TB Update 2/2/95

STATE OF ALASKA

TONY KNOWLES, GOVERNOR

DEPT. OF HEALTH & SOCIAL SERVICES

DIVISION OF PUBLIC HEALTH
SECTION OF EPIDEMIOLOGY

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INFECTIOUS DISEASES
AIDS/STD
TUBERCULOSIS
IMMUNIZATION
CHRONIC DISEASES
DIABETES
INJURY CONTROL

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Tuberculosis Control Program

Status Report
February 2, 1995

- Tuberculosis caused widespread outbreaks in several rural villages in 1994.

- Investigation of the outbreaks required a comprehensive, collaborative effort to identify individuals infected, assess presence and severity of disease, and arrange for appropriate treatment with antibiotics. The investigations are still underway.

- In seven villages with a population of 5434, field teams of community health aides, public health nurses, an x-ray technician, and medical epidemiologists have:

  Administered and interpreted tuberculosis skin tests 2,918
  Taken and interpreted chest x-rays 656
  Collected sputum for laboratory analysis 1,169
  Interviewed patients and performed clinical examinations 592

- To date, we have identified and begun antibiotic treatment for:

  38 Alaskans with active tuberculosis disease
  147 Alaskans who have been infected but have not yet developed disease (preventive antibiotic treatment)

- It will be necessary to monitor closely the status of tuberculosis in the affected villages for several years.

- Because low priority has been given to tuberculosis for at least a decade, institutional expertise and infrastructure to respond to these outbreaks of tuberculosis was inadequate. As a result, the following problems arose and impaired an effective and prompt response:

  1) Detection of the outbreaks was delayed, allowing more transmission of tuberculosis than should have occurred.

  2) Because local resources and experience were inadequate, initiation of antibiotic treatment was delayed.

3) Our ability to take x-rays in the field was compromised initially and then failed totally, a major setback to our control efforts.

- Tuberculosis can be treated effectively and successfully but requires patients to take medication for 6 months to 24 months.

- Maintenance of State laboratory expertise and capacity in tuberculosis is essential.

- Rebuilding statewide expertise and capacity will take several years.

- New legislation is needed to enable appropriate implementation of involuntary confinement and treatment of individuals infected with tuberculosis while ensuring due process safeguards for individuals.

- Many health facilities will need modification to be in compliance with new OSHA requirements that apply to diagnosis and treatment of persons infected with tuberculosis.

- Strains of tuberculosis bacteria have emerged nationally, and recently in Alaska, that are resistant to all available antibiotics.

- Prevention of tuberculosis and its spread must remain an essential responsibility of the State in full partnership with all those involved in control of tuberculosis.

Tuberculosis in Alaska, 1994-95

Outbreak investigation

		PPDs			X-Rays		Sputum		
Village	Population	Total	Converters	Reactors	Persons	Total	Persons	Total	Total Cases
Savoonga	618	318	18	26	316	364	106	279	8*
Gambell	643	431	21	33	206	221	86	216	13
St. Paul Island	665	272	1	10	70	71	58	144	5
Hooper Bay	1,257	545	2	1	0	0	33	85	2
Scammon Bay	477	316	13	11	0	0	63	149	7
Chevak	822	458	3	1	0	0	34	75	1
Mountain Village	952	578	1	6	0	0	80	221	2
Total	**5,434**	**2,918**	**59**	**88**	**592**	**656**	**460**	**1,169**	**38**

*2 cases now living elsewhere in Alaska

Section of Epidemiology
February 1, 1995

Tuberculosis Field Visit Schedule

(X = visit done, O = visit planned)

1994

	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.
Savoonga								X	X	X		
Gambell									X	X		
St. Paul										X		
Hooper Bay												
Scammon Bay												
Chevak												
Mt. Village												
Lower Kalskag												
Prince of Wales												
Aniak											X	

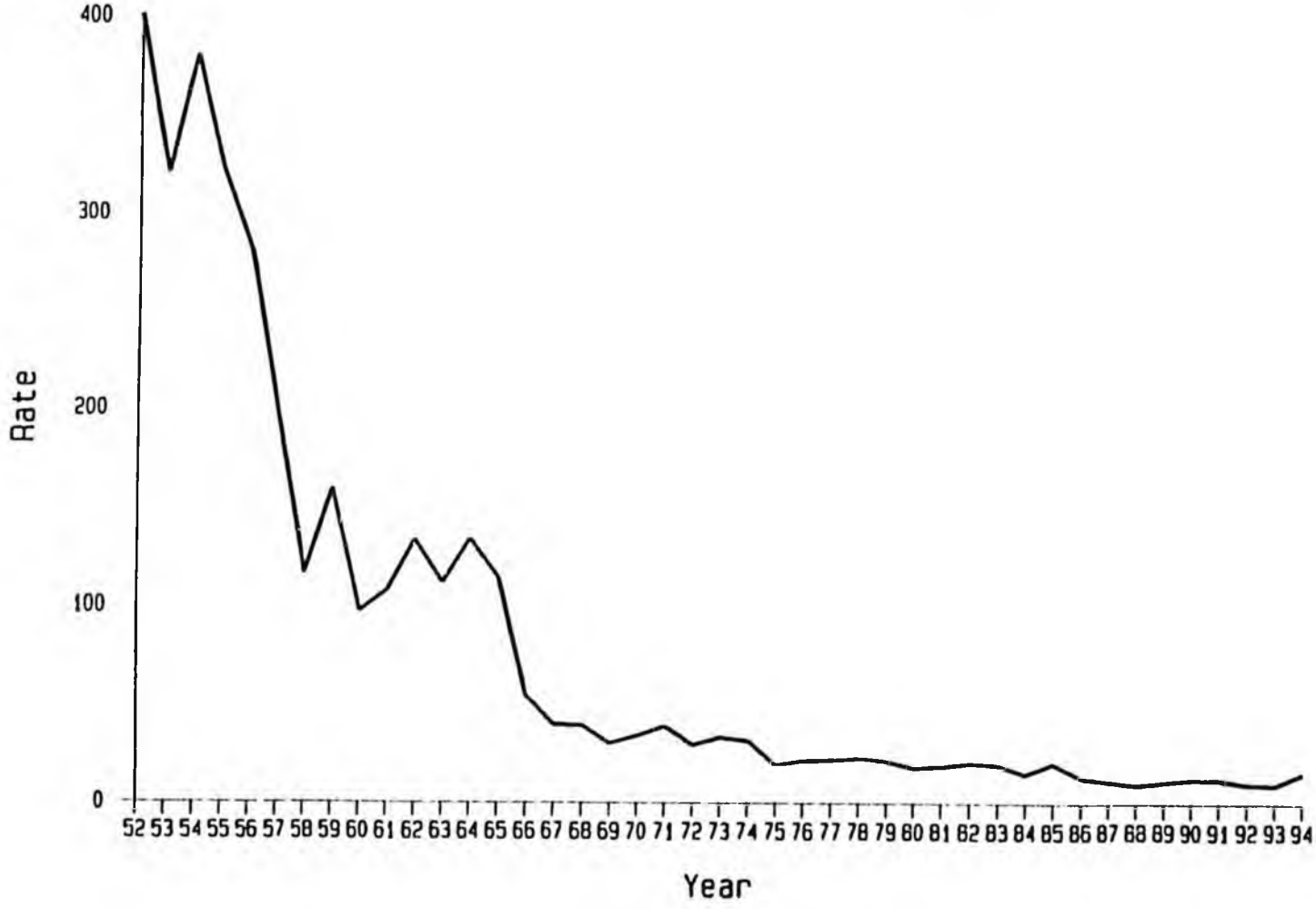
1995

	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.
Savoonga		O			O				O		O	
Gambell		O			O				O		O	
St. Paul		O							O			
Hooper Bay	X		O		O				O			
Scammon Bay	X		O		O				O		O	
Chevak	X		O		O				O			
Mt. Village	X		O		O				O			
Lower Kalskag			O		O				O		O	
Prince of Wales				O								
Aniak									O			

1/31/95

Annual Tuberculosis Case Rates, Alaska, 1952-94

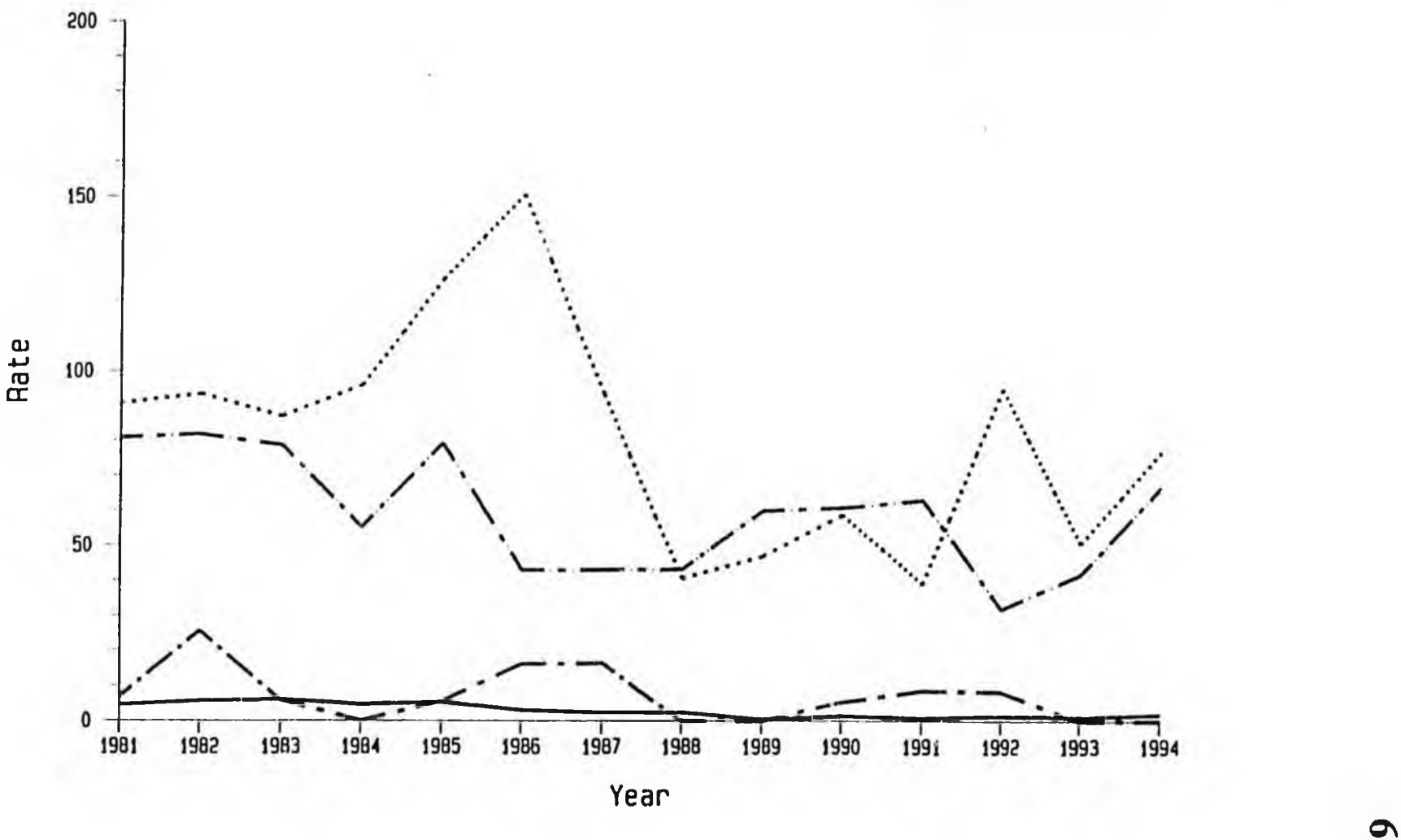
(Rate per 100,000 population)



Cases of Tuberculosis in Alaska, 1981-1994

Rate / 100,000 By Race

N=1054



Tuberculosis Cases and Incidence Rates*,By Race, Alaska, 1981-94

	White		Black		Alaska Native		Asian	
	No	Rate	No	Rate	No	Rate	No	Rate
1981	15	(4.5)	1	(6.8)	56	(80.7)	11	(90.6)
1982	20	(5.6)	4	(25.5)	60	(81.6)	12	(93.3)
1983	23	(6.0)	1	(5.9)	62	(78.5)	12	(86.8)
1984	19	(4.7)	0	(0.0)	46	(55.1)	14	(95.9)
1985	22	(5.3)	1	(5.5)	68	(79.0)	19	(126.2)
1986	13	(3.1)	3	(16.1)	33	(42.9)	23	(150.5)
1987	10	(2.4)	3	(16.4)	34	(43.0)	14	(93.3)
1988	10	(2.4)	0	(0.0)	35	(43.1)	6	(40.5)
1989	2	(0.5)	0	(0.0)	50	(59.9)	7	(46.9)
1990	6	(1.4)	1	(5.3)	52	(60.7)	9	(58.6)
1991	4	(0.9)	2	(8.6)	56	(62.9)	8	(39.0)
1992	6	(1.4)	2	(8.3)	29	(31.7)	20	(94.7)
1993	5	(1.1)	0	(0.0)	40	(41.7)	12	(50.6)
1994	8	(1.8)	0	(0.0)	66	(66.6)	19	(76.7)

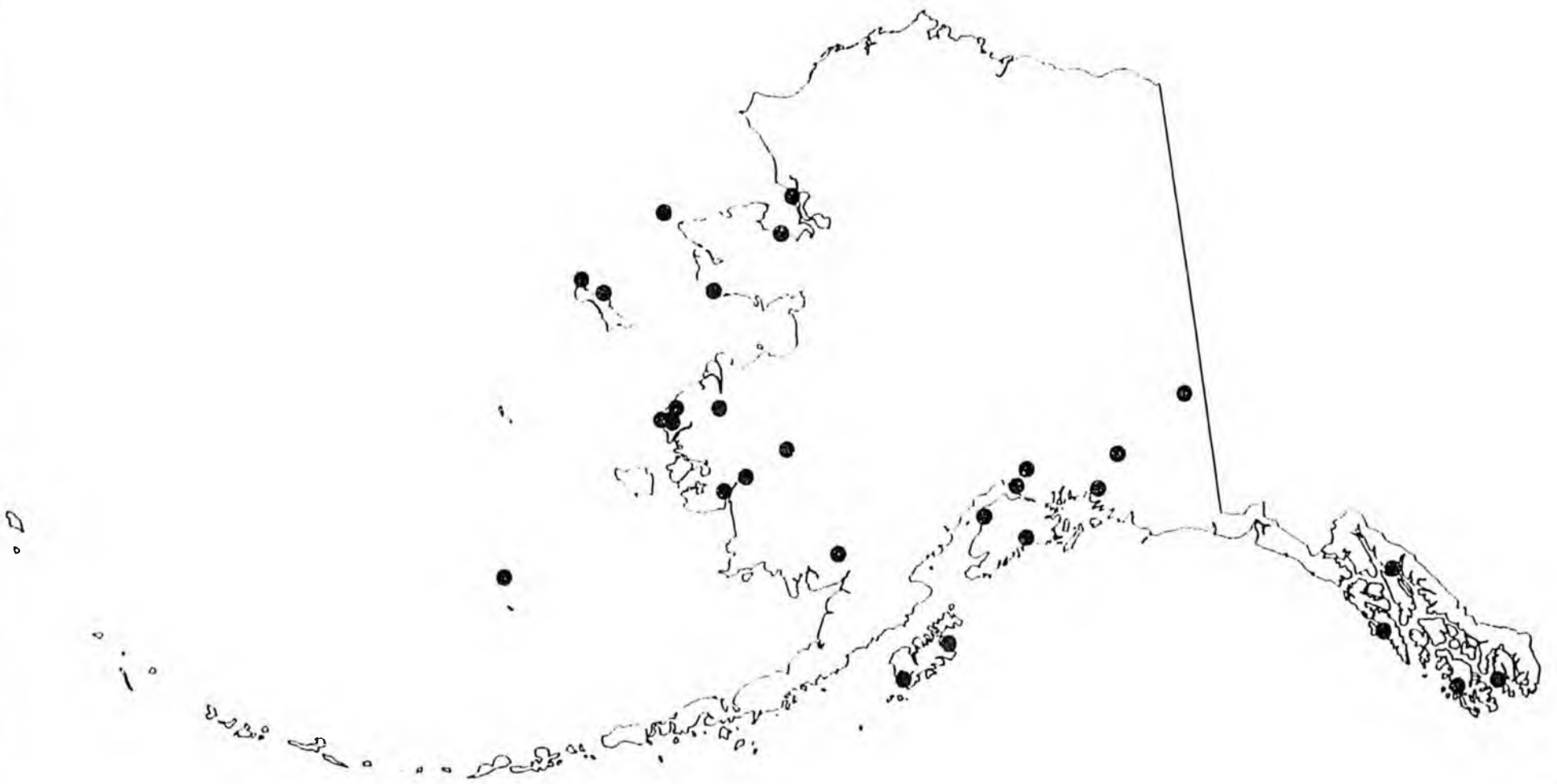
*Rate per 100,000 population

Characteristics of Tuberculosis Cases, Alaska, 1988-94

	1988	1989	1990	1991	1992	1993	1994
No of cases	51	59	68	70	57	57	93
Alaska case rate (per 100,000)	9.5	11.0	12.4	12.3	9.7	9.5	15.5
USA case rate (per 100,000)	9.1	9.5	9.6	10.4	10.5	9.8	—
No 0-14 yrs old (% of total)	9 (18%)	13 (22%)	9 (13%)	11 (16%)	4 (7%)	5 (9%)	11 (12%)
No of foreign born (% of total)	7 (14%)	7 (12%)	7 (10%)	8 (11%)	17 (30%)	12 (21%)	17 (18%)
No with drug resistant tuberculosis	2	6	0	3	4	3	2
No asscciated with outbreaks	0	13	23	26	7	0	38

