (Grimwood et al., 1995). The most common method of transmission is via the faecal–oral route, with food and water as possible vectors (Mickan and Kok, 1994). Illness lasts slightly longer than rotavirus infections, but the symptoms of diarrhoea, vomiting and fever are milder (Christensen, 1989).

Several studies covering the analysis of about 5000 faecal specimens during the period 1981–1996 indicate that adenoviruses contribute 3–9% of the gastroenteritis cases admitted to Australian hospitals (Pitson et al., 1986; Mickan and Kok, 1994; Grimwood et al., 1995; Palombo and Bishop, 1996). Diagnosis of the virus is generally based on its detection in faecal samples by electron microscopy and enzyme immunoassay. The majority of the cases are associated with young children and involve serotype 41 (40–80%) and to a lesser extent, serotype 40 (less than 20%). Seasonal patterns of the virus genotypes were evident, with type 41 being prevalent in late autumn and type 40 remaining prevalent year-round. The source of the virus and its mode of transmission were not examined in these studies.

8. Human calicivirus or Sapporo-like viruses

Human caliciviruses (HCVs), also known as Sapporo-like viruses, are a subgroup of the *Caliciviridae* and cause gastroenteritis similar to that caused by Norwalk or Norwalk-like viruses. The first reported outbreak in Australia attributed to HCV occurred in a day care centre in 1988. Fifty-three people were affected, with HCV being found in 32% of faecal samples from symptomatic adults and children, and in 8% of asymptomatic individuals. The outbreak, which affected young children more severely, lasted more than 10 weeks due to the number of asymptomatic individuals and the prolonged excretion of virus particles. The outbreak persisted despite taking many control measures such as closing the central kitchen, encouraging handwashing, and modifying diaper changing practices (Grohmann et al., 1991).

Since 1988, the cases of HCV in Australia have remained sporadic and rare (Table 1). A study of faecal samples from patients with gastroenteritis during 1980–1996 found that only 0.14% of cases were due to HCV, compared with 3.5% of cases due to Norwalk and Norwalk-like viruses (Wright et al., 1998).

9. Other viruses

Parvovirus-like particles were responsible for an outbreak of gastroenteritis involving 217 children and teachers at a primary school in Sydney, NSW, in 1977, but the source of the virus was not determined (Christopher et al., 1978). Although these viruses contribute significantly to the sporadic incidence of gastroenteritis (Table 1), their transmission in foods is not reported. Coronaviruses have been detected in the faeces of adults in Australia (Marshall et al., 1989), including aborigines where the excretion rates were higher than those for non-aboriginals (Schnagl et al., 1978). While the standard of hygiene is

thought to relate to the incidence of this virus, evidence of a direct role of foods in its transmission is not clear (Caul, 1994). Coxsackie, echo and other enteroviruses have been detected in oysters harvested from NSW estuaries (Eyles et al., 1981; Grohmann, 1997) but not implicated in outbreaks of gastroenteritis. Nevertheless, these viruses have been connected with numerous cases of gastroenteritis, the causes of which were not reported (Table 1).

10. Conclusion

Viruses, especially Norwalk-like agents and HAV, have caused the largest recorded outbreaks of foodborne microbial illness in Australia during the last 20 years. While the occurrence of such large outbreaks is infrequent, epidemiological and surveillance statistics suggest that viruses account for 30–40% of all foodborne microbial illness in this country. Despite the magnitude of this problem, our review has identified several issues that require further consideration and action to improve the surveillance, monitoring and control of viruses in foods. Compared with bacteria, epidemiological investigation and reporting of foodborne viral illnesses were frequently inadequate and, rarely, identified the source of the virus and its mode of transmission. More rigorous, standardised protocols for investigating foodborne viral disease are required. Molecular methods for the detection and tracking of viral contamination in suspect foods are now available (Richards, 1999) but greater education is needed in the use of these new technologies. However, routine monitoring for specific viruses in foods (e.g. Norwalk or HAV in oysters) is still problematical and not done because of the cost and inconvenience of the assays. Moreover, few, if any, non-government laboratories currently have this analytical expertise and, again, education is needed. Faecal coliform and *E. coli* tests are widely used and accepted as indicators of food contamination with sewage, despite their unreliability to indicate virus presence. This limitation is particularly acute for monitoring the safety of shellfish which have an established history of causing foodborne viral illness in Australia. Consequently, there would be significant interest in convenient bacteriophage tests that could reliably indicate the presence of human viruses in foods (Armon and Kott, 1996). With the absence of analytical monitoring, management of foodborne viral disease largely relies upon the proper implementation of HACCP principles. However, it is imperative that these be adapted to account for specific viral hazards.

