Conf #3
5/4-5/89

LYME DISEASE

CONFERENCE

THIS CONFERENCE IS PRESENTED BY

TEXAS DEPARTMENT OF HEALTH
1100 WEST 49TH STREET
AUSTIN, TEXAS 78756

LYME BORRELIOSIS FOUNDATION, INC.
P.O. BOX 462
TOLLAND, CONNECTICUT 06084

#3
5/4-5/89

May 4, 1989 — Moderators: Julie Rawlings/Jeff Taylor

8:00-8:15	Introductions and Welcome Dr. Robert Bernstein Karen Forschner
8:15-8:45	Overview, Lyme Borreliosis Dr. Willy Burgdorfer
8:45-9:15	Overview, Relapsing Fever Dr. Doug Hurley
9:15-9:45	Biology of Borrelia Dr. Alan Barbour
9:45-10:15	Break (Provided by Whittaker Bioproducts)
10:15-10:45	Clinical Diagnosis and Treatment Dr. John Drulle
10:45-11:15	Serologic Confirmation of Lyme Borreliosis Dr. Paul Mitchell
11:15-11:45	Epidemiology Dr. Ted Tsai
11:45-12:15	Dermatologic Manifestations Dr. Alan MacDonald
12:15-1:30	Lunch
1:30-2:00	Neurologic Manifestations Dr. Audrey Stein-Goldings
2:00-2:30	Neuro-Ophthalmologic Manifestations Dr. J. Lawton Smith
2:30-3:00	Pathology Dr. Paul Duray
3:00-3:30	Break
3:30-4:00	Isolation of Borrelia Dr. Alan MacDonald
4:00-4:30	Molecular Studies Dr. Tom Schwan
4:30-5:00	New non-antibiotic treatment for neuro-borreliosis Dr. Henry Heimlich
5:00-5:30	Reservoirs Dr. John Anderson
5:30-6:00	Ecology Dr. Edward Bosler

May 5, 1989 — Moderator: Keith Clark

8:00-8:30	Veterinary Aspects/Non-tick Transmission Dr. Elizabeth Burgess
8:30-8:45	Veterinary Research Dr. Arturo Angulo
8:45-9:00	Veterinary Research Dr. Keith Clark
9:00-10:00	Case Studies Dr. Robert Longfield Diana Eaton Dr. Paul Lavoie
10:00-10:30	Break (Provided by Wampole Laboratories)
10:30-11:00	Pediatric and Congenital Lyme Borreliosis Dr. Lawrence Zemel
* 11:00-11:15	Congenital Lyme Borreliosis-The Emerging Syndrome Karen Forschner
11:15-11:30	Treatment Alternatives Dr. Paul Lavoie
11:30-11:45	NIH Involvement Dr. Bob Quackenbush Dr. Stephen Heyse
11:45-12:00	Armed Forces Involvement Capt. George Korch
Important * 12:00-12:20	M.S. Study Dr. Willy Burgdorfer Julie Rawlings
12:20-12:30	Closing Remarks Dr. Charles Sweet
12:30-2:00	Lunch
2:00-4:00	Break Out Sessions 2:00-3:00 Demonstration of Laboratory Techniques 2:00-3:00 Veterinary Workshop 3:00-4:00 Workshop: The Treatment of Lyme Borreliosis-Cure or Control
7:00-9:00	Public Forum

Several conference participants will be available to answer questions from the public and the media.

Conference location: Howard Johnson Plaza South Hotel (448-2444)

LYME BORRELIOSIS IN TEXAS

AUDREY STEIN GOLDINGS, M.D.
JEFFERY TAYLOR, M.P.H.
JULIE RAWLINGS, M.P.H.

February 13th, 1993
Lyme borreliosis Conference
(Dallas, Texas)
Updated from the article that appeared in Texas Medicine Volume 87, Number 9, Sept, 1991

Lyme borreliosis, a spirochetal infection caused by Borrelia burgdorferi (1), has become a public health problem of international scale. In the United States alone, more than 40,000 cases were reported to the Centers for Disease Control between 1982 and 1991 (2). The most prevalent tick borne disease worldwide, this infection is present not only in North America but in Europe, Asia, and Australia (4-8). Although a provisional total of 9,469 cases were reported to the Centers for Disease Control (CDC) in 1991 (3), researchers believe that the number actually could be 5 to 10 times higher. As physicians become more aware of this disease, the number of cases will probably continue to rise. While it is not being reported in epidemic proportions Lyme borreliosis is endemic in Texas and physicians need to be familiar with it. Clinical manifestations, diagnosis, treatment, and epidemiology are reviewed herein with particular emphasis on important features of Lyme borreliosis as an endemic disease of Texas.

