

A school and community outbreak of tuberculosis in Auckland

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Abstract

Aim. To describe a school and community outbreak of tuberculosis in South Auckland in 1997/8.

Methods. Cases were diagnosed according to national guidelines at Middlemore, Green Lane and Starship Hospitals. Public health follow-up was conducted by Auckland Healthcare.

Results. Twelve cases were diagnosed during the outbreak. Nine cases were from the same South Auckland secondary school; six reported no association outside school. Three cases were in younger children who had close household contact with two of the school cases. Nine cases (including eight from the school) had identical *Mycobacterium tuberculosis* isolates on restriction fragment length

polymorphism testing. No microbiological culture was obtained from the three remaining cases. Contact investigation detected five of the cases. Chemoprophylaxis was prescribed for twenty-six school students, two adult staff, and nine household contacts.

Conclusion. This is the first published account of a tuberculosis outbreak in a New Zealand school setting for decades. Recognition of the outbreak was delayed. DNA fingerprinting played a valuable role in the investigation. The source case may have been a school student. The social impact of the outbreak and preventability with routine adolescent BCG vaccination are discussed.

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A series of tuberculosis (TB) cases were diagnosed in late 1997 and early 1998 in a South Auckland secondary school. This report describes the course of the outbreak, the clinical cases, the contact investigation, the role played by molecular typing and the control measures adopted.

Methods

Cases were diagnosed and treated at Middlemore Hospital, Green Lane Hospital and Starship Hospital in Auckland on the basis of standard guidelines.¹

DNA fingerprinting was performed according to the standard method proposed by van Embden et al.² Briefly, the technique entails the growth of *M tuberculosis* and extraction of genomic DNA, which is digested with the restriction enzyme Pvu II. A Southern blot is performed and probed with a 245 base pair fragment of insertion sequence IS6110. The reference strain of *M tuberculosis*, Mt 14323, was included as an internal control and known band size markers. Isolates from two unrelated cases and one isolate of *M bovis* were used as additional controls.

Public health investigation of household and school contacts by the Public Health Protection Service of Auckland Healthcare involved 5 tuberculin unit Mantoux testing and, if indicated, a chest x-ray.

Students were asked to take home explanatory letters and consent forms for informed parental consent for Mantoux testing and radiologic follow-up. Letters were sent and home visits made by a public health nurse to non-responding parents. If parents still failed to respond they were visited by specially trained Pacific Islands or Maori members of a community group who provided education about TB and sought consent for testing. Parents who still did not respond were visited by an out-of-hours public health nurse.

Cutting points adopted for a positive Mantoux test followed national guidelines for school teachers and household contacts.¹ For the school students, we departed from the national guidelines by adopting a single cutting point, whereby a Mantoux reaction ≥ 10 mm was considered positive. Past BCG vaccination status was considered positive if there was a scar overlying the insertion of the left deltoid muscle.

Results

Cases. Features of the cases are summarised in Table 1. The relationships between the outbreak cases are shown in Figure 1.

Cases 1-6 were diagnosed between December 1997 and February 1998. Case 1 was an adolescent at the secondary school and had no contact with case 2, a two

year old child with infectious disseminated TB. Cases 3-6 were household contacts of case 2. Cases 3 and 4 presented at Middlemore Hospital with symptomatic disease before their contact screening procedures could be completed. Cases 5 and 6 were asymptomatic and were detected by contact screening. Five of the first six cases were members of the same household. In May 1998 there were a further two notifications (cases 7 and 8) in students attending the school and one of these was sputum smear positive. Contact investigation detected three more asymptomatic cases (9, 10 and 11). All had active pulmonary TB. Case 12, who had been a casual contact of one case in school and one case outside school, had left the school in March 1998 and presented to Middlemore Hospital with severe pulmonary TB almost one year later.

The cases were all of Pacific Islands ethnicity but only two were born overseas. All nine *M tuberculosis* isolates obtained were fully susceptible to first-line antituberculous agents and shared an identical restriction fragment length polymorphism (RFLP) pattern (Figure 2), including eight of the school students (of whom only four had any association outside school). In two of the other three cases no attempt was made to isolate an organism as epidemiological and x-ray findings were thought to be adequate for a diagnosis. Only one of the 12 notified cases (case 8) was an obvious infectious risk to others, having strongly smear positive sputum for the first four weeks of antituberculous treatment and symptoms which lasted for eight weeks after treatment started. This case had extensive shadowing in both lung fields on chest radiograph. None of the cases identified during the first phase of the outbreak (cases 1 - 6) appeared to be a major infectious risk, judging by sputum smear negativity (for acid-fast bacilli), lack of cough and sputum, and minimal or no chest radiograph abnormalities. Seven of the nine school cases carried scars consistent with past BCG vaccination. All cases were treated by directly observed therapy.

