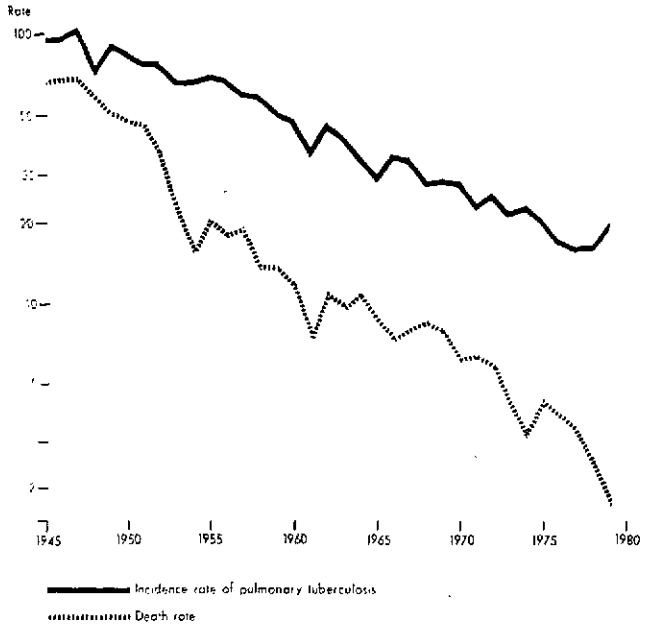
Figure 1. Incidence rates of pulmonary tuberculosis and of deaths due to tuberculosis per 100,000 population. Costa Rica, 1945-1979.



this ratio is not really a case fatality rate since the data come from different sources—records of new cases of pulmonary tuberculosis, death certificates—it does indicate a lower risk of death in the general population and in the known cases of this disease. The impact of specific therapy became evident between 1951 and 1961; the stabilization in the following years could be interpreted as late mortality in chronic cases, which was delayed but not prevented by the therapy. The ratio (which in 1965 had been 0.3) continued to fall, which points to an increase in diagnostic coverage and in the quality of the treatment of the cases detected.

In recent years, the death rate has continued to decline, but the number of cases has increased, which may be due to more intensive case detection. Extrapulmonary tuberculosis increased during the course of the program: in 1945-1949, 10 cases were reported (0.3 per cent) as opposed to 157 cases (7.5 per cent) in 1973-1977. Tuberculosis incidence fell more rapidly in children under 15 years (from 29.7 per 100,000 population in 1950 to 1.2 in 1977) than in the age group 15-44 years and in the age group 45 years and over (137.2 to 24.0 and 119.4 to 40.6 per 100,000 population, respectively).

Each year the national control program prepares an annual work program, quantified by health service and by health region (five regions). For 1980, it provides for 22,000 sputum examinations for the purpose of detecting and treating 639 cases (515 on an outpatient basis). In 1980 short course treatment will be begun, including the administration of isoniazid, rifampicin, and pyrazinamide, with complete supervision in 255 cases.

In 1977 BCG vaccination, which was begun in 1968, covered 76.7 per cent of the newborn (88.8 per cent of those born in official institutions) and 81.3 per cent of the school population, according to studies of the prevalence of scars prior to vaccination in school.

(*Source:* Tuberculosis Program, Communicable Disease Control Unit, Disease Prevention and Control Division, PAHO)

Status of Eastern Equine Encephalitis in Venezuela

Eastern equine encephalomyelitis (EEE) virus was first isolated in Venezuela in sentinel hamsters exposed to the infection in 1975 in the area of Catatumbo, Zulia State.[1] The virus was isolated at the Institute of Veterinary Research in Maracay in 1976 and in 1978, from autopsy materials taken from horses from Zulia[2] and Yaracuy States, respectively. Likewise, in 1978 the virus was found in sentinel hamsters in the swampy area at the end of Lake Maracaibo. In 1979 the virus was again recovered from horses that had died in Yaracuy. These findings

[1] Walder, R. and O.M. Suárez, Primera evidencia en Venezuela de la encefalitis equina del este (EEE) en circunstancias silentes. *Bol Div Maraliol y Saneam Ambiental* 16 (2):119-125, 1976.

