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The public health response to a case of brucellosis in Auckland

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The Auckland Regional Public Health Service investigated a blood culture confirmed case of brucellosis due to *Brucella suis* in March 2002. The case was a 43 year old male from South Auckland. The epidemiological evidence suggests that infection was locally acquired. Given that *B suis* was the pathogen, two pigs prepared for consumption by the case were a plausible source of infection. The case's exposure to the pigs' blood and body fluids via aerosols and a finger cut would have provided sufficient opportunity for infection. A total of 86 contacts were identified, counselled and offered serological testing for brucellosis. No associated cases were found. The Ministry of Agriculture and Forestry screened pigs on 42 farms. No infected animals were found. While confirmed cases of sporadic brucellosis are rare in New Zealand, the implications for both the public's health and the economy are important and necessitate a timely and thorough investigation.

Brucellosis, a disease caused by the gram-negative bacterium, is of both public health and economic significance. Only *B abortus* and *B ovis* have ever been identified in New Zealand.¹ In many developed countries, including New Zealand, *B abortus* has been controlled or eradicated by measures that have eliminated the bacteria from infected cattle herds. *Brucella* spp. of public health significance are those capable of zoonotic transmission: *B abortus*, *B melitensis*, *B canis*, and *B suis*. The animal reservoirs for *Brucella* spp. are extensive, but preferred hosts exist for each species, ie cattle for *B abortus*, goats and sheep for *B melitensis*, pigs for *B suis* and dogs for *B canis*. Transmission of brucellosis occurs through direct contact with infected animals and animal carcasses, inhalation of aerosolised organisms in the laboratory or abattoir and via the consumption of contaminated and untreated milk products. Rare instances of person-to-person transmission have been recorded.²

Illness in humans is typically severe, occurring from one to four weeks after exposure, however in some cases several months may elapse. The clinical presentation is non-specific with flu-like symptoms including fever, malaise, anorexia, headaches, myalgia and back pain. An undulant fever may occur. Sequelae include osteoarticular complications (osteomyelitis and septic arthritis), depression, endocarditis, orchitis, and epididymitis. If untreated, brucellosis can be fatal.²

The combination of clinical symptoms and a four-fold increase in serum *Brucella* antibody titres or direct isolation of *Brucella* from the infected person confirm the diagnosis.³ Antibiotic treatment of brucellosis is the mainstay of treatment and is usually prolonged.

Methods

Case investigation: The Disease Control Team (Auckland District Health Board) was notified of a case of brucellosis on March 6th 2002 in a 43-year-old male from South Auckland. *B suis* had been isolated from blood

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cultures. Public health staff interviewed the case and ascertained the most likely source of infection.

Identification of the source farm: The case's sister, accompanied by a public health nurse, identified the farm supplying the pigs. Contact was then made with the farmer who was symptom free. He agreed to undergo serological testing for *Brucella* infection and to provide information to the Ministry of Agriculture and Forestry (MAF) so that they could undertake a trace back to potentially infected herds.

Contact tracing: Follow-up was conducted on those present at the two family feasts. Information was given about why the investigation was underway, the symptoms of brucellosis and what to do if they occurred. All contacts were offered serological testing. The public health service revisited all people who subsequently tested positive on any of the three serological tests and arranged for repeat tests 2-4 weeks later.

Farm tracing: MAF conducted a traceback investigation of pigs sold to the implicated farm around the time that the case had made his purchase. This investigation identified the primary source farms of potentially infected pigs. MAF supplied the addresses of these farms to public health authorities so that human follow-up could be arranged. Farms outside

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the Auckland region were contacted by their regional public health service. Farmers were counselled about the illness, written information on the disease was provided to people in contact with the pigs on these farms and serological testing was offered.

Potentially exposed laboratory staff: Staff at the Middlemore hospital Microbiology laboratory who worked with the blood culture isolate outside a biosafety cabinet were also counselled and underwent serological testing.

Results

Case Investigation: From the interview with the case it was revealed that the most likely risk factor for infection was the preparation and consumption of two homekill pigs. There had been no exposure to unpasteurised foods or imported meat products, no recent overseas travel and while the case was not born in New Zealand, there was no past history of illness consistent with brucellosis.

In December 2001, the case had purchased two pigs for family social events. These were slaughtered in a field of the supplier's farm (the farmer having purchased them at a local sale). The carcasses were taken home and with the assistance of two other family members, dressed (gutted and the meat prepared for cooking). The case recalled that the day before dressing the pigs, he had cut his finger with a knife and a small open wound remained. A sticking plaster had been used to cover the cut but this subsequently fell off. No other personal protective equipment was worn by any of the individuals involved in dressing. The case was the main butcher and was the only one likely to be exposed to splashes of blood and body fluids from the pigs.

After preparation the dressed meat was frozen and the offal buried in the back yard. The meat was consumed at two family events that occurred at Christmas and New Year. For these events the same three people as well as the case's sister and one other relative prepared the food. This was cooked in a traditional hangi. The pork was reportedly well cooked, the juices were clear and when consumed, the meat fell off the bone. No raw or cooked meat remained from either pig after the social events.

Source farmer: The farmer was symptom free and tested negative for Brucella on serological screening. The farmer concerned had for many years sold pigs from his farm. In earlier years he had reared the pigs, however more recently he had purchased pigs from the local stock sales and then on-sold them privately.

Contact finding results: There were 26 people at the first and second family feast including those who participated in the preparation of the pork. At the second family feast, an additional four people ate hangi food. Contact was made with all 30 people associated with the two meals. Four people (including the case) reacted positively to one or more of the screening tests (Table 1). One person (in addition to the case) had symptoms consistent with brucellosis including recurrent fevers and flu-like symptoms and was immediately referred for medical assessment. Investigations excluded brucellosis.

Of the three contacts undergoing repeat serological tests, one displayed rising titres on both Coombs antiglobulin test (1:80 to 1:320) and serum agglutination test (SAT) (1:80 to 1:160). This contact was also referred for assessment. Blood cultures were negative. Repeat tests including blood cultures were performed. These were subsequently negative. Antibiotic treatment was not deemed necessary by the treating infectious disease physician, as the contact had remained symptom free and had only minimal exposure to the suspected infected meat.

