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Hepatitis A in New South Wales, Australia, from consumption of oysters: the first reported outbreak

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SUMMARY

Between 22 January and 4 April 1997, 467 hepatitis A cases were reported to the New South Wales Health Department, Australia. To identify the cause of the outbreak, we conducted a matched case-control study, and an environmental investigation. Among 66 cases and 66 postcode-matched controls, there was a strong association between illness and consumption of oysters (adjusted odds ratio 42; 95% confidence interval 5–379). More than two-thirds of cases reported eating oysters, including one third of cases and no controls who reported eating oysters in the Wallis Lake area. A public warning was issued on 14 February, and Wallis Lake oysters were withdrawn from sale. Hepatitis A virus was subsequently identified in oyster samples taken from the lake. Hepatitis A virus poses a special risk to consumers who eat raw oysters because it can survive for long periods in estuaries and cause severe disease.

INTRODUCTION

Hepatitis A is caused by infection with a small non-enveloped RNA virus, hepatitis A virus (HAV). Infection is via the faecal-oral route, except for rare cases of parenteral transmission [1]. Transmission usually follows close (household or sexual [2]) contact with an infected case. However, point source outbreaks linked to consumption of contaminated food from infected food handlers [3], sewage contamination of shellfish [4–8] and contaminated water supplies [9] are well described. Outbreaks linked to swimming pools [10] and contact with sewage [11] have also been reported. After an incubation period of 2–6 weeks, infection commonly causes a 1–2 week illness characterized by fever, malaise, anorexia, nausea and abdominal pain, followed by jaundice [12]. Full recovery is the norm. However, the spectrum of illness varies from inapparent infection [13] to severe prolonged illness and, rarely, death [14]. Recent infection is confirmed by presence of IgM antibody in sera (anti-HAV IgM).

New South Wales (NSW) is an eastern coastal state of Australia with a population of 6·04 million people. Laboratories in NSW are obliged to notify IgM positive cases to regional Public Health Units who in turn report to the NSW Health Department. In NSW over the last 5 years an average of 2–3 notifications of hepatitis A per day were received.

On Monday 10 February 1997 the AIDS/Infectious Diseases Branch of the NSW Health

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Department received reports of four cases of hepatitis A from a Sydney metropolitan Public Health Unit. Three of the four cases had travelled to the mid-north coast of NSW and two reported having consumed oysters there. All Public Health Units in NSW were asked to contact recent cases of hepatitis A, and to obtain a history of travel, food consumption and swimming. That day we learned of 18 cases notified from several different areas of the state. These cases all had symptom onset in either late January or early February, and, of those where information was available, 10 had a history of travel to the mid-north coast, and 9 had a history of oyster consumption.

By the end of the week we had been notified of 150 cases of hepatitis A. Oysters sourced from Wallis Lake, a major oyster growing estuarine lake on the mid-north coast of New South Wales were identified by press release as cause of the epidemic on Friday 14 February. Oyster harvesting ceased and farmers began recalling stock. Wallis Lake was reopened for oyster harvesting on 18 April.

This paper describes the combined epidemiologic, environmental, food and laboratory investigation that lead to the identification of oysters from Wallis Lake as the cause of the epidemic, characterization of the probable source of contamination, and methods used to determine the safety of reopening the lake to oyster harvesting.

METHODS

Epidemiologic investigation

We distributed a questionnaire to all 17 regional Public Health Units in NSW on 11 February. Individuals with serologically confirmed hepatitis A, with date of onset after 21 January, were identified as cases and were interviewed by telephone by Public Health Unit staff. Postcode matched controls for each case were selected from randomly sorted lists of telephone numbers assembled for the postcode of each case entered onto our database up to 14 February. Interviewers selected telephone numbers from the top of these randomly sorted lists and proceeded down the list until a successful telephone interview was obtained with a consenting adult. Only one call attempt was made for each number on the list. Matched controls were questioned on exposures for the period 2–6 weeks before onset of illness in their matched case. All matched controls were interviewed between 1400 and 1900 hours, 14 February. Surveillance for associated cases was continued for 7 weeks until 4 April 1997.

