DISCUSSION

Lyme disease is now the most common tick-borne infection in the United States and Europe[4,18]. In general, it is difficult to diagnose Lyme disease from the clinical picture alone. The diagnosis of this disease is generally based on clinical criteria and the results of serological tests. Because of a delay in antibody production in the first 3 to 6 weeks of the disease, serology is not reliable. In addition, *B. burgdorferi* has slow growth properties and is difficult to isolate from infected tissues[14].

Serology may be negative or equivocal in early stages of borreliosis because of a poor or delayed immune response[5]. Cross-reactive antibodies may cause false-positive results in patients with other spirochetal infections[12].

The highly sensitive and specific DNA amplification system is potentially an excellent tool for the identification of Lyme disease agent in clinical samples as well as in vectors and animal reservoirs. This system is being applied widely for the detection of pathogens in blood and body fluids, as well as in tissue samples[14].

In vitro amplification of nucleic acids by the PCR has been adapted for rapid, sensitive and specific detection of *B. burgdorferi*. Target sequences for PCR amplification were in the genes encoding the flagellin, the major outer surface protein (OspA), or 16S rRNA[1,11,15].

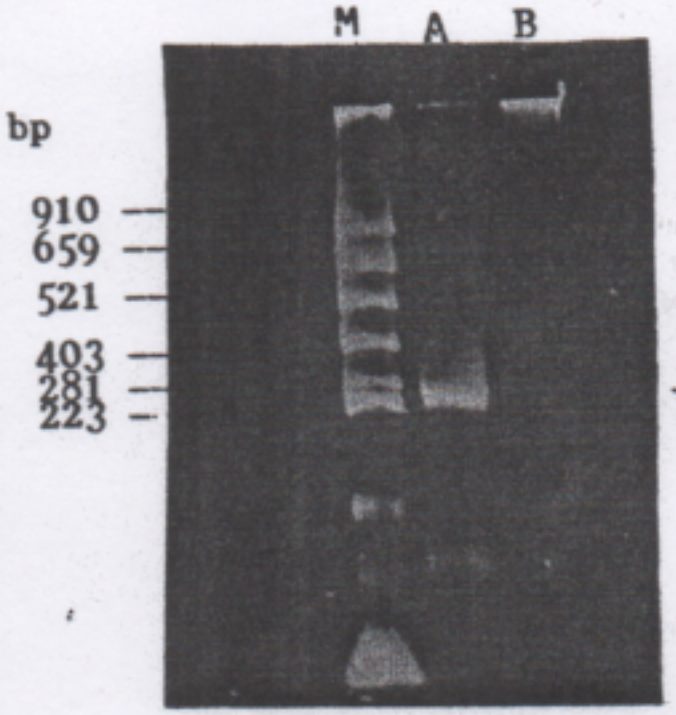

Figure 1. PCR amplification and PAGE analysis of isolates of *B. burgdorferi*. Lane A, DNA from strain B31 (ATCC) of *B. burgdorferi*; Lane B, DNA from *E. coli*; Lane M, molecular weight marker (pBR 322+ *Alu*I).

We were able to detect borrelia-specific DNA in synovial fluid from a dog with Lyme arthritis and skin biopsy samples from two patients (human beings) with acrodermatitis 1 week after beginning antimicrobial therapy. Thus, PCR may prove to be a useful and sensitive method for diagnosis of Lyme disease. This test may also help to detect *B. burgdorferi* in other specimens, e.g., urine, cerebrospinal fluid from patients with this disease.

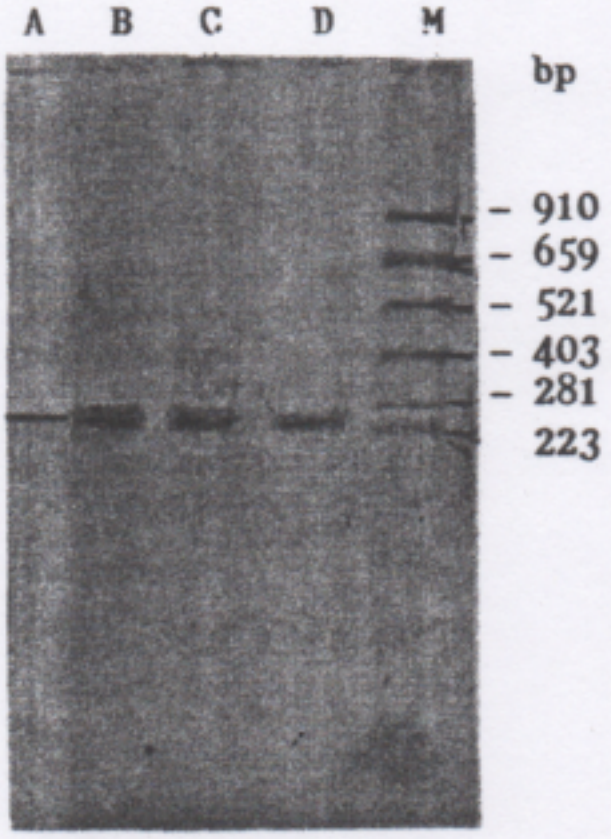

Figure 2. PCR detection of *B. burgdorferi* in skin biopsy samples and synovial fluids in 2% agarose gel. Lane A, DNA from *E. coli*; Lanes B and C, DNA from skin biopsy samples from two patients (human beings); Lane D, DNA from synovial fluid from a dog; Lane M, molecular weight marker (pBR 322 + *Alu*I)

REFERENCES

1. ADAM, T.; B. GRAF, U. NEUBERT & U.B. GÖBEL. 1992. Detection and classification of *Borrelia burgdorferi* by direct sequencing of 16S rRNA amplified after reverse transcription. *Med. Microbiol. Lett.* **1**:120-126.
2. BARBOUR, A.G. 1984. Isolation and cultivation of Lyme disease spirochetes. *Yale J. Biol. Med.* **57**:521-525.
3. BURGDORFER, W.; A.G. BARBOUR; S.F. HAYES; J.L. BENACH, E. GRUNDWALD & J.P. DAVIS. 1982. Lyme disease — a tick-borne spirochetosis? *Science.* **216**:1317-1319.
4. CIESIELKI, C.A.; E. MARKOWITZ; R. HORSLEY; A.W. HIGHTOWER; H. RUSSEL & C.V. BROOME. 1988. The geographic distribution of Lyme disease in the United States. *Ann. N.Y. Acad. Sci.* **539**:283-288.
5. CRAFT, J.E.; R.L. GRODZICKI & A.C. STEERE. 1984. The antibody response in Lyme disease: evaluation of diagnostic tests. *J. Infect. Dis.* **149**:789-795.