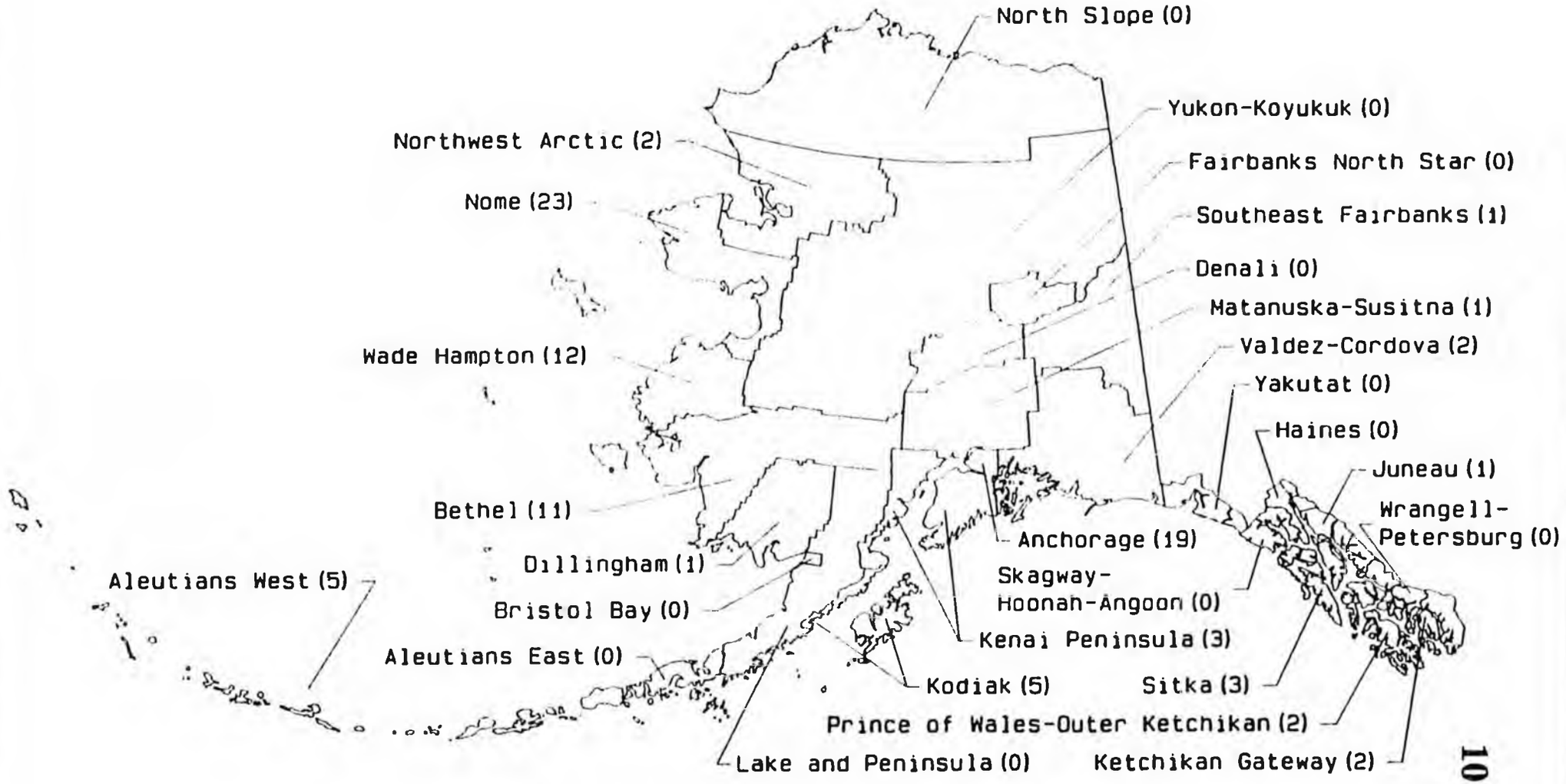
Tuberculosis in Alaska, 1994

Villages with one or more cases; N(cases) =93 N(villages) =28



Tuberculosis in Alaska, 1994

Cases by census area; N = 93



Tuberculosis in Alaska, 1988-1994

Cases by census area; N = 455

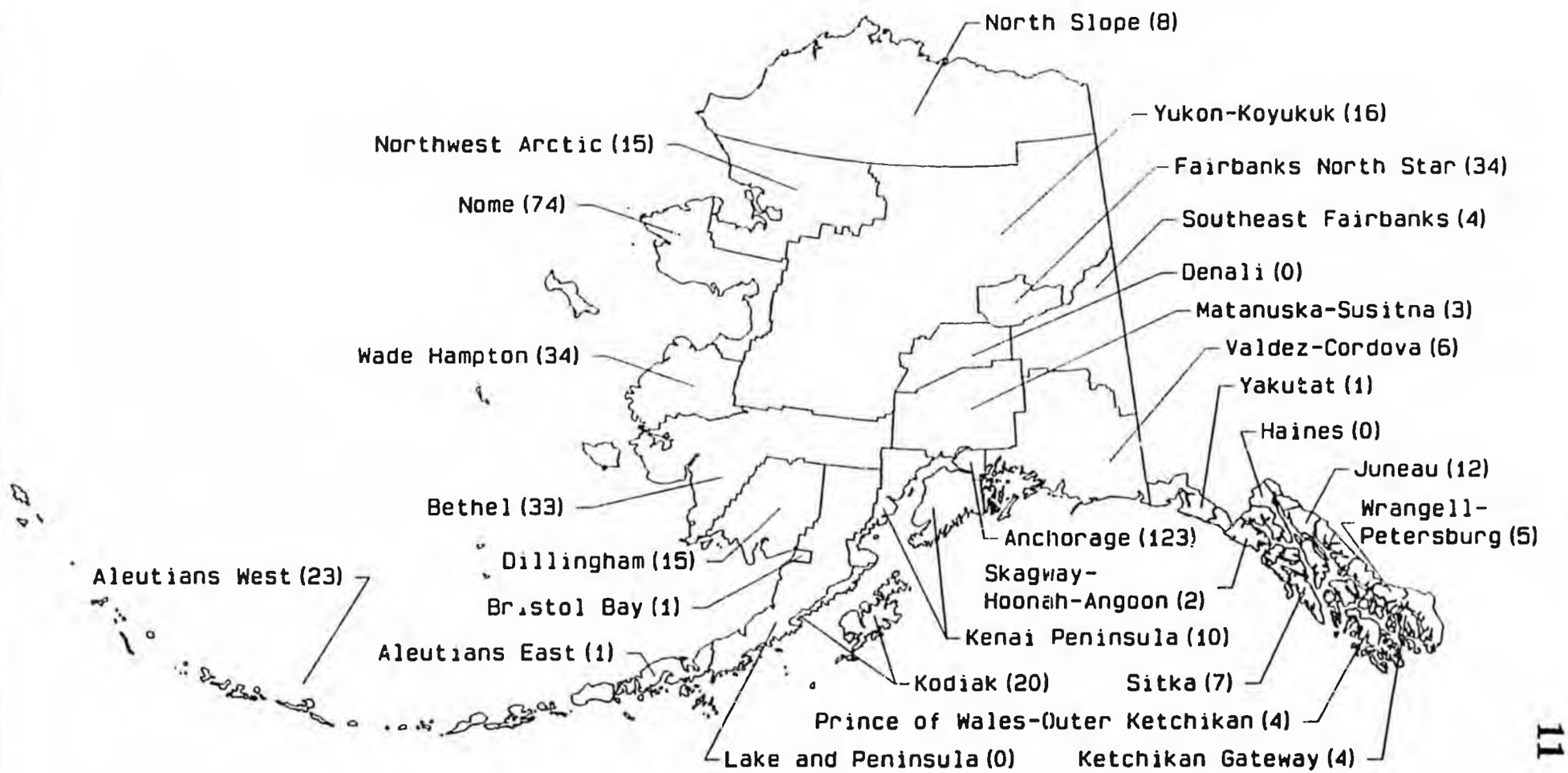
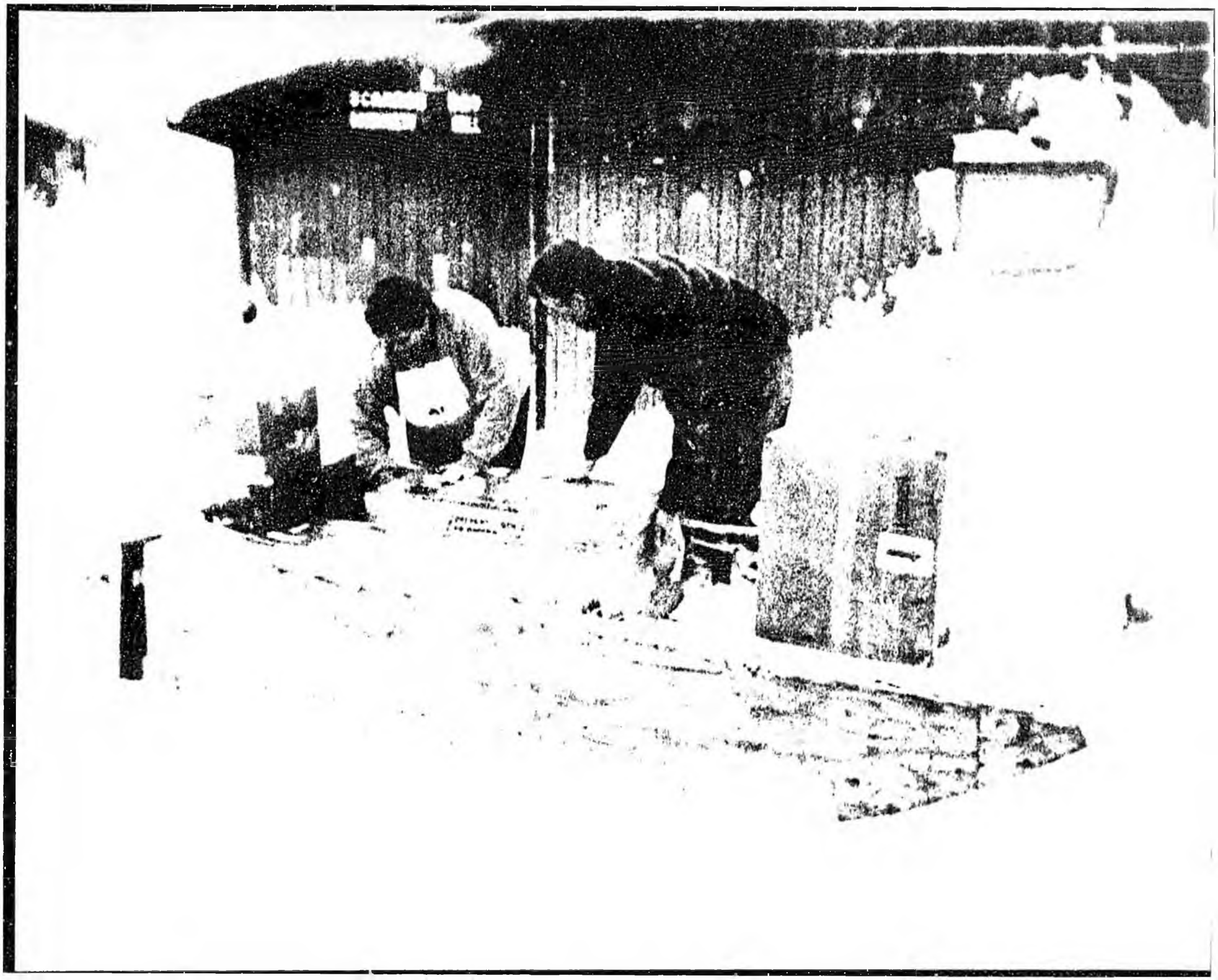
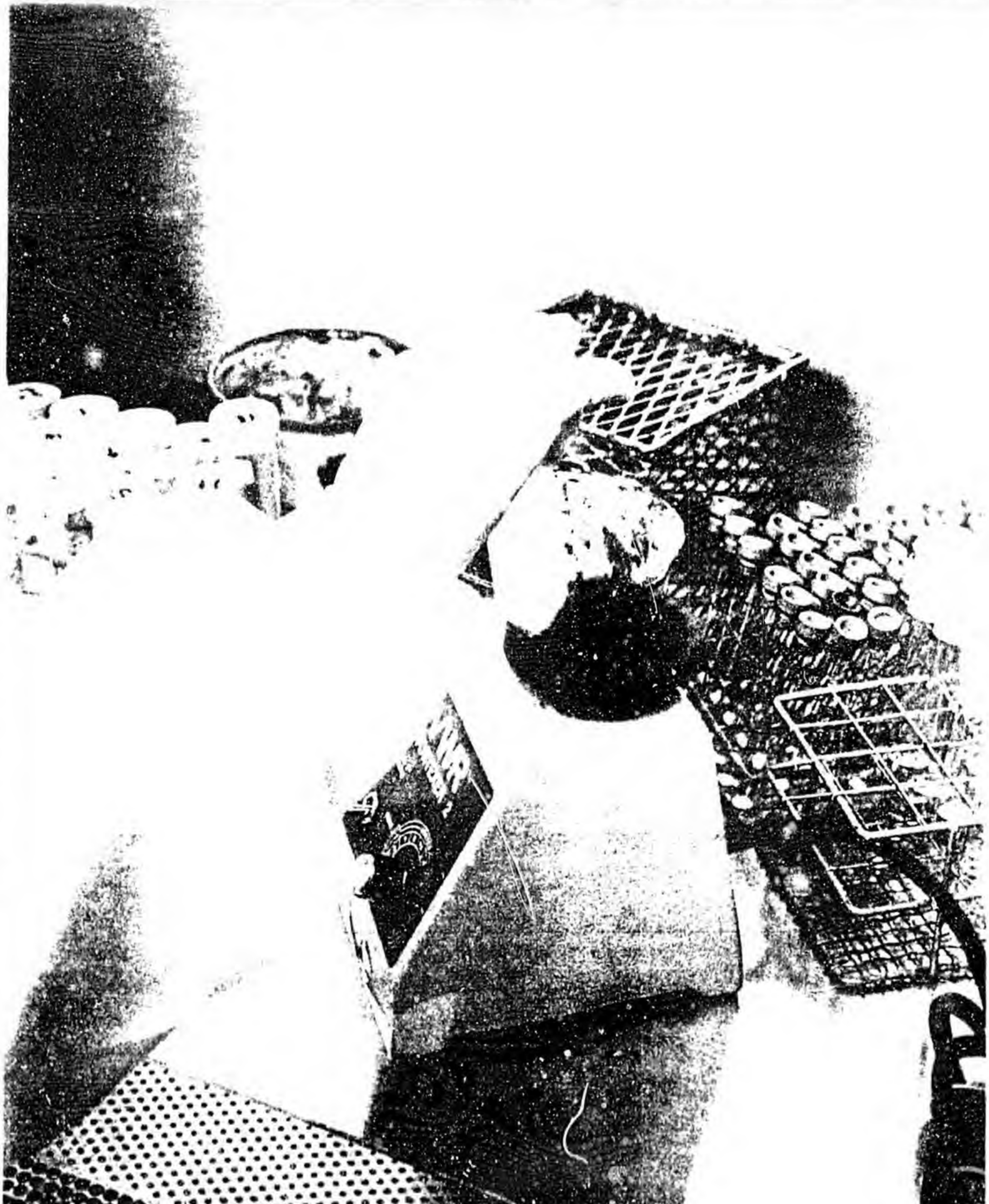
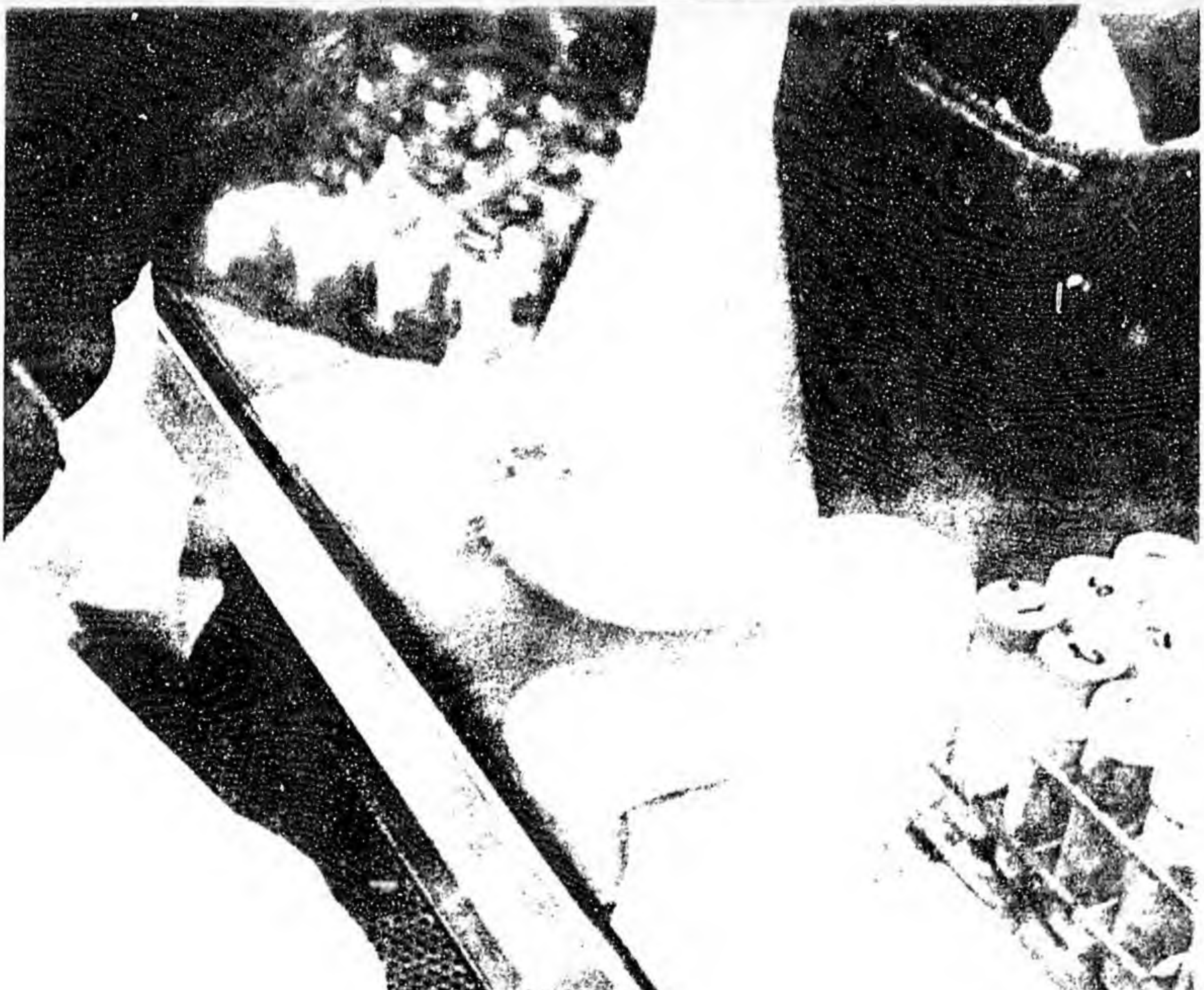


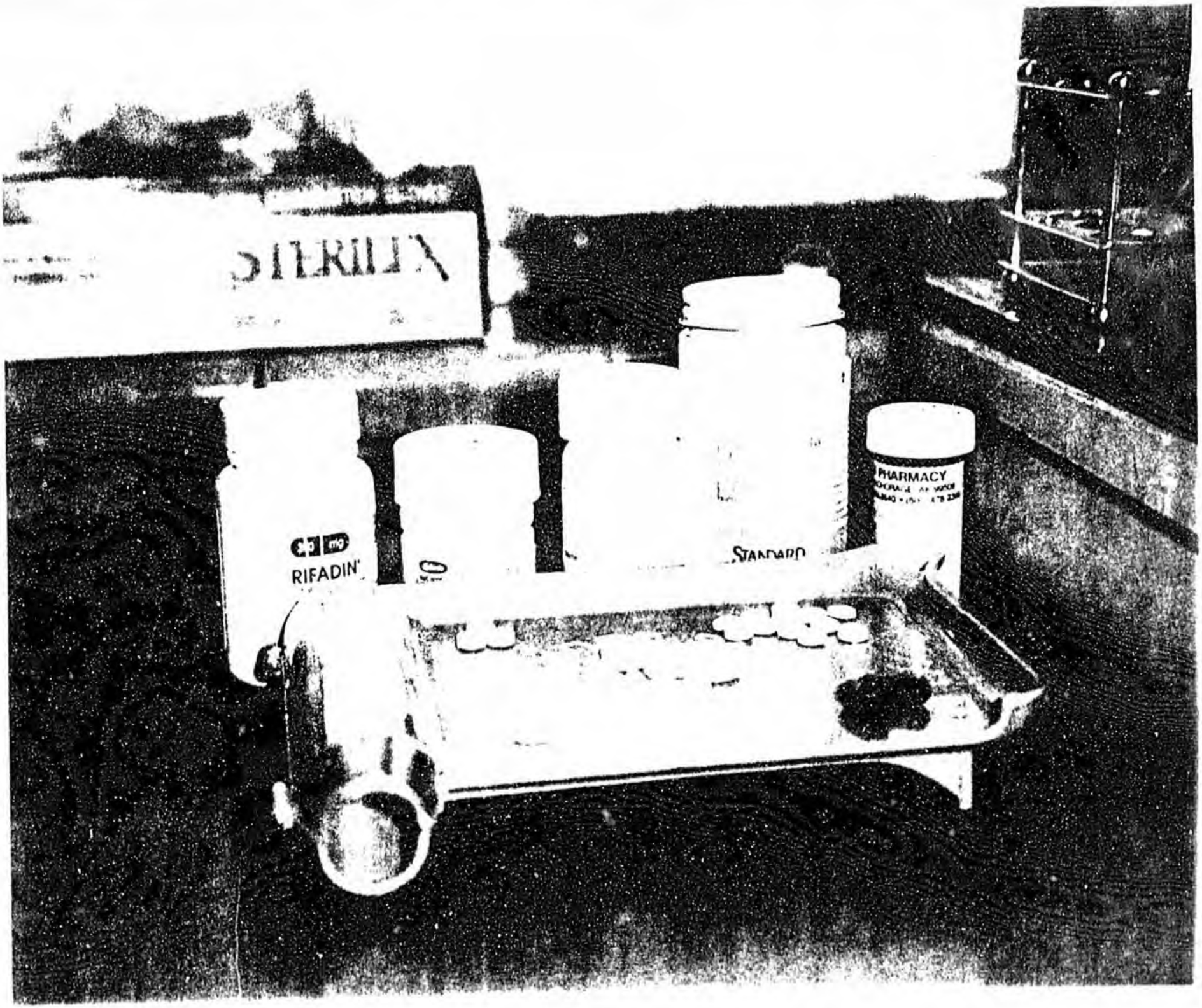
Table 6. Selected Tuberculosis Outbreaks, By Village, Alaska, 1987-91

	Holy Cross	Chevak	Savoonga	St. Paul
Year	1987	1989	1990	1990
Number of Cases	12	9	22	7
Number of Culture-Confirmed Cases	5	4	18	6
Mean Age in Years (Range)	18 (1-48)	9.6 (0.3-18)	22.1 (1-35)	28.7 (0.9-57)
Number of Tuberculin Converters	13	63	36-98	7
How Outbreak was Detected	Contact Investigation	School Screening	Contact Investigation	School Screening





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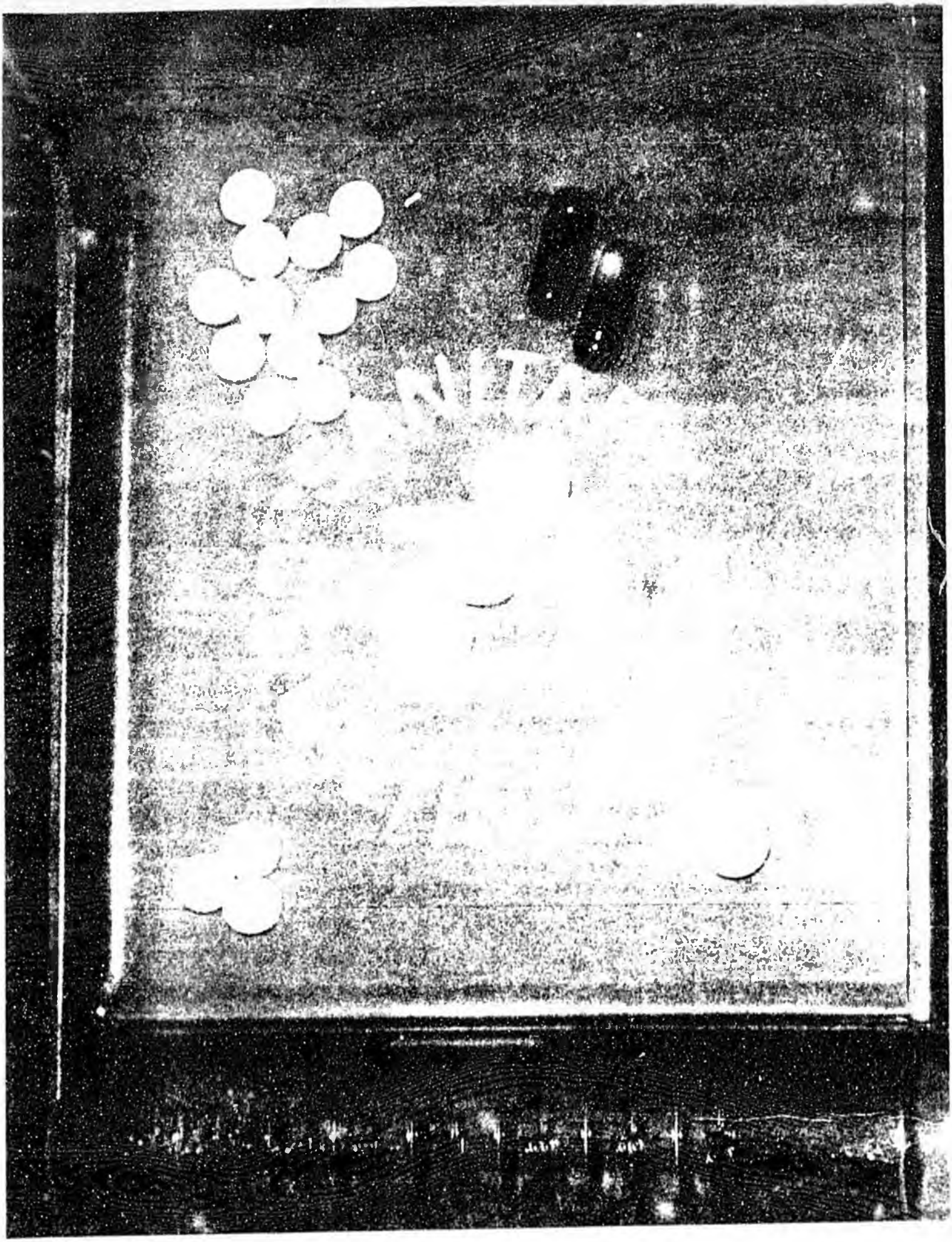

Old enemy

A new threat

"Retirement of key personnel, outdated methods and dwindling state funds have left Alaska's efforts to eradicate tuberculosis in a precarious position, according to a new study from the national Centers for Disease Control."

— A Daily News story, Oct. 9, 1987.

"Tuberculosis — once presumed headed for extinction in the state — has broken out in several western Alaska viliages, and officials say the public heaith system lacks the money and people to battle it."

— A Daily News story, Nov. 8, 1994.

Anyone who has been in Alaska for any length of time should be aware of the deadly potential of tuberculosis or TB.

Between 1920 and 1950, TB was to blame for almost half of all Native deaths. As recently as 1985, Alaska had the highest tuberculosis incidence rate of any state. More specifically, as Sylvia Carlsson, spokeswoman for the Alaska Native Medical Center, said not long ago in a Daily News story, "The epidemic wiped out whole families. I remember one family that had 12 children, and they all died. Just about every Alaska Native was affected in one way or another."

Alaskans are fortunate now to have drugs that are effective against TB; the drug-resistant strains spreading rapidly in other parts of the world aren't yet a problem in Alaska. No more are people sent to sanitariums for years at a time in hope of a cure. But we must not let complacency and shortsightedness rule or many more Alaska families could again become painfully familiar with TB.

The budget for the state's tuberculosis program has been cut almost by half in the last nine years. In 1985, the budget was $810,000. This year, it's $438,000. Medical and public health professionals who used to concentrate on detecting and treating TB cases were assigned other duties when it appeared the disease was under control in Alaska. The Indian Health Service, which formerly contributed $250,000 to $300,000 a year specifically to fight TB, no longer does so.

Now, new cases of TB have appeared in Savoonga, Gambell, St. Paul, Scammon Bay, Hooper Bay and Chevak. The Division of Public Health has spent $100,000 over its budget fighting this outbreak alone, and has prepared a supplemental budget increase request of approximately $600,000 for 1994.

There are few jobs more basic to government than controlling and preventing a deadly infectious disease that can affect a large number of people in a short time. If we don't find the resources to adequately combat TB now, the costs, in dollars as well as in human suffering, may become overwhelming.

Anchorage Daily News

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Katherine Fanning, Editor and Publisher, 1971—1983
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Founded in 1946 by Norman C. Brown

What is TB?

A few facts

The main form of the bacterial disease known as tuberculosis is pulmonary TB, or an affliction of the respiratory system — usually the lungs.

TB attacks body organs, causing the patient to lose weight and strength. In some cases, the organs fail and the person dies. People with TB may experience night sweats and persistent coughing.

A common way to check for the disease is a TB skin test. But the tests may read false negative, since the disease can escape detection for up to two months after exposure.