Table 1. Clinical and serological investigation of potential human brucellosis cases

Characteristic	Contact Type			
	Family Contacts	Farm Contacts	Laboratory Contacts	All Contacts
Number contacts (n)	30 ¹	49 ²	7	86
Proportion counselled (%)	30 (100)	49 (100)	7 (100)	86 (100)
Number (%) Symptomatic	1 (3.3)	0	0 (0)	2 (2.3)
Response rate for serology (%)	29 (96.7)	22 (44.9)	7 (100)	58 (67.4)
Positive Brucella Screen (rose bengal) (%)	4 (13.3)	0	0	4 (4.7)
Positive Coombs ³ (%)	4 (13.3)	1 (5.5)	0	5 (5.8)
Positive SAT ³	2 (6.7%)	0	0	2 (2.3%)
Greater than 4-fold rise in titre ⁴	1 (3.3%)	0	0	1 (1.2%)

¹ does not include confirmed case

² from 22 farms

³ titre of >1:40

⁴ for either Coombs or SAT

Human contacts from potential source farms: A total of 22 farms were implicated. Six of these farms were outside the Auckland region (three in Waikato and one each in Taranaki, Wellington, and Southland health districts). One clinically well person returned a positive Coombs test. This person was advised to undergo repeat serology.

Farm tracing: MAF performed serological testing on 285 animals from 42 farms.⁴ All pigs tested were serologically negative.

Laboratory contacts: No symptoms developed in the 7 laboratory staff and they all remained *Brucella* antibody negative.

Discussion

No cases of Brucellosis have been diagnosed in New Zealand cattle since 1989 and New Zealand has for some time claimed *Brucella*-free status in its cattle and pigs.⁵⁻⁸ Brucellosis is a notifiable disease in New Zealand. In humans there has been no confirmed transmission of *B abortus* in New Zealand over the last decade. A review of notified cases from 1993 to 1997 showed that they either did not meet the definition for a recent infection or that infection had been acquired overseas.^{9, 10} In New Zealand there has never been a case of locally acquired *B suis* infection in pigs or humans. The only previous human infection with *B suis* in New Zealand was attributed to infection acquired in the Pacific Islands where the disease is known to be endemic.^{11, 12} Our case was confirmed to be infected with *B suis* biovar 3 by the Veterinary reference laboratory in Weybridge, United Kingdom.

Interviews with the patient revealed that he had no known risk factors for *B suis* infection other than his exposure to the 'home killed' pig meat. However, other possibilities for the route of infection should be hypothesised, as the source was not confirmed. Such possibilities include chronic brucellosis infection obtained overseas, though the case's past medical history was negative and the case had not travelled outside New Zealand in over a decade; the consumption of infected meat following illegal importation, although the case denied this; or even infection by some hitherto unrecognised animal reservoir such as rodents.¹³

The public health response to this case was rapid and involved continuing interaction between a number of organisations including, hospitals, laboratories, public health services, and the Ministries of Health and Agriculture and Forestry. Due to the need for close collaboration and the timely transfer of information, weekly teleconference calls were held between stakeholders.

The high response rate for the serological testing of the family members potentially exposed to brucellosis (97%) was not replicated among farmers (45%). This was because in many cases MAF field workers had visited the farm, tested pigs and advised farmers of the negative serology prior to the arrival of public health staff. Farmers inferred that if their pigs were not currently infected then they would not be either. Despite efforts to explain to farmers that there still was a small potential risk of infection, many decided against serology. However, assurances were gained that if any farmers or contacts of their stock did become symptomatic then they would immediately seek medical advice. In any similar investigation in future, joint visits by MAF and public health services might be considered.

The emergence of *B suis* in New Zealand's pig stock could potentially jeopardise New Zealand's agriculture trade markets. New Zealand currently declares freedom from the disease to the Office International des Epizooties and in MAF export certification for a wide range of animal products (meat, live pigs and pig semen). If *B suis* is confirmed in New Zealand livestock then trade ramifications may include the suspension of export of live pigs, pig semen/ova and embryos and the suspension of export certification for pig meat products.

At present there is evidence that the commercial sector of the pig industry is free of the disease. The evidence includes: no cases reported in New Zealand of reproductive failure in sows accompanied by locomotor disorders and orchitis in boars; microbiological surveillance of fifty porcine abortions undertaken from 1992 to 1995;⁸ export testing of pigs for brucellosis; and no reported human infections, in veterinarians, pig farms or abattoir workers. In view of this evidence, New Zealand continues to be considered from a trade perspective to be brucellosis free.

A technical advisory group has been established to review the response to the human case of brucellosis. At present this group is deliberating on what further actions may be necessary particularly in relation to enhanced animal surveillance for brucellosis in pig herds, both commercially farmed and feral. This case raises the possibility that the disease occurs in low prevalence in the small-scale pig production sector of the industry.

Although brucellosis is a rare disease in New Zealand this case should raise awareness among GPs and hospital physicians that the infection

should be considered in the differential diagnosis of patients with undulant fever. This report highlights the significant amount of public health resource devoted to the investigation of a confirmed sporadic case of brucellosis. The epidemiological evidence suggests that infection was locally acquired. Given that *Brucella suis* was the pathogen, the two pigs were a plausible source of infection. The degree and nature of the case's exposure to these pigs, via aerosols and a finger cut during dressing, provided sufficient opportunity for infection. However, despite thorough public health and veterinary investigation the source of the case's illness could not be confirmed and no associated cases were identified.

