

antigen. So far, our attempts to use the  $\mu$ -capture technique with enzyme-conjugated antigen have resulted in lessened sensitivity of the assay, and further work is necessary to establish an enzyme immunoassay method for measuring poliovirus-specific IgM antibodies.

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## Detection of *Salmonella typhi* by PCR

In their paper, Song et al. describe a PCR-based diagnostic test for the detection of *Salmonella typhi* (3). The authors employed a nested PCR approach using as their first pair of primers an *S. typhi*-specific forward primer (ST 1) and a common flagellin gene-derived reverse primer and nested to them a second set of primers. In this second set, the forward primer (ST 3) was originally also thought to be *S. typhi* specific (1) but was later found to share the same DNA sequence with other type *d* flagellin genes (2). Therefore, it is not surprising that in the nested PCR, applied to cultured salmonella, an amplified product was obtained when *S. typhi* as well as *S. muenchen* DNA was used as template. In the Discussion section, the authors doubt (quite rightly) the significance and actual probability of finding *S. muenchen* in blood samples. However, in order to avoid this problem altogether, the authors could have used, as we are in our laboratory, a different forward primer for the first PCR which is derived from the DNA sequence located just upstream to primer ST 1 (1, 3), i.e., 5' TATGCCGCTACATATGATGAG 3' (1), together with primer ST 2 (1, 3). The use of these primers in the first round of DNA amplification should result in an amplified product using either *S. typhi* or *S. muenchen* DNA template (and also DNA from other *Salmonella* species expressing flagellar antigen *d*). Primer ST 1, which is the *S. typhi*-specific

primer, should then be used together with primer ST 4 (3) for the nested reaction. As blood PCR was positive only for the nested reactions, false-positive amplification due to sample contamination with other *Salmonella* species (like *S. muenchen*) will be avoided.

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## Treatment of Late Lyme Disease: a Challenge to Accept

We have read with great interest Liegner's guest commentary (1). We present a case which supports Liegner's opinions concerning the diagnosis and treatment of Lyme disease.

A 41-year-old male physician suffered a tick bite on his back. Forty-four hours later, the tick was traumatically removed by a dermatologist. Prophylactic oral doxycycline (200 mg/12 h) was administered for 5 consecutive days. Three months later, he complained of fatigue, febricula, and asthenia. The patient ignored these symptoms until he noted generalized and progressive muscle hypotrophy with fasciculations, accompanied by nonspecific neurologic, gastrointestinal, genitourinary, and cardiorespiratory symptomatology 27 months after the tick bite. He was initially diagnosed as having a psychiatric disorder by several specialists. Ancillary studies included complete blood count, erythrocyte sedimentation rate, biochemical pro-

filing, serum immunoglobulins (Igs), thyroid hormones, human immunodeficiency virus, *Treponema pallidum*, *Brucella melitensis*, *Salmonella typhi*, Epstein Barr virus serum antibody titers, tuberculin testing, chest X ray, electrocardiogram, electroencephalogram, echocardiography, abdominal ultrasound, and central nervous system (CNS) magnetic resonance imaging. Results were within the normal range except for a high serum IgM titer and partial IgA deficiency. Forty-one months after the tick bite, the patient was diagnosed by a neurologist as having probable encephalomyelitis due to *Borrelia burgdorferi*. Serum and cerebrospinal fluid (CSF) antibody titers to *B. burgdorferi*, as well as CSF cytology and biochemistry, were negative. Thus, no antibiotic therapy was initiated until further clinical deterioration was very evident. He received 2 g of ceftriaxone daily for 4 weeks. Marked early clinical improve-