TB can either be contagious or latent in the body. It is treatable with antibiotics, but the patient remains contagious during the first two weeks of treatment. Once diagnosed, treatment can last from six to 12 months.

In medical terminology, those with active tuberculosis infections are considered "diseased." They are the ones who spread the bacteria to others. "Infected" persons have been exposed to the bacteria and test positive for it. TB can lodge in their lungs or elsewhere, but they aren't actively contagious.

Roughly 10 percent of those infected but asymptomatic can develop active cases — even decades later — due to alcoholism, bad nutrition or damaged immune systems.

Those infected but symptomless can have TB settle in other parts of the body, too, ranging from the brain to intestinal lining. If they develop TB later in life, they are not contagious. Only those with a respiratory infection are contagious.

Pulmonary TB is spread through coughing, singing or sneezing. Contaminated droplets are expelled into the air, where they can hover for days. "One person potentially can infect hundreds," says Dr. John Middaugh, state epidemiologist.

"This is a disease where prevention is the best medicine," he concludes. "Anytime tuberculosis breaks out, it is a report card of failure of the public health system. Period. I mean we get an 'F.'"

Tuberculosis

Outbreaks are troublesome

Savoonga. Gambell. Scammon Bay. Hooper Bay St. Paul. Chevak and Mountain Village. Each of these communities has seen suspected or confirmed cases of tuberculosis. Some village outbreaks are akin to mini-epidemics. State epidemiologist Dr. John Middaugh says, for example, that one out of three Savoonga and Gambell residents has been infected with TB in the past four years.

So far, state and federal government responses to tuberculosis have been inadequate — unlike decades ago, when TB was battled with a vengeance. "We heard about an outbreak of TB in Scammon Bay (three months ago) and no one's gone there yet," explained Dr. Middaugh in mid-December. Scammon Bay and three nearby villages — while they're getting some local medical attention — won't be visited by the state's TB specialists until January.

TB was once the scourge of Alaska. In fact, the Native hospital in Anchorage — with its top-floor solarium — was established in 1953 as a TB sanitarium.

Modern antibiotics eventually brought TB under control. But strains resistant to multiple antibiotics have plagued other states. Alaska has no such cases recorded — yet. It would be wise public and fiscal policy to keep it that way.

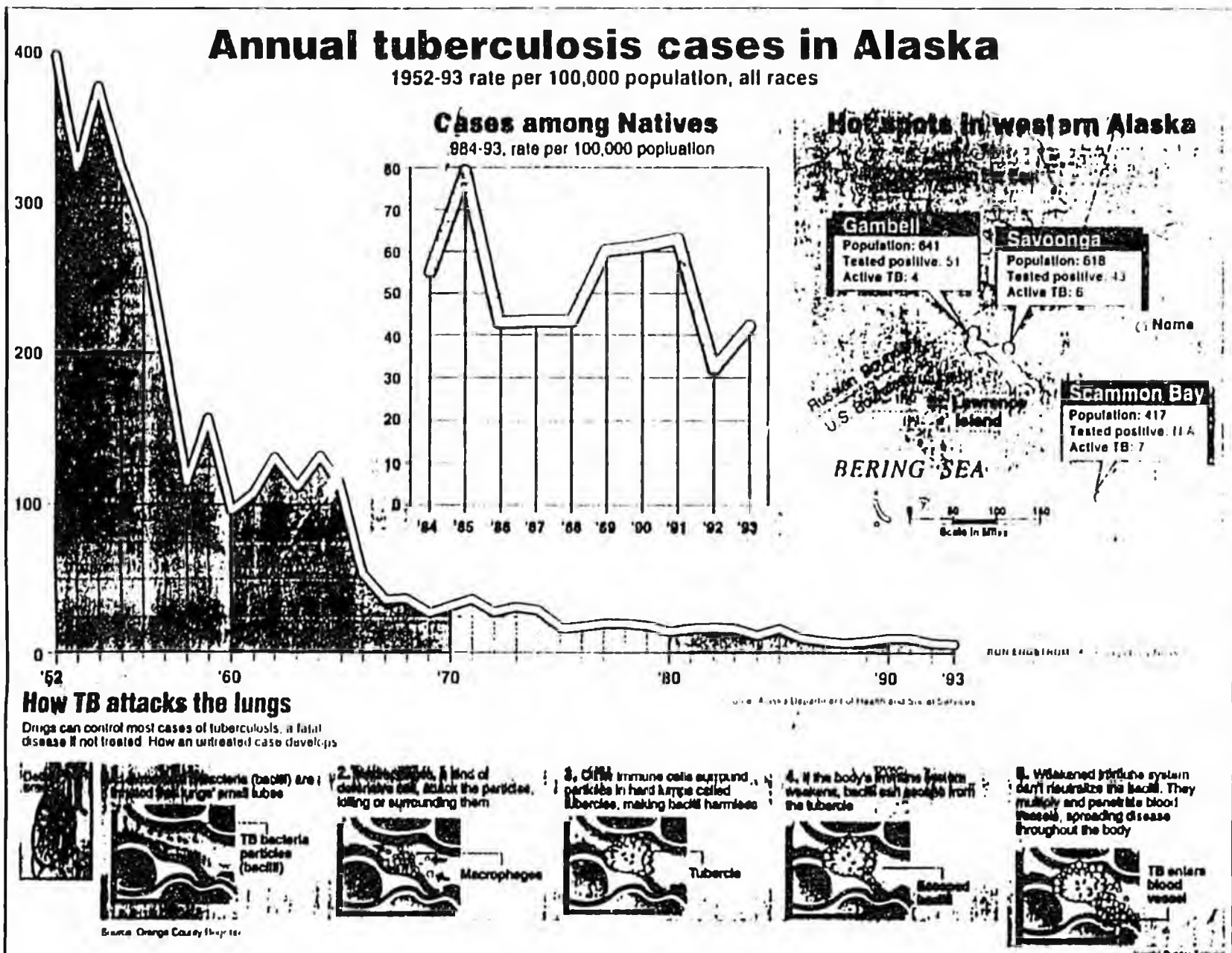
Resistant strains can develop when antibiotics aren't taken for their prescribed length. To prevent this, officials must in some rare cases turn to quarantine. A Kotzebue woman quarantined with TB recently went on a drinking binge while out on a pass. Though she was not infectious, her case is an example of the challenges Alaska faces in controlling TB.

Tuberculosis cases are now primarily occurring in rural areas among Alaska Natives and in urban areas among Asians and Pacific Islanders. From 1988 to 1991, when it resurged in Savoonga and Gambell, 171 Alaskans were diagnosed with the disease; seven of them died. Eighty percent of cases were Alaska Natives.

Does the fact that TB is largely a rural, Native disease explain why Alaska lacks the resources to fight it? If one out of three Anchorage residents was infected with TB, how would government respond?

With the cases in hand, we already have a public health problem of significant proportions. To control TB and other infectious diseases, public health officials need resources — namely money and personnel — to identify and treat the contagious and TB carriers. Alaskan lawmakers can spend the money necessary to aggressively eradicate TB, or they can sit

Rural outbreaks of TB alarm state



Funds lacking as old disease spreads anew

By TOM BELL
Daily News reporter

Tuberculosis, once presumed headed for extinction in the state, has broken out in several western Alaska villages, and officials say the public health system lacks the money and people to battle it.

"If we don't get this controlled, it will keep spreading," said Dr. John Middaugh, chief of the state's epidemiology section.

The hardest hit villages are on St. Lawrence Island, where every resident of Savoonga and Gambell has been tested for TB.

In Savoonga, population 618, doctors found six people with active cases, plus 43 others who've been infected but aren't ill. In Gambell, population 611, four people have the disease, with 51 infected. Other villages with active cases are St. Paul, Scammon Bay, Hooper Bay and Chevak.

Those people are receiving treatment that should cure them and keep the bacteria from spreading.

An active case of TB is infectious, and it usually attacks the lungs and spreads through the air. Symptoms include a persistent cough, weight loss, fever, fatigue and night sweats.

In those infected with

Please see Back Page, TB

TB: State health officials worry they lack resources to deal with disease

Continued from Page A-1

the disease but showing no symptoms, the TB bacteria is dormant. However, alcohol abuse, malnutrition or sickness can weaken the immune system and allow the bacteria to become active. Health officials say 2 to 18 percent of infected Natives will get the disease within two years if they go untreated.

State health officials say that currently most of the people with the disease are either young adults or children.

Last week, a man with an active case of TB alarmed health officials when he flew from St. Lawrence Island to Anchorage, possibly exposing other people on the plane to the disease. He was immediately taken to the Alaska Native Medical Center for treatment. The other passengers are now being tested for TB.

Vicki-Marie Colacicco, director of public health nursing for the Norton Sound Health Corp., said outbreaks will continue to occur in the state because so many older Alaska Natives were infected in the 1950s when TB was widespread.

"I don't want any village to be complacent just because they're not on St. Lawrence Island," Colacicco said. "It could happen anywhere in rural Alaska."

Middaugh said the disease's resurgence is occurring at the same time public resources for fighting TB are dwindling. Since the mid-1980s, he said, the state and the Indian Health Service have cut almost $800,000 that had been dedicated to TB control.

He said the state lacks enough experienced personnel, including X-ray technicians, health aides and nurses, to do the labor-intensive work of identifying people who have the disease and making sure they get treatment.

"The infrastructure of public health is in danger of falling apart, which threatens the ability to adequately respond to TB and other outbreaks," Middaugh said.

Funds for TB programs were cut because health officials stopped seeing the disease as a threat, said Thomas Nighswander, director of community health services at the Alaska Native Medical Center.

"It looks like we went to sleep a little bit," he said.

So far, the state Division of Public Health has spent $100,000 over its budget in fighting the outbreak in western Alaska, agency director Dr. Peter Nakamura said. He said he will ask the legislature for more money.

Unlike smallpox, typhoid and the other diseases that devastated Native communities in the 18th and 19th centuries, TB is within the memory of many Natives. Between 1920 and 1950, the disease was blamed for almost half of all Native deaths.

During that time, Alaska Natives had the highest incidence rates ever recorded in the world. The Indian Health Service built the Alaska Native Medical Center in 1953 as a sanatorium for TB patients.

The disease's resurgence is frightening news for Native Alaskans familiar with the epidemic, said Sylvia Carlsson, the hospital's spokeswoman and a Native from Sitka.

"The epidemic wiped out whole families," she said. "I remember one family that had 12 children, and they all died. Just about every Alaska Native was affected in one way or another."

Russian and American whalers first brought TB to Alaska in the 18th and 19th centuries. The disease's spread has paralleled the number of Westerners in the territory.

Since anti-TB drugs were developed more than 40 years ago, the disease has been steadily declining. The drugs were so effective, Colacicco said, that doctors once believed they could eliminate TB by the year 2000. However, recent outbreaks indicate the bacteria has already been passed on to new generations and will probably linger in Alaska for at least another 75 years.

Health officials first learned about the outbreak on St. Lawrence Island when a large number of Savoonga children who had previously tested negative for TB tested positive.

Usually, only the children's friends and family would then be tested. But in this case, the numbers were so large that officials decided to test the whole village. Then they tested residents of nearby Gambell.

The outbreak has many islanders anxious, said Rena Boolowon, the Gambell resident in charge of distributing the medicine to treat TB. "Some people are pretty scared," she said.

One of those is Savoonga resident Elsie Kava, 81, who said she remembers how TB once killed a lot of people, and she's afraid it might happen again.

"I'm worried about my grandchildren," she said.

People with TB must take several pills a week for six months. That makes stopping the outbreak difficult because people often quit taking medicine once they feel better. And if the bacteria isn't killed, these people can continue to infect others.

Several years ago the state began a program that required health aides and responsible community members to actually watch TB patients take their medicine.

Boolowon, who has that job in Gambell, says she sometimes has to chase people down if they don't show up at the health clinic for their pills.

Officials say TB is being introduced to the Anchorage community by immigrants, especially people from Asia. In fact, Asians living in Alaska now have a higher incidence rate than Alaska Natives.

Nighswander said many immigrants may be infected but go untreated because language and cultural barriers make it difficult for health care providers to do the kind of extensive interviews and testing needed for an effective TB control effort.

State of Alaska Epidemiology **Bulletin**

Department of Health and Social Services — Margaret R. Lowe, MEd, EdS, Commissioner
Division of Public Health — Peter M. Nakamura, MD, MPH, Director
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Bulletin No. 25 October 25, 1994

Tuberculosis Threatens Rural Alaskans

Detection of infectious, pulmonary tuberculosis (TB) in several rural villages has sounded a wake-up call for Alaska's medical and public health professionals. Intensive investigations are underway in Savoonga, Gambell, St. Paul and Scammon Bay. Awaiting further field work are St. George, Chevak, Hooper Bay, and Mt. Village.

1. Savoonga (population 618): Public health teams skin tested 290 persons not known to be previously TB skin test positive, obtained chest x-rays from 297 persons, and collected sputum for acid-fast bacillus (AFB) smear and culture from 122 people. Preliminary results identified 6 persons with pulmonary TB and 43 persons whose skin test changed from previously negative to positive (ie, ≥ 10 mm induration). These 41 people were infected with Mycobacterium tuberculosis at some time between the occurrence of the negative and the positive skin test--for 18 of them, the last negative skin test was within a 2 year time period. Each of the six TB patients with active disease has been started on isoniazid (INH), rifampin, pyrazinamide, and ethambutol administered as directly observed therapy (DOT). Arrangements for starting INH preventive therapy for the remaining infected persons have not yet been completed.

2. Gambell (population 641): Skin tests were placed on 387 persons, chest x-rays were obtained from 163 persons, and sputum specimens were collected from 83 persons. Four cases of active pulmonary TB and 51 newly infected persons--including 23 who appeared to be infected during the past 2 years--were identified. The TB patients with active disease were started on four drug therapy given as DOT; arrangements for INH preventive therapy are pending.

3. St. Paul (population 670): During 1993-94, four persons were identified with active tuberculosis: two subsequently died and two were started on self-administered four drug therapy. The epidemiologic investigation consisted of skin tests of 251 persons, chest x-rays of 70 persons, and sputum collection for AFB smear and culture of 61 persons. Preliminary results indicate that one person had previously unidentified active pulmonary TB and eight persons had a skin test which changed from previously negative to positive. Arrangements were made to place all three patients with active disease on DOT.

4. Yukon-Kuskowkim Delta (estimated populations: Scammon Bay 417; Hooper Bay 964; Chevak 682): Routine tuberculin skin testing of school children conducted by the Bethel Health Center identified four children with new positive tests in Scammon Bay. Follow-up medical evaluation of these children and epidemiologic investigation of their contacts in Scammon Bay and the other two above villages identified seven persons with suspected pulmonary TB and four persons with newly acquired tuberculous infection.

Discussion: During the past 25 years, TB incidence in Alaska has been steadily decreasing. For 1990-1993, an average of 63 cases were diagnosed each year (range: 57-70 per year). As of October 17, 62 cases of TB were diagnosed among Alaska residents during 1994. The increase that has occurred in 1994 illustrates the need for continued vigilance for TB among Alaska health-care providers. When undiagnosed, TB can spread to family members, friends, and other community members resulting in additional TB cases. The cardinal symptoms of TB are cough, fever, weight loss, and hemoptysis.

Routine annual screening of school-children is an essential component of TB control in Alaska. Follow-up investigations conducted in response to children with TB skin test conversions lead to discovery of unrecognized, undiagnosed, and untreated pulmonary TB cases.

All TB patients should be treated using Directly Observed Therapy. The Section of Epidemiology, in coordination with public health nurses, assures that mechanisms are in place for DOT to be carried-out. Persons needing INH preventive therapy should be strongly considered for DOT as well.

Patients hospitalized for TB should never be discharged until arrangements for DOT have been established. Arrangements for patients who are not hospitalized should be initiated as soon as TB is a likely possibility. The Section of Epidemiology will help set-up a DOT program. Patients should be provided with a minimum supply of TB medications (ie, 2 or 3 days) in order to carry them through to their first DOT visit.

Patients who are suspected to have TB must be reported to the Section of Epidemiology at 561-4406.

Village	Population	PPD Status			No. CXR	No. Sputum*	Active TB
		No. done	No. Positive (No. Converters*)				
Savoonga	618	290	43	(18)	297	122	6
Gambell	641	387	51	(23)	163	83	4
St. Paul	670	251	8	(1)	70	61	3 (4)
Scammon Bay	417	investigation underway					7

*Converter = patient with positive skin test and last known negative skin test within the past 2 years.

American Journal of Public Health

Editorials

Tuberculosis Control and Social Change

Tuberculosis and its control are manifestations of social and economic development. During the industrial revolution, crowding and other factors caused the number of tuberculosis cases to increase. Tuberculosis rates fell steadily in most developed countries in this century except during periods of social stress such as wartime, with higher rates persisting among immigrants from high-prevalence countries. The discovery of antituberculosis medications accelerated this decline and brought into focus the possibility of eliminating tuberculosis in the United States.[1]

During the past 5 years, cases of active tuberculosis have increased substantially in the United States and other industrialized countries.[2] This increase was caused by the following: the spread of tuberculosis in such congregate settings as hospitals, shelters, and correctional facilities; declining public health infrastructure; immigration from areas of the world where tuberculosis remains endemic[3]; and the human immunodeficiency virus (HIV) epidemic.[4]

Tuberculosis is caused by the bacteria from the *Mycobacterium tuberculosis complex*. Without treatment, approximately 1 in 10 persons with tuberculosis infection will develop tuberculosis disease at some point in their lives; most of the remaining persons will never have symptoms nor develop active disease or spread the infection to others.[5] Persons who are infected with both HIV and *M tuberculosis* are at much higher risk, perhaps 7% to 10% per year, for developing active disease.[6] HIV-infected persons newly infected with *M tuberculosis* face an even higher risk for active disease.[7] Antibiotics, which must be taken for at least 6 months, cure active tuberculosis in 98% of patients with drug-susceptible disease and prevent tuberculosis infection from progressing to active disease in 60% to 90% of infected persons, even those who are HIV-infected.[8]

Based on these data, the principles of tuberculosis control are (1) to identify all persons with active disease and ensure their complete treatment and (2) to identify high-risk persons with tuberculosis infection and provide them with complete preventive treatment. Groups at highest risk for tuberculosis infection and disease are HIV-infected persons, close contacts of persons with active tuberculosis, persons from countries where tuberculosis remains common, and patients whose chest radiographs show scarring due to incompletely treated tuberculosis. There is little or no public health value in identifying persons who have tuberculosis infection or disease if complete treatment cannot be offered. Although socioeconomic improvements can decrease tuberculosis incidence, effective tuberculosis control programs can reduce tuberculosis incidence even faster.[9]

Four articles in this issue of the Journal illustrate the challenges and priorities of modern tuberculosis control. Buskin and colleagues reviewed risk factors for active tuberculosis among patients in King County, Washington.[10] The authors found that HIV infection is the strongest risk factor for tuberculosis disease and that increasing age, male sex, non-White race/ethnicity, birth in a foreign country, and several medical conditions (e.g., partial gastrectomy and low body weight) are associated with tuberculosis disease. The authors also found that smoking and alcohol consumption increase the risk for

Editor's Note. See related articles by Buskin et al. (p 1750), Leonhardt et al. (p 1834), and Cieslelski et al. (p 1836) and commentary by Comstock (p 1729) in this issue.

active tuberculosis, although they did not control for the potentially confounding effects of HIV infection and socioeconomic status. As the authors note, these data provide the framework for targeting control efforts.

Tuberculosis can affect all sectors of society, but it disproportionately affects socially and economically disadvantaged communities, which require expanded outreach and services. Many tuberculosis cases in the United States went undetected 50 years ago, and it is estimated that half of tuberculosis cases are undetected in developing countries. In contrast, today in the United States, even in underserved communities, most active tuberculosis cases are known; therefore, rather than directing our efforts at case finding, we should direct our efforts at ensuring that patients with active disease complete their treatment and at finding high-risk infected persons and ensuring that they complete preventive therapy.

Leonhardt and colleagues[1] describe a cluster of tuberculosis among persons associated with a high-risk group—crack cocaine users. Of 89 identified contacts of a single source case, 46 (52%) were tuberculin positive. The authors report that 13 (15%) of these contacts had active disease, including 5 of 6 children (83%) who were 5 years old or younger and 6 of 14 children (43%) who were 6 to 18 years old. This study confirms other investigations demonstrating that a single individual can infect dozens or hundreds of people.[2,4]

Although young children are known to be at increased risk for tuberculosis disease if infected, the very high rate of active disease among children suggests either a dose-response relationship between exposure and active disease or an overdiagnosis of tuberculosis disease in children, in whom standard diagnostic criteria are difficult to apply. A dose-response relationship has not been convincingly documented as being a risk factor for the development of active disease in exposed persons. It is intriguing that Leonhardt et al. documented a 57% tuberculin positivity rate among contacts considered to have a high exposure level to the source case, compared with a 9% positivity rate among contacts who had less exposure. It would be interesting to control for HIV status and compare the rates of disease progression between these two groups of infected contacts to explore the possibility of a dose-response relationship.

What is most impressive about the report by Leonhardt and colleagues is the authors' successful outreach into a difficult-to-reach population. With persistence, sensitivity, and a mobile van, public health workers gained the trust and participation of patients and their social network. This was essential in identifying and testing contacts and in helping 74% of infected contacts complete isoniazid preventive therapy. An even more rapid public health response may have prevented 12 secondary cases believed to have been part of the cluster.

Although the incidence of tuberculosis disease has been well characterized in the United States, there is limited information on the incidence of tuberculosis infection. Case rates do not necessarily reflect infection rates. Ciesielski and colleagues present provocative data on a medically underserved community—migrant agricultural workers.[5] In 1988, the authors tested 543 workers and found a tuberculin positivity rate of more than 30%.[5] To estimate the rate of new tuberculosis infection, in 1991 the authors, using a design somewhat analogous to the "capture-recapture" method,[6] attempted to retest workers who were tuberculin negative in 1988. Forty-six such individuals were retested. Of these, 14 (30%) were tuberculin positive or had active tuberculosis.

These data suggest that the annual risk of infection in this community could be as high as 10%. Small sample size, potential selection bias, inaccuracies of tuberculin testing, and the confounding effect of tuberculin boosting, which may occur in Bacillus Calmette-Guerin (BCG)-vaccinated individuals,[7] make these data difficult to interpret. It is clear, however, that tuberculosis in migrant farmworkers warrants further investigation and intervention. The report by Ciesielski et al. also highlights the importance of improved coordination and communication among health care workers, public health programs, clinics, and other agencies in serving difficult-to-reach populations.

Finally, Dr George Comstock, a pioneer in tuberculosis control, reviews past and prospective strategies for controlling the disease.[3] Physicians once debated whether tuberculosis was caused by heredity, the environment, or infection. Comstock astutely notes that although tuberculosis in an individual is caused by infection with *M tuberculosis*, tuberculosis in society and its control depend on environmental factors and that even genetic characteristics may be important and amenable to therapeutic intervention.[8] Although many recommended measures are complex, Comstock notes that "simplicity is likely to be the key to success."

Comstock calls for renewed investigation of the epidemiology of tuberculosis. Coming from an investigator who, as much as any other, defined our understanding of tuberculosis epidemiology,[3] this call is particularly compelling. There are many unanswered questions in tuberculosis epidemiology: Where does most transmission occur? Why are some patients and organisms so effective at spreading infection and disease? How can risk of reactivation best be predicted? Are immunosuppressed patients at increased risk for infection?