Table 1. Outbreak cases.

Case	Age*	Born in NZ	Past BCG vaccination	Date of notification	Approx date of symptom onset	Site of disease	Smear positive specimen	Culture positive specimen	Susceptible to HREZS	Outbreak RFLP pattern
1	14	Y	N	29/12/97	18/12/98	Pulmonary and pleural effusion	N	Pleural aspirate	Y	Y
2	2	Y	N	20/01/98	06/01/98	Miliary	Gastric aspirate	Gastric aspirate	Y	Y
3	15	N	Y	13/02/98	06/02/98	Pulmonary	N	Sputum, gastric washings	Y	Y
4	16	N	Y	18/02/98	01/02/98	Pleural effusion	N	Pleural aspirate	Y	Y
5	5	Y	N	18/02/98	Nil	Pulmonary	Not done	Not done	NA	NA
6	9	Y	Y	18/02/98	Nil	Pulmonary	Not done	Not done	NA	NA
7	15	Y	Y	08/05/98	07/04/98	Pulmonary	N	N	NA	NA
8	15	Y	N	16/05/98	01/03/98	Pulmonary	Sputum	Sputum	Y	Y
9	16	Y	Y	22/09/98	Nil	Pulmonary	N	Sputum	Y	Y
10	16	Y	Y	04/09/98	Nil	Pulmonary	N	Sputum	Y	Y
11	16	Y	Y	18/05/99	Nil	Pulmonary	N	Induced sputum	Y	Y
12	17	Y	Y	16/02/99	01/01/99	Pulmonary, pleural effusion, nodal	Pleural biopsy	Sputum	Y	Y

Y= Yes; N= No; NA= Not applicable; HREZS= isoniazid, rifampicin, ethambutol, pyrazinamide, streptomycin; * on 29/12/97, the date of notification of the first case.

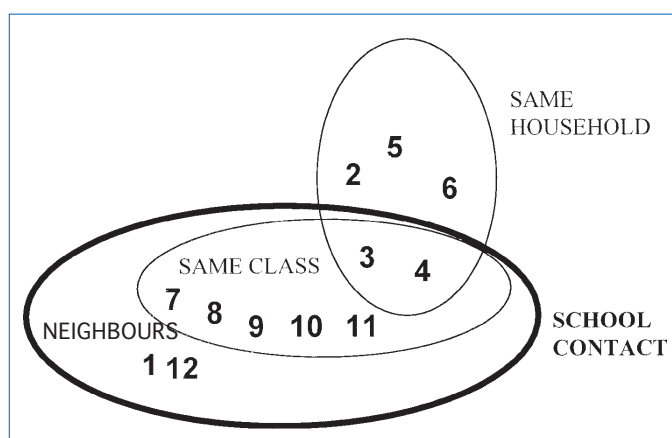


Figure 1. Relationships between the outbreak cases.

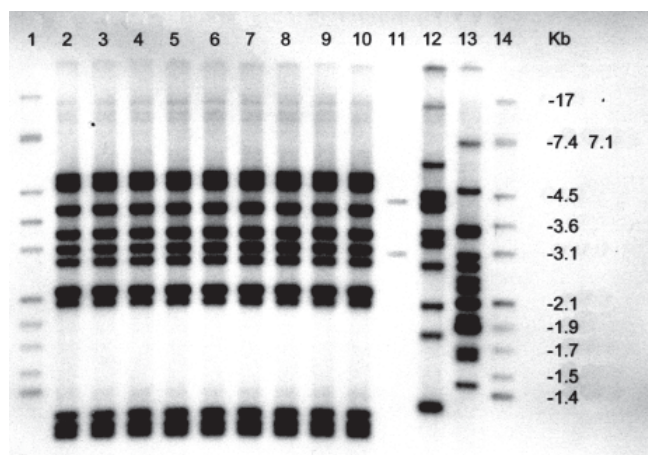


Figure 2. DNA fingerprints of *Mycobacterium tuberculosis* strains. Lanes 1 and 14 Reference strain Mt 14323; Lanes 2-10 Cases 1,2,3,4,8,9,10,11,12; Lanes 11-13 Unrelated *M. tuberculosis* isolates; Kb = kilobase.

