Hansen’s disease, or leprosy, is caused by the bacteria *Mycobacterium leprae*. Infected persons experience a variety of clinical forms attributed to the immune response.\(^1\)\(^2\) Hansen’s disease is categorized by clinical, immunologic, and histopathologic findings. Localized disease, termed tuberculoid or pauci-bacillary leprosy, is characterized by a single or few hypopigmented or erythematous skin lesions. Sensory loss, impaired sweating, and loss of hair may occur within the lesions. Few bacteria are seen in skin biopsies. Generalized disease, termed lepromatous or multi-bacillary leprosy, is characterized by numerous, symmetrically distributed skin lesions. The lesions may be macular, papular, or nodular in appearance. Nerve damage is usually present. Many bacteria are seen in skin biopsies. Between localized and generalized disease is a spectrum of disease termed borderline or dimorphic leprosy. Multi-bacillary leprosy is the most contagious form of Hansen’s disease.

Humans are the principal reservoir of *M. leprae*. The incubation period from exposure to onset of illness ranges from one to 10 years or more but is commonly 3–5 years. The bacteria are found in skin lesions and in the nasal and oral mucosa of infected persons. Transmission occurs by contact with infected skin and by inhalation of aerosolized droplets containing *M. leprae*. However, most patients have no known human contacts with leprosy.\(^3\)\(^4\) Many environmental nonhuman exposures have been suggested.\(^5\) Exposure to armadillos has been suggested as a possible risk factor for Hansen’s disease. A study of Hansen’s disease patients in Los Angeles, California, found an association with Hansen’s disease in males after exposure to armadillos.\(^6\) This association was not seen in female patients. However, no association with contact with armadillos was found for patients in Louisiana.\(^7\)

Two antibiotics, dapsona and rifampin, given together for six months, constitute the current therapy for treating paucibacillary disease. Therapy for multi-bacillary disease consists of three antibiotics, dapsona, rifampin, and clofazimine, given for two years or longer.\(^1\)

From 1991 through 1995, 136–187 Hansen’s disease cases were reported annually in the United States.\(^8\) Although only 25% of the patients in the United States are from Texas, Texas reports a majority (60%) of the indigenous cases.\(^9\) Information concerning secular trends and demographic characteristics of endemic cases in Texas and other states is limited. The last description of Hansen’s disease in Texas was published more than 40 years ago.\(^10\) The present study examines the demographic and clinical characteristics of Hansen’s disease cases in Texas and provides a detailed description of an endemic focus.

**METHODS**

Hansen’s disease cases are reported to the Hansen’s Disease Program of the Texas Department of Health by physicians, local health departments, and other medical facilities. We reviewed the following data for cases reported from January 1, 1973, through December 31, 1997: name, date of birth, country of birth, sex, race/ethnicity, date of arrival into the United States, dates of onset of disease and diagnosis, city and county of residence at diagnosis, known contact with another Hansen’s disease case, presence of deformity or insensitivity at diagnosis, disease type, initial bacteriology findings, and known drug resistance. Patients with tuberculoid or indeterminate disease were classified as pauci-bacillary. Patients with lepromatous or borderline disease were classified as multi-bacillary.

Incidence determinations were based on United States census population data for Texas from either the 1970 census for the years 1973–1977, the 1980 census for the years 1978–1987, and the 1990 census for the years 1988–1997.\(^11\)\(^–\)\(^13\) When a response to a specific question was not known, the case was excluded from analysis for that specific variable. All analyses were performed with Epi Info (Centers for Disease Control and Prevention, Atlanta, GA) software. Categorical groups were analyzed by Fisher’s exact test or by chi-square analysis. Comparisons of two means were made by Student’s *t*-test.

**RESULTS**

From 1973 through 1997, 810 Hansen’s disease cases were reported in Texas. The number of cases reported annually ranged from 18 in 1974 to 54 in 1992; 26–42 cases were reported annually during 1993 through 1997. The average annual incidence rates for various time periods ranged from 1.9 cases per million population during 1973 through 1977 to 2.4 cases per million population during 1983 through 1987.

A majority of patients were males (63%) and white (77%). Approximately one-fifth (19%) were Asian; half (51%) reported being Hispanic. Only 24 (3.0%) patients were African-American. For comparison, 2% of the Texas population
is Asian, 12% is African-American, and 75% is white. Hispanics of any race comprise 25% of the population. Patients ranged in age at onset of illness from two to 87 years (median = 44 years). Patients born in the United States were older at onset (mean age = 50.6 years) compared with foreign-born patients (mean age = 36.0 years) \( (P < 0.0001) \).

