References

1. *Statistical Abstract of the United States: 1986.* 106th ed. Washington, DC: US Bureau of the Census; 1986.
2. *Statistical Abstract of the United States: 1994.* 114th ed. Washington, DC: US Bureau of the Census; 1994.
3. Ensminger AH, Ensminger ME, Konlande JE, Robson JRK. *Foods & Nutrition Encyclopedia.* 2nd ed. Boca Raton, Fla: CRC Press; 1994.
4. Maxwell JC. Annual soft drink report. *Beverage Industry.* 1994;85:4–20.
5. Wyshak G, Frisch RE, Albright TE, Albright NL, Schiff I, Witschi J. Nonalcoholic carbonated beverage consumption and bone fractures among women former college athletes. *J Orthop Res.* 1989;7: 91–99.
6. Wyshak G, Frisch RE. Carbonated beverages, dietary calcium, the dietary calcium/phosphorus ratio, and bone fractures in boys and girls. *J Adolesc Health.* 1994;15: 210–215.
7. Barrett-Connor E, Chang JC, Edelstein SL. Coffee-associated osteoporosis offset by daily milk consumption. *JAMA.* 1994;271: 280–283.
8. Keil DP, Felson DT, Hannan MT, Anderson JJ, Wilson PWF. Caffeine and the risk of hip fracture: the Framingham study. *Am J Epidemiol.* 1990;132:675–684.
9. Smith S, Swain J, Brown EM, et al. A preliminary report of the short-term effect of carbonated beverage consumption on calcium metabolism in normal women. *Arch Intern Med.* 1989;149:2517–2519.
10. Massey LK, Strang MM. Soft drink consumption, phosphorus intake, and osteoporosis. *J Am Diet Assoc.* 1982;80:581–583.
11. Phillips MC, Briggs GM. Milk and its role in the American diet. *J Dairy Sci.* 1975;58: 1751–1753.
12. Heaney RP. The role of nutrition in prevention and management of osteoporosis. *Clin Obstet Gynecol.* 1987;30:833–846.
13. Einhorn TA, Levine B, Michel P. Nutrition and bone. *Orthop Clin North Am.* 1990;21: 43–50.
14. Willett WC, Sampson L, Stampfer MJ, et al. Reproducibility and validity of a semiquantitative food frequency questionnaire. *Am J Epidemiol.* 1985;122:51–65.
15. *SAS/STAT User's Guide, Version 6.03.* Cary, NC: SAS Institute Inc; 1988.
16. Mazariegos-Ramos E, Guerrero-Romero F, Rodriquez-Moran M, Lazcano-Burciaga G, Paniagua R, Amato D. Consumption of soft drinks with phosphoric acid as a risk factor for the development of hypocalcemia in children: a case-control study. *J Pediatr.* 1995;126:940–942.
17. Pennington JAT. *Bowes & Churches Food Values of Portions Commonly Used.* 16th ed. Philadelphia, Pa: JB Lippincott; 1994.
18. National Research Council. *Recommended Dietary Allowance.* 10th ed. Washington, DC: National Academy Press; 1989:174–194.
19. Calvo MS. The effects of high phosphorus intake on calcium homeostasis. In: Draper HH, ed. *Advances in Nutritional Research.* New York, NY: Plenum Press; 1994;9:183–207.

Transmission of *Mycobacterium tuberculosis* in a California State Prison, 1991

Denise T. Koo, MD, MPH, Roy C. Baron, MD, MPH, and George W. Rutherford, MD

ABSTRACT

Objectives. An investigation was conducted to determine whether ongoing transmission of *Mycobacterium tuberculosis* was occurring in a California state prison.

Method. Prison pharmacy records were used to identify cases of active tuberculosis (TB).

Results. Ten of the 18 cases of active TB treated at the facility during 1991 were diagnosed at the prison that same year (an incidence of 184 per 100 000). Three inmates were infectious for a total of 7 months while imprisoned. The prevalence of TB skin test–positivity among inmates was 30%, and the incidence of new infection attributable to incarceration was 5.9 per 100 inmates per year.

Conclusions. Transmission of *M. tuberculosis* may be occurring in the California prison system. (*Am J Public Health.* 1997;87:279–282)

Introduction

The control of tuberculosis (TB) within prisons and jails has been a long-standing concern.[1–5] Correctional institutions house large numbers of persons at high risk for tuberculosis, including persons of lower socioeconomic status, persons from inner cities where rates of *Mycobacterium tuberculosis* infection are higher, and persons co-infected with human immunodeficiency virus (HIV).[6–11] In addition, close quarters, poor ventilation, and overcrowding in these institutions facilitate transmission. *M. tuberculosis* infection has been found to be associated with increased time in or admission to a large city jail system.[12]

In fall 1991, three cases of active TB were diagnosed at a California state prison, two among inmates and one in a prison employee. A study was conducted to determine whether transmission of *M. tuberculosis* was occurring at this facility.