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Trichinellosis and homekill pork

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Trichinellosis is a zoonotic disease resulting from ingestion of insufficiently cooked meat, usually pork, containing *Trichinella* spp. larvae. Notification data in New Zealand suggest that the disease is very uncommon, with no locally acquired cases notified prior to 2001. Two suspected cases of trichinellosis were notified to the Medical Officer of Health, Waikato District Health Board, in September 2001. Both were farmers, living in the same geographical area, and had consumed meat from the same pig, butchered under licensed homekill conditions. Testing of remaining frozen meat from the pig confirmed the presence of *T spiralis* cysts. Investigation of the source farm revealed poor animal husbandry practices and trichinellosis infection among approximately 50% of pigs on the farm. Trichinellosis should be considered in patients presenting with fever, myalgia, and periorbital oedema with recent history of consuming non commercial pork or poorly cooked meat, and is definitively diagnosed by demonstration of *Trichinella* spp. larvae within striated muscle. Prompt notification of suspected cases to the Medical Officer of Health and the Ministry of Agriculture and Forestry allows timely investigation and appropriate public and animal health action.

Trichinellosis (also known as trichinosis) is a disease caused by *Trichinella spiralis*, an intestinal roundworm whose larvae migrate to and become encapsulated in muscle.¹ Clinical illness in humans is highly variable, ranging from asymptomatic infection to fatal disease. Clinical symptoms of trichinellosis parallel the development stages of the organism in the human host, and are grouped into intestinal, visceral and convalescent phases.² The intestinal phase corresponds to the period of larval penetration of intestinal mucosa and generally begins within seven days of ingestion or infected food. Symptoms during this phase frequently include malaise, nausea, vomiting, epigastric pain, diarrhoea, constipation, and low grade fever.

Visceral involvement manifests itself as early as the second week or as late as the eighth week of illness and represents newborn larval dissemination to striated muscle and other organs. Symptoms include headache, fever, chills, cough, muscle pain, and tenderness, and characteristic signs include periorbital or facial oedema, conjunctivitis, subconjunctival haemorrhages, peripheral oedema, and a variety of non-specific skin rashes. Major neurological symptoms occur in up to a quarter of serious cases and may include deafness, seizures and focal motor deficits. Death may occur due to myocardial failure. The convalescent phase typically occurs after three or four weeks and is characterised by a reduction in fever and muscle pain. Recovery is

usually complete within a few months, although fatigue, weakness and diarrhoea can persist for many months. Serologic tests and marked eosinophilia may aid diagnosis, although biopsy of skeletal muscle taken more than ten days after infection provides conclusive evidence of infection by demonstrating the uncalcified parasitic cyst.¹

Trichinellosis has made a dramatic re-emergence in many areas around the world over the past two decades, in spite of a century of veterinary and public health control and eradication efforts. The re-emergence of trichinellosis is due to human manipulation of ecosystems, war and political turmoil, rapidly changing food distribution and marketing systems, and rising affluence in developing countries.³ The reported incidence of trichinellosis is likely to be an underestimate of the actual burden of disease, as the wide range of symptoms makes diagnosis difficult in low incidence countries in non-outbreak situations,⁴ and many infections may remain asymptomatic or mildly symptomatic.

Trichinellosis was made a notifiable disease in New Zealand in 1988. The sole statutory notification of trichinellosis prior to 2001 was a 1992 case, confirmed by muscle biopsy due to *T pseudospiralis*, related to transmission thought to have occurred in Tasmania.⁵ This report describes the first notified, locally acquired human cases of trichinellosis in New Zealand, and the public and animal health investigations undertaken to determine the source and prevent further cases.

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Methods

Investigation of the cases commenced after notification to the district Medical Officer of Health and the Ministry of Agriculture and Forestry (MAF). The cases and their family members were carefully questioned about consumption of potentially contaminated meat. All persons who had consumed the implicated product were traced and evaluated for signs and symptoms of illness. The butcher who had processed the meat was interviewed to establish the extent of product distribution. Samples of leftover frozen product were collected and tested, as described below.

The farm source for the implicated product was traced and investigated thoroughly by MAF staff and contractors. A site visit was conducted to assess the environment of this index farm, and to identify other potentially infected animals. Serum was collected from all pigs on the farm testing for the presence of antibodies to *Trichinella* antigens. Regardless of test results, the pigs on the farm were then destroyed and post mortem muscle samples were collected from diaphragm pillars, the site likely to have the highest concentration of larvae in infected animals. The local spread of infection from the farm was investigated by trapping feral animals in the vicinity, including at a closed refuse disposal site in the same general area as the farm. Muscle samples collected from trapped animals were also sent for testing.

Trace forward and trace back investigations of pig movements to and from the index farm were undertaken to identify other 'at risk' properties, focusing on the six month period prior to notification of the human cases. Serum for antibody testing was collected from all adult pigs on farms identified in this way. Pigs with positive test results were killed and muscle specimens collected. This testing regimen followed protocols prescribed by the World Organization for Animal Health.⁶ Serum specimen collection to detect late seroconversion were repeated three to four weeks later on pigs who had left the index farm.

Samples collected from leftover product, destroyed farm animals and trapped wildlife were tested using pepsin digest tests⁸ by AgriQuality Ruakura Animal Health Laboratory to detect larvae. Pepsin digest, using a 1g muscle sample, will detect *Trichinella* larvae at concentrations greater than three larvae per gram. DNA sequencing was performed by an international reference laboratory to speciate larvae detected by pepsin digest. Enzyme-linked immunosorbent assay (ELISA) tests were performed on serum specimens to identify antibodies to *Trichinella* antigens. The results of pepsin digest and ELISA tests for all pigs tested on the index farm were compared using the Kappa test for correlation.⁷

Results

Clinical details of the cases: On 31 August 2001, a previously healthy 41-year-old woman (Case 1), an office worker and dairy farmer, presented with a 24 hour history of fever, headache and vomiting. The provisional diagnosis of leptospirosis was made and she was hospitalised and commenced on penicillin and ceftriaxone. Despite antibiotics she remained pyrexial with drenching night sweats, presenting a clinical picture of septicaemia. On 2 September she was noted to have periorbital oedema.

The eosinophil count was normal on admission (0.19, 2%) but began to rise at the time when periorbital oedema was first noted. The eosinophil count peaked at 1.89 (20%) on her tenth hospital day, and subsided over the subsequent weeks. Apart from a transiently low lymphocyte count, haematology remained otherwise normal. The patient's liver enzymes rose steadily from admission, with serum alkaline phosphatase peaking at 206 on day eight. Her creatine kinase (CK) was not checked until the diagnosis of trichinellosis was suspected on day six, when it was 1119u/l, although myalgia was not clinically prominent. She was discharged, afebrile, on the twelfth hospital day.