Oyster trace back

Information about oyster purchase location and date obtained from cases was passed on to Public Health Unit Food Inspectors who gathered from retailers invoice details, oyster lease numbers and batch dates corresponding to the date of oyster purchase of each case.

Environmental investigation

The NSW Health Department together with the Great Lakes Shire Council, NSW Fisheries, NSW Environment Protection Authority, NSW Department of Land and Water Resources, NSW Maritime Services Board, NSW Public Works Department and NSW Shellfish Quality Assurance Program conducted a wide ranging food and environmental investigation. All depuration plants on the lake were inspected for compliance with NSW Health Department guidelines. Oyster samples were taken from growing areas throughout the lake, and from a depuration plant post-depuration from 11 February. Additional oyster samples were supplied by the NSW Shellfish Quality Assurance Program. Sediment samples were taken from the lake bed under major oyster growing areas. Rainfall data and other environmental data were collated. A detailed audit of possible sources of sewage contamination both private and commercial was commenced. Human waste disposal units were assessed. Method of effluent disposal, proximity to the water course, evidence of structural defects, and evidence of current pollution were used to assign waste disposal units into four categories: actual polluters, high potential polluters, medium potential polluters, and low potential polluters.

Laboratory investigation

Samples of oysters and sediment were tested for a range of common human enteric viruses, HAV, enteroviruses, adenoviruses, reoviruses and Norwalk viruses, by nucleic acid amplification techniques (PCR or RT–PCR) using published primers and methods as below. Virus was recovered from oysters by allowing oysters under test to depurate in 5 l of sterile sea water for a 24 h period and testing water after ultrafiltration

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and precipitation for viral nucleic material by PCR. These methods are described in detail elsewhere [15]. Oyster flesh (post depuration) was tested by conventional techniques [16,17]. Sediment samples were prepared for PCR testing by mixing with 2 l of carbonate buffer (pH 9·0) and stirred at 40 °C for 4 h and bench centrifuged. The supernatant was treated with PEG 6000 to give a volume of 30 ml and treated with 'genetron'. Samples were then subjected to sucrose gradient ultra centrifugation.

Mass testing of oysters for HAV

A trial was conducted to establish if oysters in Wallis Lake (both pre- and post-depuration) were free of HAV and therefore safe to harvest and market. Approximately 20000 oysters of marketable quality were harvested from six different growing areas on Wallis Lake on 26 March and 4 April 1997. Oysters were placed in two 8000 l depuration tanks. One tank was run with ultraviolet (u.v.) disinfecting process intact and the other without u.v. Samples of oysters (n = 24) were taken from each tank at 0 and 36 h during the depuration process and tested for enteric viruses by nucleic acid amplification techniques as described above. A 1000 l water sample was taken from each tank and concentrated on-site to approx. 5 l using an ultrafiltration unit and ultrafiltration membrane and then processed and tested for viral nucleic material as described above. After draining each tank approx. 100 g of sediment (pseudo faeces) from the base of each tank was sampled and processed as for other sediment samples.

RESULTS

Descriptive epidemiology

There were 467 cases of hepatitis A in NSW with date of symptom onset after 22 January notified up to 4 April. Cases began to increase in the week beginning 20 January and peaked on 3 February, with 34 cases, and declined to background levels in early March (Fig. 1). Sixty-four percent of cases resided in metropolitan Sydney.

We obtained exposure information by questionnaire on 422 (90%) of these cases. These cases were predominantly male (61%), and had a mean age of 35·3 years. Sixty-four percent of these cases reported a history of oyster consumption. A history of oyster consumption was most commonly elicited from those cases during the peak of the epidemic when up to 85% of cases ate oysters (Fig. 1). The most common oyster purchase dates were 31 December and 4 January (Fig. 2). Amongst those cases who reported a single purchase date, the median length of time from oyster purchase to symptom onset was 29 days. The last purchase date recorded was 14 February. This case developed symptoms on 12 March and was notified 19 March. The last case with a history of oyster consumption had onset of illness on 16 March. No further cases with a history of oyster consumption were detected over our period of surveillance to 4 April.