Methods

Because individual medical records do not remain at the prison, we identified cases of active TB at the state prison in 1991 by checking pharmacy records for inmates taking at least two anti-TB medications and checking laboratory culture results for specimens positive for *M. tuberculosis.* TB cases were defined by using the Centers for Disease Control and Prevention surveillance case definition for

At the time of the study Denise T. Koo and Roy C. Baron were with the Division of Field Epidemiology, Epidemiology Program Office, Centers for Disease Control and Prevention, Atlanta, Ga. George W. Rutherford is with the School of Public Health, University of California, Berkeley.

Requests for reprints should be sent to Denise T. Koo, MD, MPH, Division of Surveillance and Epidemiology, MS C-08, Epidemiology Program Office, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, Atlanta, GA 30333.

This paper was accepted July 15, 1996.

TABLE 1—Race and HIV Status of the 18 Patients with Active Tuberculosis in a California State Prison, 1991

	No.	%
Race		
African American	11	61
Caucasian	4	22
Latino	2	11
American Indian	1	6
HIV status		
Positive	7	39
Negative	6	33
Unknown	5	28

TABLE 2—Sites of Tuberculosis and Time of Diagnosis for 18 Patients in a California State Prison, 1991

			Time of Diagnosis	
Type of Tuberculosis	No.	%	Before Incarceration	After Incarceration[a]
Pulmonary	11[b]	61	5	6
Mesenteric	2	11	0	2
Knee osteomyelitis	2	11	0	2
Pleural	1[c]	6	1	0
Disseminated	1	6	1	0
Central nervous system	1[d]	6	0	1
Total	18	100	7	11

[a]One case was diagnosed after incarceration, during 1990.
[b]Includes two culture-negative cases.
[c]Culture negative.
[d]Also with pulmonary involvement.

clinically or laboratory-confirmed TB.[13] We defined incident cases of TB as those diagnosed during 1991 while an inmate was at the prison. We defined infectiousness among persons with TB by (1) a sputum smear positive for acid-fast bacilli, (2) the presence of cough and culture-positive sputum, or (3) the presence of a cavity on chest x-ray and a sputum culture positive for *M. tuberculosis*. To estimate the point prevalence of tuberculin skin test positivity among the general prison population, we used the results of prisonwide skin testing, conducted by the California Department of Corrections for the first time in November 1991. Prison policy had previously consisted of administering a tuberculin skin test to inmates only on (each) entry into the prison system. We defined a positive tuberculin skin test as a reaction of 10 mm or greater induration. We defined tuberculin skin test conversion as an increase in the reaction of at least 10 mm.[14] To estimate the risk of acquiring *M. tuberculosis* infection in the prison, we calculated the incidence of skin test conversions occurring within the previous 2 years among inmates at risk for new *M. tuberculosis* infections.

We presumed that *M. tuberculosis* infection was acquired in the state prison system if an inmate had a documented negative tuberculin skin test on or after entry into the state prison system and if he became ill with TB or his skin test converted without his having left prison. For recent converters, we calculated the time in the prison system until conversion as half the number of days in the conversion interval, since it could not be determined at what point in the interval the inmate became infected.

Results

Incidence of Active Tuberculosis

Case finding revealed 18 inmates with active TB diagnosed or treated at this state prison during 1991. Race and HIV status are listed in Table 1.

TB sites are listed in Table 2. Of the 15 culture-confirmed cases, 12 (80%) involved *M. tuberculosis* infections that were sensitive to all drugs tested and 3 each were infected with organisms resistant to only one of the following: isoniazid, streptomycin, or ethambutol.

Eight of the 18 cases were diagnosed prior to the individual's prison entry or before 1991 (Table 2). The remaining 10 cases were incident at the prison during 1991. On the basis of the midyear population of 5421 inmates, the estimated annual incidence of TB at this prison was 184 cases per 100 000 inmates.

As a result of delayed sputum conversion, 3 of the 18 inmates with TB (17%) were presumed to have been infectious for a collective total of 7 person-months while imprisoned in 1991.