A previously well 27-year-old male pig farmer (Case 2), a neighbour of Case 1, presented with a similar illness on 4 September 2001. He also remained febrile, with drenching night sweats, and was distressed by headache, vomiting, and increasingly painful legs. Case 2 was also provisionally diagnosed with leptospirosis, and was treated with doxycycline. On his third hospital day he developed diarrhoea, and an itchy macular rash on his back which later became painful. Over the next few days he experienced muscle cramps involving his legs, although had a history of a deep venous thrombosis following calf trauma four years earlier. His fever eventually subsided and he was discharged on the eighth hospital day.

This patient also had a marked eosinophilia, rising from 1.32 (18.9%)

at presentation, to 2.88 (33%) at discharge on day eight. His CK was also elevated (maximum CK level 349 on day seven) but did not reach the high levels of Case 1, despite his muscle symptoms being more prominent. His liver enzymes remained normal.

The severity of the headache, fever and systemic upset, and the persistence of the symptoms in both cases were consistent with leptospirosis, which remains common in Waikato District Health Board. Although the cases lived on neighbouring properties, Case 2 worked some distance from his home, and the nature of their farming activities were different. In particular, Case 1's farming activities were limited as she had employment away from the family farm. They knew of each other's illness, and themselves identified consumption of home killed pork as the only common factor. Without this history, the diagnosis of trichinellosis might not have been considered. Muscle biopsy was considered, but was not undertaken because neither had evidence of acute inflammation of a specific muscle group, and also because of the severity of their systemic upset at the time. Neither patient received antihelminthic treatment. Case 1 subsequently had serological confirmation of trichinellosis, while Case 2's serology remained negative. At review both patients were improving steadily but convalescence was protracted. Both cases were notified to the Medical Officer of Health on the basis of reasonable clinical suspicion, as required by the Health Act 1956.

Case investigation: Both cases reported that they had recently consumed pork from a domestic pig raised in a small non-commercial pig farm managed by Case 2, and that the pork they had consumed had been well cooked. Neither had prepared meat in a manner that could have contaminated other food. Both cases reported eating much less of the pork than other members of their households, who remained asymptomatic. Pepsin digest testing of remaining samples of frozen pork found within the freezers of both cases was positive for parasite cysts, confirmed as *T spiralis* through DNA sequencing.⁸

The pig had been butchered and processed by a local butcher, registered as a homekill service provider under the Animal Products Act 1999. The butchered meat was therefore not a regulated animal product. This meat had been divided up between the families of the two cases. Another family who received one package of bacon did not report illness consistent with trichinellosis, although two of the children had a diarrhoeal illness shortly after eating well-cooked bacon. This was confirmed as cryptosporidiosis in the one child who was tested. There was no evidence that infected meat had been distributed commercially.

Investigation of index farm: The index farm was declared a Restricted Place under the Biosecurity Act 1993. Until revoked, this declaration prevents any further movement of meat or animals from a farm. At the time of investigation, the index farm was running five captured wild pigs, 50 domestic pigs of mixed ages, two horses, and approximately 100 cattle and 100 sheep. The wild pigs had been added to the herd following live capture on the adjoining Department of Conservation estate. It was noted that the housing and fencing for farmed pigs were in disrepair, allowing pigs to roam free. Pigs were fed uncooked off-cut meat from other butchered pigs. The index farm was near a refuse recycling station, at the site of a closed sanitary landfill. The state of fencing, general housekeeping and hygiene practices observed on the farm in question had attracted rodents, feral cats and feral pigs.

Of the 55 pigs present on the index farm, samples were collected from 31 (all adult pigs) for testing. Of these 14 were positive by ELISA, and 16 were positive by pepsin digest. In total, 55% (17/31) of the pigs tested were infected with *Trichinella* spp. The results of testing are set out in Table 1. The kappa statistic for test comparison was 0.74, and significantly different from zero, showing a good correlation between the two tests for disease status.

Table 1: ELISA and pepsin digest test results on the 31 pigs tested from the index farm

ELISA test result	Pepsin digest test result		
	Negative	Positive	Total
Negative	14	3	17
Positive	1	13	14
Total		15	16

Kappa coefficient = 0.74, 95% confidence interval 0.51-0.98

Wildlife trapping was undertaken for one night only on the index farm and immediate vicinity, including the refuse station. Five feral cats, 22 rats, nine possums and one hedgehog were trapped over this period. Of these, four cats (80%) and seven rats (32%) had positive pepsin digest tests. Full results of pepsin digest tests on trapped animals are shown in Table 2.

Table 2: Results of pepsin digest testing of animals trapped in the vicinity of the index farm

Animals	Pepsin digest test		Total
	Negative	Positive	
Feral cats	4	1	5
Rats	7	15	22
Possums	0	9	9
Hedgehog	0	1	1

Trace back and trace forward: Four farms were identified as sources of live pigs for the index farm. Two of these farms no longer had pigs. The remaining two farms held nine pigs, all of whom tested negative by ELISA. Three farms were identified as being destinations for live pigs from the index farm, and of these farms two still had live pigs at the time of the investigation. On the first of these farms, four pigs that had been moved from the index farm in May 2001 were tested with ELISA and found to be negative, both on initial testing and subsequent re-testing. On the second farm, four pigs that had been moved from the index farm in September 2001 were negative on initial ELISA testing. One of these four was positive on re-testing on 29 October 2001. This pig was presumed to have been infected while in the index farm, and was destroyed. Frozen pig meat samples were collected from the destination farm without live pigs, were tested by pepsin digest and found to be negative. The owner of the index farm informed MAF that he would not farm pigs in future. MAF recommended that the district council control vermin at the refuse dump.