In 1980, many people incorrectly assumed that tuberculosis in the United States had been controlled; the case rate of tuberculosis in Central Harlem in New York City was 50 per 100 000 persons. In 1991, there was widespread concern about tuberculosis in New York City—the case rate for all of New York City was 50 per 100 000, and Central Harlem's rate had increased to 221 per 100 000. If we had been as concerned about Central Harlem in 1980 as we were about all of New York City in 1991, much of the city's epidemic might have been avoided. Similarly, if we were as concerned today about tuberculosis in the developing world as we are about tuberculosis in the United States, we could prevent cases here and abroad for decades to come. The tuberculosis bacterium infects approximately 1.7 billion people, causing about 8 million cases and 2 million to 3 million deaths annually worldwide, more than any other infectious agent.[9] With nearly 1 in 3 US tuberculosis cases occurring in foreign-born persons, we cannot afford to continue our policy of public health isolationism.

In the 1970s and 1980s, tuberculosis declined, and the programs that had been established in the United States for its control were disbanded.[4] As a result, we have seen a dramatic increase in tuberculosis and drug resistance in recent years. In response, federal, state, and local efforts have begun to reestablish effective tuberculosis control programs. As the disease once again begins to decline in the United States and leaves the front pages, our challenge will be to persevere. We must expand effective outreach programs such as those described by Leonhardt et al., provide services to underserved populations such as those described by Ciesiel-

...et al. Target services to groups identified by epidemiological studies such as the study by Buskin et al., conduct the epidemiologic investigations called for by Comstock, and work to improve the social and economic environment that provides the substrate for the tuberculosis epidemic in the United States and abroad.

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Toward an Epidemiology of Disablement

Finding out how many people with disabilities live in a population, what the preventable or intractable determinants of disability are, and what problems and unmet needs exist is not a straightforward task. The epidemiology of disablement, a global term covering impairment, disability, and handicap,[1] has developed slowly from multiple origins and within multiple disciplines. The articles devoted to disablement in this issue of the Journal illustrate the use of large data sets and epidemiological surveys to investigate its prevalence, distribution in the population, and consequences.[2–5] National surveys and large population catchment-area studies provide the data for analyses. In some instances,[4] other data sources provide contextual variables reflecting the social and economic conditions of the population.

Not surprisingly, these researchers have found a pattern for the distribution of disability or the restrictions in activity that is consistent with previous research on correlates of mortality and other indicators of health status.[6] Disability, measured as rates of activity limitation, increases with age and worsening socioeconomic conditions, and is unequally distributed by race and sex. Specific types of impairments, such as back pain, hand discomfort, and dermatitis, are more prevalent in some occupations than in others.[2] Specific combinations of disabilities and impairments, such as hip fracture in association with cognitive impairment, are associated with higher mortality.[5]

These findings prompt increased attention to health promotion and disease prevention focused on the needs of persons with disabilities.[7] Primary prevention activities are targeted to the occurrence of the disabling condition itself. Persons already affected by the condition, as illustrated in the articles in this issue,

Editor's Note. See related articles by Behrens et al. (p 1780), Bruce et al. (p 1796), Lafata et al. (p 1813), Marottoli et al. (p 1807), and Wagner et al. (p 1800) in this issue.

Commentary

Tuberculosis: Is the Past Once Again Prologue?

ABSTRACT

Tuberculosis has been considered the result of hereditary susceptibility, miasmas in the environment, and contact with contagious patients. During most of the latter half of this century, tuberculosis control efforts have concentrated almost exclusively on contagion by treating patients to make them noninfectious, treating latent tuberculosis to prevent reactivation, and in some countries, vaccinating uninfected persons to protect them from the consequences of infection. With the resurgence of tuberculosis in 1985, interest in all methods of tuberculosis control has been rekindled. Much remains to be discovered and much needs to be done. If renewed efforts succeed in again forcing tuberculosis rates downward, will we have the wisdom to eliminate tuberculosis in the United States, or will we relax and bring about another resurgence? (*Am J Public Health.* 1994;84:1729–1731)

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Diagnosis, treatment, prevention, and control of any disease depend to a large extent on what we think we know about the disease and our attitudes toward those afflicted with it. This certainly has been true of tuberculosis since it was first described by early medical writers. Throughout its long history, there have been three major theories regarding the etiology of tuberculosis. The etiologic factors involved in these theories are hereditary, environment, and contagion.

Two hundred years ago, western opinion was divided as to the cause of consumption or phthisis. The belief that there was an overriding hereditary predisposition was widely held throughout northern Europe and America. The major argument in favor of its hereditary nature was familial aggregation. Even though concentration of a disease in families can also result from a common environment of exposure, it was difficult to explain why many members of a family developed tuberculosis as they made the transition into adulthood and developed the disease at widely different times, when exposure to an infectious patient had occurred many years earlier. Consider the literary Bronte family of six children. All six died of tuberculosis, the first to die did so at the age of 11 years and the last, Charlotte, at age 39.[1] Although all of them were apparently infected by their father, the development of disease at long and variable intervals after exposure was so different from the fixed and relatively short incubation periods of other communicable diseases that it did not seem possible that contagion was the cause.

During this time, many tuberculosis patients from northern Europe went south for a cure, only to run into unforeseen difficulties. To the patients' surprise, much of Mediterranean Europe considered tuberculosis communicable. For example, Chopin and his lover, George Sand, ran into difficulties when he took Chopin to Majorca for tuberculosis treatment.[2] When Majorca residents discovered the nature of his illness, Chopin and Sand were evicted. No one could take them in, and even their rich friends refused to provide them with a carriage to take them to their ship. They and their goods had to make the trip in wheelbarrows.

Although reliable data are lacking, neither the hereditary nor the contagion theory of tuberculosis as understood at that time appears to have had an appreciable impact on tuberculosis during the 1700s. To attack the disease under the hereditary theory would have required proper selection of mates, a practice not widely adopted even during the heyday of eugenics. Isolation of infectious patients might have had some effect if the isolation had been more than sporadic and not directed mostly at those regarded as strangers.

The 1800s brought remarkable changes, as evidenced by the decline in British tuberculosis death rates from approximately 450 per 100 000 in 1810 to 250 per 100 000 in 1890.[3] This impressive improvement probably resulted from changes brought about by the sanitary reform movement, a reaction to the adverse effects of the Industrial Revolution. While the growth of industries

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Editor's Note. See related editorial by Frieden (p 1721) and articles by Buskin et al. (p 1750), Leonhardt et al. (p 1834), and Ciesielski et al. (p 1836) in this issue.

brought remarkable benefits, the displacement of workers and their families into cities ill-equipped to handle such an influx resulted in appalling living conditions. Sanitation was primitive. In many cities, pigs, dogs, and goats roaming the streets were the accepted form of garbage disposal. In some cities, water was available only from a pump in the street. Rooms were small, poorly lit, and poorly ventilated, and crowding within them often was extreme. Under these conditions, it is not surprising that such infectious diseases as measles, diphtheria, tuberculosis, and typhoid fever flourished. These diseases struck hardest at the inner-city poor, but did not spare the upper classes. Because of their endemicity and familiarity, they were accepted as being one of the normal hazards of life and provoked little public reaction.

It was the great epidemics, notably cholera and yellow fever, that struck fear in the hearts of all levels of society. Dead bodies in the streets and the fetid crowded living quarters were believed to create miasmas, which could spread disease throughout the air. Leading citizens began to see that improving the health and housing of the working classes was essential for protecting the rest of society.[4] Health efforts, public and private, focused largely on controlling the sources of miasmas. None of the sanitary reform measures were thought to be related to tuberculosis, which was still considered to involve a hereditary predisposition. Nevertheless, it now seems certain that measures to ensure fresh air and sunshine, relieve overcrowding, and encourage people to cover their mouths when coughing or sneezing must have been beneficial by reducing both the frequency and intensity of exposure to infectious patients.

The discovery of bacterial causes for many diseases in the 1880s sounded the death knell for the environmental miasma theory and relegated the hereditary theory of tuberculosis to the background. Attention was focused on sick individuals as prime sources of contagion. State and local governments had scientific reasons to become involved as part of their mandate to protect public health. By 1913, every state and territory in the United States had a health department.[4]

One of the earliest tuberculosis control programs in which state and local health departments were involved, and one of the most successful, was to keep milk from spreading tuberculous infection. This program, combined with pasteurization of milk, has been so successful that it has been called "man's greatest victory over tuberculosis."

Tuberculosis control efforts directed at human populations understandably were not as draconian as the test and slaughter program among cattle. A major component was the sanatorium movement. As the realization grew that tuberculosis was infectious and that adequate treatment of cases and protection of contacts was difficult to obtain at home, state and local governments began to provide sanatoria or tuberculosis units in general hospitals. The peak capacity of nearly 120,000 beds was reached in 1954 for an estimated 150,000 known active tuberculosis cases.[4] Although it can be argued that the majority of infections of tuberculosis contacts occur before the disease is diagnosed, the isolation of two-thirds of the cases after diagnosis must have had an appreciable beneficial impact by reducing the subsequent tuberculosis caseload.[4]

The role of local health departments in tuberculosis control was largely confined to home nursing visits to reduce the transmission of infection from patients to their household contacts. To provide this service, compulsory reporting was essential. First introduced by the Michigan State Board of Health in 1893,[4] compulsory reporting of tuberculosis cases had become virtually universal in the United States by 1917.

Major improvements were associated with the official establishment of the Division of Tuberculosis in the US Public Health Service in 1944.[4] The agency's budget included substantial amounts to help state and local health departments establish screening, diagnostic, and treatment facilities. By 1960, there were nearly 1200 tuberculosis clinics in the United States; two thirds of them were operated by local and state health departments.[4] Even so, this number represented an average of less than one clinic for every three counties.

In keeping with the interests of nonmedical people in the sanitary reform movement and the social and economic implications of tuberculosis, attempts were made to establish a national tuberculosis organization.[4] Medical interests in treatment and the public health belief in prevention were combined with the founding of the National Association for the Study and Prevention of Tuberculosis in 1904.[4,13] At first, the association believed that the tuberculosis movement should remain a private affair, but gradually it became a strong supporter of official tuberculosis control, reserving for itself the functions of demonstration, education, and legislation.

The Arden House Conference in 1950, with its slogan "Chemotherapy is the key," focused more attention on the individual patient with tuberculosis.[4] The remarkable efficacy of multiple drug chemotherapy came close to guaranteeing cures if patients completed recommended regimens. Outpatient chemotherapy for patients and preventive chemotherapy for their infected contacts became the mainstay of tuberculosis control.

Unfortunately, adherence to medication regimens that lasted for months proved easier to recommend than to attain. The homeless, immigrants, drug addicts, and working poor, all unduly susceptible to tuberculous infection and disease, often had problems that were much more important to them than their tuberculosis. The pervasive decrease in funds for tuberculosis control during the 1980s led to a fallback position of dropping all control efforts that were not maximally cost-effective. In some populations, even the most cost-effective procedures were seriously curtailed.[4]

In the 1990s, reports of tuberculosis outbreaks in various health care settings initiated another shift in policy.[4,14] Several of these outbreaks involved transmission of tubercle bacilli that were resistant to first-line drugs, and in some instances to several second-line drugs as well. Although the high rates of infection and disease, accompanied by high case fatality rates, were concentrated among persons infected with the human immunodeficiency virus, these rates caused tremendous concern, especially among health care workers.

As a result, more attention is being paid to the environment and the modern miasma, airborne droplet nuclei. As often happens, there is little emphasis on primary prevention. Having patients cover their mouths when coughing is rarely accorded more than a line in published recommendations;[4,15] virtually nothing is being done to instill this habit in the general public. Emphasis is given to removing infected air from spaces occupied by infectious patients, along with air sterilization by ultraviolet irradiation. High-efficiency masks, which obscure facial expressions and interfere with speech, are being recommended for health care workers. Such recommendations, however, come with little concern for the

potentially negative effect on patient-provider communication and rapport.

Well-intentioned as these recommendations are, it is tragic that we know so little about the need for them and their effectiveness in practice. Are the relatively few reported tuberculosis outbreaks the tip of the iceberg? Are there other equally hazardous situations that are outbreaks waiting to happen? Or are the reported outbreaks truly rare events? How do situations that have resulted in outbreaks differ from those that have not? An epidemiologic investigation of clinics, with and without outbreaks, could provide some useful answers.

The general decrease in epidemiologic studies of tuberculosis during the past 2 decades leaves many other pertinent questions unanswered. Most estimates of the risks of becoming infected with tubercle bacilli and of subsequently developing tuberculosis are based on data collected in the 1950s and 1960s. Current studies of the risks of tuberculous infection and disease are needed. Routine data systematically collected and analyzed by health departments, hospitals, and clinics could provide the knowledge we need to solve some current problems of tuberculosis control.

All aspects of tuberculosis control, however, need to be considered. More progress might be made if we could broaden and keep in balance all three of the historic points of attack—heredity, contagion, and environment. It now seems likely that hereditary resistance to tuberculosis depends on the cellular immune system. Recent advances in this area raise hopes of increasing resistance through a variety of immune mechanisms. Improved methods of rendering tuberculosis patients noncontagious are also needed. Not only are more efficacious treatment regimens desirable, but equally important are behavioral changes that make uncovered coughs socially unacceptable and social changes that alleviate the problems that now make tuberculosis a secondary problem to many people. It seems likely that sanatorium treatment again may be needed for patients whose multidrug-resistant organisms make chemotherapy difficult or even impossible. Perhaps there should be hospicelike shelters for homeless tuberculosis patients.

Environmental needs are related to controlling the miasma of infectious droplet nuclei. Interest in air sterilization is reawakening after decades of dormancy. Personal protection by masks must be evaluated under practical conditions in the field. In all these areas, simplicity likely will be the key to success. Above all, we must find a way to bring prevention methods to persons who need them most—the socially and economically disadvantaged. Solving their tuberculosis-related problems and reducing the consequent risks to others remain a major challenge for the future.

A detailed strategic plan to accomplish these goals and reduce the annual tuberculosis case rate in the United States to less than one case per million by the year 2010 was published more than 5 years ago.[18] Its wide-ranging recommendations include those related to heredity, contagion, and environment. Some important features of the plan have been implemented, notably increased funding for tuberculosis control and research, improved surveillance, wider application of directly observed treatment, and respiratory protection of health care workers. Many other recommendations have yet to be implemented. Their future, like the future of many public health programs and research, remains clouded as the debate on health care reform concentrates almost exclusively on illness care reform.

Additional uncertainty arises from our questionable ability to learn from the past. If increased tuberculosis control efforts succeed in sending tuberculosis case rates downward, as happened in 1993,[19] will we maintain a level of control that is long and intensive enough to reach our goal of eliminating the disease and reaping subsequent attendant social and financial benefits? Or will we once again be lulled into a false sense of security, relax our efforts, and thereby allow tuberculosis to make another comeback?

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Editorials

The U-shaped Curve of Concern

Tuberculosis (TB) is back with a vengeance. New case rates are rising precipitously: New York City, 38%; California, 16%; Texas, 17%; USA, 9.4% (1990). This is a paradox because TB is relatively easily diagnosed, treated, controlled, and prevented. Recognizing that even in the face of this rise TB occurs in well-defined population groups, the Secretary of Health and Human Services has endorsed a national plan for the elimination of TB by the year 2010 (1). To support this goal many diverse health care organizations have, in an unprecedented manner, adopted and endorsed a core curriculum on the proper way to diagnose and treat this disease (2,3).

In this issue of the REVIEW, Brudney and Dobkin analyze what may be responsible for the rising rates (4). Their experience is so bleak that at first reading one might mistake it for a theoretical worst-case scenario for TB control or at least a description of TB control in a developing country. However, they present a careful, prospective analysis of a cohort of 224 consecutive patients recently admitted to a large, sophisticated university affiliated urban hospital center in the USA. Their patients, reflecting the usual urban practice of tuberculosis, were 53% alcoholic, 64% intravenous drug and/or crack cocaine abusers, 68% homeless or unstable housing, and 50% human immunodeficiency virus (HIV) infected. Ultimately 178 patients were discharged on TB treatment, but 159 (89%) were lost to follow-up, failing to complete therapy. Of these, 48 patients were readmitted within 12 months with confirmed active TB; 40 of these were discharged on treatment, and another 35 were lost.

The findings of Brudney and Dobkin are extremely important in that they show, in a well-documented manner, that the rise of TB in New York City, and by analogy other urban areas of the US, is not due only to the HIV or homeless problems usually cited by the media, but it reflects the total failure of a public health system, even in the face of previous experience and warnings. These previous experiences and warnings are the most fascinating aspects of their report. They document the year by year promises and recommendations by august study groups and task forces compared with the stark reality of piecemeal slacking of the program's resources that are directly related to the rise in cases.

The Brudney-Dobkin experience precisely reflects what seems to be a ubiquitous occurrence in public health practice. First, evaluation indicators of a public health program show improvement leading to diminishment of compelling need. Then resources providing fuel and direction for the program are removed. Finally, the incidence of the disease "controlled" begins to rise in proportion to the diminished resources. This phenomenon has been called "the U-shaped curve of concern" (5). One can picture the letter "U" and then compare it with a graph of actual US TB case rate data in figure 1. For this figure, one should recognize that in 1969 Federal Project Grants for TB Control of over $20 million were provided. From 1970 to 1972 these were completely phased out in favor of block grants to the states which were allocated to TB only if a local priority. Therefore, total TB spending diminished heavily over the next 20 yr. By 1982, Federal Project Grants were again appropriated but only for $1 million. In 1983, the appropriation was raised to $5 million, which was continued. Finally, in 1991, a total of only $9.1 million was appropriated, although Congress had authorized $36 million. For the past several years, the American Lung Association has requested the full $36 million authorized, but this request has had to compete with numerous other (political) priorities; the results have been so far unfavorable.

These data can be compared with those of Brudney and Dobkin (4) redrawn for comparison in figure 2, which also is a U-shaped curve. When considering this figure, one should recognize that a major new thrust in TB control through the Lindsay task force occurred in 1968. However, the New York City fiscal crisis followed in 1974 to 1978, and termination of the New York State TB Control Contract, which previously supported 50% of the program, occurred in 1979 (4). Annual TB spending in New York was $40 million between 1968 and 1973, and $23 to 25 million in 1978, not including inflation. Direct Federal Public Health Service support to New York was $1.4 million in 1979 and $283,000 in 1980 (4).

TB obviously is a serious worldwide problem. One-third of the world's population is infected with the TB bacillus; 8 million new cases occur annually with 2.5 million deaths caused by the disease (6). The worldwide leading cause of death of *any* infectious disease, TB respects no national or state boundary. Technology for diagnosis, treatment, care, and control are easily available, but these *require* several modalities beyond drugs: directly administered, supervised therapy; careful contact follow up; use of community health workers for surveillance and treatment; hospitalization when necessary; medication provided without cost to the patient; and compliance enhancements and enablers to assure drug taking and follow-up (3). Even including all these critical aspects, treatment of TB, recently described by the World Bank as "one of the most cost effective health interventions available" (7), is cheap. Unfortunately, these critical factors are usually the first to be eliminated for budgetary reasons.

When such support is removed, case rates rise, and increased disease spread occurs. Because medication taking without supervision becomes inconsistent and sporadic, a rise in multidrug resistance with transmission to contacts can be anticipated and indeed has recently and alarmingly been described (8–10) even with

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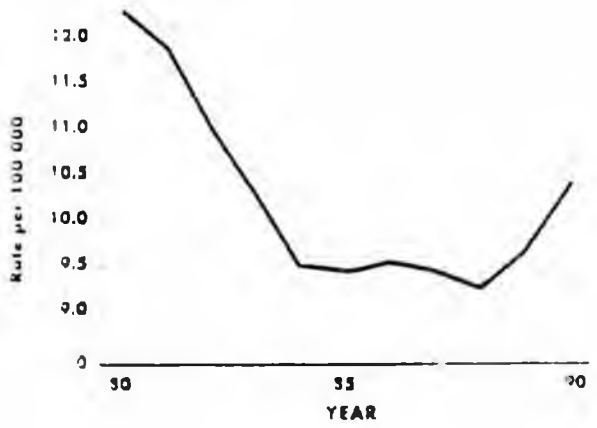

Fig 1. USA National Tuberculosis Rate/100 000 population 1980 to 1990 (see text)

Fig 2. New York City Tuberculosis Rate/100 000 population 1969 to 1989 adapted from (4) (see text)

documented transmission of multidrug-resistant TB to health care workers (9).

Brudney and Dobkin document that the rise in TB rates in their center is largely caused by the failure of the system although the knowledge and technology to reverse this trend have been readily available. But this is the state of TB control in the United States in the 1990s. The problem is amply defined and well documented, and the solution attainable with dedication, resources, and commitment. Many programs have long demonstrated they have the dedication. Unfortunately and urgently, we still await the resources and commitment.

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Tuberculosis in Alaska Natives

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INTRODUCTION

In the 1940s and 1950s an epidemic of tuberculosis devastated Alaska's indigenous people. A special health survey team reviewed the health status of Alaskans in 1954 and found, "Alaska is unfinished business. It is our laxity as citizens which is reflected in congressional indifference. There has been tolerated for too long the disgraceful burden of disease we know how to prevent."(1) The most pressing health problems fell disproportionately on Alaska Natives. "In terms of his productive life or his degeneration and death, the more urgent of these questions concerns the Native. Upon him falls the greatest burden of preventable disease, unnecessary crippling and premature death in all Alaska."(1) Finding severe deficiencies in many areas, the team documented absence of safe water, proper disposal of sewage, and adequate housing, severe problems of dental disease, inadequate nutrition with widespread malnutrition, and inadequate support for the mentally ill. But chief among its findings was the epidemic of tuberculosis, ...the Alaskan scourge."(1)

As a result of the devastating conditions documented by the health survey team, emergency programs were initiated by the territorial government of Alaska and by the United States government to address the most pressing and urgent problems of public health in Alaska. During the 45 years since publication of this report, almost miraculous improvements have occurred in the health status of Alaska Natives. But tuberculosis remains a serious health problem in the 1990s and will continue to be a formidable challenge in the future.

BACKGROUND

Alaska, with a land area of more than 586,000 square miles, is about one-fifth the size of the contiguous United States. The 1990 population (550,043) comprised about 84% non-Natives and

16% Natives. Non-Native Alaskans include white, black, hispanic, Asian/Pacific islander, and other racial groups. Alaska Natives include Eskimo (Inupiat and Yupik), American Indian (Athapaskan, Tlingit, Haida, Tsimshian, and other North American tribes), and Aleut. Eskimos are comprised of two major groups: the Inupiat who inhabit the northern and coastal areas of Alaska and the Yupik Eskimos who live in the southwest area of Alaska. Indians comprise the Athapaskan Indians who occupy the vast areas of the interior Alaska and the Tlingit, Haida, and Tsimshian who occupy the coastal area of the panhandle. Aleuts include the residents of the Aleutian Chain of Alaska and the Pribilof Islands. (2,3)

Alaska has the youngest population in the United States: the median age is 26.5 years and more than half the population is 15- 39 years of age. About 62% of Alaskans live in three cities: Anchorage (44%), Fairbanks (13%), and Juneau (5%). Of the 85,698 Alaska Natives, 70% live in 171 villages with populations ranging from 30 to 3,500. (4)

Health care facilities are located in the larger population hubs throughout the state. The Indian Health Service (IHS) maintains a tertiary care hospital in Anchorage and supports 11 small hospitals in rural cities. The IHS provides medical care for Alaska Natives and other U.S. Public Health Service (USPHS) beneficiaries as well as some non-Natives in rural areas. There are four major referral hospitals in Alaska: the Alaska Native Medical Center (IHS) in Anchorage, two private hospitals in Anchorage, and one private hospital in Fairbanks. Fourteen community hospitals provide care for persons living in smaller towns.