At least 2 of the 18 inmates with active TB may have acquired the disease at this state prison. Neither man left the state prison in the 8 months between the negative tuberculin skin test on reentry to prison and the diagnosis of TB. There were no known associations between the 3 infectious inmates and these latter 2 inmates.

A US-born correctional counselor developed culture-negative pulmonary TB after counseling inmates in the prison HIV unit for 16 months. This employee recalled exposure to one of the three infectious inmates and denied known exposure to *M. tuberculosis* outside the prison. The first positive tuberculin skin test was noted during this employee's diagnostic evaluation for TB; the most recent prior negative tuberculin skin test occurred 1 year before the individual's employment at the state prison.

Incidence of New Infection

Among 2944 inmates with known tuberculin skin test results, 873 had positive results, corresponding to an estimated point prevalence of 30%. Of these 873 inmates, 324 tested positive for the first time during the November 1991 testing. Of this group of 324, 148 had documented prior negative tests, 155 had no recorded prior skin tests, and 21 tested positive, but the number of millimeters of induration was not recorded.

Among the 148 identified converters, the mean age was 35 years. Fifty-three (36%) were foreign born, and 56 (38%) were Latino; 40 were both foreign born and Latino. Approximately 25% of the overall prison population was foreign born, and 25% of the population was Latino. Prisoners were often grouped for housing in the prison by racial or ethnic background. In addition, 143 converters (97%) had a documented prior negative result recorded as 0 mm or negative, and 127 (86%) had an increase in purified protein derivative induration of 12 mm or greater.

Of the 148 inmates with documented conversions, 105 had been incarcerated

within the prison system during the entire interval between negative and positive TB skin tests. Of these individuals, 97 had converted within the previous 2 years. The conversion incidence (risk of acquisition of tuberculosis infection) for the previous 2 years in the state prison system was 5.9 per 100 person-years spent in prison.

Discussion

The findings of this investigation suggest that transmission of *M. tuberculosis* has occurred in the California state prison system. The high incidence of new infection associated with incarceration supports this conclusion. In addition, at least two inmates with active TB may have become infected and developed the disease at the prison. Acquisition of *M. tuberculosis* infection was probably a result of exposure to incompletely treated or unrecognized cases of active pulmonary or laryngeal TB among the prison population. The prolonged infectiousness involved with the three active cases illustrates the potential for *M. tuberculosis* exposure in prison.

On the basis of the number of known cases, the minimum estimate of the incidence of active TB in this prison (184 per 100 000 persons per year) was more than 10 times that reported among the general population in California during 1991 (17.4 per 100 000 persons per year; California Department of Health Services, Tuberculosis Control Branch). The 10 new cases were 3.3 times the number expected among California residents of the same race, age, and gender composition as the prison population (3.0 expected cases, adjusted with data from the Tuberculosis Control Branch). The yearly incidence of new *M. tuberculosis* infection (5.9 per 100 person-years in prison) was comparable in scale to that found in other prison investigations.[1,3,8,15,16]

Our calculations of the prevalence and incidence of *M. tuberculosis* infection probably underestimate the magnitude of the problem in the state prison system. The lack of prior skin test results forced us to exclude 155 inmates with positive tuberculin skin tests in November 1991. In addition, we do not know the prevalence of HIV infection among the prisoners; in 1988, 5.3% of inmates entering the California correctional system from the predominant catchment area of this prison were HIV positive.[17] Currently, an estimated 17% to 20% of New York State inmates are HIV positive,[18] and the usefulness in HIV-infected persons of skin tests for documenting recent *M. tuberculosis* infection has been questioned.[19] HIV-related anergy or defining tuberculin skin test positivity as 10 or more mm (rather than 5 mm) may have led to further underestimation of the prevalence and incidence of *M. tuberculosis* infection.

Some of the conversions observed might have been due to the boosting phenomenon. Boosting occurs when serial tests are required in order to "boost" a waned immune response to *Mycobacteria.* The high prevalence of foreign-born Latino inmates among the converters raises this concern, because boosting has been found to occur more frequently among racial and ethnic minorities and the foreign born.[20] It increases with age,[21,22] and it also occurs more often among people with an initial slight reaction than among those with an initial negative reaction.[22,23]

It is unlikely that boosting played a substantial role in the estimation of conversion incidence, however. More than half of the converters were neither Latino nor foreign born, and the mean age of the converters was 10 years younger than that of persons exhibiting boosting in one study.[22] In addition, 97% of the converters had a documented prior negative result, and 86% had an increase in purified protein derivative induration of 12 mm or greater. Stead and To[24] and Narain[25] suggested that a 12-mm increase in induration be used as the conversion criterion, contending that the larger the purified protein derivative reaction, the more likely the reaction is due to a new infection (and not to boosting). Purified protein derivative conversions among inmates are therefore more likely to be manifestations of new *M. tuberculosis* infections.