Discussion

This report describes two cases of trichinellosis that appear to have resulted from consuming home-killed pork. Although the results of the investigation provide no definitive evidence of the sequence of events in this outbreak, it is likely that infection cycling in wildlife (for example, feral pigs, rats and cats) was introduced into the index farm through poor on-farm biosecurity, and the prevalence of infection among the farm animals was then amplified through feeding practices. Infection of the human cases must then have resulted from consumption of insufficiently cooked infected meat, despite the history of thorough cooking in both households.

Muscle biopsy testing of the human cases was not undertaken to confirm the diagnosis of trichinellosis. The classical clinical presentation of the cases and the finding of *T spiralis* cysts in samples of remaining meat, from a pig that both had consumed, provides strong corroborative evidence for the diagnosis. Although muscle biopsy testing is an expensive and invasive test, overseas experience suggests that it is highly specific and useful in cases where there is no epidemiological link.

Prompt notification of the illness on suspicion to the Medical Officer of Health and subsequent notification of MAF allowed immediate initiation of investigation of the remaining frozen meat, and of the live animals on the implicated property. Serology results were not available for some months, but the confirmation of *Trichinella* larvae in the meat allowed reasonable confirmation of the diagnosis in the humans, and rapid intervention to limit further risk to both humans and animals.

Investigations on the index farm revealed poor animal husbandry practices, including poor isolation of domestic from feral animals, and feeding of uncooked pork offcuts to pigs. High levels of infection in pigs on the index farm were demonstrated by pepsin digest and serum testing. Further, there was evidence of an informal system of meat distribution that had exposed others to the hazard. The extent of poor animal husbandry and illegal distribution of homekill meat in the production of non-commercial pork in New Zealand is known to be common but difficult to quantify.

Trichinella infection in domestic and feral pigs slaughtered at licensed meat premises produced within New Zealand is monitored through an ongoing surveillance system operated by the MAF Food Assurance Authority.⁹ No infections have been identified through this system. Infection among wildlife is monitored through active surveillance around properties where pig infections have been diagnosed, and data from this programme suggests that a cycle of *Trichinella* transmission exists in New Zealand involving feral pigs, cats and rats.^{10, 11} Occasional transmission of infection to pigs reared in non-commercial backyard operations has been demonstrated, posing a risk to persons dressing or consuming homekill or feral pig meat. Capture and consumption of wild pig is common and may expose many people to the risk of *Trichinella* infection.

The risk of trichinellosis can be reduced by improvements in animal husbandry of pigs, and by taking precautions in preparing meat potentially infected with *Trichinella*. Pigs should be prevented from eating uncooked meat, including uncooked kitchen waste, and should be restricted from contact with rodents and other wildlife that may carry *Trichinella*.¹² Hunters should ensure that offal and unwanted meat is not eaten by other pigs or livestock. People who dress or consume wild pork should be informed of the risks and the importance of thorough cooking to render it safe and careful preparation to prevent cross contamination.

The following processes are recommended by the International Commission on Trichinellosis (ICT) to render pig meat safe for consumption.¹¹

- **Cooking:** *Trichinella* in meat is inactivated if the meat is cooked thoroughly. A change in colour of the meat from pink to grey throughout and a change in the texture such that muscle fibres are easily separated from each other indicate that meat has been rendered safe for eating.
- **Freezing:** *Trichinella* is inactivated in frozen meat. Cuts of meat up to 15cm in thickness should be frozen solid (to at least -15°C) for no less than three weeks, and cuts of meat up to 69cm in thickness should be frozen solid (to at least -15°C) for no less than 4 weeks. These temperatures are unlikely to be attained in a domestic freezer.
- **Irradiation:** *Trichinella* is also inactivated by irradiation. However, this process is not available in New Zealand.

Curing or smoking is not recommended for killing *Trichinella* cysts in pork or game meats. Curing should only be used after extensive validation and with strict process controls. The ICT recommends that only inspected meats certified *Trichinella*-free should be used in the preparation of cured or smoked products. The ICT does not recommend the use of microwave ovens for cooking potentially infected meat.

The symptoms of trichinellosis are often non-specific and may be overlooked if there is a low parasite load, few symptoms and a lack of awareness of the disease. Trichinellosis should be considered in any patient presenting with a combination of fever, myalgia, periorbital oedema, and eosinophilia, and with a history of recent consumption of poorly cooked wild or domestic pork. Suspected cases of disease should be notified to the Medical Officer of Health and to MAF.

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Surveillance data

National surveillance data - April to June 2002

Disease ^{1,2}	Current year - 2002 ³			Previous year - 2001			Trends - April to June 2002	
	2nd Quarter 2002 cases	Cumulative total since 1 January	Current rate ⁴	2nd Quarter 2001 cases	Cumulative total since 1 January	Previous rate ⁴	Percentage change ⁹	
AIDS	5	10	0.6	5	12	0.6		
Campylobacteriosis	2042	5711	318.0	1468	3974	215.8		***
Cholera	1	1	0.1	0	0	0		
Cryptosporidiosis	88	189	23.4	274	522	31.2	***	
Dengue fever	31	39	3.4	4	5	0.3		*** 1033
Gastroenteritis ⁵	279	505	28.6	170	379	20.3		***
Giardiasis	428	854	43.7	446	823	42.9		
<i>H influenzae</i> type b disease	3	3	0.2	5	7	0.4		
Hepatitis A	34	88	3.2	13	29	2.6		
Hepatitis B (acute) ⁶	20	36	1.6	14	33	1.9		
Hepatitis C (acute) ⁶	15	27	1.6	13	26	1.8		
Hydatid disease	0	0	0.1	1	2	0.1		
Influenza ⁷	197	206	17.4	207	220	12.2		***
Lead absorption	25	48	2.8	36	72	3.7		
Legionellosis ⁷	12	23	1.1	21	40	2.2	**	
Leprosy	1	1	0.1	1	2	0.2		
Leptospirosis	41	78	3.5	29	52	2.7		
Listeriosis	1	7	0.5	2	7	0.4		
Malaria	17	38	1.6	13	34	3.0	**	
Measles	5	14	1.8	12	30	1.5		
Meningococcal disease ⁸	148	232	17.0	138	243	14.1		*
Mumps	17	31	1.6	13	26	1.3		
Paratyphoid	6	9	0.7	9	14	0.9		
Pertussis	254	496	24.6	238	909	91.8	***	
Rheumatic fever	6	43	2.4	37	71	4.8	***	
Rickettsial disease	2	2	0.2	0	0	0.1		*
Rubella	20	24	1.1	6	14	0.9		
Salmonellosis	337	1175	66.4	446	1112	52.9		***
Shigellosis	35	67	3.5	35	94	3.9		
Tetanus	1	1	0.1	3	3	0.1		
Tuberculosis	79	167	9.5	87	188	9.9		
Typhoid	6	17	0.7	6	16	0.7		
VTEC/STEC infection	23	41	2.0	23	42	1.9		
Yersiniosis	106	263	12.9	79	209	10.2		**