The place of birth was known for 784 patients. More than half (53%) of the patients were born in the United States. Table 1 shows demographic characteristics for United States-born and foreign-born patients by time period of report. The percentage of patients born in the United States ranged from 46% for 1983–1987 to 59% for 1973–1977. From 1993 through 1997, 57% of the patients were born in the United States.

For patients born in the United States, the percentage of Hispanic patients decreased over time \( (P = 0.001, \) by test for trend). For foreign-born patients, the percentage of Asian patients has increased while the percentage of patients born in Mexico has decreased. Mean age at onset of illness for both United States-born and foreign-born patients did not change notably over time. The mean age at onset was younger for foreign-born persons compared with those born in the United States for each of the time periods examined.

Overall, a human source was identified for 23% of the patients. The percentage of patients with a human source identified was similar for patients born in the United States (21%) and those born outside the United States (24%). The percentage of foreign-born patients with a known source decreased over time \( (P = 0.003, \) by test for trend). This decrease was not seen with patients born in the United States.

Most (56%) patients were diagnosed within one year from onset of illness. For foreign-born patients, the time between onset and diagnosis was longer (mean = 3.2 years) compared with patients born in the United States (mean = 2.6 years) \( (P = 0.003) \). Most (76%) patients were diagnosed with multi-bacillary leprosy. Deformity was noted at diagnosis for 10%. At diagnosis, loss of sensation was noted for 45.7%. Foreign-born patients (82%) were more likely than to have multi-bacillary disease at diagnosis compared with patients born in the United States (71%) \( (\text{odds ratio } [\text{OR}] = 1.81, 95\% \text{ confidence interval } [\text{CI}] = 1.27–2.58) \).

Of the 254 counties in Texas, 101 had at least one Hansen's disease patient residing in the county. Harris County (Houston) and Dallas County were the counties of residence of 129 and 81 patients, respectively. A total of 582 patients were residing in Texas at onset of disease, and most (91%) resided in Texas for at least five years. Figure 1 shows the average annual incidence rates (cases/million population) by county. Goliad, Bee, and Refugio Counties had the highest average annual incidence rates of 96.1, 38.8, and 37.0 cases per million population, respectively. An endemic focus of 12 counties with high average annual incidence rates was located east and southeast of San Antonio, Texas. A total of 144 patients resided in this endemic focus. From 15% to 20% of the cases reported annually in Texas were in patients residing in this area. Patients residing in this endemic focus were more likely to be born in Texas (85%) compared with patients residing outside the area (35%) \( (\text{OR} = 10.6, 95\% \text{ CI} = 6.28–18.1) \). The percentage of patients with a known human contact was similar for patients residing in the endemic focus compared with those outside the focus.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No. born in United States*</td>
<td>64</td>
<td>77</td>
<td>77</td>
<td>102</td>
<td>93</td>
<td>413</td>
</tr>
<tr>
<td>White</td>
<td>95.3%</td>
<td>92.2%</td>
<td>97.4%</td>
<td>97.1%</td>
<td>93.5%</td>
<td>95.2%</td>
</tr>
<tr>
<td>Hispanic</td>
<td>59.4%</td>
<td>55.8%</td>
<td>58.4%</td>
<td>34.3%</td>
<td>41.9%</td>
<td>48.4%</td>
</tr>
<tr>
<td>Male sex</td>
<td>60.9%</td>
<td>61.0%</td>
<td>62.3%</td>
<td>57.8%</td>
<td>65.6%</td>
<td>61.5%</td>
</tr>
<tr>
<td>Mean age at onset (years)</td>
<td>49.1</td>
<td>49.6</td>
<td>48.6</td>
<td>54.2</td>
<td>50.4</td>
<td>50.6</td>
</tr>
<tr>
<td>Born in Texas</td>
<td>82.5%</td>
<td>88.3%</td>
<td>90.9%</td>
<td>81.2%</td>
<td>77.4%</td>
<td>83.7%</td>
</tr>
<tr>
<td>Multi-bacillary</td>
<td>81.2%</td>
<td>70.1%</td>
<td>63.6%</td>
<td>74.2%</td>
<td>68.5%</td>
<td>71.3%</td>
</tr>
<tr>
<td>Known source</td>
<td>23.8%</td>
<td>31.9%</td>
<td>25.4%</td>
<td>15.3%</td>
<td>17.3%</td>
<td>21.4%</td>
</tr>
<tr>
<td>No. foreign-born</td>
<td>44</td>
<td>78</td>
<td>89</td>
<td>90</td>
<td>70</td>
<td>371</td>
</tr>
<tr>
<td>Asian</td>
<td>27.9%</td>
<td>42.3%</td>
<td>43.8%</td>
<td>42.2%</td>
<td>44.3%</td>
<td>41.3%</td>
</tr>
<tr>
<td>Hispanic</td>
<td>72.1%</td>
<td>53.8%</td>
<td>53.9%</td>
<td>55.6%</td>
<td>48.6%</td>
<td>55.4%</td>
</tr>
<tr>
<td>Male sex</td>
<td>61.4%</td>
<td>71.8%</td>
<td>58.4%</td>
<td>64.4%</td>
<td>72.9%</td>
<td>65.8%</td>
</tr>
<tr>
<td>Mean age at onset (years)</td>
<td>35.3</td>
<td>34.0</td>
<td>33.4</td>
<td>39.2</td>
<td>38.4</td>
<td>36.0</td>
</tr>
<tr>
<td>Born in Mexico</td>
<td>65.9%</td>
<td>50.0%</td>
<td>50.6%</td>
<td>51.1%</td>
<td>44.3%</td>
<td>51.2%</td>
</tr>
<tr>
<td>Multi-bacillary</td>
<td>84.1%</td>
<td>88.5%</td>
<td>80.7%</td>
<td>73.0%</td>
<td>85.5%</td>
<td>81.8%</td>
</tr>
<tr>
<td>Known source</td>
<td>39.3%</td>
<td>38.6%</td>
<td>20.0%</td>
<td>18.6%</td>
<td>17.1%</td>
<td>24.2%</td>
</tr>
</tbody>
</table>