All states in Australia reported additional cases of hepatitis A. Queensland had 138 cases over approximately the same period, 90 with a history of oyster consumption; Victoria 161 cases, 58 with a history of oyster consumption; South Australia reported 8 cases of hepatitis A with a history of oyster consumption; the Australian Capital Territory reported 9 cases with history of oyster consumption; Western Australia reported 3 cases with history of oyster consumption; and both Tasmania and the Northern Territory reported single cases who had consumed oysters interstate. During the epidemic period at least 64 of the 467 cases of hepatitis A in NSW were hospitalized, and there was one death. This death occurred in a 77-year-old male who developed acute liver failure 2 weeks after symptom onset. He had a history of consumption of oysters on the NSW mid-north coast around 31 December.

If all cases with a history of oyster consumption in the relevant period were attributable to consumption of Wallis Lake oysters then this epidemic resulted in 274 reported cases in NSW and 170 in other states, a total of 444.

Case control study

We included 66 cases and their matched controls in the case control study. Differences between cases and controls and odds of exposure are presented in Table 1. Cases were younger than controls (mean age of 35·1 years compared with 50·8 years), more likely to be male, more likely to be Australian-born, and more likely to be from professional and managerial occupational groups. Oysters were consumed by 71% cases compared with 21% controls. The odds of hepatitis A disease were more than seven times higher

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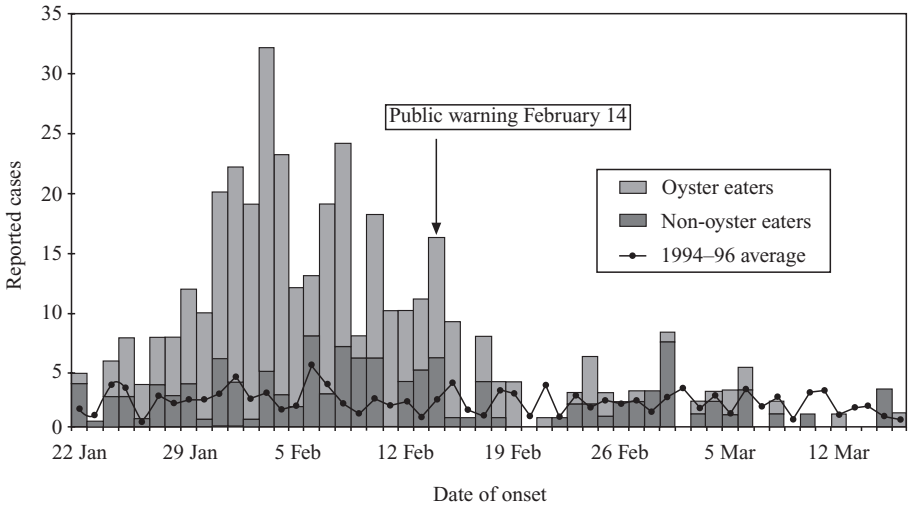

Fig. 1. Reported cases of hepatitis A in New South Wales with onset from 22 January to 16 March 1997 by date of symptom onset and history of oyster consumption. Historical background 1994–6 included.