Notes: 1 Data on surveillance of Creutzfeldt-Jacob disease have been removed from the quarterly surveillance data tables, and replaced by rickettsial disease surveillance data. Annual summary statistics for Creutzfeldt-Jacob disease will be available in the Annual Surveillance Summary.

2 Other notifiable infectious diseases reported in April to June 2002: Nil

3 These data are provisional

4 Rate is based on the cumulative total for the current year (12 months up to and including June 2002) or the previous year (12 months up to and including June 2001), expressed as cases per 100 000

5 Cases of gastroenteritis from a common source or foodborne intoxication eg, staphylococcal intoxication or toxic shellfish poisoning

6 Only acute cases of this disease are currently notifiable

7 Surveillance data based on laboratory-reported cases only

8 Totals and rates are based on the EpiSurv report date as opposed to the earliest available date used in the meningococcal disease section

9 Percentage change is the difference between the number of cases in the current year (12 months up to and including June 2002) and the previous year (12 months up to and including June 2001).

This difference is expressed as a percentage of the number of cases in the previous year

Surveillance data

Surveillance data by health district - April to June 2002 quarter

Cases this quarter Current rate²

Disease ¹	Cases for Quarter, ³ and current rate ^{2,3} by health district ^{4,5}																							
	Northland	NW Auck	Central Auck	South Auck	Waikato	Tauranga	Eastern BOP	Gisborne	Rotorua	Taupo	Taranaki	Ruapehu	Hawkes Bay	Wanganui	Manawatu	Wairarapa	Wellington	Hutt	Nelson-Marl	West Coast	Canterbury	South Cant	Otago	Southland
AIDS ⁴	0	2	1	0	0	0	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
	0	0	0	0.3	0.8	0	0	1.6	0	0	0	0	0	0	0	0	4.9	0	0	0	0.5	0	0	0
Campylobacteriosis	65	355	329	222	135	57	14	23	10	39	1	57	20	34	11	157	64	24	14	168	60	106	72	
	206.9	374.2	371.4	266.8	364.2	281.1	171.2	191.2	263.6	361.8	326.7	126.0	310.0	250.0	219.5	264.0	457.2	383.0	146.3	257.1	316.7	395.4	298.0	287.9
Cholera	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0.5	0.3	0.3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cryptosporidiosis	0	7	2	4	5	2	0	0	1	1	0	0	6	0	5	0	18	5	5	2	14	2	5	4
	17.1	9.1	4.9	9.9	55.7	20.9	14.3	9.1	34.1	53.9	18.4	14.0	37.6	30.8	32.6	18.3	24.8	12.1	11.4	36.3	15.9	65.3	42.7	64.8
Dengue fever	0	6	9	5	2	1	1	0	0	0	0	0	0	1	2	0	0	4	0	0	0	0	0	0
	2.9	4.9	8.4	4.8	2.3	2.3	2.0	0	1.6	12.7	1.0	0	0	1.7	4.1	0	2.0	6.1	0.8	0	2.5	1.3	1.2	1.9
Gastroenteritis	0	27	28	8	3	1	0	0	0	0	3	0	2	1	32	3	15	8	9	1	125	0	7	6
	3.6	21.4	31.8	10.7	5.5	3.1	8.2	63.7	18.6	34.9	22.3	0	8.4	20.6	34.7	20.9	30.4	25.0	22.1	16.5	100.8	0	43.3	12.0
Giardiasis	9	46	65	46	44	11	1	2	4	6	3	0	41	13	13	6	23	21	8	5	41	4	11	5
	22.1	47.0	62.8	41.8	49.9	47.2	22.4	66.0	35.7	47.6	16.5	21.0	91.9	41.1	30.6	31.4	53.2	44.0	43.3	52.7	34.9	28.2	27.7	15.7
H influenzae type b disease	0	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0
	0	0.2	0	0	0	0	0	0	0	0	1.0	0	1.4	0	0	0	0.4	0	0	0	0.5	0	0	0
Hepatitis A	0	6	11	3	4	0	0	0	0	1	0	0	4	0	1	0	0	1	2	0	1	0	0	0
	0.7	4.0	7.9	7.5	5.8	0	0	4.6	1.6	3.2	0	0	2.8	0	0.7	2.6	2.0	5.3	1.6	0	0.5	0	0	0.9
Hepatitis B	1	1	4	3	2	0	0	2	0	0	0	0	1	0	1	0	0	0	0	0	4	0	1	0
	1.4	1.2	3.0	1.1	1.9	2.3	0	4.6	0	3.2	0	0	2.8	0	1.4	0	2.0	0	1.6	0	2.2	0	1.8	0
Hepatitis C	0	0	0	1	0	5	0	0	1	1	0	0	1	0	0	0	3	0	0	1	0	2	0	0