HISTORY OF TUBERCULOSIS IN ALASKA

It is commonly accepted that tuberculosis was introduced to a susceptible population of Alaska Natives in the eighteenth and nineteenth centuries where it swept through the population with devastating effect. Whether tuberculosis existed in Alaska Natives prior to contact with European populations is unknown and may never be proved. (5) The devastating impact of tuberculosis among Alaska Natives was documented for the first time in the 1930s. During 1926

to 1930, 982 (35.5%) of 2,767 deaths among Alaska Natives were recorded as due to tuberculosis, an average annual rate of 655 per 100,000. In comparison, tuberculosis was recorded as the cause of death for 80 (4.7%) of 1,704 deaths among Alaskan whites, an average annual rate of 56 per 100,000. (6) In 1946 tuberculosis was listed as the cause of death on 43% on all death certificates for that year. (7)

In 1950 the population of Alaska was approximately 130,000 of which 35,000 were Alaska Natives. Alaska Natives were widely dispersed throughout the state: an estimated 8,300 persons lived in villages of less than 100 people, and only 1,933 Natives lived in urban areas.(1)

In 1950 the death rate from tuberculosis in Alaska Natives was 673.3 per 100,000 compared to 15.1 per 100,000 in Alaskan whites and 17.9 per 100,000 in whites in the United States. In 1954 the state of Alaska recorded 2,606 persons, with active or probably active tuberculosis for an overall prevalence rate of 1,785 per 100,000. Of these 2,606 persons 2,363 were Alaska Natives for a prevalence rate of 6,474 per 100,000, or greater than 6% of the Native population. In 1953 chest x-ray surveys of 200 Alaska national guardsmen, Alaska Native men between the ages of 18 and 43 years, provided evidence of tuberculosis in 40% and probably active tuberculosis in 10%. (1)

In 1952 a survey of PPD skin test results recorded from 1948 to 1951 was conducted of children through age 14 throughout rural Alaska. (8) The proportion of children in the 5 to 8-year age group with positive PPD skin test results ranged from 89 percent for Eskimos to 22 percent for Southeast Indians. (Table 1)

In response to these staggering rates of tuberculosis and their devastating impact on Alaska Natives, an emergency program was initiated in 1953 and 1954. Emphasizing principles of case finding, isolation, and treatment, a dramatic reduction in new cases of tuberculosis occurred over the following years. (9-11) Key to these efforts were innovative programs using portable chest x-ray surveys in rural villages, institution of ambulatory chemotherapy using INH and PAS with supervised home care, and initiation of chemoprophylaxis of household contacts. (12) From

1948 to 1951 the Alaska Department of Health engaged in a limited program of BCG immunization. The BCG program was abandoned because of administrative difficulties and failure to induce delayed hypersensitivity skin test reactions in vaccine recipients. In 1957 a survey was done to examine skin test positivity rates among Alaska children. Extensive record reviews were done to identify those who had received BCG immunization.

Among 2,211 individuals between the ages of 0 and 19 years only 350 (15.8%) had received BCG vaccination. Among children age 5 to 9 years, 171 (39%) had been vaccinated against BCG. Of the 1,861 individuals who had not been vaccinated with BCG, 77% were tuberculin skin test reactors (PPD $\geq$5 mm). Among those older than 10 years, 94% were tuberculin skin test reactors. (13)

Intensive case finding using statewide x-ray screening surveys, supervision of treatment of persons with tuberculosis infection or disease using INH and PAS, and vigorous use of isolation resulted in a rapid decline in tuberculosis morbidity and mortality rates and in rates of infection in children. A series of tuberculin skin test surveys in southwest Eskimo children, 3 years of age or younger, demonstrated rapid declines in tuberculin reactivity among successive birth cohorts. Of the first sample of children under age 4 tested during 1949-51, 34.4% were tuberculin reactors. (7) By 1957, only 14.4% of children in this age group were tuberculin positive; and by 1960 the infection rate in children born since the previous survey had fallen to 1.7% (8) (Table 3).

By 1970, the rate of new active cases of tuberculosis among Alaska Natives had fallen to 154 per 100,000. The rate dropped further to 79 cases per 100,000 by 1985. The number of newly identified tuberculin reactors also declined to low levels. Of 71,316 Alaskan children of school age and younger who were not known to be tuberculin positive and who were tuberculin skin tested in 1986, only 152 (0.21%) were tuberculin reactors. In the same year only 937 (4.83%) of 18,431 adults not previously known to be tuberculin reactors were found to be tuberculin positive. In 1990 only 5 (0.22%) of 2,300 children in southwest Alaska were tuberculin reactors.

TUBERCULOSIS IN ALASKA – 1990s

In 1952, Alaska's tuberculosis incidence rate was 400 cases per 100,000 population (Figure 1), a level undoubtedly matched or exceeded during earlier years of the century. The incidence rate among the Native population for that year was a staggering 1,854 cases per 100,000; or, stated differently, nearly 2% of the Native population in 1952 was diagnosed as having tuberculosis disease.

Incidence rates dropped sharply during the next six years to slightly more than 100 cases per 100,000. Rates dropped by half again to below 50 per 100,000 in the late 1960s.

The succeeding two decades were marked by a continuous, slow decline in annual tuberculosis morbidity. (Figure 2) In 1988, only 51 cases of tuberculosis (84% of which were confirmed by culture) were reported, yielding a crude rate of 9.5 cases per 100,000, the lowest ever recorded in the state.[1] During the five year period from 1986 through 1990, Alaska's mean age-adjusted incidence rate exceeded that of the entire United States. (Table 4)

Alaska Natives still bear a burden of tuberculosis infection and disease that is disproportionate to their number. Although they comprise only 16% of the population, they have accounted for 50 to 90 percent of all tuberculosis cases reported in Alaska in recent years. Race-specific tuberculosis case rates (per 100,000) during 1990 were as follows: whites, 1.4; blacks, 5.3; and Alaska Natives, 60.7, SEA/PI, 58.6. The age-adjusted rate for the United States as a whole was 9.1. The persistently high rate of tuberculosis disease among Natives has been, in part, a legacy of the remarkably intense transmission of M. tuberculosis during the first half of this century. However, Alaska's resident Asians and Pacific Islanders, most immigrants from South Korea

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[1]Tuberculosis case definition: A case that (1) is laboratory-confirmed or (2) in the absence of laboratory confirmation, meets certain clinical criteria, including (a) a positive tuberculin skin test, (b) presence of clinical evidence compatible with tuberculosis, (c) treatment with two or more antituberculosis medications, and (d) completed diagnostic evaluation. (14)

and the Philippines who account for only about 3-4% of the state's population, had tuberculosis rates very nearly as high as those of Alaska Natives: 58.6 cases per 100,000 during 1990. (Table 5)

During 1988-90, the mean age of 178 reported tuberculosis cases was 36.0 years. Slightly fewer than half the cases were under the age of 35 years. If they had been identified before progression to tuberculosis disease, they would have been eligible for isoniazid preventive therapy and their disease was, therefore, potentially preventable. It is of special concern that 48 (27%) of 178 cases identified during 1988-90 were under 20 years of age, implying active transmission to children and adolescents; 43 (90%) of the 48 were Alaska Natives. (Table 4)

In general, the geographic localization of tuberculosis disease has reflected the varying prevalence of tuberculosis infection/disease in Alaska during the epidemic period of the first half of this century. Nearly half of all tuberculosis cases identified during 1988-90 were residents of western or far northern Alaska (a broad area extending from the Yukon-Kuskokwim Delta north through the Seward Peninsula and Kotzebue Basin, and including the Arctic Slope). Twenty-three percent of the cases were reported from Southcentral Alaska, where more than half the population of the state lives; 20% from Interior Alaska; 8% from Southeast Alaska; and 2% from the Aleutian Chain.

Pulmonary tuberculosis was the only or predominant form of the disease reported in 157 (88% of the) cases reported during 1988-90. The frequency of extrapulmonary tuberculosis cases during the 3-year period was as follows: pleural, eight (4.5%); lymphatic, five (3%); genitourinary, four (2%); pericardial, one; peritoneal, one; bone/joint, one; and soft-tissue, one. Meningeal tuberculosis is rare in Alaska: six cases, two of whom were children, were reported during the 11-year period from 1980 through 1990.

Of the 178 patients reported during 1988-90, 25 (14%) had histories of prior tuberculosis disease and had either failed to complete recommended therapy or had been treated with now-obsolete antibiotic regimens.

Antibiotic-resistant strains of M. tuberculosis were cultured from only eight individuals (4.5%). Five patients had strains which were resistant to isoniazid alone; of these, four were related to an outbreak of isoniazid-resistant tuberculosis in the village of Chevak in 1989. Two cases had isolates resistant to streptomycin alone; and one patient, an immigrant from South Korea, had organisms resistant to both isoniazid and rifampin.

From 1987 through late 1991, thirteen villages (excluding the major population centers of Anchorage, Fairbanks, and Juneau)--all located in western Alaska--had clusters of three or more tuberculosis cases (range, 3-22) believed to be related to one another. During 1990, three villages, each with fewer than 800 residents, accounted for 23 (34%) of the 68 tuberculosis cases identified in Alaska during that year.

Investigations of outbreaks in four western Alaska villages during 1987-91 have yielded useful insights into village-based tuberculosis clusters. (Table 6) These four outbreaks accounted for 50 cases, 33 (66%) of which were culture-confirmed. Persons affected in these outbreaks were young, the mean age of cases ranged from 9.6 years in the Chevak outbreak to 28.7 years in the St. Paul outbreak.

Two of the outbreaks (in Chevak and St. Paul) were detected through routine annual tuberculin skin-testing of school children, and two (in Holy Cross and Savoonga) were detected through investigation of contacts of an identified tuberculosis case.

The 1989 Chevak outbreak, caused by an isoniazid-resistant strain of M. tuberculosis, was school-based and affected primarily school children. The index case, a 14-year-old boy with a cavitary pulmonary infiltrate, was initially overlooked because his respiratory symptoms were not prominent and because investigators expected to find an adult index case. Of 150 previously tuberculin-negative students in the school, 50 (33%) were infected during the outbreak; four developed tuberculosis disease. One hundred four persons were offered preventive therapy with rifampin, and 85 (82%) completed it under supervision. No additional cases have occurred during more than two years of follow-up.

The 1990-91 Savoonga outbreak, resulting in 18 culture-confirmed tuberculosis cases, had as its index case a young adult male with a past history of pulmonary tuberculosis. At the time of his diagnosis in mid-1990, he reported a two-year history of cough which had been misdiagnosed as bacterial bronchitis in October 1989 and in March 1990. The index case in the 1990 St. Paul Island outbreak was a documented tuberculin reactor who had had a productive cough for at least six months prior to diagnosis in late 1990. During that period he had been evaluated for his respiratory symptoms on two occasions by health-care providers, neither of whom considered the diagnosis of tuberculosis disease.

DISCUSSION

Public health programs implemented in Alaska in the 1950s to control the epidemic of tuberculosis in Alaska Natives largely succeeded. Tuberculosis remains a persistent problem, however, complicated by the formidable logistics of delivering medical and public health services in the vast areas of the arctic.

The goal of the Alaska Division of Public Health to eradicate tuberculosis remains elusive. The epidemiology of tuberculosis in Alaska has changed. Although Alaska Natives account for more than 80% of cases, their disease rates are similar to those of Southeast Asian immigrants. New cases of tuberculosis continue to occur in Alaska Natives who are less than 35 years of age. Because preventive therapy with isoniazid is effective, these cases all are potentially preventable.

Cases of infectious, pulmonary tuberculosis continue to occur, primarily in two groups -- younger adults aged 20-35 years who have severe dependency on alcohol and extremely poor compliance with treatment recommendations, and Alaska Natives older than 50 years who have long standing tuberculosis infection and who may have received incomplete treatment or treatment with drug regimens less effective than those now available.

Alaska Native children now have an extremely low prevalence of tuberculin skin test reactivity. Our recent experience with several village outbreaks has demonstrated the potential for infectious

cases to infect large numbers of susceptible individuals. The index cases in these outbreaks have been individuals in the two high risk groups. Village outbreaks have been discovered through routine, periodic skin testing of school children with epidemiologic investigation of converters, or through epidemiologic investigation of reported infectious cases.

As the incidence of tuberculosis disease has declined, control of the disease has become a problem that is less technical in nature than logistical. Current antibiotic regimens, the use of supervised therapy, thorough contact investigations, and tuberculin skin-testing programs have been effective in reducing substantially the gross burden of tuberculosis disease in Alaska. However, pulmonary tuberculosis in residents of rural Alaska villages--the great majority of which have clinics without x-ray facilities--is not infrequently diagnosed belatedly, either through misdiagnosis as "acute bronchitis" or through failure to consider tuberculosis in the differential diagnosis. Consequently, patients with pulmonary tuberculosis may remain symptomatic and infectious for months before a correct diagnosis is made. Delays in diagnosis result in greater numbers of contacts with tuberculosis infection and tuberculosis disease. This has the effect of producing clusters of tuberculosis cases, or localized outbreaks.

Experience in controlling tuberculosis in Alaska led to innovative strategies to implement programs. Critical was the development in 1950s of ambulatory treatment under supervision by selected friends in remote villages. The benefit of this program was not only the improved compliance with treatment, but also the evolution over 20 years of the Alaska community health aide program that has become an integral component of Alaska's medical and public health delivery system.

Because of the vast distances, remote villages, limited transportation, and high costs, reliance on village-based supervision of treatment is a key component of successful tuberculosis control. Community health aides are paid to provide directly observed treatment to all individuals with active disease and to those receiving isoniazid preventive treatment.

Routine use of chest x-ray surveys was discontinued in Alaska in 1986 when a review determined the lack of efficacy and high cost. (13) Essential is the availability of portable radiography to enable chest x-rays to be obtained in villages during investigations of infectious cases.

A critical component to tuberculosis control is routine annual tuberculin skin testing of school children. Because BCG vaccination was used in Alaska for only a short time before being abandoned in 1951, routine tuberculin skin testing provided a reliable and inexpensive surveillance system for detection of disease transmission. Trained nurse and physician epidemiologists investigate all cases of tuberculin skin test conversion to find the index case and establish treatment regimens.

Today's clinicians are far less experienced in recognizing tuberculosis. High turnover among primary care health providers in rural villages complicates the absence of familiarity with diagnosis and management of tuberculosis.

The Tuberculosis Control Program of the Alaska Division of Public Health has fully adopted and implemented recommendations of the United States National Centers for Disease Control, "Strategy for Elimination of Tuberculosis in the United States." (14-16) Key components are:

1. Rapid diagnosis and meticulous treatment under direct observed therapy of all culture-confirmed cases of pulmonary tuberculosis.
2. Identification, through epidemiologic investigation of all cases of pulmonary tuberculosis, of individuals who have been infected and need appropriate chemotherapy in order to prevent future tuberculosis disease.
3. Effective surveillance to detect new transmission of tuberculosis through routine, periodic, annual skin testing of school children, and vigorous application of epidemiologic investigation to identify cases actively transmitting tuberculosis whenever a skin-test converter is identified.

4. Directly observed therapy of all individuals to ensure meticulous compliance with drug treatment recommendations.
5. Vigorous use of prophylactic INH to prevent tuberculosis among those recently infected.

The devastating epidemic of tuberculosis that ravaged Alaska Native people in the 1940s and 1950s has been largely controlled. Only vigorous application of available public health strategies can be expected to reduce further the transmission of Mycobacteria tuberculosis. Elimination of tuberculosis from Alaska is a realistic goal but will be a slow process requiring patient and thoroughly applied public health interventions.

Table 1. Skin Test Positivity (8)	
Inupiat Eskimos	67%
Yupik Eskimo	89%
Athapaskan Indian	65%
Southeast Indian	22%
Aleut	36%

Table 2. Extent of BCG Vaccination in Alaska (13)

Age Group	BCG Number Vaccinated	Total Children	Percent (%)
0-4	71	460	15.4
5-9	171	436	39.2
10-14	79	333	23.7
15-19	23	220	10.5
0-19	350	2,211	15.8

Table 3. Prevalence of tuberculin sensitivity, by age group, Yukon-Kuskokwim Delta, 1949-1990 (9)

Age (years)	1949-51	1957	1960	1990
3-4	80.0%	49.0%	8.5%	0.7%
5-6	90.2%	74.2%	33.0%	0.4%
7-8	91.8%	83.3%	68.8%	0.9%

Table 4. Mean tuberculosis incidence rates in Alaska and the United States, by age group, 1986-1990

Age Group (Years)	Alaska Native	Total Alaska	U.S.A.
0-4	22.9	7.0	3.4
5-9	30.8	9.5	1.7
10-14	24.5	8.1	1.3
15-19	45.8	13.3	2.8
20-24	24.8	7.2	6.4
25-34	53.1	9.6	9.8
35-44	55.6	8.0	11.0
45-54	100.2	17.2	12.5
55-64	120.7	24.4	13.5
65+	112.5	38.7	20.4
Total	61.8*	14.7*	9.1*

*Rate age-adjusted to 1980 U.S. population.

Table 5. Tuberculosis Cases and Incidence Rates* in Alaska, by Race, 1981-1990

	White		Black		Native		Asian	
Year	No.	(Rate)	No.	(Rate)	No.	(Rate)	No.	(Rate)
1981	15	(4.5)	1	(6.8)	56	(80.7)	11	(90.6)
1982	20	(5.6)	4	(25.5)	60	(81.6)	12	(93.3)
1983	23	(6.0)	1	(5.9)	62	(78.5)	12	(86.8)
1984	19	(4.7)	0	(0.0)	46	(55.1)	14	(95.9)
1985	22	(5.3)	1	(5.5)	68	(79.0)	19	(126.2)
1986	13	(3.1)	3	(16.1)	33	(42.9)	23	(150.5)
1987	10	(2.4)	3	(16.4)	34	(43.0)	14	(93.3)
1988	10	(2.4)	0	(0.0)	35	(43.1)	6	(40.5)
1989	2	(0.5)	0	(0.0)	50	(59.9)	7	(46.9)
1990	6	(1.4)	1	(5.3)	52	(60.7)	9	(58.6)

*Rate per 100,000 population

Table 6. Selected Tuberculosis Outbreaks, By Village, Alaska, 1987-91

	Holy Cross	Chevak	Savoonga	St. Paul
Year	1987	1989	1990	1990
Number of Cases	12	9	22	7
Number of Culture-Confirmed Cases	5	4	18	6
Mean Age in Years (Range)	18 (1-48)	9.6 (0.3-18)	22.1 (1-35)	28.7 (0.9-57)
Number of Tuberculin Converters	13	63	36-98	7
How Outbreak was Detected	Contact Investigation	School Screening	Contact Investigation	School Screening

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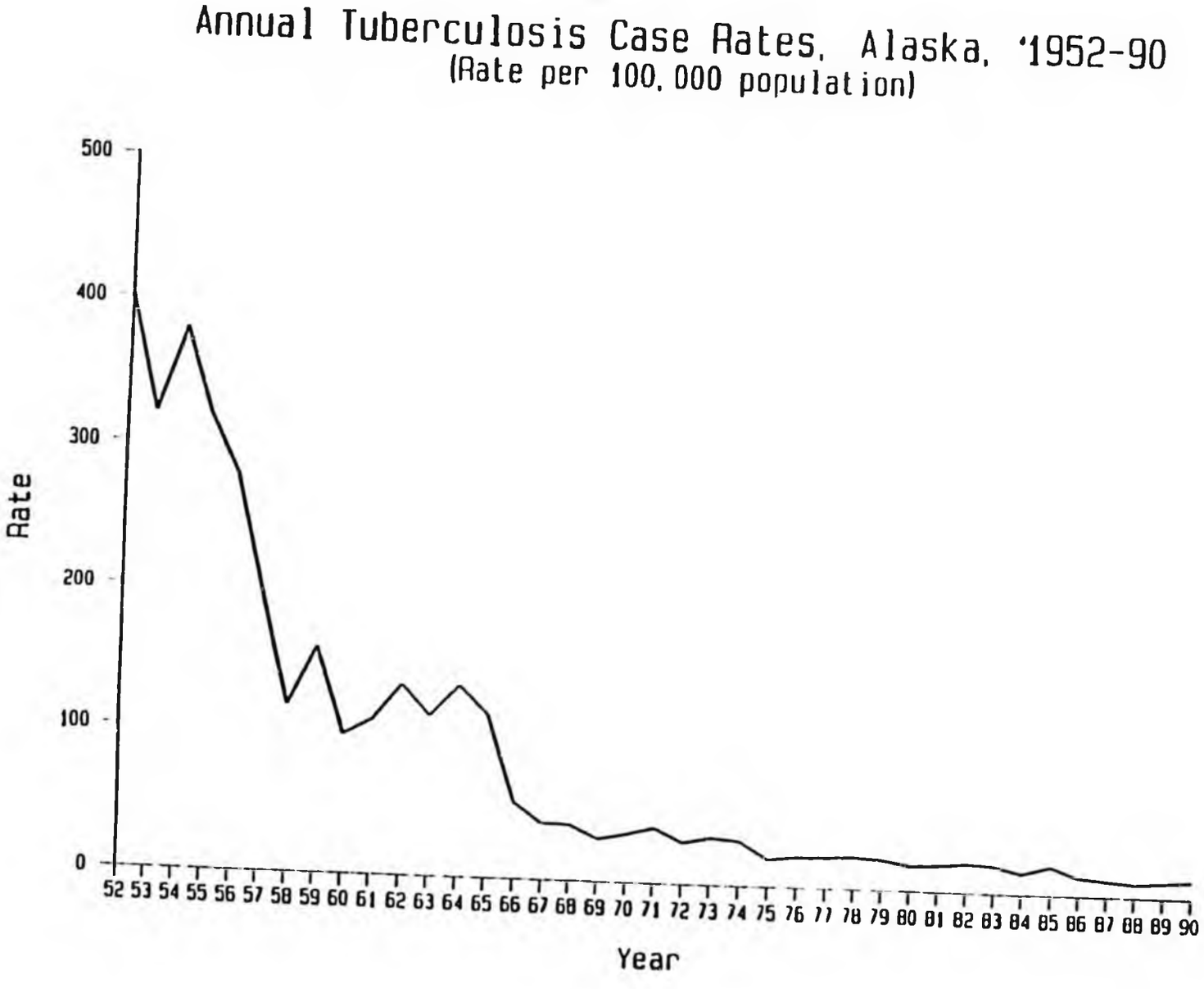
FIGURE 1


Annual Tuberculosis Case Rates, Alaska, 1952-90
(Rate per 100,000 population)
Rate
500
400
300
200
100
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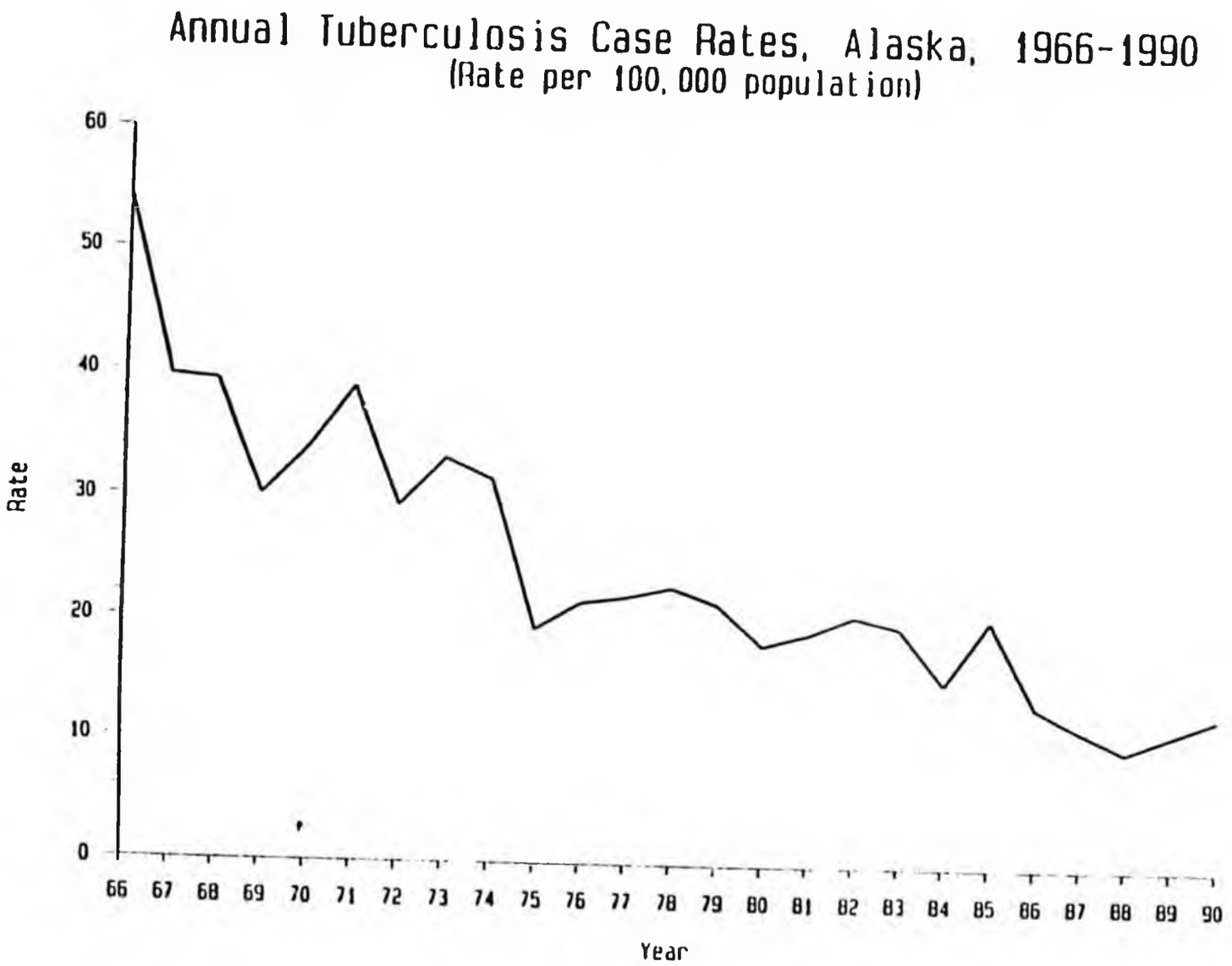
52 53 54 55 56 57 58 59 60 61 62 63 64 65 66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90
Year

FIGURE 2


Annual Tuberculosis Case Rates, Alaska, 1966-1990
(Rate per 100,000 population)
Rate
60
50
40
30
20
10
0
66 67 68 69 70 71 72 73 74 75 76 77 78 79 80 81 82 83 84 85 86 87 88 89 90
Year

The Epidemiology of Tuberculosis in Alaska, 1987

by Michael E. Jones, M.D.[1]
John P. Middaugh, M.D.[1]

ABSTRACT

Alaska's tuberculosis morbidity and mortality rates have declined dramatically since the 1950's. At the same time, the epidemiologic characteristics of the disease in Alaska have changed. The tuberculosis incidence rate is now highest among persons of Asian ancestry, particularly the foreign-born. The high rate of disease in Alaskan children indicates that active transmission of *Mycobacterium tuberculosis* to previously uninfected persons is still occurring. The large proportion of cases among persons under the age of 35 implies a failure in the detection of tuberculosis infection and/or in the use of isoniazid chemoprophylaxis. Alaska's tuberculosis control efforts must focus on ensuring that persons with tuberculosis disease are appropriately and fully treated, on detecting tuberculosis infection in persons eligible for isoniazid preventive therapy, and on applying aggressively the preventive therapy recommendations of the American Thoracic Society and the Centers for Disease Control.