[2] de Siger, J., N. Metter, and J. Castañeda. First isolation of eastern equine encephalomyelitis virus from a horse in Venezuela. 19th Annual Proceedings of the *American Association of Veterinary Laboratory Diagnosticians*, 229-236.

and those of the study of horse sera obtained between 1972 and 1979 in various states provide some idea of the distribution of EEE in Venezuela.

Figure 1 showing the political subdivisions of Venezuela indicates the areas where the sera were obtained.

Sera were subjected to hemagglutination-inhibition, complement-fixation, and neutralization tests using Venezuelan equine encephalitis (VEE) and EEE antigens. This report is based on the findings related to EEE. In each case a careful analysis was made to determine whether the findings could be attributed to EEE or VEE alone, to dual infection, or to serologic crossings. Sera

Figure 1. Zones from which sera were collected to determine the presence of hemagglutination-inhibiting antibodies to eastern equine encephalitis in horses. Venezuela, 1972-1979.

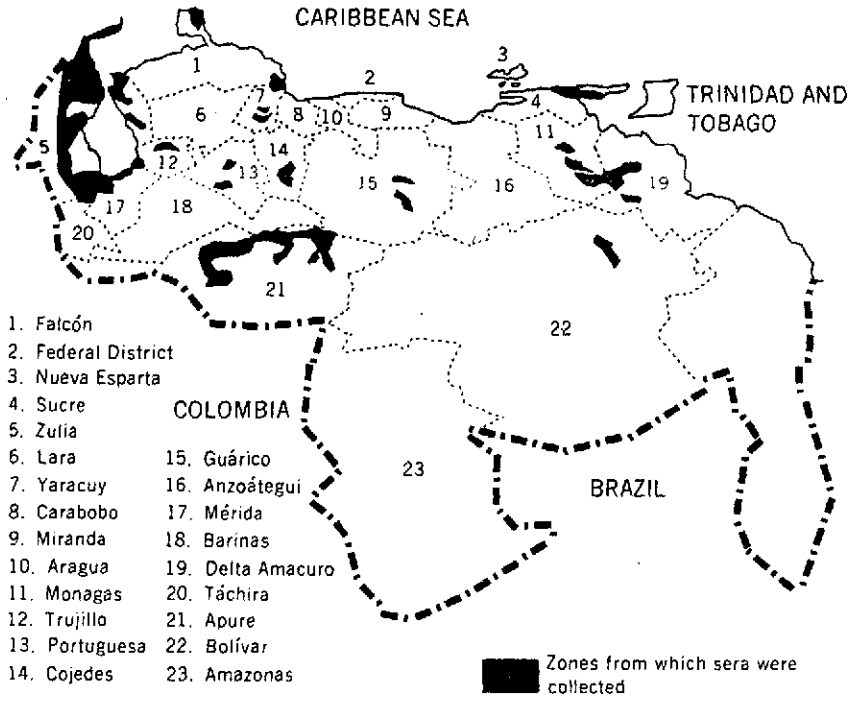


Table 1. Findings of the serologic survey in states of Venezuela, 1972-1979.

State	Year	Isolation of the virus	Presence of antibodies	No. of serum samples tested	Endemicity
Bolívar	1978	—	—	42	—
Delta Amacuro	1973	—	[b]	71	High
Monagas	1972, 1977	—	[b]	150	Moderate
Sucre	1972	—	—	76	—
Guárico	1976	—	[b]	79	(Present and past activity)
Apure	1975, 1978	—	[b]	220	Low
Trujillo	1979	—	—	43	—
Portuguesa	1975	—	—	4	—
Cojedes	1978	—	[b]	208	(Presence of the virus)
Yaracuy	1976, 1979	—	[b]	81	(Present and past activity)
Mérida	1978	[a]	[b]	17	(Recent activity)
Táchira	1978	—	[b]	107	(Recent intense activity)
Falcón	1972, 1979	—	[b]	69	(In 1972 [59 samples] no activity)
Zulia	1973, 1974	—	[b]	...	North, very high. West of Lake, high
	1976, 1978	[a]	[b]	353	East of Lake, moderate to intense

[a] Isolated.
[b] Antibodies present.

from Zulia, Yaracuy, Apure, and Guárico were tested also with western equine encephalitis (WEE) antigen, but no antibodies against this virus were found.