CLINICAL MANIFESTATIONS

Lyme borreliosis is often compared to syphilis, another spirochetal disease known for its multisystem involvement, its occurrence in three stages (early, disseminated, and chronic), and its tendency to imitate other disorders (9,10). Because patients may exhibit the symptoms of one, two, or three of the stages and because considerable overlap may occur, some investigators have divided the disease into simply early and late stages. (See attached)

I. Early Stage

The early stage may occur within 3 to 30 days of the arthropod bite and in about 50% of cases involves the production of a localized erythema migrans (EM) (11). This lesion is often found on the axilla, thigh, or groin, common sites for tick attachment (11). Malar as well as urticarial rashes have been seen instead of EM (13). Skin manifestations are often accompanied by flulike symptoms (14).

B. burgdorferi can be cultured from peripheral blood of rodents several days after inoculation, but thereafter, the spirochetes can be found only in organs such as the bladder, kidney, liver, or spleen. Similarly, in the case of humans, the spirochete appears to spread to remote sites of the body within days of initial inoculation so that later, there is a paucity of identifiable organisms (15).

II. Disseminated Stage

The disseminated stage occurs days or weeks after exposure. The infection may localize in the nervous system, causing meningitis, cranial or peripheral neuropathy, or radiculopathy (16-19). Mild encephalopathy may occur. Patients may complain of short-term memory loss, disorientation, difficulty concentrating, and emotional lability. When given formal psychometric examinations, these patients often have measurably abnormal results (20). Spinal fluid may remain normal although some cases may have a lymphocytic pleocytosis (15,18,20).

B. burgdorferi has been cultured from cerebrospinal fluid on rare occasions (22). Bell's palsy is relatively common with Lyme borreliosis (23). In fact, bilateral facial palsy in endemic areas can almost always be attributed to Lyme borreliosis. Cardiac complications include atrioventricular nodal block, myopericarditis, and pancarditis (24,25). In severe cases, patients may require temporary pacemakers or steroids. Conjunctivitis, iritis, choroiditis, retinal hemorrhage, retinal detachment, optic neuritis, and panophthalmitis have been reported (26-29). In addition, secondary annular skin lesions, migratory join pain, generalized lymphadenopathy, hepatitis, splenomegaly, hematuria or proteinuria, and orchitis may be seen as a part of the clinical syndrome associated with dissemination of the organism (11-30).

III. Chronic Stage

During the chronic stage of the infection, which occurs months to years after exposure, an arthritis involving large and small joints may develop (11-31). In severe cases, erosion of cartilage and bone can cause permanent joint disability. Leukocytes (predominantly polymorphonuclear) in joint fluid may range from .5 to 110 x 10 9/L (500 to 100,000 per cubic millimeter) but typically joints may appear normal.

Because B. burgdorferi is a neurotropic organism, a variety of chronic neurologic disorders may become manifest. For example, progressive encephalomyelitis has been reported during this stage (32). Symptoms and signs include spastic paraparesis, bladder dysfunction, ataxia, or cognitive impairment including dementia. Diagnosis has occasionally been proved by demonstrating intrathecal production of antibody to B. burgdorferi (33). Or, there may be nervous system involvement with intermittent distal parathesias or radicular pain. Findings on physical examination may be normal, although changes may be found on electromyographic examination of axonal neuropathy (19,34). Entrapment phenomena, such as carpal tunnel syndrome, may appear secondarily. Finally, a syndrome that suggests multiple sclerosis, accompanied in some cases by hyperintense areas compatible with demyelination seen on magnetic resonance imaging, has been reported (35).

Acrodermatitis chronica atrophicans (ACA) is a skin manifestation that appears late in Lyme borreliosis (36). The lesion, usually found on an extremity, begins insidiously as a swollen area of bluish-red discoloration. It may last for years, leading to gradual atrophy of the skin. B. burgdorferi has been cultured from ACA as long as 10 years after onset of the infection, illustrating the spirochete's ability to persist in the human host.

Although transmission of B. burgdorferi from mother to fetus is possible, most women in whom Lyme borreliosis has been documented deliver normal children. Gestational Lyme borreliosis may be similar to prenatal syphilis in its ability to cause a multitude of congenital defects (37). Fetal death, hydrocephalus, and sudden infant have all been associated with gestational Lyme borreliosis, but whether or not they are coincidentally or causally related remains to be determined.

DIAGNOSIS

If EM is present, Lyme borreliosis can be diagnosed immediately. Some laboratories have successfully isolated B. burgdorferi from skin lesions (43, 44). However, inspection of other tissue biopsy specimens has shown that chronically infected humans typically have few organism in the blood or vital organs. Thus, attempted isolation of B. burgdorferi for routine confirmation of Lyme borreliosis after the rash phase is impractical. If the skin lesion is atypical in appearance or absent, the physician may request serologic testing to aid in the diagnosis. An indirect fluorescent antibody procedure and enzyme-linked immunosorbent assay are the most commonly available tests (38), but unfortunately, both tests may remain negative for 6 to 8 weeks.

Limitations of serologic testing for Lyme borreliosis include cross-reactivity with antibodies to other spirochetes such as Treponema pallidum, the agent of syphilis, and the lack of standardization of tests between laboratories (40-42). Negative rapid plasma reagin and treponemal microhemagglutination procedures are useful in ruling out false-positive test for Lyme borreliosis.