INTRODUCTION

Historically, tuberculosis in Alaska predominated among Alaska Natives (Aleuts, Eskimos, and Indians). The mean annual death rate among Alaska Natives from tuberculosis during the years 1926-1930 was 655 per 100,000 (1), and tuberculosis-attributable death rates among Alaska Natives during the second quarter of this century "were among the highest ever recorded for the disease" (2). As recently as 1952, the incidence rate of tuberculosis among Alaska Natives was 1,554 cases per 100,000 (2), and the death rate from tuberculosis was 501 deaths per 100,000 (3).

Intensive case finding using statewide x-ray screening surveys, a short-lived BCG vaccination program, and intensive direct supervision of treatment of persons with tuberculosis infection or disease following the introduction of chemotherapy in the early 1950's resulted in a rapid decline in tuberculosis morbidity and mortality rates and in rates of infection in children.

Extensive tuberculin skin-testing conducted from 1948-1951 as part of the Alaska Department of Health's BCG vaccination program showed that 89% of a sample of 360 Eskimo children 5-8 years of age living in the Yukon-Kuskokwim Delta were tuberculin reactors (4). The same survey demonstrated lower rates of tuberculin reactivity among Northwest Alaska Eskimo children (67%) and Interior Alaska Indian children (65%) of the same age group; in contrast, only 0.6% of 363 white children from Southeast Alaska were found to be tuberculin reactors.

A series of tuberculin skin-test surveys of Yukon-Kuskokwim Eskimo children three years of age or younger, conducted during the period of the most intensive tuberculosis control efforts, demonstrated a rapid decline in tuberculin reactivity among successive birth cohorts (5). Of the first sample of children under age four tested during 1949-1951, 24.4% were tuberculin reactors. By 1957, only 14.4% of children in this age group were tuberculin-positive; and in 1960 the infection rate in children born since the previous survey had fallen to 1.7%.

By 1970, the rate of new active cases of tuberculosis among Alaska Natives has fallen to 154 per 100,000. It dropped further to 79.0 cases per 100,000 in 1985 (Table 1).

Moreover, the number of newly identified tuberculin reactors has declined to low levels. Of 71,316 Alaskan children of school-age and younger who were not known to be tuberculin-positive and who were tuberculin skin-tested in 1986, only 152 (0.21%) were tuberculin reactors (6). In the same year only 937 (4.83%) of 19,431 adults not previously known to be tuberculin reactors were found to be tuberculin-positive (6).

The massive public health interventions begun in the 1940's and 1950's have dramatically changed the epidemiologic characteristics of tuberculosis in Alaska. This paper will describe the epidemiology of tuberculosis in Alaska during recent years.

Tuberculosis Case Definition

For the purposes of case-counting and reporting, the American Thoracic Society and the Centers for Disease Control (7) define a case of tuberculosis as an individual from whom a sample of tissue or body fluid has yielded *Mycobacterium tuberculosis* on culture. In the absence of bacteriologic confirmation, an individual may be counted as a case of tuberculosis if diagnostic evaluation has been completed and if the following other criteria have been fulfilled: (a) the individual has (or has had) a significant tuberculin skin test reaction or has previously had culture-confirmed tuberculosis disease; (b) there are signs and/or symptoms compatible with tuberculo-

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Table 1. Tuberculosis Cases and Incidence Rates* in Alaska, by Race**, 1981–1987

YEAR	WHITE		BLACK		NATIVE		ASIAN	
	No.	(RATE)	No.	(RATE)	No.	(RATE)	No.	(RATE)
1981	15	4.5)		0.8)	56	80.7)	1	0.6)
1982	20	5.6)	4	25.5)	80	81.6)	12	93.3)
1983	22	6.0)		5.9)	92	78.5)	12	86.8)
1984	19	4.7)	0	0.0)	46	55.1)	4	45.9)
1985	22	5.3)	1	5.5)	58	79.0)	29	126.2)
1986	12	2.8)	2	10.7)	35	40.1)	23	150.5)
1987	11	2.7)	3	16.4)	31	36.1)	16	106.6)

*Rate per 100,000 population.

**Population estimates for each racial group are based on the assumption that the proportion of the population attributable to each group has remained constant since the 1980 census. Annual state-wide population estimates used for these calculations were supplied by the Alaska Department of Labor.

is, such as clinical evidence of current disease or an abnormal, unstable (worsening or improving) chest x-ray; and (c) a decision has been made to treat the individual with two or more anti-tuberculosis medications.

Reported Cases and Incidence Rates

Figures 1 and 2 depict the tuberculosis incidence rates per 100,000 population reported annually by the Alaska Department of Health & Social Services during the past thirty-six years (1952–1987). Clearly, there has been a steady downward trend in tuberculosis incidence rates during the period. The incidence rate for 1987, 11.3 new active cases per 100,000 residents, was less than half the rate for 1978. Nevertheless, Alaska's rates during 1985 and 1986 were higher than the tuberculosis incidence rates for the entire United States, which were 9.3 and 9.4 per 100,000, respectively (8). The 110 new cases of tuberculosis reported in Alaska in 1985 yielded a rate of 20.4 cases per 100,000, the highest among the 50 states for that year (9). During the following year there were 72 cases reported, a rate of 13.2 per 100,000, high enough to rank Alaska eighth among the states (9).

Only about half of the tuberculosis cases diagnosed in Alaska during 1985–1987 were confirmed by culture (Table 2), but the incidence rate of bacteriologically confirmed cases—as high as 8.7 per 100,000 in 1985—is substantial when compared to overall national rates during the same period.

Only five (9.3%) of the 54 cases of tuberculosis diagnosed in children 0–14 years of age during 1985–87 were confirmed by culture, compared to 111 (59%) of 189 cases in persons older that 14 years of age. Seven of the eight patients in the under 15 year age group reported during 1987 were younger than eight years; of these seven, only one was cultured. In 1987, 75% of cases older than 14 years were culture confirmed, compared with 54% and 49% in 1985 and 1986, respectively.

Distribution By Age and Sex

Sixty-one cases of tuberculosis were diagnosed among Alaska residents in 1987 (Figure 3). Their ages ranged from 10 months to 82 years (mean, 42.1 years; median, 41 years). Eleven (18%) of the cases were less than 20 years of age. Five cases were younger than 5 years, and three were aged 5–9 years. The finding of tuberculosis in children, in whom disease is not the

Table 2. Bacteriologically confirmed cases of tuberculosis, by age group, 1985–1987

YEAR	0–14 YEARS			15+ YEARS		
	No. of Confirmed Cases	Total No. of Cases	% of all Cases in Age Group	No. of Confirmed Cases	Total No. of Cases	% of all Cases in Age Group
1985	2	27	7%	45	83	54%
1986	3	19	16%	26	53	49%
1987	0	8	0%	40	53	75%
Total	5	54	9%	111	189	59%

Figure 1. Annual Tuberculosis Case Rates, Alaska, 1952-87
(Rate per 100,000 population)

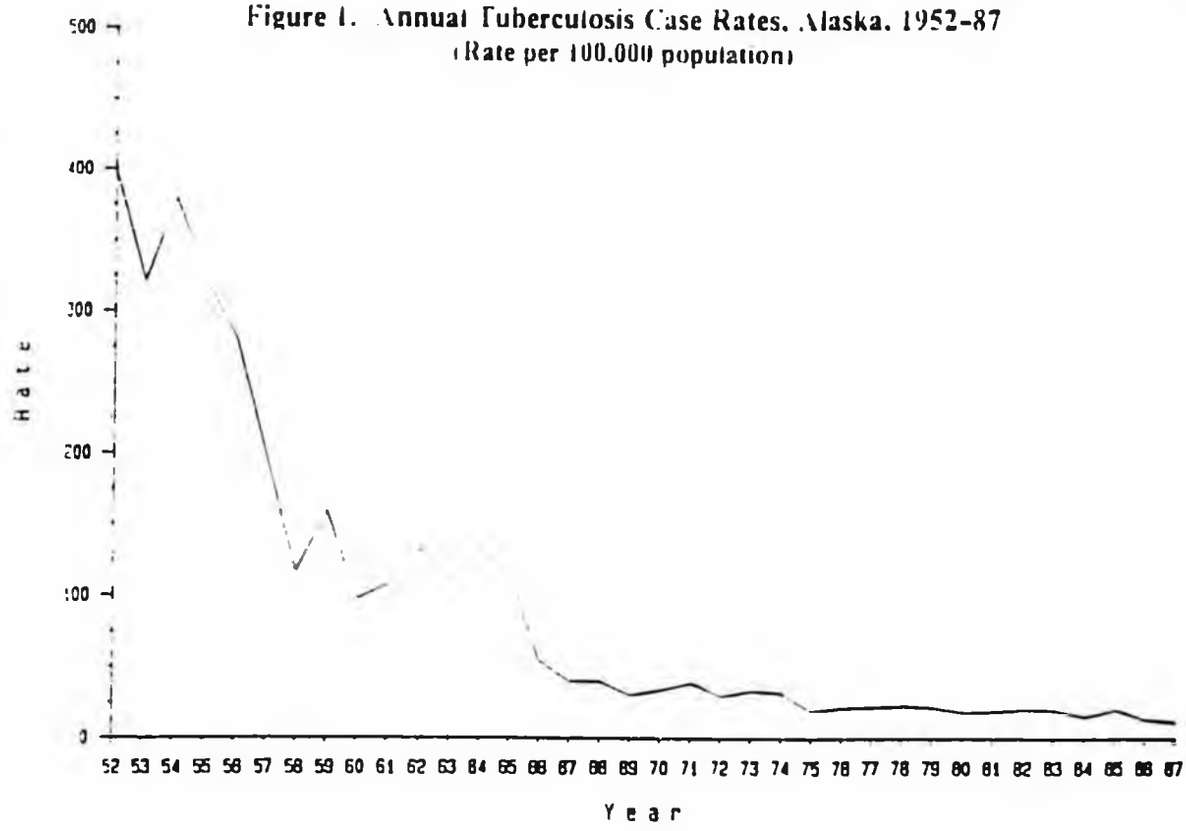
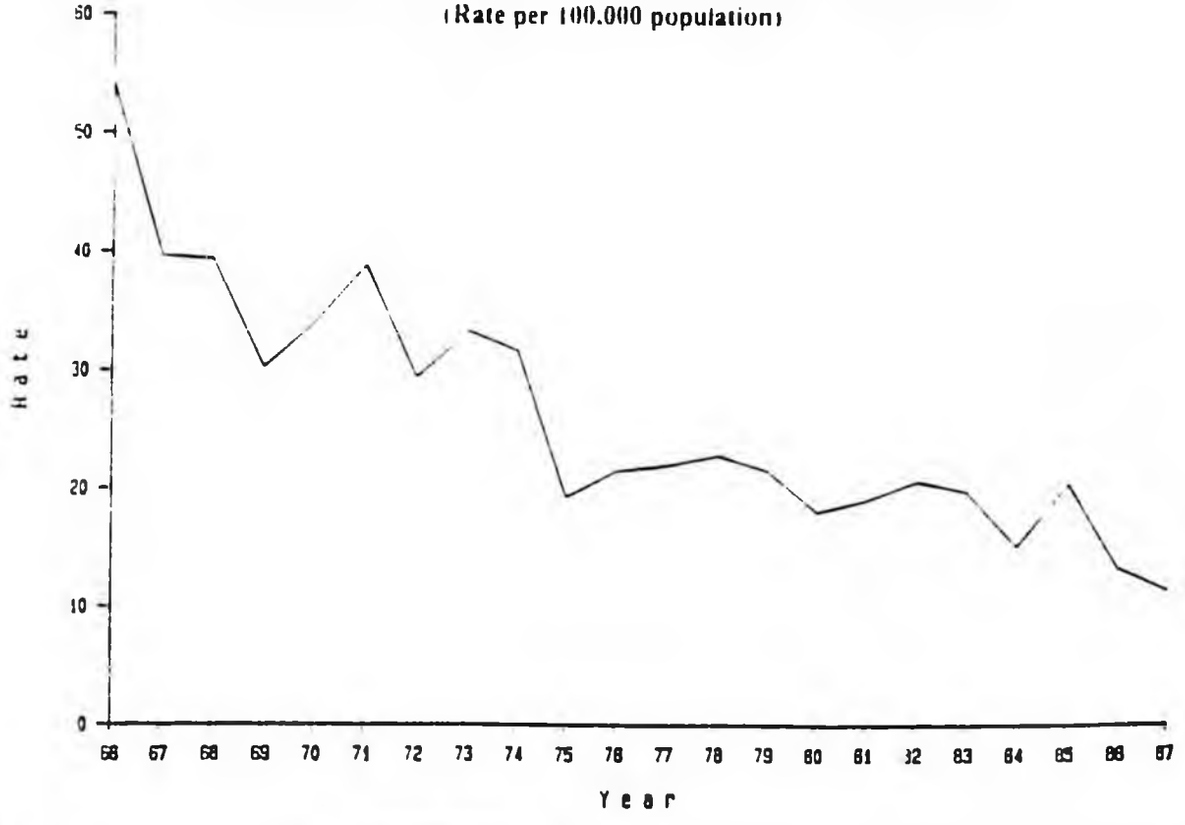


Figure 2. Annual Tuberculosis Case Rates, Alaska, 1966-87
(Rate per 100,000 population)



result of activation of "old" infection, is proof of active transmission of tuberculosis from adults with infectious pulmonary tuberculosis to uninfected children.

The age-group distribution of cases is different for each racial/ethnic group (Figure 3). Only 3 cases occurred among blacks; the three oldest age groups had one case each. For whites, there was a direct correlation between numbers of cases and increasing age; 10% of white cases were in the 0-19 age group, whereas 40% occurred in persons older than 49 years. Of the 14 Asian cases, half were in persons under 35 years of age; six (43%) were in the oldest age category. In contrast, the majority of cases among Alaska Natives were among the middle aged and elderly; sixty-two percent were 35 years of age or older—evidence of the persisting effect of high rates of tuberculosis infection and disease during the past several decades.

During 1985 and 1986 greater numbers of cases were diagnosed among younger age groups, whereas in 1987 cases were more evenly distributed. It is noteworthy that cases within the under 35-year age group accounted for 122 (50.2%) of the 243 tuberculosis cases reported during 1985-1987 (Table 3). These are cases which should be considered potentially preventable, since the American Thoracic Society and the Centers for Disease Control currently recommend that all tuberculin reactors under the age of 35 years be offered isoniazid chemoprophylaxis (10).

The greatest and most consistent differences between Alaska and U.S. rates in 1985 and 1986 are in the 5- to 14-year age range and in the 55-year-plus age group (Table 4). There were substantial differences in Alaska's age-group incidence rates for the two years, as might be expected in a relatively small population; the largest differences are in the 0-4, 35-44, 45-54, and 65+ age groups. The U.S. rates, on the other hand, show much more year-to-year consistency. The U.S. incidence rate declines from ages 0-4 years to 10-14 years, then gradually increases with age to a maximum among persons over 64 years of age. Alaska's trend is similar except for high rates in the 5- to 14-year age range, which has the lowest rates among the U.S. population. The U.S. rate for the 5-14 age group was 1.4 per 100,000 for both 1985 and 1986. Alaska's age-adjusted rates for that age group for those years were 25.6 and 19.4, respectively—rates 14-18 times as high as U.S. rates. Thus, both Alaska and the U.S. have high incidence rates among the elderly (though Alaska's rates are higher), and Alaska's rates in the 5-14 age category substantially exceed the low U.S. rates in that age category.

During the past three years (1985-1987), 153 (63%) of the 243 reported tuberculosis cases were men. Women accounted for only 37% of the cases. This is nearly identical to the sex distribution of tuberculosis cases reported for the United States during 1985 and 1986: 65% of all U.S. cases were males; 35% were females (8).

Figure 3. Cases of Tuberculosis in Alaska, 1987
By Race within Age Group
N = 61

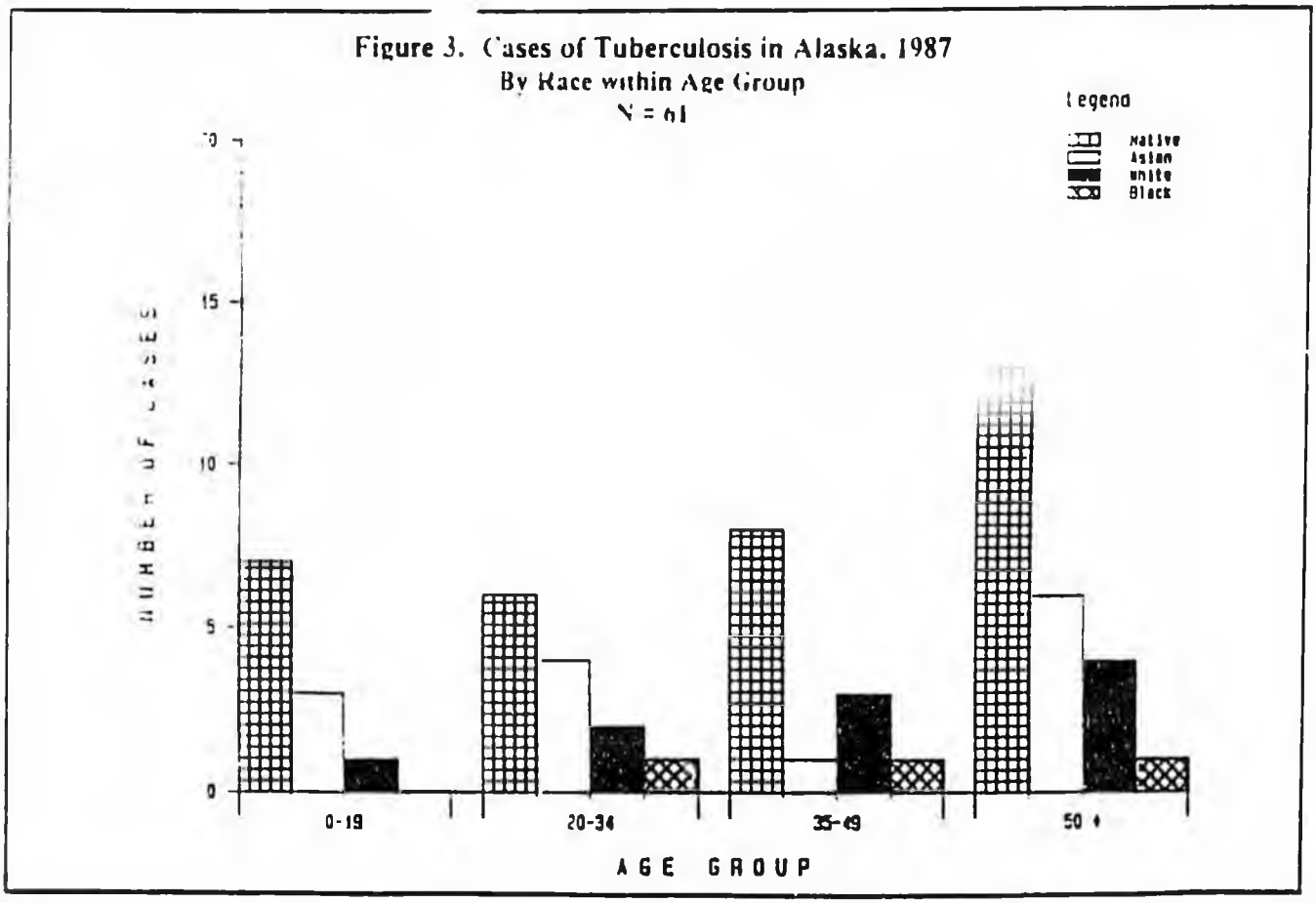


Table 3. Tuberculosis cases in Alaska, by age group, 1985–1987

AGE GROUP (YRS.)	1985		1986		1987	
	No.	(%)	No.	(%)	No.	(%)
0-19	34	(31)	24	(33)	11	(18)
20-34	23	(21)	17	(24)	13	(21)
35-49	23	(19)	11	(15)	13	(21)
50-64	18	(16)	16	(22)	12	(20)
65+	14	(13)	9	(12)	12	(20)
TOTAL	110	(100)	72	(100)	61	(100)

Distribution By Race

The incidence rate of tuberculosis among Alaska's whites is consistently low, varying from 0.0 cases per 100,000 in 1983 to 2.7 cases per 100,000 in 1987 (Table 3). For comparison, incidence rates for whites (Hispanic and non-Hispanic combined) in the United States as a whole during 1985 and 1986 were 5.6 and 5.7 cases per 100,000, respectively (8).