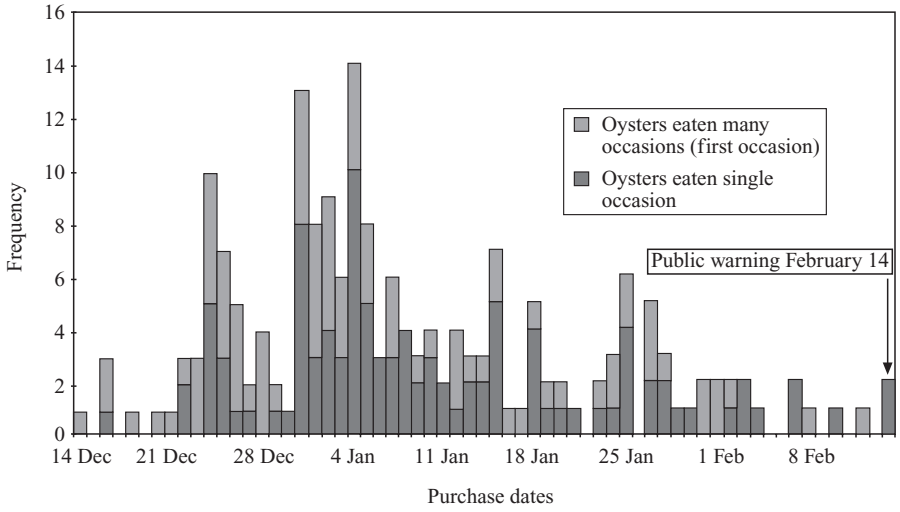

Fig. 2. Frequency of oyster purchase dates nominated by cases of Hepatitis A in New South Wales with symptom onset from 22 January notified up to 4 April 1977.

for oyster consumers compared with non-oyster consumers (unadjusted matched odds ratio 7·6, 95% CI: 3·0–24·7). One third (22/66) of cases consumed oysters in the immediate Wallis lake area; no controls consumed oysters in this area. There was no significant association between illness and consumption of mussels or fish, drinking untreated water, exposure to sewage or exposure to children aged 5 years or less. Weak associations were found between illness and consumption of prawns and illness and swimming.

Case/control status was modelled against demographic variables and possible causal variables using conditional logistic regression methods. The final model included oyster consumption, country of birth and age. The odds ratio for the risk of hepatitis A for oyster consumers was 42·4 (95% CI 4·7–379). In this model age and overseas birth remained significant independent predictors of hepatitis A. Those less than 40 years of age were many times more likely to develop hepatitis A, and overseas birth provided more than 20-fold protection (Table 2).

Trace-back investigation

Oyster supply was traced for 126/274 cases who nominated oyster consumption prior to their illness. Receipts and invoices held by oyster retailers and wholesalers frequently did not itemise the movement of particular oyster batches beyond wholesale level. For 44 cases oysters consumed were traced to

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Table 1. *Frequencies of examined risk factors for hepatitis A in cases and controls*

	Cases ($n = 66$)	Controls ($n = 66$)	Matched OR	95% CI
Demographic variables				
Age < = 40	42/66 (64)	24/66 (36)	3·2	1·5–7·3
Male gender	43/66 (65)	24/65 (37)	3·0	1·4–7·3
Overseas birth	8/66 (12)	22/66 (33)	3·0	0·9–12·8
Occupational group 1 or 2*	15/66 (23)	7/66 (11)	4·5	1·5–18·3
Foods				
Seafood	61/66 (92)	44/66 (67)	5·2	1·8–21·0
Oysters	47/66 (71)	14/66 (21)	7·6	3·0–24·7
Oysters Wallis	22/66 (33)	0/66 (0)	—†	(7·6‡)
Lake area§				
Prawns	42/65 (65)	29/66 (44)	2·6	1·1–6·3
Mussels	10/65 (15)	6/66 (9)	1·8	0·5–6·8
Fish	38/65 (59)	37/66 (56)	1·1	0·5–2·5
Other exposures				
Swimming	38/62 (61)	18/62 (29)	3·7	1·6–10·1
Swimming	16/48 (33)	0/51 (0)	—†	(5·5‡)
Wallis Lake area‖				
Sewage exposure	7/60 (12)	3/62 (5)	2·3	0·5–14·0
Untreated water	7/55 (13)	3/65 (5)	2·3	0·5–14·0
Children¶	7/63 (11)	7/66 (11)	1·2	0·3–5·0

Percentages are given in parentheses.
* ABS occupational groups 1 and 2 (managers and professionals).
† Odds Ratio is infinity or incalculable because of zero denominator.
‡ Maximum likelihood estimate of lower bound of 95% confidence interval for crude association.
§ Oysters purchased at Wallis Lake or in Forster or Tuncurry.
¶ Children 5 years or under living in household.
‖ Wallis Lake, Forster or Tuncurry.