In some cases, early treatment with antibiotics can curtail or abort the antibody response; in other cases, the antibody titer may rise after treatment. In a 1985 study conducted at the Texas Department of Health, the sera of patients from whom B. burgdorferi had been isolated were tested (39). Only one half of the specimens contained detectable antibody to the spirochete and, even then, the titers were not always high enough to be diagnostic. Therefore, negative antibody tests do not necessarily rule out disease.

Recent proficiency testing done in the midwest involving 45 laboratories showed high variability in interlaboratory and intralaboratory performances especially with lower levels of anti-Borrelia burgdorferi antibody in the serum. Over one half of laboratories could not identify a case-defined serum. Many laboratories could not reproduce their results on split samples from the same patient. A striking number of laboratories could not identify serum samples from patients with late or even chronic Lyme disease. In addition, the false positivity rate may approach 27 % depending on the assay used (42).

Fortunately new procedures such as the newly developed polymerase chain reaction hold promise for the near future (43). Until then, given the current difficulty with laboratory analysis of Lyme borreliosis, clinical acumen remains the most essential factor in its diagnosis.

TREATMENT

Recommendation for therapy should be viewed as tentative, pending the completion of case-control studies and long term follow up. Early infection (less than 6 weeks) may be treated with (a) 500-1000mg of amoxicillin three times a day with or without 500mg of probenecid three times a day; (b) 100mg of doxycycline two to three times a day; or (c) 100mg of minocycline twice a day (44-47). These drugs provide better serum and tissue levels when compared to low doses of oral penicillin. Because doxycycline given at doses of 200mg twice a day achieves cerebrospinal fluid concentrations above the minimum inhibitory concentration for B. burgdorferi, higher doses may be preferable, if tolerated, to prevent central nervous system sequelae (48). Use of cefixime administered in doses of 400mg to 800mg a day has been used effectively but in vivo studies have yet to be completed (47).

Patients who have symptoms of moderate to severe degree may need to have therapy extended. Individuals older than 60 years, those who have been on steroids, or those who have high sedimentation rate with synovitis are more likely to fail on oral therapy (49).

Intravenous antimicrobials are indicated for central nervous system involvement or for high degrees of atrioventricular block (45-47). Also, those patients who have not responded to oral therapy should be given a trial of intravenously injected antibiotics. Penicillin administered intravenously has shown a failure rate as high as 50%; ceftriaxone or cefotaxime often produces better results (50,51).

Though the optimal length of therapy for late neurologic abnormalities is not known, the previously recommended 2 weeks of therapy may be inadequate (45,52) and longer courses are currently being recommended (53).

The tendency toward relapse suggests that B. burgdorferi may remain dormant in the host for prolonged periods. In fact, B. burgdorferi has been cultured from patients following standard courses of antibiotics. Therefore, longer courses of therapy seem particularly justifiable in refractory cases. Retreatment may be necessary (52, 53).

As mentioned above, no test is currently available that can reliably indicate when B. burgdorferi has been eliminated from the host. Further, whether lingering symptoms result from persistent infection, permanent tissue damage, or some yet to be defined immune reaction is not clear. Therefore, the decision to stop therapy must be based on clinical factors.

Interestingly, patients usually have a symptomatic flare, resembling the Jarisch-Herxheimer reaction, with initiation of therapy. Severe myalgias, arthralgias, headaches, dyspnea, diarrhea, and fever have been reported. With oral antibiotics, the Jarisch-Herxheimer reaction begins on the fourth or fifth day of therapy; with intravenous therapy, on the first to the third day. The reaction lasts from 1 to 2 weeks. Antibiotic therapy may need to be withheld for 1 to 3 days for those patients who experience severe reactions (49).

EPIDEMIOLOGY

The Lyme spirochete is transmitted most effectively by Ixodes tick species (53): Ixodes dammini in the northeastern and midwestern states, Ixodes pacificus in the western United States, Ixodes ricinus in Europe, and Ixodes persulcatus in Asia. However, B. burgdorferi has been detected from at least 15 different tick species, several flea species, mosquitoes, and biting flies (54,55). In Texas, this organism has been isolated from Amblyomma americanum (the lone star tick), Ixodes scapularis (the black-legged deer tick), and Ctenocephalidis felis (the cat flea).

Transmission studies must be completed to determine the competence of arthropods other than Ixodes ticks as vectors of Lyme borreliosis. Nevertheless, the wide distribution of reported cases (see attached map) and information gathered from epidemiological investigations suggest that additional vectors must be involved.

Of 763 possible Lyme borreliosis cases reported to the Texas Department of Health between 1990 and 1992 214 met the current CDC case definition. The majority of patients resided in the north central portion of the state with sporadic cases occurring throughout the state. Forty percent of the patients remembered a tick or flea bite within the month before onset. About 32% had an EM seen by a physician equal to or greater than 5cm. in diameter. Most cases had onset of EM between April and June although onset may occur in any month.