Rates for blacks are somewhat higher than for whites, but the numbers of cases among blacks have been consistently low. Excluding 1984 (during which no cases among blacks were recorded), incidence rates have ranged from 5.5 to 25.5 per 100,000, compared with national rates of 26.7 and 27.6 per 100,000 in 1985 and 1986, respectively (3).

During 1985–1987, members of ethnic/racial minorities comprised 81.5% of all cases of tuberculosis reported in Alaska. The tuberculosis incidence rate has in the past been highest among Alaska Natives. However, during the period 1981–1987, the incidence rate was highest among Asians during each of the seven years. While the rate among Natives declined from 80.7 cases per 100,000 at the beginning of the period to 36.1 per 100,000 in 1987, the incidence rate among Asians increased from 40.6 cases per 100,000 in 1981 to rates well in excess of 100 cases per 100,000 during 1985–1987. These rates are more than twice the incidence rates reported for Asians and Pacific Islanders in the United States during 1985 and 1986 (8). Although the incidence rate of bacteriologically confirmed disease among Asians in Alaska during 1987 was only 60.0 cases per 100,000 (9 culture-confirmed cases among an estimated population of 15,005 Asians and Pacific Islanders), this rate exceeded the case rates cited for this group in the United States for 1985 and 1986 (8).

Fifteen (25%) of Alaska's tuberculosis cases reported in 1987 were immigrants. The diagnosis of tuberculosis was made after their arrival in the United States in all but one person. Of these 15 individuals, one was from the Dominican Republic and fourteen were from Asian

Table 4. Tuberculosis incidence rates in Alaska and in the United States, by age group, 1985–1986

Age Group (Yrs.)	1985		1986	
	Alaska	U.S.A.	Alaska	U.S.A.
0–4	9.9	4.4	4.9	4.0
5-9	26.1	1.6	21.5	1.6
10-14	25.1	1.2	17.3	1.3
15-19	15.6	2.5	10.2	2.8
20-24	13.3	5.8	9.4	5.9
25-34	12.6	8.5	9.3	9.2
35–44	16.2	10.1	8.0	10.2
45-54	32.0	12.8	15.8	13.1
55-64	39.5	14.6	38.9	14.0
65+	77.2	22.3	32.6	21.9
Total	27.1*	9.3	16.5*	9.4

*Rate age-adjusted to same-year U.S. population.

countries (Philippines, 6; Korea, 5; China, 2; and India, one). All of the immigrant cases lived in major cities or towns: nine (60%) in Anchorage; three (20%) in Fairbanks; and one each in Kodiak, Juneau, and Ketchikan.

Geographic Distribution

Thirty-two (52%) of the 61 cases reported in 1987 lived in a total of eight communities with populations greater than 2,500 persons; twenty-nine (48%) were residents of sixteen villages, all with populations of fewer than 1,000. Seventeen (28%) were Anchorage residents, and six (10%) resided in Fairbanks. Twenty-seven other cases were distributed among 21 communities, with no more than two cases in any one community (Figure 4).

The remaining eleven cases, however, all occurred in a rural western Alaskan village with a population of approximately 250. The investigation of this outbreak led to the screening (tuberculin skin testing and/or chest x-rays) of 227 persons in three villages. Besides the 11 active tuberculosis cases, 13 newly infected individuals were discovered. The index case was a 24-year old woman with cavitary pulmonary tuberculosis. Three cases, including the index case, required quarantine and hospitalization for medical treatment under direct supervision for eight weeks. Exclusive of hospitalization expenses for these three patients, the estimated total cost of investigating this outbreak, of medically evaluating infected persons, and of treating those with tuberculosis infection or disease was $21,000.

Site of Disease

Of the 61 cases of tuberculosis diagnosed during 1987, 50 (82%) had pulmonary disease. Five of these 50 cases also had disease in at least one additional site: two had pleural involvement; one, lymphatic disease; one, genitourinary; and one, disseminated disease.

Eleven cases (18%) had extrapulmonary disease only. Six had cervical and/or intrathoracic lymphatic involvement; two had genitourinary disease; two had pleural tuberculosis; and one had peritoneal involvement only.

One patient—a person with extrapulmonary disease confined to the intrathoracic lymph nodes—was the first individual in Alaska reported to have both tuberculosis and AIDS.

Drug-Resistant Tuberculosis

In Alaska's public health laboratories, routine antibiotic susceptibility testing is performed with fixed concentrations of isoniazid, rifampin, and streptomycin. Eight (7.3%) of all tuberculosis cases reported in Alaska in 1985 were associated with organisms resistant to anti-tuberculosis drugs, and four (5.6%) of all cases in 1986 were caused by such organisms. In all instances, organisms were resistant to only one of the three antibiotics against which they were tested. Mycobacteria from 9 of the 12 cases showed varying degrees of resistance to rifampin; those from the other 3 patients were resistant to streptomycin. There were no instances of isoniazid resistance. Eight of the 12 patients with drug-resistant organisms were Alaska Natives; two were of Asian descent; and two were Caucasian. There was no record of any of these patients' having been previously treated with the antibiotic to which their tubercle bacilli were resistant. Of the nine patients with rifampin-resistant organisms, two were treated with regimens of three or more drugs. The other seven patients, treated with a combination of isoniazid and rifampin despite known resistance to rifampin, had radiographic improvement and/or conversion of mycobacterial cultures to negative.

There were no known instances of drug-resistance among cases diagnosed in 1987. No information regarding antibiotic susceptibility was available for two patients with culture-confirmed tuberculosis.

Recurrent Tuberculosis

Eight (13%) of the 1987 cases had at least one prior diagnosis of tuberculosis. The time intervals since each patient's most recent previous diagnosis of tuberculosis ranged from 6 to 33 years (mean, 27.5 years; median, 31.0 years). All of these patients were more than 35 years old. In addition, all had been treated with some anti-tuberculosis chemotherapy for their prior episode of tuberculosis. Five patients had taken regimens of two or more drugs (isoniazid/PAS, 4 cases; isoniazid/rifampin/ethambutol, one case). The regimens of three patients were not specified. Details regarding the exact duration of, or patients' compliance with, medical therapy were not recorded.

DISCUSSION

(1) Though the greatest number of tuberculosis cases in Alaska are Alaska Natives, the highest race-specific incidence rate in Alaska is among Asians, especially among the foreign-born. Fourteen (88%) of the 16 Asian tuberculosis cases reported in Alaska in 1987 were foreign-born. Tuberculosis is relatively uncommon among whites and blacks in Alaska. It is reasonable, then, to concentrate Alaska's tuberculosis control efforts among its Native and Asian populations.

(2) Alaska's older citizens (those 55 years of age and over) have high rates of tuberculosis, as do the elderly in the general U.S. population. However, Alaskans aged 5–14 years have much higher rates of disease than their counterparts in the general U.S. population. High disease rates in children indicate active transmission of tuberculosis to previously uninfected children from adults with pulmonary tuberculosis.

(3) Since 39%–56% of cases reported during 1985–1987 occurred among persons under age 35, and since isoniazid chemoprophylaxis is recommended for all tu-

percutin reactors in that age group, appropriate screening and prescription of preventive therapy for all tuberculosis-infected persons under age 35 will result in a substantial reduction in the number of tuberculosis cases occurring annually in Alaska.

(4) Recurrent tuberculosis is not uncommon in Alaska. The majority of persons reported to have recurrent tuberculosis in 1987 had been treated previously with chemotherapeutic regimens less effective than those now available. The anti-tuberculosis therapeutic regimens currently recommended by the American Thoracic Society and the Centers for Disease Control are associated with relapse rates of under 5% (11,12). However, in order for these regimens to achieve this level of effectiveness, they must be appropriately prescribed, and efforts must be made to ensure patients' compliance with prescribed medical therapy.

(5) Focal epidemics of tuberculosis still occur in Alaska. It is important to conduct epidemiologic investigations of close contacts of all cases of pulmonary tuberculosis (in order to identify additional cases and infected contacts) and to investigate intensively the associates of young children with tuberculosis infection (in order to identify the source of infection).

(6) Drug resistant tuberculosis is not a major problem in Alaska. Resistance to a single drug—most often rifampin—is the most common pattern.

CONCLUSION

Efforts to control the epidemic of tuberculosis in Alaska that ravaged Alaskan Natives through the 1950's achieved near-miraculous results. Now, by applying new control strategies based upon careful epidemiologic analysis of tuberculosis, further major reductions in morbidity and mortality can be expected. Indeed, there are realistic hopes that one day tuberculosis can be eradicated.

The Division of Public Health is committed to implementing the "Strategic Plan for the Elimination of Tuberculosis" developed by the national Centers for Disease Control (13,14). Key elements are presented in an accompanying article in this issue of *Alaska Medicine*.

ACKNOWLEDGMENTS

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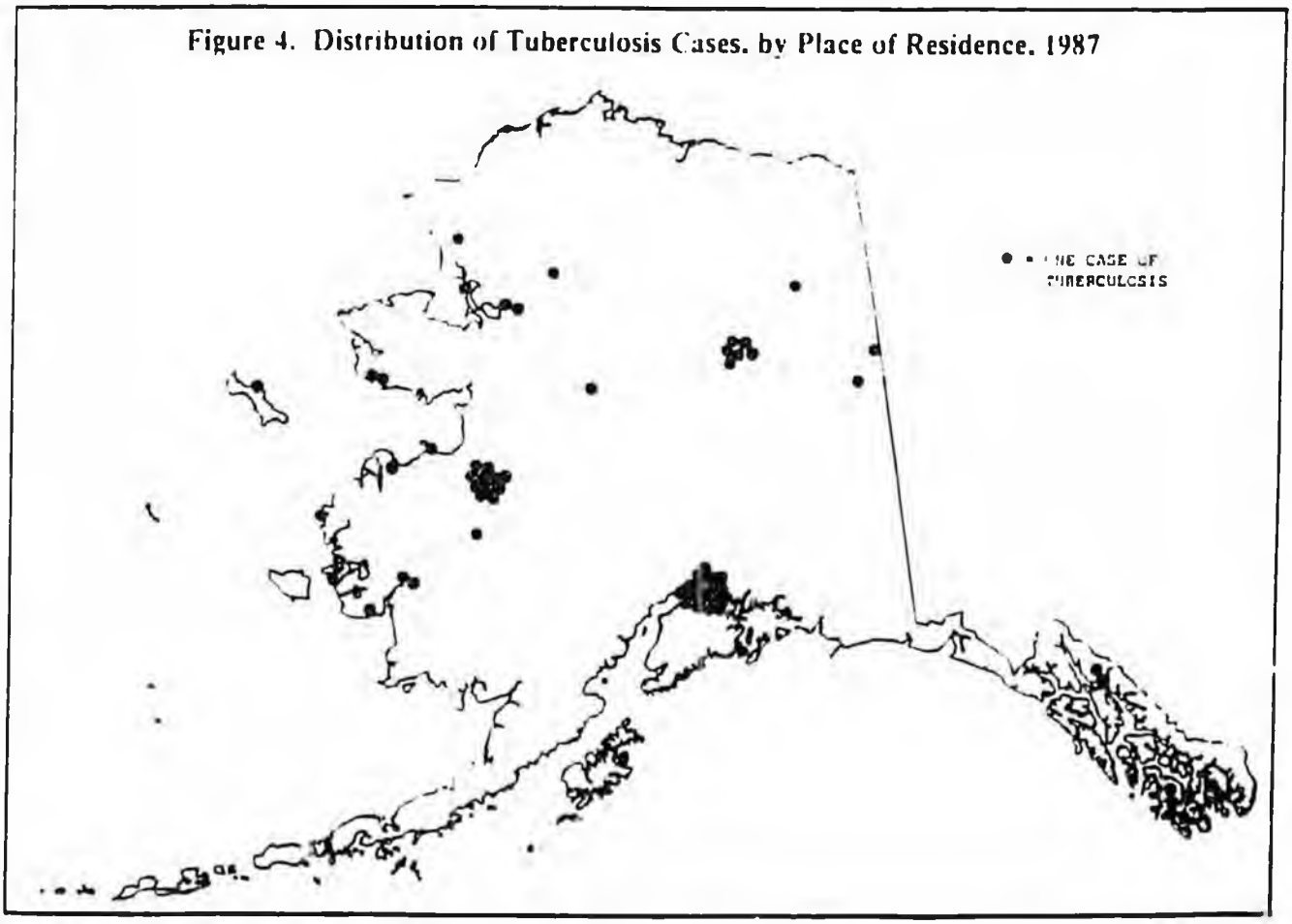
Figure 4. Distribution of Tuberculosis Cases, by Place of Residence, 1987



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Strategy for the Elimination of Tuberculosis in Alaska

Section of Epidemiology
Division of Public Health

January, 1988

INTRODUCTION

In June 1987, epidemiologists from the Division of Tuberculosis Control, Centers for Disease Control (CDC), conducted a review of the Alaska State Tuberculosis Control Program and submitted their observations and recommendations to the Section of Epidemiology. Section of Epidemiology and CDC reviewers consulted extensively with individuals and agencies involved in controlling tuberculosis. We are delighted with the widespread support that has been expressed for implementing program changes to improve our efforts to control tuberculosis in Alaska. We thank all those who contributed their valuable time and wisdom.

THE GOAL OF THE ALASKA DIVISION OF PUBLIC HEALTH IS TO ELIMINATE TUBERCULOSIS IN ALASKA.

Strategies for the Elimination of Tuberculosis

1. ***Diagnosis and Treatment of all Culture-Confirmed Cases of Pulmonary Tuberculosis.*** These are the patients most likely to transmit infection. Prompt identification and appropriate treatment of these cases are essential to limiting the spread of tuberculosis.

2. ***Epidemiologic Investigation of Close Contacts of all Cases of Pulmonary Tuberculosis.*** Infected contacts should receive appropriate chemotherapy in order to prevent future tuberculosis disease.

3. ***Effective Skin-Test Screening Programs*** to identify (and to treat, when appropriate) other individuals infected with *M. tuberculosis*.

   Periodic Skin-Testing of School Children.

   - Routine skin-testing by grades. (Skin-testing of students in pre-elementary school and in grades K,1,3,7, and 11 is required by state law.)
   - More extensive testing in schools with a large Native/Asian population or with a significant proportion of tuberculin reactors among the student population.
   - Preventive therapy for all infected children.
   - Screening of close contacts of newly infected children in order to identify the source of infection and other infected associates.

   Screening of High Risk Adult Populations. (For example, Asians, Natives, the homeless.)

4. ***Assistance to Insure Meticulous Compliance with Drug Treatment.***

These efforts should reduce the transmission of *M. tuberculosis* and shrink the population of infected persons at risk of developing tuberculosis disease. Any actions on the part of patients, health care providers, or public health personnel which result in the failure of these strategies will impede the eradication of tuberculosis from our state.

Components of Alaska's Tuberculosis Control Program

Tuberculosis Case Register

The case register contains data on patients with active or inactive tuberculosis disease and on investigated contacts of those cases. It enables public health personnel to keep track of diagnostic information on each patient with tuberculosis, their clinical management, and their compliance with recommended therapy.

2. ***Provision of Anti-Tuberculosis Drugs***

   The Section of Epidemiology supplies anti-tuberculosis chemotherapy **free of charge**, for the duration of treatment, to patients being treated for tuberculosis infection or disease.

3. ***Laboratory Services***

   The Section of Laboratories provides, **free of charge** to the patient, level 1 and 2 laboratory services, including AFB smears, mycobacterial cultures and antibiotic susceptibility testing of mycobacterial isolates.

4. ***Expert Consultation is available to physicians and all other health care providers regarding:***

   a. Clinical Diagnosis

b. Treatment and management of tuberculosis disease
c. Preventive therapy

5. *Disease Reporting and Surveillance*

The goal of surveillance is the detection of all persons with tuberculosis infection or disease, for the following purposes:

a. Treatment and control of disease.
b. Program planning and evaluation.
   - Data analysis—determination of incidence rates by age groups, sex, race, geographic area, and subpopulation (e.g., school children).
   - Identification of high risk groups.
   - Provision of outreach programs to persons at increased risk of developing tuberculosis disease, infection (for example, the Asian community, the urban homeless).
   - Formulation of State Tuberculosis Control policies.
   - Evaluation of effectiveness of program activities.

IMPORTANT CHANGES IN PROGRAM POLICIES/RECOMMENDATIONS

1. *Patient Care*

The Tuberculosis Control Program will no longer be involved in direct clinical patient care or management. Evaluation and treatment of patients with suspected or documented tuberculosis disease or infection should be conducted by a primary care provider of each patient's choice. The Tuberculosis Control Program will always assist in any circumstance to see that all patients have a "medical home," where their clinical needs can be optimally provided for.

2. *Elimination of the Use of Chest X-Rays for Screening*

Chest x-ray examinations of unselected populations are not sufficiently productive as a screening device for tuberculosis, and their routine use for such purpose cannot be recommended. Chest x-ray examinations should be obtained only when a specific medical indication exists (e.g., relevant history, symptoms, and/or significant tuberculin skin test reaction).

The use of chest x-rays for pre-employment screening, for routine follow-up of tuberculosis patients who have completed treatment, as a periodic screening tool in long-term care facilities, or as a periodic evaluation tool for tuberculin reactors should be discontinued.

As an indicator of response to tuberculosis treatment, chest x-ray examinations are less reliable than results of sputum smear, culture and assessments of symptoms and clinical status; thus, routine periodic chest x-ray examinations during tuberculosis treatment are not recommended. Chest x-rays performed during initial evaluation for tuberculosis, at 2-3 months after initiation of treatment for tuberculosis disease, and at completion of treatment, would be reasonable.

The Section of Epidemiology will provide reimbursement for chest x-rays only if authorization for payment has been pre-approved and if the patient does not have health insurance coverage which would pay for the x-ray. If pre-approved, total reimbursement for a chest x-ray and its interpretation will not exceed $40.00. The Section of Epidemiology will not pay for routine chest x-rays.

3. *Preventive Therapy*

Certain groups of people are known to be at high risk of developing tuberculosis. The benefits of preventive therapy with isoniazid in these individuals far outweigh the risks. However, isoniazid preventive therapy among these groups is underused in Alaska. These groups are as follows:

a. Household members and other close associates of potentially infectious tuberculosis cases.
b. Newly infected persons (those with tuberculin skin test conversion within the past two years, because the risk of developing disease is greatest during the 1-2 years immediately following infection).
c. Persons with significant reactions to tuberculin skin test and abnormal chest x-ray. Preventive therapy should be given to tuberculin skin test reactors with radiographic findings consistent with nonprogressive (previous) tuberculosis disease (negative bacteriology and stable parenchymal lesions).
d. Persons with significant reactions to a tuberculin skin test and who have any of the following clinical conditions which are associated with an increased risk of developing tuberculosis:
   - Silicosis.
   - Diabetes mellitus.
   - Prolonged treatment with adrenocorticosteroids.
   - Immunosuppressive therapy.
   - Some hematologic and reticuloendothelial dis-

cases, such as leukemia and Hodgkin's disease.

- Acquired immunodeficiency syndrome and persons with antibodies to the AIDS virus.
- End-stage renal disease.
- Clinical situations associated with substantial rapid weight loss or chronic undernutrition (e.g., intestinal bypass surgery, postgastrectomy state, chronic ulcer disease, malabsorption syndromes, etc.).

c. Tuberculin skin test reactors under 35 years of age with none of the above risk factors.

4. ***More Intensive Follow-up for Compliance with Treatment***

A major objective of the program is to insure that all patients complete recommended treatment for tuberculosis disease or infection. With the assistance of other public health personnel, the program will monitor and encourage patients' compliance with medical treatment (both preventive and therapeutic) and with clinical follow-up. Directly observed therapy will be employed, whenever possible, to assure patient compliance. Incentives (for example, provision of room and board during therapy) may be needed and will be provided for patients who resist treatment or whose lifestyles make compliance with treatment regimens difficult.

5. ***Increased Epidemiologic Investigations***

The Tuberculosis Control Program will conduct and/or coordinate epidemiologic investigations surrounding all potentially infectious cases of pulmonary tuberculosis (in order to identify persons infected by these cases) and newly infected individuals (in order to identify the source of infection and/or other infected persons).

B. RECENT TRENDS AND PROGRAM NEEDS

1. RECENT TRENDS IN ALASKA

The extent of Alaska's tuberculosis problem and recent epidemiologic trends in tuberculosis incidence are described below.

Reported Cases and Incidence Rates: During the period 1988-1993, the number of reported cases of tuberculosis disease in Alaska ranged from a high of 70 (in 1991) to an all-time low of 51 (in 1988). Corresponding annual incidence rates (per 100,000 population) ranged from 12.4 to as low as 9.5. In 1992, for the first time, Alaska's annual TB morbidity rate--9.7 cases per 100,000 (the 15th highest rate among the 50 states)--fell below the same-year U.S. rate, which was 10.5 per 100,000.

Bacteriologically-Confirmed Cases: During 1985-1986, fewer than 50% of tuberculosis cases were confirmed by mycobacterial culture. During 1988-1993, the proportion of bacteriologically-confirmed cases varied from 61% to 84%, probably as a result of strict application of a tuberculosis case-definition. Considering only culture-confirmed cases, Alaska's annual incidence rates (per 100,000) for the years 1988-1993 would have been 8.0, 7.8, 10.2, 9.6, 8.0, and 8.0.

Children 0-14 Years of Age: During 1988-1993, an average of 8.5 tuberculosis cases (range: 4-13) were reported annually among children under 15 years of age: rates of 2.5-9.4 cases per 100,000. During 1993, 9% of all reported tuberculosis cases in Alaska occurred in this age group.

Racial/Ethnic Minorities: Alaska Natives, traditionally the population in Alaska most severely afflicted with tuberculosis, had an incidence rate during 1993 of 41.7 cases per 100,000--over 40-fold less than their rate during the early 1950's. Estimated annual incidence rates among Asians and Pacific Islanders (A/PIs) exceeded those of Alaska Natives in 1993 and during 5 years of the 1982-1991 decade; their rate in 1993 was 50.6 cases per 100,000. Substantial annual changes in the TB incidence rates among Alaska Natives and A/PIs undoubtedly reflects only year-to-year variation among these small populations rather than real trends in morbidity. Tuberculosis is uncommon among both whites (< 3.0 cases per 100,000 annually since 1986) and blacks (a total of five cases reported during 1988-1993) in Alaska.

Foreign-Born Persons: During the 6 years from 1988 through 1993, 58 Alaska residents born outside the United States were diagnosed as having tuberculosis (mean, 9.7 cases/year; range, 7-17). Foreign-born individuals accounted for 12 (21%) of the 58 tuberculosis cases reported in Alaska during 1993. Eleven (92%) originated from Asian countries (Philippines, 8; Korea, 2; Thailand, 1); one was from Central America. Of the 12, 9 (75%) had entered the U.S. within the past 5 years.

Homeless Persons: During 1989-1993 there were a total of 26 cases (mean, 5.2 per year; range, 3-7/year) which occurred among persons considered to be homeless (i.e., individuals who lacked a stable address or who lived in a shelter for the homeless). Twenty-two (85%) were male. Nineteen (73%) were residents of Anchorage; six (23%) lived in Fairbanks; and one, in Ketchikan. Although no reliable denominator data are available, it is estimated that, on an annual basis, Anchorage has a homeless population of about 4,000. Thus, one could estimate an annual tuberculosis incidence rate of about 95 cases per 100,000 Anchorage homeless.

Of these 26 case-patients, 1 died of causes unrelated to tuberculosis before completing therapy; 1 moved out of state during therapy; 6 (23%) were lost to follow-up; and 18 (69%) completed anti-TB therapy. All nine homeless TB case-patients identified in 1992 and 1993 completed their anti-TB therapy on DOT.