Table 2. *Variables and their odds ratios with 95% confidence intervals in final model.*

Variable	Chi-square (likelihood ratio)	df	*P* value	Odds ratio	95% CI
Oyster	33·0	1	< 0·001	42·4	4·7–379
Australian-born	13·6	1	< 0·001	28·6	2·5–337
Age (all terms)	25·2	3	< 0·001	—	—
< 25 years	—	—	—	85·9	5·0–1467
25–39 years	—	—	—	56·5	2·9–1107
40–45 years	—	—	—	9·5	0·7–123
> 55 years	—	—	—	1·0*	—

* Referent.

depuration plants on Wallis Lake. For another 57 cases the oysters consumed came from suppliers that sourced some (but not all) of their oysters from Wallis Lake. A further 19 cases nominated purchase from locations in towns on the estuary which were unlikely to have oysters sourced from other locations. For six cases oysters consumed were probably from sources other than Wallis Lake. The earliest date of depuration identified for any of the 44 oyster batches traced back to depuration plants was 12 December.

Environmental (site) investigation

Wallis Lake is the largest growing area for Sydney Rock oysters (*Saccostrea commercialis*) in Australia. It is a large, shallow estuarine lake on the mid-north

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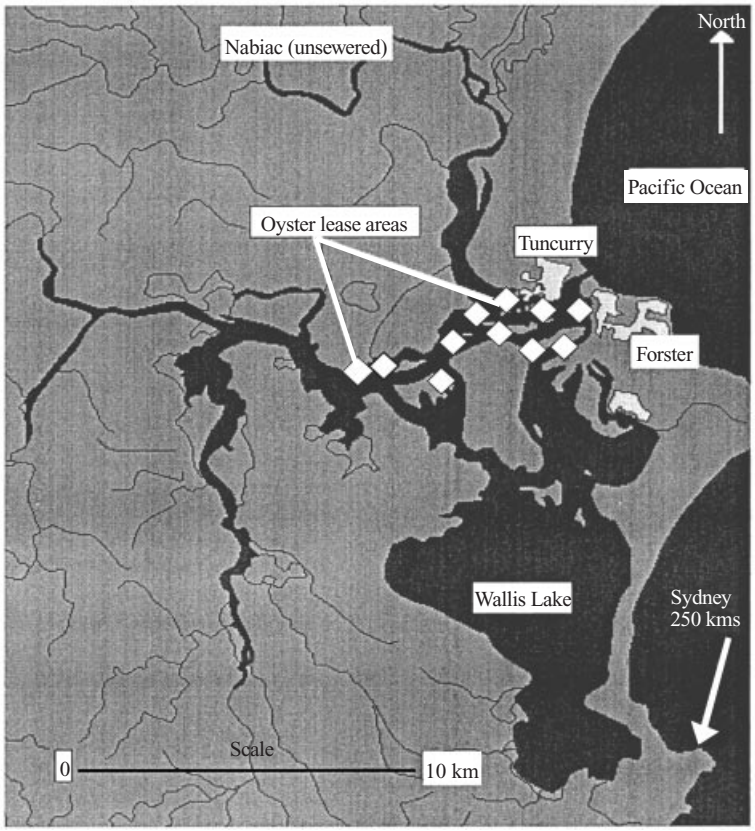

Fig. 3. Map of Wallis Lake showing oyster lease areas.

coast of NSW (Fig. 3). Two rivers, the Wallamba and the Coolongolook feed into the lake. There were 48 aquaculture permit holders on the lake and 32 oyster depuration plants. Approx. 5 million oysters were harvested from Wallis Lake from November 1996 up until the closure on February 14. Nearly all oysters were bagged and sold live to distributors. Some oysters were opened and sold raw for immediate consumption.

There are two towns on the either side of the mouth of the estuary, both sewered. One town discharged treated sewage to sea 7 km south of Wallis Lake, the other discharged into sand percolation beds at the edge of Wallis Lake and the Wallamba River. Test bore samples at the edge of the Wallamba River in proximity to the sand percolation beds failed to detect faecal coliforms. There was an unsewered town with a population of 560 located 20 km up the Wallamba river served by septic tanks. Of the 319 residences inspected, 7 were actually polluting at time of inspection with drainage into storm-water channels, 16 were considered high risk polluters on the basis of major malfunction or fault, 44 medium risk and 252 low risk. Four caravan parks were located on the shores of the lake all of which disposed of effluent by spray irrigation. There were 12 picnic and camping areas in proximity to the lake. some without toilets and others with pit type toilets. Three boats operated on the lake, only two of which had appropriate disinfected holding tanks. About 40 houseboats used the lake none of which had adequate holding and pump-out facilities. Approx. 90 boats visited the lake between November and March with an unknown capacity for appropriate sewage disposal.