PREVENTION

The prevention of Lyme borreliosis includes prudent avoidance of tick infested areas. When this is not possible, clothing should be worn so as to prevent arthropods from gaining access to the skin, and insect repellents containing DEET can be applied to skin and permethrin to clothing. Any attached ticks should be removed promptly. Dogs and cats should be inspected regularly for ticks. Besides bringing ticks and fleas into the home environment, these household pets can also acquire the infection (56).

SUMMARY

Lyme borreliosis is a protean infection caused by B. burgdorferi, a recently recognized arthropod-borne spirochete. The disease is generally acquired during warm weather, and its onset is characterized by a skin lesion, EM, and flulike symptoms. Neurologic, cardiac, and/or rheumatologic abnormalities may emerge weeks, months, or years later. In the absence of the pathognomonic skin lesion, determination of antibody response is currently the most practical laboratory aid in diagnosis. However, clinical judgment is necessary for the correct interpretation of laboratory results because false positive and false negative results are common. Antibiotics remain the mainstay of therapy. Longer courses of antibiotic therapy than those previously recommended may be needed to obtain a cure, particularly in later stages of the illness.

REFERENCES

1. Johnson RC, Hyde FW, Schmid GP, Brenner DJ. Borrelia burgdorferi: The etiological agent of Lyme disease. Int J Syst Bacteriol. 1984;34:496-497.

2. Dennis DT. Epidemiology in: Coyle PK, ed. Lyme disease St. Louis: Mosby. Year Book, 1993:27-37.

3. CDC/Lyme Disease Surveillance Summary. Vol. 3, No. 3, Sept. 1992

4. Schmid GP. The global distribution of Lyme disease. Rev Infect Dis 1985;7:41-50.

5. Kryuchehnikov VN. Erythema chronicum migrans (Lyme disease)-a new tick-borne spirochetosis. Zh Mikrobiol Epidemiol Immunobiol. 1985;9:101-108.

6. Kawabata M, Baba S, Iguchi K, Yamaguti N, Russell N. Lyme disease in Japan and its possible incriminated vector Ixodes persulcatus. J Infect Dis. 1987;156:854.

7. Ai CX, Wen YX, Zhang YG, et al. Clinical manifestations and epidemiological characteristics of Lyme disease in Hailin county, Heilongjiang Province, China. Ann N Y Acad Sci. 1988;539:302-313

8. Dekonenko EP. Clinical manifestations of tick-borne erythema in the USSR. Ann N Y Acad Sci. 1988;539:452-453.

9. Steere AC, Bartenhagen NH, Craft JE, et al. The early clinical manifestations of Lyme disease. Ann Intern Med. 1983;99:76-82.

10. Asbrink E, Hovmark A. Early and late cutaneous manifestations in Ixodes-borne borreliosis (erythema migrans borreliosis, Lyme borreliosis). Ann N Y Acad Sci. 1988;539:4-15.

11. Steere AC, Taylor E, Wilson ML, et al. Longitudinal assessment of the clinical and epidemiological features of Lyme disease in a defined population. J Infec Dis. 1986;120:1017-1021.

12. Berger BW. Erythema chronicum migrans of Lyme disease. Arch Dermatol. 1984;120:1017-1021.

13. Eichenfield AH. Diagnosis and management of Lyme disease. Pediatr Ann. 1986;15:583-594.

14. Steere AC, Malawista SE, Bartenhagen NH, et al. The clinical spectrum and treatment of Lyme disease. Yale J Biol Med. 1984;57:453-461.

15. Duray PH, Steere AC. Clinical pathologic correlations of Lyme disease by stage. Ann N Y Acad Sci. 1988;539:65-79

16. Reik L, Steere AC, Bartenhagen NH, Shope RE, Malawista SE. Neurologic abnormalities of Lyme disease. Medicine. 1979;58:281-294

17. Steirnstedt GT, Granstrom M, Hederstedt B, Skoldenberg B. Diagnosis of spirochetal meningitis by enzyme-linked immunosorbent assay and indirect immunofluorescent assay in serum and cerebrospinal fluid. J Clin Microbiol. 1985;21:819-825.

18. Steirnstedt GT, Gustafsson R, Karlsson M, Svenungsson B, Skoldenberg B. Clinical manifestations and diagnosis of neuroborreliosis. Ann N Y Acad Sci. 1988;539:46-55.

19. Pachner AR, Steere AC. The triad of neurological manifestations of Lyme disease: meningitis, cranial neuritis, and radiculoneuritis. Neurology. 1985;35:47-53.

20. Halperin JJ, Pass HL, Anand AK, Luft BJ, Volkman DJ, Dattwyler RJ. Nervous system abnormalities in Lyme disease. Ann N Y Acad. Sci. 1988;539:24-34.

21. Ackerman R, Horstrup P, Schmidt R. tick-borne meningopolyneuritis (Garin-Bujodox, Bannwarth). Yale J Biol Med. 1984;57:485-490.

22. Preac Mursic V, Wilske B, Schierz G, Pfister HW, Einhaupl K. Repeated isolation of spirochetes from the cerebrospinal fluid of a patient with meningioradiculitis Bannworth. Eur J Clin Microbiol Infect Dis. 1984;3:564-565.