* The place of birth was unknown for 26 cases.
from 1.7 to 2.3 cases per million population in Texas. From 1930 through 1940, average annual incidence rates ranged from 1.7 to 2.3 cases per million population; the rate for these two counties is now greater than 30 cases per million population. This persistent endemic focus may be explained by a large population of genetically susceptible persons or an environmental exposure to M. leprae or both. The persistent occurrence of cases and the high percentage of cases with no known human contact suggest continual environmental exposures. Knowledge of the exposures could be used to prevent additional cases.

The incidence and demographic characteristics of Hansen’s disease in Texas have changed little over time. From 1930 through 1940, average annual incidence rates ranged from 1.7 to 2.3 cases per million population in Texas. These rates are similar to those reported during 1973 through 1997. Annually the percentage of cases in males, approximately 60%, and the percentage in African-Americans, approximately 3%, have remained the same since 1930. A demographic characteristic that has changed over time is country of birth. From 1930 through 1939 and from 1950 through 1961, 61% and 62% of the cases reported in Texas were in patients born in Texas. Fewer cases (42–48%) reported from 1973 through 1997 were in patients born in Texas. However, more patients are now born outside the United States and Mexico. During 1973 through 1997, 23.1% of the patients were born in other counties besides the United States and Mexico. Fifty years ago, less than 1% of patients were born outside the United States and Mexico. Differences in country of birth are also seen when reviewing cases reported from other states. In New York, only 1% of the patients are born in the United States and only 1% of the foreign-born patients are born in Mexico.

In Texas, the patient’s clinical types and age are similar to types and age reported for patients in the United States. Lepromatous and dimorphous types represent 65% of the cases in the United States. These types represent 74% of the cases reported in Texas. For indigenous cases in Texas, the mean age at onset of illness for patients is 50 years. For indigenous cases in the United States from 1971 through 1988, the mean age was 49 years.

Texas is one of a few states reporting indigenous cases of Hansen’s disease. From 1950 to 1955, more than 10 indigenous cases were reported in Hawaii each year. However, from 1990 to 1995, fewer than two cases were reported each year. From 1950 through 1969, 27 indigenous cases were reported in Florida. However, from 1971 through 1988, no indigenous cases were reported. While the occurrence of indigenous cases in other states has decreased, no decrease is apparent in Texas.

Texas now reports more than 60% of the indigenous cases in the United States compared with approximately 20% from 1950 through 1969. Within Texas, an endemic focus exists along the Gulf of Mexico coast. This focus has existed for more than 50 years. The factors responsible for the persistence of indigenous cases in Texas are a mystery.

Acknowledgments: We thank the physicians and local public health department staff throughout Texas for their efforts in collecting demographic and clinical information from Hansen’s disease patients, and Susan Hammock for reviewing the manuscript.

Authors’ addresses: Jeffery P. Taylor and Jeffrey W. Smedley, Tuberculosis Elimination Division, Texas Department of Health, 1100 West 49 Street, Austin, TX 78756. Isabel Vitek and Virginia Enriquez, Hansen’s Disease Program, Texas Department of Health, 1100 West 49 Street, Austin, TX 78756.

Reprint requests: Jeffery P. Taylor, Tuberculosis Elimination Division, Texas Department of Health, 1100 West 49 Street, Austin, TX 78757.

REFERENCES