Tuberculosis Cases with a Diagnosis of AIDS: Seven (0.4%) of the 1,803 tuberculosis cases reported during the years 1972 through 1993 were diagnosed during the same year or during a later year as having AIDS. These seven tuberculosis cases were diagnosed in 1982, 1985, 1987, 1988, 1992, and 1993. No instances of TB/AIDS were reported during 1989-1991. In 1992, two TB/AIDS case-patients--including the first Alaska Native with TB/AIDS--were reported. In 1993, a second Alaska Native with TB/AIDS was reported.

Persons Living in Institutions: Data are available for 1988-1993 only. During these 6 years, 15 TB cases were reported among persons living in institutions: 6 in correctional facilities, 5 in nursing homes, and 4 in hospitals. Five of the 15 cases among institutional residents occurred in 1992. No secondary cases are known to have occurred in these facilities.

Drug-Resistant Tuberculosis: Isolates of drug-resistant *M. tuberculosis* are not common in Alaska. Fifteen (5.7%) of 265 culture-confirmed cases reported during 1986-1991 had isolates resistant to one or more anti-TB drugs. Of 46 *M. tuberculosis* isolates in 1992 for which susceptibility information was available, 4 (8.7%) were drug-resistant. Two were resistant to isoniazid alone; a third (from a recent immigrant from the Philippines) was resistant to isoniazid and rifampin; the fourth was resistant to pyrazinamide. During 1993, among 44

isolates with drug susceptibilities. 3 (6.8%) with drug resistance were identified. One was resistant to isoniazid alone, another (from a Filipino immigrant) was resistant to isoniazid, rifampin, and ofloxacin, and the third (from a Korean immigrant) was resistant to isoniazid, rifampin, streptomycin, and capreomycin.

Cases Related to Outbreak Situations: Outbreak-associated cases ($\geq$ 3 related cases) have accounted for a substantial proportion of Alaska's tuberculosis morbidity. Twelve outbreak-associated cases were reported during 1987; 13 during 1989; 23 in 1990; 26 during 1991; and 7 in 1992. No such case-clusters were found during 1986 or 1988 or 1993.

In 1994 (thru September), the Tuberculosis Control Program initiated extensive field investigations in three isolated villages: Savoonga, Gambell, and St. Paul. These investigations were necessary because skin test converters--but no active TB cases--had been identified as a result of routine testing and follow-up. These investigations are still in progress and have already identified several pulmonary TB cases and many converters. In addition, routine school TB skin-test screening in September led to recognition of an outbreak in the Western part of the state. To date, six probable cases have been identified.

Pulmonary tuberculosis in residents of rural Alaska villages--the great majority of which have clinics without x-ray facilities--is not infrequently diagnosed belatedly, either through misdiagnosis as "acute bronchitis" or through failure to consider tuberculosis in the differential diagnosis. Consequently, patients with pulmonary tuberculosis may remain symptomatic and infectious for months before a correct diagnosis is made Delays in diagnosis result in greater numbers of contacts who develop tuberculous infection and tuberculosis disease. This has the effect of producing clusters of tuberculosis cases, or localized outbreaks.

Geographic Distribution: The geographic localization of tuberculosis disease has reflected the varying prevalence of tuberculosis infection/disease in Alaska during the epidemic period of the first half of this century. One hundred thirty-six (38%) of the 362 tuberculosis cases identified during 1988-1993 were residents of western or northern Alaska. One hundred thirty-three (37%) of the cases were reported from Southcentral Alaska (which includes the Anchorage Borough), where more than half the population of the state lives; 48 (13%), from Interior Alaska (of which Fairbanks is the major population center); 26 (7%), from Southeast Alaska; and 19 (5%), from the Aleutian Chain or Pribilof Islands.

Close living conditions and extensive intra-village social interactions greatly complicate contact investigations surrounding tuberculosis cases in some remote villages. The full extent of transmission in these instances may be practically impossible to determine. Periodic tuberculin screening of children and young adults and appropriate evaluation of persons with persistent respiratory symptoms is critical in these "high-risk" villages..

AIDS in Alaska: From 1982 through December 31, 1993, a total of 204 Alaska residents had been diagnosed as having AIDS. One-hundred and twenty-two (60%) were known to have died. One hundred and eighty-two (89%) of the cases were male. The mean age of patients at the time of diagnosis was 35 years (range, 0-75 years); 185 (91%) were between the ages of 20 and 49 years. Their distribution by race was as follows: white (71%), Alaska Native (17%), black (8%), Hispanic (3%), and Asian (1%). Sixty-nine percent were residents of Anchorage; 7% lived in Fairbanks; 5% in Juneau. The remaining 19% resided in small cities, towns or villages. Hierarchical distribution of the 204 cases by AIDS risk factors is as follows: male-male sex (66%), transfusion (5%), heterosexual contact of high-risk individual (7%), IV drug use (6%), hemophiliac (2%), perinatal infection (1%), and other (6%).

AIDS Cases with Diagnosis of TB: Seven (3.4%) of the 204 AIDS cases diagnosed during the years 1981 through 1993 were diagnosed during the same year or had been diagnosed in an earlier year as having tuberculosis. These seven AIDS cases were diagnosed in 1985 (2 cases), 1987 (1), 1988 (1), 1992 (2), and 1993 (1). One of the AIDS cases diagnosed in 1985 had been diagnosed in 1982 as having tuberculosis. In the other five instances, the diagnoses of AIDS and tuberculosis were made during the same year. No cases of AIDS/TB were identified during 1989-1991. Of the two persons with AIDS were diagnosed as having culture-confirmed pulmonary TB during 1992, one was an Alaska Native woman with a history of IV drug use.

Prevalence of HIV Infection in Alaska: HIV infection is not a reportable condition in Alaska, and HIV antibody-testing at the State Public Health Laboratory, Fairbanks (SPHL-F) is conducted anonymously. For most individuals tested, information regarding gender, race/ethnicity, age group, and risk category is available.

Through June 30, 1994, sera from a total of 70,182 individuals had been tested for HIV antibody at the SPHL-F. Four hundred and fifty-four (1.3%) of 34,155 males had positive test results; 62 (0.2%) of 35,679 females had positive results. Three hundred and forty-four (0.8%) of 45,582 white persons had positive results, as did 79 (0.5%) of 15,733 Alaska Natives, 52 (1.3%) of 3,960 blacks, 33 (2.1%) of 1,563 Hispanics, and 7 (0.4%) of 1,671 persons of "Other" race. By risk category, 264 (13.8%) of 1,914 homosexual/bisexual males tested were HIV antibody-positive, as were 32 (1.1%) of 2,831 IV drug users tested. Twenty-five (31.6%) of the 79 seropositive Alaska Natives were categorized as homosexual/bisexual males; 5 were heterosexual contacts of high-risk persons; 4 had received transfusions; and 45 were designated as "Other" risk category.

CHARACTERISTICS OF TUBERCULOSIS CASES, ALASKA, 1988-93

YEAR	1988	1989	1990	1991	1992	1993
No of cases	51	59	68	70	57	57
Alaska rate (per 10^5) USA rate (per 10^5)	9 5 9 1	11 0 9 5	12 4 9 6	12 3 10 4	9 7 10 5	9 5 9 8
No bacteriologically confirmed (% total)	43 (84%)	42 (71%)	56 (82%)	55 (79%)	47 (82%)	48 (84%)
No 0-14 yrs old (rate/10^5)	9 (7%)	13 (9%)	9 (6%)	11 (7%)	4 (3%)	5 (9%)
No foreign-born (% total)	7 (14%)	7 (12%)	7 (10%)	8 (11%)	17 (30%)	12 (21%)
No homeless	-	4	7	6	3	6
No with AIDS diagnosis	1	0	0	0	2	1
No with drug-resistant tuberculosis	2	6	0	3	4	3
No associated with outbreaks	0	13	23	26	7	0

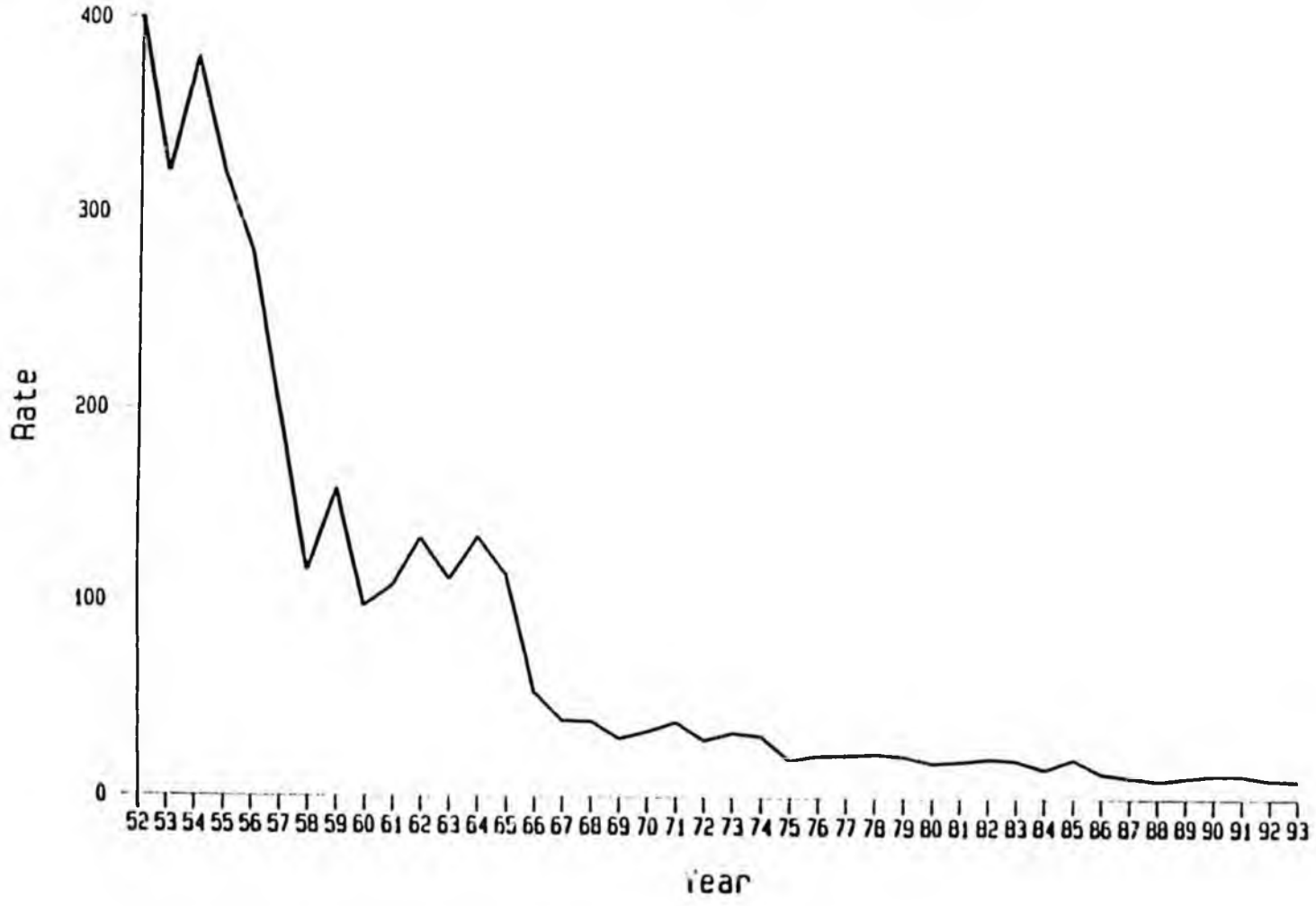
TUBERCULOSIS CASES AND INCIDENCE RATES*, BY RACE, ALASKA, 1981-1993

	WHITE		BLACK		AK NATIVE		ASIAN	
YEAR	No.	(Rate)	No.	(Rate)	No.	(Rate)	No.	(Rate)
1981	15	(4.5)	1	(6.8)	56	(80.7)	11	(90.6)
1982	20	(5.6)	4	(25.5)	60	(81.6)	12	(93.3)
1983	23	(6.0)	1	(5.9)	62	(78.5)	12	(86.8)
1984	19	(4.7)	0	(0.0)	46	(55.1)	14	(95.9)
1985	22	(5.3)	1	(5.5)	68	(79.0)	19	(126.2)
1986	13	(3.1)	3	(16.1)	33	(42.9)	23	(150.5)
1987	10	(2.4)	3	(16.4)	34	(43.0)	14	(93.3)
1988	10	(2.4)	0	(0.0)	35	(43.1)	6	(40.5)
1989	2	(0.5)	0	(0.0)	50	(59.9)	7	(46.9)
1990	6	(1.4)	1	(5.3)	52	(60.7)	9	(58.6)
1991	4	(0.9)	2	(8.6)	56	(62.9)	8	(39.0)
1992	6	(1.4)	2	(8.3)	29	(31.7)	20	(94.7)
1993	5	(1.1)	0	(0.0)	40	(41.7)	12	(50.6)

*Rate per 100,000 population

Annual Tuberculosis Case Rates, Alaska, 1952-93

(Rate per 100,000 population)



Tuberculosis

Mission Statement

The goal of the Tuberculosis (TB) Control Program is the elimination of TB from Alaska. This can b accomplished through efforts targeted at preventing transmission of the causative organism of tuberculosi identifying persons with TB infection and TB disease, and working to assure appropriate and comple treatment of persons for whom anti-tuberculous therapy is indicated.

Work Plan

Recent investigations of cases of tuberculosis have revealed severe deficiencies in the Division's ability t control tuberculosis. Several years of successive budget reductions, diminished priority given to training an field response, staff turnover, and loss of institutional knowledge of program procedures have combined t compromise the integrity of this critical program.

- Outbreaks in Savoonga, Gambell, St. Paul Island, and Nome exposed the inadequacy of our program. Investigation and implementation of treatment of all persons infected is the highest priority of the Section.
- Once acute epidemic investigations are completed, a major program to increase knowledge of, an commitment to, tuberculosis control will be essential.
- We will work aggressively to enhance PHN training and personnel, lab capacity including DNA-finge printing of isolates, and re-establishing knowledge of mechanisms and ability to implement quarantine c patients who do not take necessary medications.

Program staff will place particular emphasis on detection and epidemiologic investigation of clusters of TI cases. Such investigations, especially when they are conducted in remote villages, are time- and labor intensive, requiring extensive tuberculin skin-testing, collection of specimens for culture, and taking of ches x-rays using a portable machine.

Because TB cannot be diagnosed unless it is suspected, the Program Nurse Consultant will continue to make presentations to regional gatherings of Community Health Aides/Practitioners about TB, its clinical presentation, and the means by which it can be diagnosed. Program staff will continue to work to develop methods to assure compliance of patients--particularly those with active TB disease--with recommended anti-TB therapy.

- Conducting active and passive surveillance for new cases of TB disease; acting as the central point to which all cases of TB disease in Alaska residents are reported; and maintaining an up-to-date register of all TB cases currently under treatment;
- Annually analyzing characteristics of cases of TB disease in order to identify trends in TB morbidity in Alaska;
- Directing and/or conducting timely investigations of contacts of patients with infectious TB disease in order to identify and to offer appropriate treatment to other persons with TB infection or TB disease;
- Conducting investigations of TB outbreaks/clusters and recommending appropriate interventions and control measures;
- In conjunction with the Section of Public Health Nursing, monitoring medical compliance of patients receiving anti-TB chemotherapy in order to assure full and appropriate therapy; and administering a program through which "chemotherapy aides" provide directly-observed or closely-supervised anti-TB chemotherapy to selected patients;

- Ordering quarantine of patients who are non-compliant with medical therapy and who are believed to pose a threat to the health of the public;
- Providing consultation, information, and education about TB and its diagnosis and treatment to health-care providers (CHAs, PHNs, mid-level practitioners, physicians) throughout Alaska;
- Establish state-of-the-art clinical care for patients by supporting clinical services at the Anchorage Neighborhood Health Center.
- Monitoring compliance of preschools and schools with statutory requirements for periodic tuberculin skin-testing of school-children, and annually tabulating and analyzing reports of these skin-test results;
- Providing itinerant chest x-ray services, where indicated; and
- Providing tuberculin skin-testing materials and anti-TB medications free-of-charge.
- Maintain current program activities as a part of our initiative to implement "Strategies for the Eradication of TB in Alaska."
- Improve patient compliance with prescribed treatment.

4) Potential problems (see Attachment 2)

Current village outbreaks of tuberculosis are requiring large expenditures of unbudgeted resources that impact Nursing, Labs, and Epi. No IHS funds are being contributed at this time. Capacity to deal with tuberculosis has eroded to unacceptable levels. Compacting raises concerns about future IHS involvement in TB control. Proposed OSHA regulations could result in requirements for massive redesign of health facilities.

5) Actions

A high level meeting with IHS should focus on allocation of personnel and resources along with clear responsibilities for tuberculosis control activities including patient transport; x-rays; clinical diagnosis, treatment, and management; quarantine; and payment for epidemic field investigations.

We suggest that representatives from the A.G. meet with IHS attorneys to be briefed on legal ramifications for public health program issues under compacting.

An FY96 increment is needed to restore State capacity to control TB in Alaska.

6) Collaboration

Epi and Labs will collaborate on submitting a consolidated TB grant to CDC.

Epi and Nursing will collaborate to mobilize staff needed to control village outbreaks and to conduct appropriate training.

TUBERCULOSIS TRANSMISSION IN THE 1990s

Two studies reported in this issue of the *Journal*[1,2] use new techniques of molecular epidemiology to extend our understanding of the transmission and control of an ancient disease — tuberculosis. The conventional wisdom has been that 90 percent of active cases arose from foci of infection first acquired years or decades ago. In the United States alone, the pool of persons with latent tuberculous infection is estimated at 10 to 15 million.[3] But where do today's active cases come from? The reports in this issue suggest that at least in San Francisco and New York City, a third or more result from recent person-to-person transmission rather than from the reactivation of latent infection.

Small et al.[1] and Alland et al.[2] both use restriction-fragment–length polymorphism (RFLP) analysis to determine the genetic relatedness of organisms cultured from patients with active tuberculosis.[4,5] RFLP analysis has been a powerful tool for confirming the results of standard epidemiologic investigations in hospitals,[6] shelters for homeless persons,[7] and prisons.[8] Both studies applied RFLP analysis to large samples of cases and found remarkable genetic diversity among isolates of *Mycobacterium tuberculosis*, suggesting that most active tuberculosis continues to result from the reactivation of remote infection. But both groups also found substantial proportions of patients with organisms that had the same RFLP pattern ("DNA fingerprint"), and each group argues that these clusters of identical isolates indicate recent transmission.

Despite some differences in methods, the findings of the two studies are remarkably similar. In both, patients who were older or foreign-born were less likely to have clustered isolates, suggesting that in these patients disease is generally due to reactivation of infection rather than to recent transmission. Alternatively, foreign-born patients may have been recently infected by persons living abroad whose isolates were unavailable for DNA analysis. Both studies found that indigent patients were more likely to have identical isolates and that certain racial and ethnic groups had more clustering, although the particular groups were different in the two studies, suggesting that transmission is related to social and economic factors rather than to race itself. Alland et al. found that clustering was more common among patients with drug-resistant isolates, possibly because such patients remain infectious longer.

Most dramatically, in both studies nearly two thirds of the patients infected with the human immunodeficiency virus (HIV) or who had AIDS had clustered strains of *M. tuberculosis*. Thus, it appears that in most HIV-infected persons in the two urban areas, tuberculosis may be due to recent transmission rather than to the reactivation of remote infection.

There was also remarkable similarity in the proportion of patients considered to have clustered isolates, although the two studies differed slightly in their manner of performing this calculation. Small et al. appropriately did not include the first (index) patient in a cluster among the patients who had recently transmitted infection, whereas Alland et al. did not consider isolates with only two identical RFLP bands to be definitive evidence of recent transmission. (The more bands, the lower the likelihood of a match by chance alone.) After adjustment for these differences, the studies both found that from one fourth to one third of patients had molecular evidence suggestive of recent transmission. This may substantially underestimate the true extent of clustering, because not all related patients were included. On the other hand, the proportion of cases due to recent transmission may be overestimated if clustering sometimes represents remote rather than recent spread.

Interestingly, the authors found similar ratios of clusters to cases (1:9 and 1:11), suggesting that approximately 1 in 10 patients with tuberculosis may be highly infectious and may account for most instances of transmission. One patient who was infectious for a prolonged period spread the disease widely and was responsible for 6 percent of all the cases in San Francisco.

These investigations raise many questions. Once infected with HIV, are patients more susceptible to infection with *M. tuberculosis*, as well as to progression to active disease? Are CD4 cell counts higher in HIV-infected patients with reactivated *M. tuberculosis* infection than in those with recently transmitted disease? Why was the proportion of patients with disease due to recent transmission so similar in the two cities, when at the time of the study the rate of completion of treatment was more than 95 percent in San Francisco but less than 40 percent in New York City (unpublished data)? Are long delays common in initiating treatment for tuberculosis? When they are, treatment can be 100 percent complete, but transmission can still be extensive. Finally, why are certain patients such effective disseminators of infection and disease?

These studies have important implications for tuberculosis control. A single patient can account for dozens of active cases, as well as hundreds of tuberculosis infections. Combined with increasing drug resistance[9] and the well-documented inability of health care providers to predict compliance with therapy,[10] this underscores the importance of expanding the use of directly observed therapy, in which a health worker watches as patients ingest their antituberculous medications. Recent reports[11,12] demonstrate that the use of directly observed therapy can turn back the tide of drug-resistant tuberculosis. If a large percentage of new cases result from recent infection, then we must also improve contact investigation and follow-up.

That a substantial proportion of cases are due to recent transmission is an indictment of the current

health care system. But it is also a message of hope, because it implies that improved treatment could rapidly decrease the number of active cases. This appears to be occurring in New York City. The number of patients receiving directly observed therapy in New York increased from fewer than 100 in 1991 to more than 1200 in 1993, and the proportion completing treatment increased substantially; the number of new cases decreased by 15 percent in 1993. This decrease did not occur among the elderly and foreign-born (unpublished data), whose disease is mostly due to reactivation; this suggests that the decrease was attributable to an interruption in the ongoing transmission of disease as a result of improved treatment.

These studies also have important implications for the practicing physician. First, physicians should "think TB" when any patient has chronic cough and fever, regardless of the results of the tuberculin skin test, even if the radiographic findings are not typical of tuberculosis, and especially if the patient may be infected with HIV. Second, physicians should ensure prompt, effective, directly observed treatment for patients with tuberculosis. Third, they should work closely with the local health department to conduct sensitive and complete contact tracing, guided by an understanding of our increasingly complex social patterns. Fourth, physicians should ensure effective infection control in congregate settings, particularly hospitals, correctional facilities, and shelters. And fifth, they should target preventive treatment to persons at high risk, such as close contacts of patients with active tuberculosis, HIV-infected patients, and foreign-born persons from countries with a high prevalence of tuberculosis, in order to stop infection from progressing to active, infectious disease.[3]

Finally, these studies have important implications for our society. Interrupting transmission may be much easier than preventing the cases that result from a reactivation of latent infection. Unless effective public health measures continue and expand, the aptly named "U-shaped curve of concern"[13] will describe our efforts, and the recent encouraging downward trends will be replaced by rising numbers of cases. Before the studies in this issue of the *Journal* appeared, we could comfortably believe that the vast majority of cases of clinical tuberculosis arose from infection acquired many years ago. We must now recognize that much of today's tuberculosis results from recent failures of treatment and public health measures. This should heighten our sense of accountability to our patients and to society. We know that poverty, overcrowding, and HIV infection accelerate the spread of this disease. It is our responsibility both to ensure appropriate treatment for our patients and to improve the troubled social conditions that allow tuberculosis to spread.

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