23. Clark JR, Carlson RD, Sasaki CT, Pachner AR, Steere AC. Facial paralysis in Lyme disease. Laryngoscope. 1985;95:1341-1345.

24. Steere AC, Batsford WP, Weinberg M, Alexander J, Berger HJ, Wolfson S, Malawista SE. Lyme carditis: cardiac abnormalities of Lyme disease. Ann Intern Med. 1980;93:8-16.

25. Olson LJ, Okafor EC, Clements IP. Cardiac involvement in Lyme disease: manifestations and management. Mayo Clin Proc. 1986;61:745-749.

26. Steere AC, Duray PH, Kauffman DJ, Wormser SG. Unilateral blindness caused by infection with the Lyme disease spirochete, Borrelia burgdorferi. Ann Intern Med. 1985;103:382-383.

27. Bertuch AW, Rocco E, Schwartz EG. Eye findings in Lyme disease. Conn Med. 1987;51:151-152.

28. MacDonald AB. Lyme disease: a neuro-opthslmologic view. J Clin Neuro Ophthalmol. 1987;7(4):185-190.

29. Winward MD, Lawton Smith J, Culbertson WW, Paris-Hamelin A. Ocular Lyme borreliosis. Am J Ophthalmol. 1989;108:651-657.

30. Steere AC, Malawista SE, Hardin JA, Ruddy S, Askenase PW, Andiman WA. Erythema chronicum migrans and Lyme arthritis. The enlarging clinical spectrum. Ann Intern Med. 1977;86:685-698.

31. Steere AC, Gibofsky A, Pattarroyo ME, Winchester RJ, Hardin JA, Malawista SE. Chronic Lyme arthritis: clinical and immunogenetic differentiation from rheumatoid arthritis. Ann Int Med. 1979;90:896-901.

32. Ackerman R, Rehse-Kupper B, Gollmer E. Progressive Borrelia encephalomyelitis. Zentralb Bakteriol Hyg. 1986;263:297-300.

33. Wilske B, Schierz G, Preac Mursic V, et al. Intrathecal production of specific antibodies against B burgdorferi in patients with lymphocytic mengoradiculitis (Bannwarth's syndrome). J Infect Dis. 1986;153:304-314.

34. Halperin JJ, Little BW, Coyle PK, Dattwyler RJ. Lyme disease: cause of treatable peripheral neuropathy. Neurology. 1987;36:1700-1706.

35. Halperin JJ, Luft BJ, Volkman DG, et al. Lyme neuroborreliosis-central nervous system manifestations. Neurology. 1989;39:753-759.

36. Asbrink E, Hovmark A, Olsson I. Clinical manifestations of acrodermatitis chronica atrophicans in 50 Swedish patients. Zentralb Bakteriol Microbiol Hyg. 1986;263:253-261.

37. MacDonald AB. Gestational Lyme borreliosis: implications for the fetus. Rheum dis Clin North Am. 1989;15:657-677.

38. Mangarelli LA, Anderson JF. Enzyme-linked immnuosorbent assays for the detection of class-specific immnuoglobulins to Borrelia burgdorferi. Am J Epidemiol. 1988;127:818-825.

39. Rawlings JA, Fournier PV, Teltow GA. Isolation of Borrelia spirochetes from patients in Texas. J clin Microbiol. 1987;25:1148-1150.

40. Barbour AG. Laboratory aspects of Lyme borreliosis. Clin Microbiol Rev. 1988;1:399-414.

41. Magnarelli LA, Anderson JF, Johnson RC. Cross-reactivity in serological tests for Lyme disease and other spirochetal infections. J Infect Dis. 1987;156:183-188.

42. Rosa PA, Schwan TG. A specific and sensitive assay for the Lyme disease spirochete B burgdorferi using the polymerase chain reaction. J Infect Dis. 1989;160:1018-1029.

43. Stanek G, Wewalka G, Groh V, Neumann R. Isolation of spirochetes from the skin of patients with erythema chronicum migrans in Austria. Zentralb Bakteriol Microbiol Hyg. 1985;260:88-90.

44. Berger BW, Kaplan MH, Rothenberg IR, Barbour AG. Isolation and characterization of the Lyme disease spirochete from the skin of patients with erythema chronicum migrans. J Am Acad Dermatol. 1985;13:444-449.

45. Sterre AC. Medical progress: Lyme disease. N Engl J Med. 1989;321:586-596.

46. Luft BJ, Dattwyler RJ. Treatment of Lyme borreliosis. Rheum Dis Clin North Am. 1989;15:747-755.

47. Burrascano, J. Lyme disease treatment guidelines. 1990 National Lyme Borreliosis Scientific forum; March 29-30, 1990; Orlando, Fla; 49-54.

48. Dotevall L, Hageberg L. Penetration of doxycycline into cerebrospinal fluid in patients treated for suspected Lyme neuroborreliosis. Antimicrob Agents Chemother. 1989;33:1078-1080.

49. Burrascano J. Late stage Lyme disease: treatment options and guidelines. Int Med for the Specialist. 1989;10(12):102-107.