References

Amin, J., Heath, T., Morrell, S., 1999. Hepatitis A in Australia in the1990s: future directions in surveillance and control. Comm. Dis. Int. 23, 113–119.

Anonymous, 1997a. Australian Food Standards Code. Australian Government Publishers Service, Canberra, Australia.

Anonymous, 1997b. Foodborne disease. Towards reducing foodborne illness in Australia. Technical Report Series Number 2 from the Foodborne Diseases Working Party for the Commonwealth Diseases Network Australia and New Zealand, Legislative Services AusInfo, Canberra, Australia.

Anonymous, 1997c. Hepatitis A Outbreak Traced to Consumption of Wallis Lake Oysters. NSW Pub. Health Bull. 8, 1,5.

Anonymous, 1997d. Hepatitis A outbreak linked to a Sydney restaurant. NSW Pub. Health Bull. 8, 51.

Anonymous, 1999a. Food Safety Standards, Costs and Benefits. Australia – New Zealand Food Authority, Commonwealth Government of Australia, Canberra, pp. 1–154.

Anonymous, 1999b. Discussion paper on viruses in food. CX/FH 99/11. Joint FAO/WHO food standards programme codex committee on food hygiene. Codex Alimentarius Commission, Rome

Appleton, H., 1994. In: Hui, Y.H., Gorham, J.R., Murrell, K.D., Cliver, D.O. (Eds.), Foodborne Disease Handbook. Norwalk virus and the small round viruses causing foodborne gastroenteritis, Vol. 2. Marcel Dekker, New York, pp. 57–79.

Armon, R., Kott, R., 1996. Bacteriophages as indicators of pollution. Crit. Rev. Environ. Sci. Technol. 26, 299–335.

Ayres, P.A., 1991. The status of shellfish depuration in Australia and south east Asia. In: Otwell, W.S., Rodrick, G.E., Martin, R.E. (Eds.), Molluscan Shellfish Depuration. CRC Press, Boca Raton, pp. 287–322.

Barnes, G.L., Uren, E., Stevens, K.B., Bishop, R.F., 1998. Etiology of acute gastroenteritis in hospitalized children in Melbourne, Australia, from April 1980 to March 1993. J. Clin. Microbiol. 36, 133–138.

Bell, J.C., Crewe, E.B., Capon, A.G., 1994. Hepatitis A in a community of persons with developmental disabilities in western Sydney. Comm. Dis. Int. 18, 5–8.

Bishop, R.F., 1994. Natural history of human rotavirus infections. In: Kapikian, A.Z. (Ed.), Viral Infections of the Gastroentestinal Tract, 2nd Edition. Marcel Dekker, New York, pp. 131–167.

Bishop, R.F., Barnes, G., 1996. Rotavirus infection – the need for a vaccine. Comm. Dis. Int. 20, 296–298.

Bishop, R.F., Davidson, G.P., Holmes, I.H., Ruck, B.J., 1974. Detection of a new virus by electron microscopy of faecal