Rainfall data for Forster from the Bureau of Meteorology showed 225·8 mm in November 1996, 38·6 mm in December, and 110·4 mm in January 1997. The November rainfall was close to the historical record for that month. Rainfall in the 3 months prior to November had been average. More than half of November's rainfall, 122·4 mm or approx. 4 inches, fell from November 23 to 25. This rainfall was accompanied by anecdotal reports of turbid flows down the Wallamba river after which high *E. coli* counts (30·5 MPN per g) were found by the local oyster farmer quality assurance program in a single sample of oysters taken from near the river mouth on 26 and 28 November. There was voluntary cessation

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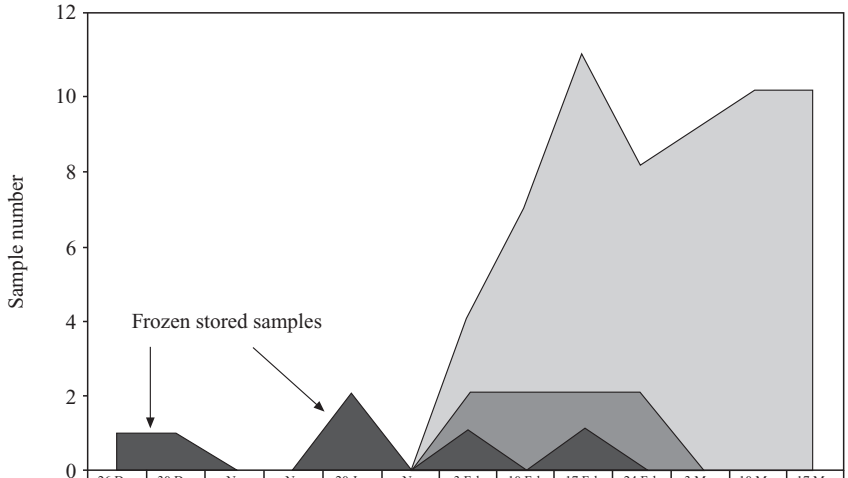

	26 Dec 96	30 Dec 96	No sample	No sample	20 Jan 97	No sample	3 Feb 97	10 Feb 97	17 Feb 97	24 Feb 97	3 Mar 97	10 Mar 97	17 Mar 97
Nil detected	0	0	0	0	0	0	2	5	9	6	9	10	10
Enteric only	0	0	0	0	0	0	1	2	1	2	0	0	0
HAV and Enteric	1	1	0	0	2	0	1	0	1	0	0	0	0

Week of collection

Fig. 4. Detection of HAV and enteric viruses in oyster samples from Wallis Lake by week of collection.

of harvesting by oyster farmers during this rainfall event.

Laboratory investigation

Sixty-three (dozen) samples of oysters and 82 (200 g) sediment samples were sent to the Virology Unit, Department of Veterinary Anatomy and Pathology, the University of Sydney for viral examination. Samples were taken of oysters harvested between 24 December 1996 and 20 March 1997 and returned by retailers and wholesalers in response to the recall. Samples of oyster harvested before 10 February had been frozen and stored [18]. Oyster samples were taken from Wallis Lake approximately weekly after this date. A total of six oyster samples were positive for HAV. The first positive sample was collected on 24 December, and the last collected on 18 February. All these samples were also positive for other viruses. Six samples were positive for other viruses without HAV being detected, the last collected on 27 February. The other viruses detected were enteroviruses, adenoviruses and reoviruses. All oyster samples collected after 24 February were negative for HAV and other enteroviruses (Fig. 4). Sediment samples were collected between 18 February and 4 June 1997. Thirty-one of 82 sediment samples were positive for enteroviruses and a single sediment sample from 15 April was positive for HAV. Sediment samples remained positive for enteroviruses up until final sampling in June.