50. Dattwyler RJ, Halperin JJ, Volkman DJ, Luft BJ. Treatment of late Lyme borreliosis-randomized comparison of ceftriaxone and penicillin. Lancet. 1988;1:1191-1194.

51. Pfister HW, Preac Mursic V, Wiske B, Einhaupl KM. Ceftriaxone vs penicillin G for acute neurologic manifestations in Lyme borreliosis: a prospective randomized study. Arch Neurol. 1989;46(11):1190.

52. Logigian EL, Kaplan RF, Sterre AC. Chronic neurologic manifestations of Lyme disease. N Engl J Med 1990;1438-1444.

53. Rahn DW, Malawista SE. Lyme disease: Recommendations for Diagnosis and treatment. Annals of Int Med. 1991;114:472-482.

54. Preac-Mursic V, Weber K, Pfister HW, et al. Survival of B burgdorferi in antibiotically treated patients with Lyme borreliosis. Infection. 1989;17:355-359.

55. Burdorferi W. Discovery of the Lyme disease spirochete and its relation to tick vectors. Yale Journal of Biological Medicine. 1984;57:65-70.

56. Magnarelli LA, Anderson JF, Barbour AG. The etiologic agent of Lyme in deer flies, horse flies, and mosquitoes. J Infect Dis. 1986;154:355-358.

57. Rawlings JA. Lyme disease in Texas. Zentralb Bakteriol Microbiol Hyg. 1986;263:483-487.

58. Rawlings JA. Tick-borne aoonoses in Texas. Texas Veterinary Medical Journal. 1990;52:21-22.

CLINICAL MANIFESTATIONS OF LYME

EARLY (within 1 month of exposure)

- EM
- flu like symptoms

DISSEMINATED (typically days or weeks after exposure)

Neurologic
- meningitis
- neuropathy (cranial (Bell's)/ peripheral /radiculopathy)
- mild encephalopathy

Cardiac
- AV block, pericarditis, pancarditis

Arthritic
- migratory joint pain, especially knees

Opthalmologic
- conjunctivitis, iritis, optic neuritis etc.

Skin
- secondary annular skin lesions

CHRONIC (typically months to years after exposure)

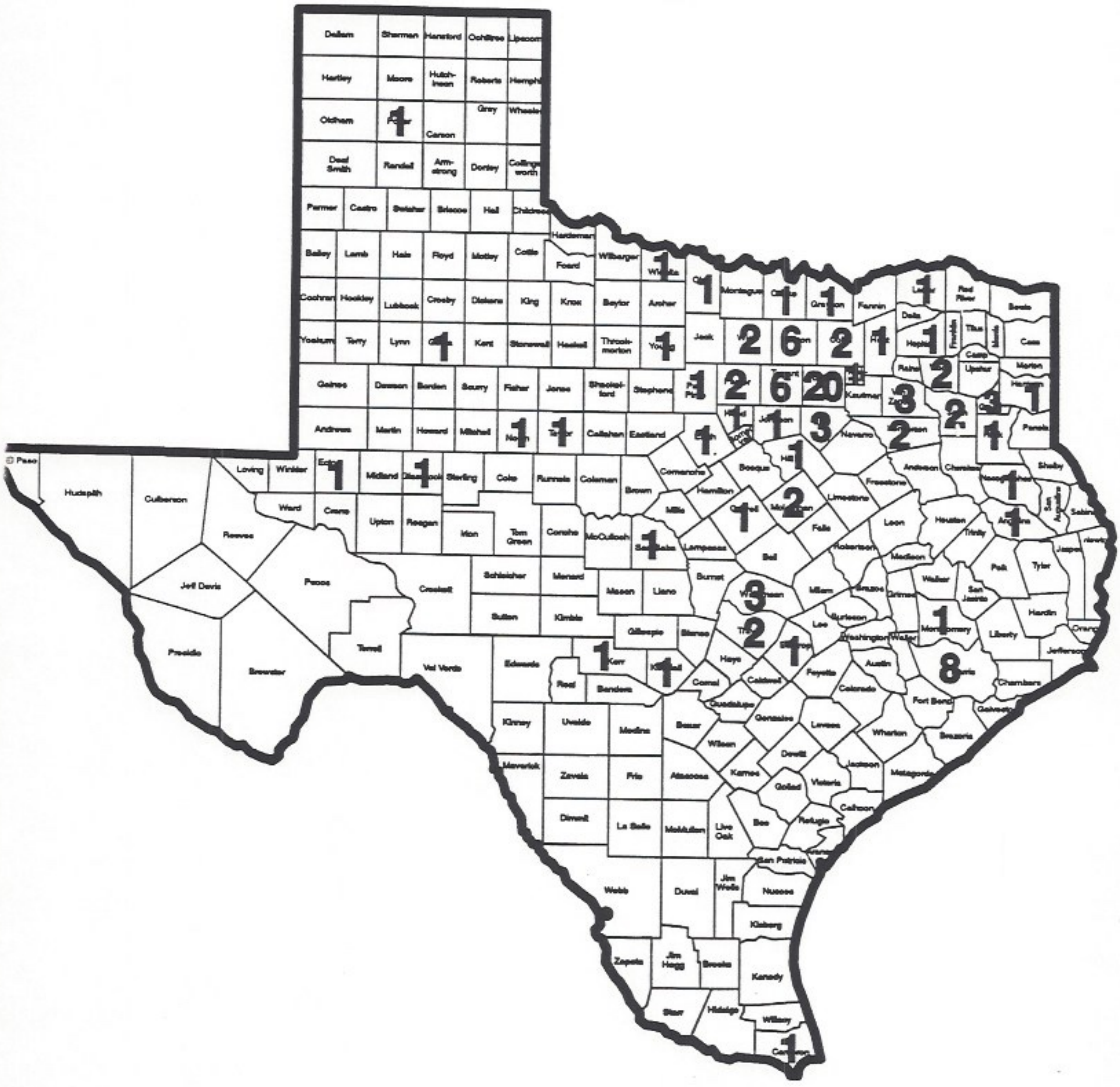
Neurologic
- mild encephalopathy, progressive encephalomyelitis, neuropathy/radiculopathy, MS-like illness