extracts from children with acute gastroenteritis. Lancet 2, 149–151.
Carlin, J.B., Chondros, P., Masendycz, P., Bugg, H., Bishop, R.F., Barnes, G.L., 1998. Rotavirus infection and rates of hospitalisation for acute gastroenteritis in young children in Australia, 1993–1996. Med. J. Aust. 169, 252–256.
Caul, E.O., 1994. Human coronaviruses. In: Kapikian, A.Z. (Ed.), Viral Infections of the Gastroentestinal Tract, 2nd Edition. Marcel Dekker, New York, pp. 603–625.
Christensen, M.L., 1989. Human viral gastroenteritis. Clin. Microbiol. Rev. 2, 51–89.
Christopher, P.J., Grohmann, G.S., Millsom, R.H., Murphy, A.M., 1978. Parvovirus gastroenteritis – a new entity for Australia. Med. J. Aust. 1, 121–125.
Crerar, S.K., Dalton, C.B., Longbottom, H.M., Kraa, E., 1996. Foodborne disease: current trends and future surveillance needs in Australia. Med. J. Aust. 165, 672–675.
Desselberger, U., 1998. Viral gastroenteritis. Curr. Opin. Infect. Dis. 11, 565–575.
Dick, A., Beezley, C., Scott, J., 1994. Persisting hepatitis A infection in a central Queensland town. Comm. Dis. Int. 18, 83–89.
Dienstag, J.L., Gust, I.D., Lucas, C.R., Wong, D.C., Purcell, R.H., 1976. Mussel-associated viral hepatitis, type A: serological conformation. Lancet 1, 561–564.
Diwarkala, C.S., Palombo, E.A., 1999. Genetic and antigenic variation of capsid protein VP7 of serotype G1 human rotavirus isolates. J. Gen. Virol. 80, 341–344.
Eyles, M.J., 1978. Viruses in food. Food Technol. Aust. 30, 291–294.
Eyles, M.J., 1980. Accumulation and elimination of viruses by oysters. Food Technol. Aust. 32, 89–91.
Eyles, M.J., 1989. Viruses. In: Buckle, K.A., Davey, J.A., Eyles, M.J., Hocking, A.D., Newton, K.G., Stuttard, E.J. (Eds.), Foodborne Microorganisms of Public Health Significance, 4th Edition. CSIRO Food Science and Technology, North Ryde, NSW, Australia, pp. 335–346.
Eyles, M.J., Davey, G.R., Huntley, E.J., 1981. Demonstration of viral contamination of oysters responsible for an outbreak of viral gastroenteritis. J. Food Protect. 44, 294–296.
Ferson, M.J., 1995. Direct and indirect costs of an outbreak of rotavirus in a child-care centre. Comm. Dis. Int. 19, 4–6.
Ferson, M.J., Young, L., Robertson, P.W., Whybin, L.R., 1994. Hepatitis A outbreak in a preschool in Eastern Sydney. Comm. Dis. Int. 18, 82–83.
Ferson, M.J., Young, L.C., Stokes, M.-L., 1998. Changing epidemiology of hepatitis A in the 1990s in Sydney, Australia. Epidemiol. Infect. 121, 631–636.
Fleet, G.H., 1996. Microbiological methods – the Australian perspective. Food Control 7, 41–46.
Grimwood, K., Carzino, R., Barnes, G.L., Bishop, R.F., 1995. Patients with enteric adenovirus gastroenteritis admitted to an Australian pediatric teaching hospital from 1981 to 1992. J. Clin. Microbiol. 33, 131–136.
Grohmann, G.S., 1997. Viruses, food and environment. In: Hocking, A.D., Arnold, G., Jenson, I., Newton, K., Sutherland, P. (Eds.), Foodborne Microorganisms of Public Health Significance, 5th Edition. CSIRO Food Science and Technology, North Ryde, NSW, Australia, pp. 603–620.
Grohmann, G.S., Glass, R.I., Gold, J., James, M., Edwards, P., Borg, T., Stine, S.E., Goldsmith, C., Monroe, S.S., 1991. Outbreak of human calicivirus gastroenteritis in a day-care center in Sydney, Australia. J. Clin. Microbiol. 29, 544–550.
Grohmann, G.S., Greenberg, H.B., Welch, B.M., Murphy, A.M., 1980. Oyster-associated gastroenteritis in Australia. The detection of norwalk virus and its antibody by immune electron microscopy and radioimmunoassay. J. Med. Virol. 6, 11–19.
Grohmann, G.S., Murphy, A.M., Christopher, P.J., Auty, E., Greenberg, H.B., 1981. Norwalk virus gastroenteritis in volunteers consuming depurated oysters. Aust. J. Exp. Biol. Med. Sci. 59, 219–228.
Hanna, J., 1992. An endemic of rotavirus diarrhoea in far north Queensland. Comm. Dis. Int. 16, 425–426.
Hanna, J., Brookes, D., 1993. Absenteeism from a child day-care centre during a rotavirus outbreak. Comm. Dis. Int. 17, 350–351.
Hanna, J., Brookes, D., 1994. Some occupational exposures to hepatitis A. Comm. Dis. Int. 18, 3–5.
Hathaway, S., 1999. Management of food safety in international trade. Food Control 10, 247–253.
Hedberg, C.W., Osterholm, M.T., 1993. Outbreaks of foodborne and waterborne viral gastroenteritis. Clin. Microbiol. Rev. 6, 199–210.
Hellard, M.E., Findlay, C.K., 1997. Gastroenteritis in Australia: who, what, where, and how much? Aust. NZ J. Med. 27, 147–149.
Jouve, J.L., 1998. Principles of food safety legislation. Food Control 9, 75–81.
Lester, R., Stewart, T., Carnie, J., Ng, S., Taylor, R., 1991. Air travel-associated gastroenteritis outbreak, August 1991. Comm. Dis. Int. 15, 292–293.
Lew, J.F., Moe, C.L., Monroe, S.S., Allen, A.R., Harrison, B.M., Forrester, B.D., Stine, S.E., Woods, P.A., Hierholzer, J.C., Herrmann, J.E., Blacklow, P.R., Bartlett, A.V., Glass, R.I., 1991. Astrovirus and adenovirus associated with diarrhea in children day care settings. J. Infect. Dis. 164, 673–678.
Linco, S.J., Grohmann, G.S., 1980. The Darwin outbreak of oyster-associated viral gastroenteritis. Med. J. Aust. 1, 211–213.
Locarnini, S.A., Gust, I.D., 1978. Food and hepatitis A. Food Technol. Aust. 30, 295, 297–298.
McAnulty, J.M., Rubin, G.L., Carvan, C.T., Huntley, E.J., Grohmann, G., Hunter, R., 1993. An outbreak of Norwalk-like gastroenteritis associated with contaminated drinking water at a caravan park. Aust. J. Pub. Health 17, 36–41.
Marshall, J.A., Danielson, G., Doultree, J.C., Gunesekere, I.C., Seah, E.L., Wright, P.J., 1997. Norwalk-like virus associated with gastroenteritis in babies. Med. J. Aust. 166, 276.
Marshall, J.A., Thompson, W.L., Gust, I.D., 1989. Coronavirus-like particles in adults in Melbourne, Australia. J. Med. Virol. 29, 238–243.
Marshall, J., Wright, P., 1999. Norwalk-like viruses: prevalence and control. Vic. Infec. Dis. Bull. 2, 1–3.
Masendycz, P.J., Palombo, E.A., Barnes, G.L., Bishop, R.F., 1999. Rotavirus diversity: what surveillance will tell us. Comm. Dis. Int. 23, 198–199.
Merritt, A., Symons, D., Griffiths, M., 1999. The epidemiology of