Contact investigation. Since no family contacts of case 1 were infected, school contacts were not screened. A number of household contacts of case 2 were found to be infected. After notification of cases 3 and 4 limited screening was conducted in the school. Testing of four students and 15 adult staff yielded no cases and only one Mantoux-positive

person. Cases 5 and 6 were children and screening was confined to household contacts. After the notification of cases 7 and 8, we extended the contact investigation in the school to cover all students who had been exposed to any school case. We also requested RFLP typing, which had just become available in Auckland Healthcare, of the *M tuberculosis* isolates. The extended contact investigation in the school revealed three further cases (9, 10 and 11). The contact investigation was extended further to encompass all un-screened students in the school and the remainder of exposed staff. The community group which assisted with the investigation obtained 123 of 148 outstanding consents (83%) within two weeks. The results of the contact investigation are shown in Table 2. Twenty-one household contacts and 40 school contacts were found to be Mantoux positive. No adult source case of lung disease was discovered despite screening of 42 adult household contacts and 45 adults among school staff. Ten school staff were not screened (one refused and nine were lost to follow-up).

Chemoprophylaxis of contacts. Thirty-seven people were placed on antituberculous chemoprophylaxis. For Mantoux-positive students this was carried out by directly observed preventive therapy (DOPT). Duration of chemoprophylaxis varied depending on the public health staffing available and the proximity of the start of treatment to the summer vacation. Three students received six months' uninterrupted isoniazid (H); 19 students received four months' uninterrupted rifampicin and isoniazid (RH); four students received five months' RH interrupted by the summer vacation (eight weeks). Standard doses of R and H were prescribed for twice-weekly treatment.¹

Discussion

We have described a school- and community-based outbreak in which twelve cases of TB and 61 Mantoux-positive people were identified.

In retrospect, recognition of the school outbreak was delayed. Initially only close household and very few school contacts were tested. No household contacts of case 1 were infected, indicating low infectivity of this case. Therefore no school contacts were examined. Cases 2, 3 and 4 shared close household or social relationships outside the school and only case 2, a preschool child, was smear positive. Limited screening of staff and student contacts of cases 3 and 4 yielded only one Mantoux-positive person, while there was extensive tuberculosis infection and disease in the household. At this stage we were unconvinced of transmission within

Table 2. Outcome of the contact investigation of twelve cases of tuberculosis.

		Number of contacts identified	Number (%) tested [†]	Number (%) refusing testing	Number (%) lost to follow-up	Number (%) not tested as already known to have clinical disease	Number (%) not tested owing to past disease
School contacts	Students	491	445 (91)	3 (1)	33 (7)	5 (1)	5 (1)
	Adults	55	45 (82)	1 (2)	9(16)	0	0
Household contacts	Children	34	34(100)	0	0	0	0
	Adults	43	42 (98) [‡]	0	0	0	0
		Number (%) [§] Mantoux negative	Number (%) Mantoux Positive	Number (%) identified with clinical TB disease	Number (%) on chemoprophylaxis	Number (%) followed up with serial chest X-rays	
School contacts	Students	415 (93)	30 (7)	3 (0.7)	26 (6)	1(0.2)	
	Adults	35 (78)	10 (22)	0	2 (4)	8 (18)	
Household contacts	Children	27 (79)	7 (21)	2 (6)	4 (12)	0	
	Adults	28 (67)	14 (33)	0	5 (12)	8 (19)	

[†] Percentages do not add up to 100% due to rounding; [‡] One person not tested due to concurrent illness; [§] % = percentage of those tested.

the school, even though school staff were expressing concern about the unusual number of cases. This initial cluster of cases was in a community with a high proportion of Pacific Islands students, an ethnic group with known high rates of TB infection. It was only with the appearance of two further school cases, one of whom was sputum smear positive (case 8), and the availability of RFLP testing that an outbreak could be confirmed.