Arthritic
- large and/or small joints

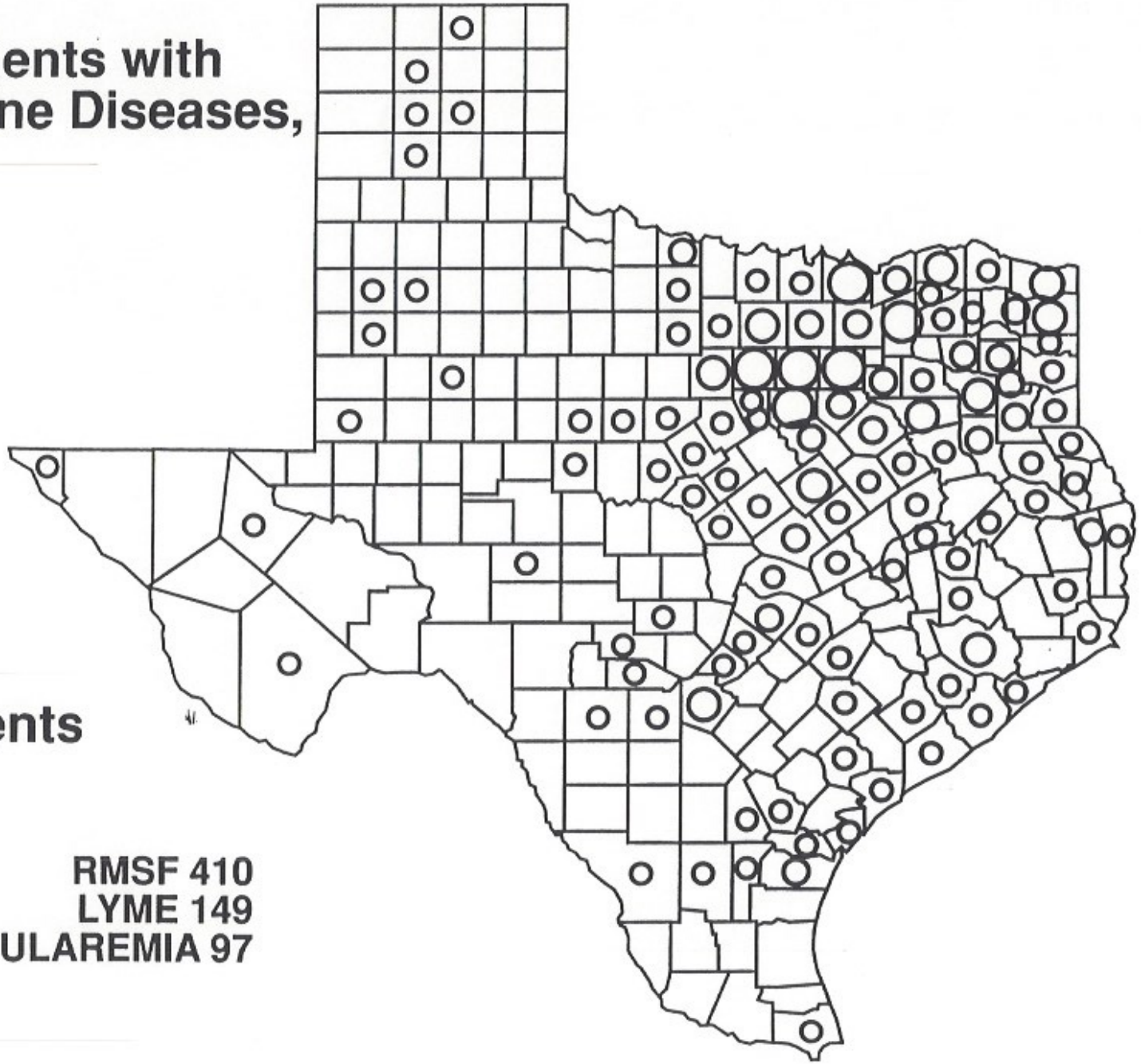
Skin
- acrodermatitis chronica atrophicans (ACA)