Table 1 describes the EEE situation in the states that were visited for the purpose of conducting the serologic survey or establishing the etiology of cases of encephalitis in equines.

Predictably, the above information indicates that EEE activity is related to the presence of surface waters such as swamps, marshes, and ponds, and possibly to waterfowl and mosquito populations; this situation is observed in Delta Amacuro and in the southwestern part of Lake Maracaibo. The opposite occurs in semidesert areas, where stagnant surface waters, if they exist at all, are found only during brief periods in the short rainy season; the same is true along the Caribbean coastal area of Zulia and Falcón, where EEE does not exist.

Epidemiological differences between EEE and VEE are marked in these arid zones where recurrent and explosive VEE epidemics are observed. Between the two above-described extremes, a range of ecological combinations prevails with varying grades of EEE activity, which includes small local outbreaks. To date there is no evidence of EEE infection in man.

(*Source: Boletín Epidemiológico Semanal 7,* 1980. Ministry of Health and Social Welfare of Venezuela)

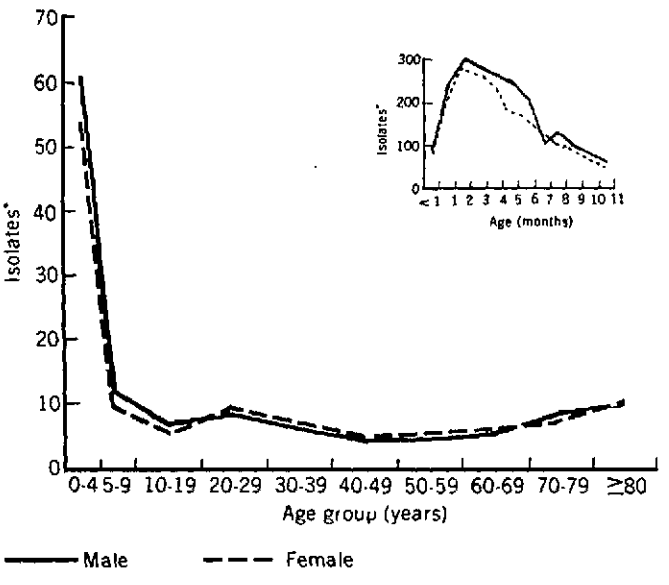
Human *Salmonella* Isolates—United States, 1979

In 1979, 31,123 isolations of salmonellae (including *Salmonella typhi*) from humans were reported to CDC—an increase of 8.3 per cent over 1978.

The increase in isolates was not confined to one state or region. However, five states—Connecticut, Massachusetts, Maryland, Washington, and Illinois—accounted for two-thirds of the 8.3 per cent increase. *S. enteritidis* alone accounted for over one-fourth of the increase; most of this occurred in Connecticut and Massachusetts. *S. enteritidis, S. heidelberg, S. saint-paul,* and *S. infantis* accounted for almost two-thirds of the increase. These additional isolates were not concentrated in any single age group. The 10-19 year age group sustained the largest percentage increase, but increases were also seen for the age groups 30-39 years and 50-79 years.

The age distribution of persons from whom isolates were obtained (Figure 1) followed a well-established pattern: the rate was highest for infants approximately 2-3 months of age, decreased rapidly through early childhood, and then held fairly constant from approximately age 7 through the adult years. Isolation rates for those under 20 were higher for males than for females, but for

Figure 1. Rate of reported isolates of *Salmonella*, by age, United States, 1979.



*Per 100,000 population