	0.7	0.2	0.5	0.8	0.3	14.7	2.0	0.0	7.8	9.5	0.0	0.0	0.7	0.0	0.0	0.0	3.5	2.3	0.0	6.6	1.2	2.6	0.6	0.9
Hydatids	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0.7	0.2	0	0	0	0	0	2.3	0	0	0	0	0	0	0	0	0.4	0	0	0	0.2	0	0	0
Lead absorption	1	1	2	1	6	0	0	1	0	0	1	0	0	1	2	0	1	1	0	0	4	0	2	1
	3.6	1.2	2.2	0.5	5.2	3.1	2.0	9.1	3.1	0	3.9	7.0	1.4	1.7	5.4	2.6	0.4	0.8	2.5	0	3.5	9.0	7.8	2.8
Legionellosis ⁶	0	0	1	0	1	0	0	0	0	2	0	0	0	0	0	0	1	0	1	0	5	1	0	0
	2.1	0.5	0.5	0.5	1.6	0	0	0	0	6.3	0	0	0.7	1.7	0	2.6	1.2	2.3	1.6	0	2.5	2.6	0.6	0
Leprosy	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	0	0.5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Leptospirosis	3	2	0	0	3	2	0	0	0	0	0	0	13	1	2	0	0	0	4	2	2	4	2	1
	9.3	1.9	0	0.3	5.8	5.4	2.0	15.9	1.6	0	1.9	14.0	20.2	1.7	6.8	2.6	0.8	0	4.9	6.6	1.2	14.1	1.8	0.9
Listeriosis	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0.7	0.5	0.5	0.3	0.3	1.5	0	2.3	0	1.0	0	0	0	0	0	0	0.4	0.8	0	0	0.5	1.3	1.2	0
Malaria	0	1	2	1	4	0	0	0	0	1	0	1	0	0	2	0	1	0	1	0	1	1	1	0
	0.7	0.7	0.8	0.8	1.9	1.5	0	0	3.1	3.2	1.0	14.0	0.7	0	7.5	0	0.8	0.8	2.5	0	2.5	2.6	2.4	0
Measles	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	0
	1.4	0.2	0.8	1.3	0.6	5.4	0	4.6	0	0	1.9	0	2.8	0	0.7	0	2.4	0	6.5	3.3	2.7	1.3	1.8	7.4
Meningococcal disease ⁷	8	12	17	34	8	11	4	1	8	4	2	0	8	1	4	1	3	1	0	1	11	0	7	2
	25.0	10.2	19.3	30.9	19.4	20.9	34.7	18.2	48.1	38.1	10.7	7.0	18.8	8.6	15.6	28.7	8.3	13.7	4.9	6.6	6.0	5.1	30.7	11.1
Mumps	0	2	1	3	0	0	0	0	0	0	0	1	1	0	0	0	0	3	1	1	0	1	1	3
	2.9	1.4	0.8	1.3	0.3	0.8	4.1	0	1.6	0	0	0	2.1	1.7	0	0	1.6	2.3	3.3	3.3	2.0	0	6.0	3.7
Paratyphoid	0	2	0	2	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0
	0	0.9	1.4	1.3	0.6	0.8	0	0	0	1.0	0	1.4	0	0	0	0	1.2	0.8	0.8	0	0.2	1.3	0	0
Pertussis	1	11	8	4	11	0	0	1	2	2	2	0	3	4	3	1	19	5	8	43	50	61	1	14
	10.7	15.4	10.3	9.3	41.8	9.3	2.0	6.8	4.7	9.5	6.8	21.0	9.8	10.3	7.5	5.2	26.8	35.6	123.4	187.9	30.9	97.3	4.8	38.9
Rheumatic fever	2	0	0	0	1	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	6.4	0.7	3.8	9.6	2.6	3.1	8.2	4.6	0	3.2	1.0	7.0	1.4	1.7	0	2.6	0.4	0	0	0	0.2	0	0	0
Rickettsial disease	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	0	1.4	0	0	0.3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rubella	1	1	0	0	0	1	0	0	0	0	0	0	10	0	0	1	1	2	1	0	0	0	0	0
	0.7	0.5	0.3	0.5	0	0.8	0	0	0	0	1.0	0	9.8	0	0	2.6	1.6	0.8	2.5	3.3	1.5	0	0	0.9
Salmonellosis	17	29	30	26	38	5	7	4	4	7	5	0	16	5	18	4	23	5	14	1	37	14	14	14
	54.9	47.2	57.9	48.5	59.0	44.9	63.2	66.0	65.1	92.0	60.1	35.0	98.2	71.9	58.4	73.2	67.0	53.8	154.4	95.6	63.2	116.5	99.3	94.4
Shigellosis	1	7	6	7	0	0	0	0	1	0	1	0	0	0	0	0	4	0	0	0	2	4	1	1
	0.7	5.6	7.6	7.5	1.0	1.5	0	0	3.1	3.2	3.9	0	4.2	3.4	0	1.6	0.8	1.6	0	3.0	5.1	3.0	0.9	
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
	0	0	0	0	0	0	0	1.6	0	0	0	0	0	0	0	0	0	0	0	0	0	1.3	0	0
Tuberculosis	3	6	18	9	6	2	0	0	1	2	0	0	7	2	0	4	6	6	1	0	3	1	2	0
	11.4	7.7	22.0	16.0	6.5	8.5	4.1	2.3	6.2	25.4	0	0	11.1	8.6	4.8	18.3	12.6	12.1	3.3	0	5.0	6.4	3.0	2.8
Typhoid	0	0	1	4	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
	0	1.4	1.9	2.4	0	0	0	0	0	0	0	0	0	0	0.7	0	1.6	0	0	0	0.2	0	0	0
VTEC/STEC infection	1	1	3	0	6	1	2	0	2	0	1	0	0	0	1	0	0	0	0	0	4	0	0	1
	1.4	0.9	1.9	0	5.8	2.3	10.2	4.6	4.7	0	2.9	0	1.7	2.0	0	0.8	0.8	0	0	3.2	0	3.0	2.8	
Yersiniosis	0	25	16	12	4	4	0	1	1	2	0	2	1	0	8	0	5	0	1	3	10	1	7	3
	5.7	14.4	17.1	12.2	11.7	19.4	8.2	13.7	12.4	22.2	5.8	14.0	10.4	12.0	10.9	5.2	11.8	10.6	4.1	33.0	14.2	20.5	15.0	12.0