Chronic Fatigue Syndrome/Fibromyalgia

County of Residence for 101 Lyme Borreliosis Cases Texas, 1990-1991



Residence of Patients with Common Tickborne Diseases, 1980-1989



Number of Patients

- ◯ 20-57
- ◯ 11-19
- ○ 6-10
- ○ 1-5

RMSF 410
LYME 149
TULAREMIA 97

REPORTABLE DISEASES IN TEXAS

The Communicable Disease Prevention and Control Act (Texas Civil Statutes, Articles 4419b-1) requires physicians, dentists, and veterinarians to report, after the first professional encounter, each patient examined who is suspected of having a reportable disease. Also required to report are certain individuals from hospitals, laboratories, and schools. Detailed rules on the reporting of notifiable diseases and conditions and the duties of local health authorities may be found in Article 97, Title 25, Texas Adminstrative Code.

DISEASES REPORTABLE IMMEDIATELY BY TELEPHONE

BY NAME, CITY, AGE, SEX, RACE/ETHNICITY, DISEASE, TYPE OF DIAGNOSIS, DATE OF ONSET, AND PHYSICIAN:

To the Infectious Diseases Program,
Texas Department of Health, in Austin
(CALL TOLL-FREE 1-800-252-8239)

- Botulism (adult)
- Cholera
- Plague
- Rabies in Man
- Yellow Fever

To the Immunization Division,
Texas Department of Health, in Austin
(CALL TOLL-FREE 1-800-252-9152)

- Diphtheria
- Measles
- Pertussis
- Polio, paralytic
- Rubella

All of the other diseases listed below are to be reported to your local health authority, who will in turn report them to the Texas Department of Health.[1]

DISEASES REPORTABLE ON A WEEKLY BASIS

BY NAME, CITY, AGE, SEX, RACE/ETHNICITY, DISEASE, TYPE OF DIAGNOSIS, DATE OF ONSET, AND PHYSICIAN:

- Acquired Immune Deficiency Syndrome (AIDS)
- Acute Occupational Pesticide Poisoning[2]
- Amebiasis
- Anthrax
- Asbestosis[2]
- Botulism (infant)
- Brucellosis
- Campylobacteriosis
- Chlamydia trachomatis infections (laboratory confirmed only)[3]
- Coccidioidomycosis
- Dengue
- Elevated Blood Lead in Adults (blood lead ≥40 mcg/dl in persons ≥15 years of age)[2]
- Encephalitis (specify etiology)
- Gonorrhea[3]
- Haemophilus influenzae Infections (systemic)[4]
- Hansen's disease (leprosy)
- Hepatitis, Viral
  - Type A
  - Type B
  - Type D (delta agent)
  - Non-A, Non-B
  - Unspecified
- Histoplasmosis
- Legionellosis
- Leptospirosis
- Listeria Infections
- Lyme Disease
- Malaria
- Meningitis
  - Aseptic/Viral
  - Bacterial (specify etiology)
  - Fungal
  - Other
- Meningococcal Infections
- Mumps
- Psittacosis
- Q Fever
- Reye Syndrome
- Rocky Mountain Spotted Fever
- Rubella, Congenital Syndrome
- Salmonellosis
- Shigellosis
- Silicosis[2]
- Syphilis[3]
- Tetanus
- Toxic Shock Syndrome
- Trichinosis
- Tuberculosis[5]
- Tularemia
- Typhoid Fever
- Typhus Fever
  - Endemic (murine)
  - Epidemic
- Vibrio Infections (specify species)
- Viral Hemorrhagic Fever

BY NUMBER ONLY:

- Influenza & Flu-like Illnesses

BY NUMBER AND AGE GROUP ONLY:

- Chickenpox

BY NUMBER, AGE GROUP, AND SEX ONLY:

- Human Immunodeficiency Virus (HIV) Infections[6]

In addition to the requirements of individual case reports, any unusual outbreak of disease of public health concern shall be reported to the Texas Department of Health in Austin through the local health authority or to the State Epidemiologist directly by the most expeditious means.

---

[1] The local health authority or regional director shall collect reports of disease and transmit them at weekly intervals to the Texas Department of Health. Transmittal may be by telephone, mail, or electronic transmission.

[2] The Occupational Disease Reporting Act, Article 5182c, Texas Civil Statutes, requires physicians and directors of laboratories to report these occupationally related diseases to the Texas Department of Health.

[3] Syphilis, gonorrhea, and laboratory-confirmed Chlamydia trachomatis infections are reportable in accordance with Sections 97.132, 97.134, and 97.135 of 25 TAC. Form STD-27, "Confidential Report of Sexually Transmitted Disease," shall be used to report these sexually transmitted diseases.

[4] Includes meningitis, septicemia, cellulitis, epiglottitis osteomyelitis, pericarditis, septic arthritis, and pneumonia.

[5] Report tuberculosis on form TB-400, "Report of Case and Patient Services."

[6] Reported by physician only once per case, following initial physician diagnosis.