acute hepatitis A in North Queensland, 1996–1997. Comm. Dis. Int. 23, 120–124.

Mickan, L.D., Kok, T.-W., 1994. Recognition of adenovirus types in faecal samples by southern hybridization in South Australia. Epidemiol. Infect. 112, 603–613.

Moe, C.L., Gentsch, J., Ando, T., Grohmann, G., Monroe, S.S., Jiang, X., Wang, J., Estes, M.K., Seto, Y., Humphrey, C., Stine, S., Glass, R.I., 1994. Application of PCR to detect norwalk virus in fecal specimens from outbreaks of gastroenteritis. J. Clin. Microbiol. 32, 642–648.

Murphy, A.M., Grohmann, G.S., Christopher, P.J., Lopez, W.A., Davey, G.R., Millsom, R.H., 1979. An Australia-wide outbreak of gastroenteritis from oysters caused by Norwalk virus. Med. J. Aust. 2, 329–333.

Murrell, W.G., 1986a. Microbial safety of food in Australia: a critical overview. Part 1. Food Technol. Aust. 38, 320–325.

Murrell, W.G., 1986b. Microbial safety of food in Australia: a critical overview. Part 2. Food Technol. Aust. 38, 366–373.

Oliver, B., Ng, S., Marshall, J., Greenberg, H., Gust, I.D., Creswell, V., Ward, B., Kennett, M., Birch, C., 1985. Prolonged outbreak of Norwalk gastroenteritis in an isolated guest house. Med. J. Aust. 142, 391–395.