Notes: 1 Data on surveillance of Creutzfeldt-Jacob disease have been removed from the quarterly surveillance data tables, and replaced by rickettsial disease surveillance data. Annual summary statistics for Creutzfeldt-Jacob disease will be available in the

Hepatitis C risk from sharing drug equipment other than syringes

The risk for hepatitis C virus (HCV) transmission through shared syringes is well established. A recent cohort study in the United States has measured the risk of HCV seroconversion associated with sharing injection equipment other than syringes. Seronegative injection drug users (IDUs) aged 18-30 were tested for HCV antibodies at regular intervals. During 290 person-years of follow-up, 29 participants seroconverted, an incidence of 10 per 100 person-years. After adjusting for syringe-sharing, sharing "cookers" (containers used to mix and heat drugs) was the strongest predictor of seroconversion (adjusted relative hazard (RH) = 3.54, 95% confidence interval (CI): 1.26 - 9.94), followed by sharing rinse water (adjusted RH = 2.29, 95% CI 1.01 - 5.20). The authors conclude that sharing injection equipment other than syringes may be an important cause of HCV transmission between IDUs (Thorpe EL, Ouellet LJ, Hershov R, et al. Risk of hepatitis C virus

infection among young adult injection drug users who share injection equipment. *Am J Epidemiol* 2002; 155: 645-53.)

Editorial note: Between 60 and 100 cases of hepatitis C are notified annually in New Zealand, however disease modelling suggests that the true rate of new infections is likely to be closer to 1280 cases annually. As injecting drug use is the most common route of transmission of HCV in New Zealand, minimisation of harm associated with injecting drug use is central to reducing the burden of disease associated with hepatitis C. Data on risk behaviours among IDUs in New Zealand are scant, however responses to one survey of IDUs suggested that 54% of respondents had shared spoons used for preparing drugs for injection [*N Z Med J* 1998; 111: 50-3]. The Ministry of Health is currently developing a hepatitis C prevention action plan which, combined with a review of the Needle Exchange Programme, will inform further policy development in this area.

Human error was found contributing to 67% of mercury spills

Schools and Universities (20%), private residences (17%), and health care facilities (17%) were the most frequent locations involved, and human error (67%), the contributing factor for most of the mercury releases in the United States (US). These results were based on analysis of data from the Hazardous Substances Emergency Events Surveillance System (HSEES) from 1993 to 1998. The HSEES is an active, state based surveillance system to collect and analyse information on hazardous substances emergency events. It covers ten states in the US. HSEES events are defined as sudden uncontrolled, or illegal releases of at least one hazardous substance that had to be removed, cleaned up, or neutralised according to federal, state, or local law. This analysis was restricted to events in which mercury was the only chemical released and included 413 releases between 1993-1998. No fatalities resulted, however 14 people experienced adverse health effects and an additional 31 had elevated blood mercury levels. Evacuations were ordered in 22% of the events and ranged from one hour to 46 days. The authors suggest that a switch to mercury free alternatives, training people in the safe handling and disposal of mercury, and secure storage of mercury could potentially lessen the consequences of mercury spills (Zeitz P, Orr ME, Kaye WE. Public Health consequences of mercury spills: hazardous

substances emergency events surveillance system, 1993-1998. *Environ Health Perspect* 2002; 110: 129-32).

Editorial Note: Several organisations in New Zealand collect data on mercury injuries and spills including OSH, ERMA, the New Zealand Fire Service, and regional councils. ERMA has primary responsibility for the management of hazardous substances through its enforcement agencies. No notifications have been received by ERMA involving mercury since the hazardous substance provision of the HSNO Act commenced on 2 July 2001. The National Poisons Centre fielded 1061 calls relating to mercury from 1997 to 2001. Of these, 61% concerned poisoning/exposures and 26% were about spills. Mercury thermometers were the main source of poisoning/exposure calls (89%) and spill related calls (69%). Other sources of mercury included barometers, sphygmomanometers, mercury amalgams, fixative solutions, mercury in fish and occupation exposure (jewellery). A national chemical injury surveillance system, a joint initiative between ESR and the Ministry of Health, is being developed to record hazardous substances injuries resulting in hospitalisations. No chemical injuries involving mercury were notified during a pilot study of the surveillance system between July and December 2001 in six Public Health Services.

Travel health

Water disinfection for international and wilderness travellers

Waterborne disease is a constant hazard for travellers to developing countries and for wilderness travellers who drink surface water in any country. The safety of water cannot reliably be assessed based on look, smell and taste. In developing countries tap water must be assumed to be contaminated. Bottled water is generally safer, but it is important to check the seal has not been broken which may indicate it has been tampered with or refilled. Disinfection aims to make water potable, ie, ensuring that it contains only a minimal microbial hazard and that the likelihood of illness is reduced to an acceptable level. The choice of water treatment in such situation depends on a number of factors including space and weight constraints and individual taste preferences.

Heat is an effective one-step disinfection process. Enteric pathogens are killed within seconds by boiling water. Bringing water to the boil should be adequate, even at high altitude. Chemical disinfection with halogens (mainly chlorine and iodine) is commonly used for water disinfection. These agents may be obtained from pharmacies and camping shops. Their use is limited by their poor effectiveness against *Cryptosporidium* oocysts. Concentrations need to be increased if water is cloudy. Halogens are also useful to prevent contamination when water has to be stored for an extended period. There is a wide range of water filtration devices. Some of these will remove larger pathogens such as *Giardia* but few completely remove viruses. Filtration may be adequate for relatively unpolluted water encountered in wilderness areas. (Backer H. Water disinfection for international and wilderness travelers. *Clin Infect Dis* 2002; 34: 355-64.)

Editorial note: The incidence of waterborne disease in those returning from overseas and wilderness travellers in New Zealand is difficult to measure. All of the notifiable enteric diseases have some association with overseas travel, particularly typhoid and hepatitis A. The Ministry has produced a health education pamphlet (*Enjoying Yourself in the Great Outdoors*) that includes advice for wilderness travellers on safe water supplies. The Ministry also has a pamphlet (*Health Advice for Overseas Travellers*) that provides advice for international travellers to reduce their risks from unsafe water supplies and other hazards.

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