Mass testing of oysters for HAV and lake reopening

HAV was not able to be detected in the 20000 oysters harvested from representative areas of Wallis lake on 26 March and 4 April. The concentrated water samples from both depuration tanks (u.v. and not u.v. treated) both before and after depuration were all negative for HAV. Similarly HAV was not detected in the samples of sediment or pseudo-faeces from the bottom of the tanks. After the results of these tests became available the lake was reopened to harvesting on 18 April.

DISCUSSION

This is the first reported outbreak of hepatitis A in Australia linked to consumption of oysters. Wallis lake was identified publicly as the source of the epidemic on 14 February on the basis of epidemiologic evidence alone. Our case control study established a clear association between oyster consumption and the epidemic of hepatitis A. The epidemiological evidence identifying Wallis Lake as the source of the oysters was strong: one third of our cases (and no controls) ate oysters purchased at Wallis Lake or from the two towns on either side of the mouth of the estuary. Subsequent trace back, environmental and laboratory evidence, particularly the identification of HAV in oysters samples, provided overwhelming evidence that Wallis Lake was the source of the epidemic.

Our case control study was conducted rapidly. Selection bias may have been introduced by con-

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ducting the survey on the same afternoon with only single attempts made to obtain interviews with most households. For example, controls were more likely to be female and more likely to be elderly than we would expect from a random population sample presumably because of the greater chance of finding females and elderly at home. Adjusting our analysis for sex and age strengthens the estimate of effect for the oyster–hepatitis A association. It is unlikely that other biases introduced at selection (that cannot be adjusted for) would alter the robust association between oyster consumption and hepatitis A. We found no evidence of any other possible cause for this epidemic–weak associations between case status and prawn consumption and case status and swimming were not significant in a multivariate model.

We were not able to identify any cluster of hepatitis A in the Wallis Lake area in the final quarter of 1996 and we were not able to identify any exceptional or major pollution episode affecting Wallis Lake. However, there were 973 cases of hepatitis A notified in NSW in 1996, the highest number of notifications for 5 years. The absence of a local cluster of hepatitis A could well be due to subclinical disease or disease in visitors to the area residing elsewhere. Despite not identifying a major pollution event or major source, we identified multiple possible sources of sewage or faecal pollution in our environmental audit.

Morbidity was high in this epidemic with life-threatening illness and one death. Other food-borne outbreaks of hepatitis A in developed countries affecting adult groups have also been associated with high mortality [19], a mortality similar to that found in hospital series [20]. This highlights the changing epidemiology of hepatitis A that has occurred with improved sanitation [21–23]. As hepatitis A becomes an uncommon disease in childhood because of improved sanitation, the risk of epidemic and severe disease may increase because of the larger pool of susceptible adults. This is particularly relevant in epidemics of food-borne disease which often affect adult groups.

The timing of events in this outbreak strongly points to contamination of oysters occurring in or just before early December. Sydney Rock oysters (*Saccostrea commercialis*) are able too survive live in the sack for up to 6 weeks. After harvest they are required to be depurated for 36 h. After being sold to distributors they can be stored live, shelled and delivered on demand. Time from harvest to consumption depends on demand, but is typically 10 days. As Figure 2 demonstrates, the bulk of the epidemic (which, by symptom onset peaked on 3 February) seems due to consumption of oysters over the Christmas/New Year period. The first date of oyster purchase, where there was no other competing explanation for hepatitis A and a single purchase date, was 16 December 1996. Despite limited trace back of implicated oyster batches because of mixing of stock at the retail level and uncertainties over relevant purchase details, the earliest date of depuration of implicated oysters is 12 December 1996, supporting first harvest of contaminated oysters in early December.

The precise cause of contamination of oysters in Wallis Lake with HAV remains unknown. However, two pieces of evidence suggest a well dispersed pollution source or event resulting in almost lake-wide contamination with HAV. First, multiple oyster lease areas were implicated in this epidemic. Implicated leases were located at the mouth of the Wallamba river, near the central island, and towards the mouth of the estuary. Secondly, the recovery of a wide range of human enteric viruses in a large number of oyster and sediment samples suggests significant sewage or faecal contamination in the lower part of the lake.