Oram-Miles, W., 1999. Imported food inspection in Australia. Food Aust. 51, 286–288.

Palombo, E.A., Bishop, R.F., 1995. Genetic and antigenic characterization of a serotype G6 human rotavirus isolated in Melbourne, Australia. J. Med. Virol. 47, 348–354.

Palombo, E.A., Bishop, R.F., 1996. Annual incidence, serotype distribution, and genetic diversity of human astrovirus isolates from hospitalized children in Melbourne, Australia. J. Clin. Microbiol. 34, 1750–1753.

Palombo, E.A., Bugg, H.C., Masendycz, P.J., Coulson, B.S., Barnes, G.L., Bishop, R.F., 1996. Multiple-gene rotavirus reassortants responsible for an outbreak of gastroenteritis in central and northern Australia. J. Gen. Virol. 77, 1223–1227.

Peters, R.E., 1998. The broader application of HACCP concepts to food quality in Australia. Food Control 9, 83–89.

Peters, R.E., 1999. Developing and implementing HACCP certification in Australia. Food Control 10, 307–309.

Pitson, G.A., Grimwood, K., Coulson, B.S., Oberklaid, F., Hewstone, A.S., Jack, I., Bishop, R.F., Barnes, G.L., 1986. Comparison between children treated at home and those requiring hospital admission for rotavirus and other enteric pathogens associated with acute diarrhea in Melbourne, Australia. J. Clin. Microbiol. 24, 395–399.

Richards, G.P., 1999. Limitations of molecular biological techniques for assessing the virological safety of foods. J. Food Protect. 62, 691–697.

Rodger, S.M., Bishop, R.F., Birch, C., McLean, B., Holmes, I.H., 1981. Molecular epidemiology of human rotaviruses in Melbourne, Australia, from 1973 to 1979, as determined by electrophoresis of genome ribonucleic acid. J. Clin. Microbiol. 13, 272–278.

Scott, J., Sheridan, J., 1994. Review of hepatitis A in Queensland, 1983 to 1993. Comm. Dis. Int. 18, 86–89.

Schnagl, R.D., Holmes, I.H., Mackay-Scollay, E.M., 1978. Coronavirus-like particles in aboriginals and non-aboriginals in Western Australia. Med. J. Aust. 1, 307–309.

Selden, S., Cameron, S., Jarrett, P., Clark, C., 1993. An outbreak of gastroenteritis at a brownie camp. Comm. Dis. Int. 17, 355–359.

Selden, S., Cameron, S., Walker, C., 1994. The epidemiology of hepatitis A in South Australia 1983 to 1993 and implications for national surveillance. Comm. Dis. Int. 18, 138–140.

Stafford, R., Strain, D., Heymer, M., Smith, C., Trent, M., Beard, J., 1997. An outbreak of Norwalk virus gastroenteritis following consumption of oysters. Comm. Dis. Int. 21, 317–320.

Souness, R.A., Fleet, G.H., 1991. Bacterial agents in shellfish depuration. In: Otwell, W.S., Rodrick, G.E., Martin, R.E. (Eds.), Molluscan Shellfish Depuration. CRC Press, Boca Raton, pp. 59–69.

Tallis, G., Veitch, M., Harries, B., 1996. Hepatitis A associated with a child-care centre. Comm. Dis. Int. 20, 116–118.

Taylor, K., Murphy, A., 1994. Norwalk-like virus outbreak in a hostel for the aged. Victoria Comm. Dis. Int. 18, 92.

White, D.O., Fenner, F.J., 1994. Medical Virology, 4th Edition, Academic Press, New York, pp. 306–314, 382–417.

Wilby, R., Ferriera, C., 1995. An outbreak of gastroenteritis at a hostel for the aged and at a private hospital caused by a Norwalk-like virus. Comm. Dis. Int. 19, 117–118.

Wilcox, J., 1999. Ryan vs. Great Lakes Council FCA 177 (5 March 1999). NSW District Registry, Federal Court of Australia.

Wright, P.J., Gunesekere, I.C., Doultree, J.C., Marshall, J.A., 1998. Small round-structured (Norwalk-like) viruses and classical human caliciviruses in southeastern Australia, 1980–1996. J. Med. Virol. 55, 312–320.