The transmission of *M. tuberculosis* in this prison indicates the need to stringently enforce infection control practices in the state prison system. Such enforcement is particularly important now given the increasing prevalence of HIV infection[18] in prisons and the threat of the introduction of multidrug-resistant TB into this environment. Insufficient adherence to infection control guidelines has also been implicated in nosocomial transmission of multidrug-resistant TB in hospitals.[26,27] The California Department of Corrections is addressing infection control issues in its facilities, with special emphasis on routine screening of all inmates and employees for TB infection, early acid-fast bacilli isolation and appropriate treatment of persons with suspected TB, timely and accurate contact investigations, and tracking systems to improve continuity of care for both inmates and former inmates. □

Acknowledgments

We thank the California Department of Corrections and the California Department of Health Services for inviting our participation in this investigation, which could not have proceeded without their help. We also thank Dr George Cauthen of the Division of Tuberculosis Elimination, Centers for Disease Control and Prevention, for his technical advice.

References

1. Spencer SS, Morton AR. Tuberculosis surveillance in a state prison system. *Am J Public Health.* 1989;79:507–509.
2. King L, Geis G. Tuberculosis transmission in a large urban jail. *JAMA.* 1977;237:791–792.
3. Stead WW. Undetected tuberculosis in prison: source of infection for the community at large. *JAMA.* 1978;240:2544–2547.
4. Abeles H, Feibes H, Mandel E, et al. The large city prison—a reservoir of tuberculosis. *Am Rev Respir Dis.* 1970;101:706–708.
5. Snider DE, Hutton MD. Tuberculosis in correctional institutions. *JAMA.* 1989;261:436–437.
6. Centers for Disease Control. Tuberculosis morbidity in the United States: final data, 1990. *MMWR Morb Mortal Wkly Rep.* 1992;40:23–27.
7. Selwyn PA, Hartel D, Lewis VA, et al. A prospective study of the risk of tuberculosis among intravenous drug users with human immunodeficiency virus infection. *N Engl J Med.* 1989;320:545–550.
8. Braun MM, Truman BI, Maguire B. Increasing incidence of tuberculosis in a prison inmate population. *JAMA.* 1989;261:393–397.
9. Morse DL, Truman BI, Hanrahan JP, et al. AIDS behind bars: epidemiology of New York state prison inmate cases, 1980–1988. *NY State J Med.* 1990;90:133–138.
10. Hutton MH, Cauthen GM, Bloch AB. Results of a 29-state survey of tuberculosis in nursing homes and correctional facilities. *Public Health Rep.* 1993;108:305–314.
11. Centers for Disease Control. Transmission of multidrug-resistant tuberculosis among immunocompromised persons in a correctional system—New York, 1991. *MMWR Morb Mortal Wkly Rep.* 1992;41:507–509.
12. Bellin EY, Fletcher DD, Safyer SM. Association of tuberculosis infection with increased time in or admission to the New York City jail system. *JAMA.* 1993;269:2228–2231.
13. Centers for Disease Control. Case definitions for public health surveillance. *MMWR Morb Mortal Wkly Rep.* 1990;39(RR-13):39–40.