RFLP testing played a valuable role in confirming the findings of the epidemiologic investigation. Experience to date with RFLP testing of approximately sixty *M tuberculosis* isolates in Auckland indicates a high degree of strain heterogeneity (personal communication, Arthur Morris, 4/3/99). Therefore the identical DNA pattern found in the isolates in this outbreak provides powerful evidence of person-to-person transmission within the school, particularly since five of the nine school cases reported no contact with each other outside school. RFLP testing could not be conducted on three of the cases (5,6,7) from whom no *M tuberculosis* was isolated. We are however, highly suspicious on epidemiological grounds that they were part of the outbreak.

Accounts have been published of outbreaks in primary³⁻⁷ and secondary⁸⁻¹³ schools in several countries. In these reports an adult source case was often implicated. We did not discover an unequivocal source case for this school outbreak. Case 8 is the most likely source. It is puzzling that the reported onset of symptoms in case 8 was ten weeks after the onset in case 1. However, case 8 had extensive chest radiographic shadowing on presentation, slow radiographic improvement and prolonged sputum smear positivity despite aggressive antituberculous treatment. We believe that active tuberculosis must have been present for many weeks before presentation. Alternative sources include one of the other school cases who may have spontaneously remitted to a less infectious stage by the time of diagnosis, an undetected adult source case in one of the students' households, or a source among staff or students at the school whom we were unable to screen. No such case, however, has presented spontaneously since the end of the outbreak. Case 12, who presented in February 1999, had no symptoms until ten months after leaving school and was an unlikely source.

National guidelines¹ would have required us to adopt different cutting points for a positive Mantoux test for different students depending on age, closeness of contact and BCG status. In a departure from the national guidelines, we adopted $\geq 10\text{mm}$ as the cutting point for a positive Mantoux test for all students in the school investigation. Our

reasons were twofold: to increase the sensitivity of the test for detecting infection, thus increasing our chances of controlling the outbreak; and because of the administrative complexity of determining and explaining different cutting points for each student during a large testing programme.

A second Mantoux test was not carried out to test for conversion because more than twelve weeks had elapsed between the departure of the last infectious case and the commencement of testing.¹ Some of the positive Mantoux results would therefore have been due to remote infection or vaccination, rather than exposure during this outbreak.

We wanted to ensure compliance with chemoprophylaxis by Mantoux-positive students. We did not consider that self-medication was suitable and instituted DOPT at school. The usual regimen used in New Zealand is six months uninterrupted isoniazid taken daily.¹ Since community-based DOPT for all students was beyond our staffing resources during the summer vacation, we adopted alternative prophylactic regimens for some students: five months' RH, with interruption of the regimen during the vacation; or four months' uninterrupted RH. Evidence for the effectiveness of shorter regimens has been published.¹⁴

The school's routines were heavily disrupted by the public health investigation and follow-up. Intense local and national media attention stretched the resources of the school and the public health service during the outbreak. The publicity had an adverse affect on the morale of the school, generating shame among the students and alarm amongst staff. This caused considerable distress in the school community, which may in turn have led to the difficulties we experienced in conducting contact screening. Obtaining consent was time-consuming. The loss-to-follow-up rate of 7% among students was achieved only by a substantial input of resources. Our employment of a community group to obtain outstanding consents for screening demonstrated that it is possible to train community members rapidly even if they have no health background. Stigmatisation of cases and their families is a continuing obstacle to effective TB control. The difficulties encountered in this investigation suggest that in-depth education about TB should be planned with affected communities.

This outbreak should be viewed with concern. In the experience of public health offices, tuberculosis transmission in New Zealand usually occurs in family groups as a result of household contact. We could find no reports of school outbreaks, (i.e. transmission within the school setting), in New Zealand since 1966, in either Medline or the records of

the New Zealand Communicable Disease Centre (personal communication, Yvonne Galloway). Only two of the cases were born outside New Zealand, which raises the possibility that declining social conditions in New Zealand were a factor.

Seven of the nine school cases carried scars consistent with past BCG vaccinations. These must have been received earlier in life since routine vaccination in secondary school was discontinued in Auckland in the 1980s. Further outbreaks of this magnitude will raise the question of whether routine tuberculin screening and BCG vaccination and/or chemoprophylaxis of adolescents should be reintroduced in schools which have high numbers of Pacific Island, Asian and African students, given that in New Zealand these ethnic groups have high rates of TB.

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