The high rainfall in late November, both because of its timing and its capacity to widely disperse a pollution source, may have been a key event. The rainfall resulted, according to local reports, in a highly turbid flow of water down the Wallamba river and high *E. coli* counts in oyster meat. This rainfall and accompanying turbid flow plausibly resulted in transportation of virus adsorbed particles down the river and lead directly to oyster contamination just before time of harvest in December.

The distribution of purchase dates also suggests that contaminated product was on the market for an extended period; the last date of oyster purchase recorded was 14 February 1997 – the day of our public warning, and HAV was found in an oyster sample taken from the lake as late as 18 February. The detection of HAV in oyster samples more than 2 months after the presumed contamination event was surprising.

Bivalves certainly can concentrate and retain virus [24]. Oysters have been demonstrated to concentrate enteric viruses such as poliovirus up to 60-fold [25]. Uptake of poliovirus to a maximum generally occurs rapidly over a few hours under feeding conditions [26, 27]. Elimination of poliovirus occurs over a longer

period of 24–120 h. Hepatitis A is less studied. However, virus may be taken up to a greater extent and be eliminated more slowly. Sobsey and colleagues found that poliovirus was eliminated efficiently from the Eastern Oyster within 3 days, but HAV was eliminated relatively slowly with as much as 18% virus persistence at 5 days [27]. In other experiments on mussels HAV was accumulated 100-fold and persisted for 7 days [28]. In field conditions the period of time that oysters may remain contaminated with HAV and able to transmit disease has not been well characterized. Portnoy and colleagues describe an epidemic of oyster-borne hepatitis A in the southern USA in 1988 after flooding on the Mississippi River. Oysters were harvested at least 4 weeks after coliform counts had fallen to acceptable levels [4].

Nevertheless, a 2-month period of contamination of oysters without elimination is long. The best explanation for the prolonged period of contamination evidenced in this outbreak may be recontamination of oysters from sediment. HAV can survive a long time in the environment. In experimentally seeded groundwater, effluent, seawater and estuarine water exceeds that of poliovirus and frequently exceeds 1 month [27]. It can also be adsorbed onto small diameter particles which can be resuspended by storm and other turbulence [24].

This long period of apparent oyster consumption made it difficult to decide when oysters from the lake would again be safe to harvest. This was especially so as sediment samples remained positive for enteric viruses. Novel methods developed to test large and representative numbers of oysters in bulk for HAV and other viruses were important in contributing to the decision to reopen the lake to harvesting on 18 April.

In NSW depuration based on ultra-violet light technology is the primary method relied upon to reduce the risks associated with eating raw oysters. Oysters are allowed to filter feed in optimal conditions of salinity and temperature for at least 36 h using estuary water subjected to ultra-violet disinfection. This is the only mandatory control. In New South Wales it is illegal to sell oysters sourced from New South Wales that are not depurated, and depuration plants require annual licencing. Other safeguards are industry based and typically involve cessation of harvesting after rainfall events and testing of oyster meat for indicator organisms. A water classification system was generally not used to restrict harvest from potentially hazardous estuaries/waters.

The above measures to ensure safe product were inadequate to eliminate the risk of hepatitis A. The apparent long environmental survival of HAV and/or the failure of the oyster to clear this virus is a problem without clear technical solution.

Whilst hepatitis A remains a prevalent condition in our community, the only way that future outbreaks from eating uncooked oysters grown in our estuaries can be avoided is by minimizing pollution of our estuaries. New legislation has recently been introduced in NSW to tighten controls over maintenance of human waste disposal units (septics), a programme of connecting small villages to sewers has been reinvigorated, funding has been provided for councils to develop sewerage management plans, and a system of mandatory notification to the NSW Health Department of sewerage overflows introduced. In addition membership of industry based Shellfish Quality Assurance Programs has become mandatory, and a system of classification of estuaries introduced that restricts harvest in some of them.

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