14. California Department of Health Services/ California Tuberculosis Controllers Association. Guidelines for determination of tuberculosis skin test conversion in California. *In: TB Policy Guide;* April 17, 1991.
15. *1991 Tuberculosis Statistics in the United States.* Atlanta, Ga: Centers for Disease Control; 1993.
16. Centers for Disease Control. Prevention and control of tuberculosis in correctional institutions: recommendations of the Advisory Committee for the Elimination of Tuberculosis. *MMWR Morb Mortal Wkly Rep.* 1989;38:313–320, 325.
17. Singleton JA, Perkins CI, Trachtenberg AI, Hughes MJ, Kizer KW, Ascher M. HIV antibody seroprevalence among prisoners entering the California correctional system. *West J Med.* 1990;153:394–399.
18. *HIV Disease in Correctional Facilities.* Washington, DC: National Commission on Acquired Immune Deficiency Syndrome; 1991.
19. Glaser JB, Aboujaoude JK, Greifinger R. Tuberculin skin test conversion among HIV-infected prison inmates. *J Acquir Immune Defic Syndr.* 1992;5:430–431.
20. Fujiwara P, Steinhart R, Schecter G, Hopewell P, Cohen P. Two-step PPD testing in new employees at San Francisco General Hospital, 1982–1989. Presented at the annual meeting of the American Thoracic Society, May 1992, Miami, Fla.
21. Comstock GW, Woolpert S. Tuberculin conversions: true or false? *Am Rev Respir Dis.* 1978;118:215–217.
22. Thompson NJ, Glassroth JL, Snider DE, Farer LS. The booster phenomenon in serial tuberculin testing. *Am Rev Respir Dis.* 1979;119:587–589.
23. Narain R, Nair SS, Rao GR, Chandrasekhar P, Lal P. Enhancing of tuberculin allergy by previous tuberculin testing. *Bull WHO.* 1966;34:623–638.
24. Stead WW, To T. The significance of the tuberculin skin test in elderly persons. *Ann Intern Med.* 1987;107:837–842.
25. Narain R. Tuberculin conversions: true or false? *Am Rev Respir Dis.* 1979;119:1039–1040.
26. Rudnick JR, Kroc K, Manangan L, Banerjee S, Pugliese G, Jarvis W. Are U.S. hospitals prepared to control nosocomial transmission of tuberculosis? Presented at the 42nd Annual Epidemic Intelligence Service Conference, April 1993, Atlanta, Ga.
27. Pearson ML, Jereb JA, Frieden TR, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis:* a risk to patients and health care workers. *Ann Intern Med.* 1992;117:191–196.

Motor-Vehicle Crash Fatalities among American Indians and Non-Indians in Arizona, 1979 through 1988

Doug Campos-Outcalt, MD, Dimitri Prybylski, MPH, Arleen J. Watkins, DEd, Gary Rothfus, MPH, and Alan Dellapenna, MPH

ABSTRACT

Objectives. This study evaluated the contributions of rural residence, alcohol use, and pedestrian fatalities to the high American Indian motor-vehicle crash mortality rate in Arizona.

Methods. Records from the Fatal Accident Reporting System were used to examine mortality rates between 1979 and 1988.

Results. American Indians had increased relative risks in all motor-vehicle crash categories in all residence–gender groups. The percentage of excess mortality associated with alcohol varied from 36.8% to 66.7%, and the percentage associated with pedestrian deaths ranged from 27.2% to 55.4%.

Conclusions. Efforts to reduce excess motor-vehicle crash mortality among American Indians should concentrate on preventing pedestrian and alcohol-related fatalities. (*Am J Public Health.* 1997;87:282–285)

Introduction

In Arizona, as in the entire United States, motor-vehicle crashes are a major public health problem for American Indians.[1–7] In 1990, the age-adjusted motor-vehicle crash mortality rate was 23.8/100 000 for all ethnic groups and 81.6 for American Indians.[3] A high proportion of pedestrian deaths among American Indian motor-vehicle crash fatalities has been documented in several locations.[8–10]

Several hypotheses have been proposed or investigated to explain the elevated motor-vehicle crash fatality rate among American Indians. Some studies have indicated that alcohol intake is a partial explanation.[8,10,11] It has also been hypothesized that geographic factors are important, especially in western tribes where reservations are often located in isolated, rural areas.[11] It has been demonstrated that motor-vehicle crash mortality rates are higher in communities with low population densities.[12,13] These higher rates may be due to road characteristics, travel speeds, patterns of seat belt use, types of vehicles, and availability and quality of emergency services.[14] Other hypothesized causes of high American Indian motor-vehicle crash mortality include lax law enforcement, unlicensed drivers,[15] and social stress due to modernization.[16]

The purpose of this study was to examine motor-vehicle crash fatalities of American Indians and non-Indians in the state of Arizona and to study the relative

Doug Campos-Outcalt is with the Department of Family and Community Medicine, Maricopa Medical Center, Phoenix, Ariz. Dimitri Prybylski is with the Maricopa County Public Health Department, Phoenix. Arleen J. Watkins is with the Department of Family and Community Medicine, University of Arizona College of Medicine, Tucson, Ariz. At the time this paper was written, Gary Rothfus was with the Division of Environmental Health Services, Tucson Area Indian Health Services. Alan Dellapenna is with the Office of Environmental Health and Engineering, Phoenix Area Indian Health Services, Phoenix.

Requests for reprints should be sent to Doug Campos-Outcalt, MD, Department of Family and Community Medicine, Maricopa Medical Center, 2601 E Roosevelt, Phoenix, AZ 85008.

This paper was accepted May 17, 1996.

Note. The views expressed here are the authors' and not necessarily those of the Indian Health Service.