The crude *T. gondii* seropositivity rate within the sample population was 9.6% (95% CI, 8.5%, 10.7%). Seropositivity rates by the recruit’s regional home of record varied from 3.2% to 13.3%. Adjustment of the crude overall seropositivity rate to the 1990 United States population distribution by Census Region (excluding 42 recruits from U.S. territories or outside the United States) yielded an adjusted seropositivity rate of 9.9% (95% CI = 8.8%, 11.0%). This rate is significantly lower than the similarly adjusted rate for the 1962 data (14.9%; 95% CI = 13.6%, 16.2%; P < .01) (table 1).

Results of multivariate analysis when home of record (excluding 42 recruits from U.S. territories or outside the United States), primary childhood environment (data available only for army recruits), and educational level were simultaneously controlled for an indication that recruits from the Mountain Region were less likely to be seropositive than recruits from the other eight census regions (OR = 0.13; 95% CI = 0.02, 0.96), while those reporting a rural childhood environment were more likely to be seropositive than those who reported an urban or suburban environment (OR = 4.0; 95% CI = 2.3, 6.9). Recruits who reported that they grew up in a small town were also more likely to be seropositive (OR = 2.1; 95% CI = 1.2, 3.7). No other statistically significant differences were found.

We have shown that the adjusted rate of seropositivity for *T. gondii* has decreased by one-third over the past 30 years. The number of recruits studied and the manner and purpose for which the sera were originally collected are similar to those in Feldman’s study [4]. Although we used the direct agglutination assay and Feldman used the dye titer test, the two tests have demonstrated excellent correlation [8]. However, we cannot completely eliminate sampling bias as one possible explanation of our findings. Because cat ownership has actually increased during the past 30 years [9], the most likely explanation for our findings is a declining prevalence of *T. gondii* viable oocysts in meat products [10]. This declining prevalence is likely related to the increased use of previously frozen meat. Further studies of the prevalence of *T. gondii* in the United States are needed to define the usefulness of prenatal screening.

**References**


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**Clostridium paraputrificum** Bacteremia in a Patient with AIDS and Duodenal Kaposi’s Sarcoma

*Clostridium paraputrificum* is a rare cause of disease in humans. We discuss a case of *C. paraputrificum* bacteremia in a patient with AIDS and Kaposi’s sarcoma.

A 32-year-old male with AIDS and a baseline CD4 cell count of 8/mm$^3$ was admitted to the hospital on 5 June 1995 with a 2-week history of crampy abdominal pain, emesis, and weight loss. Findings on physical examination were unremarkable except for papules of Kaposi’s sarcoma on both arms. Upper gastrointestinal endoscopy revealed an obstructive duodenal mass; examination of a biopsy specimen revealed Kaposi’s sarcoma.

The patient received 3,000 rads of external radiation to his duodenum (treatment was completed on 7 July), which necessitated the use of total parenteral nutrition. His hospital course was complicated by numerous episodes of thrombocytopenia and neutropenia, which were treated with intravenous immunoglobulin and granulocyte colony-stimulating factor. Persistent fevers did not respond to empirical broad-spectrum antibacterial and antifungal therapy. Multiple blood cultures were sterile. A urine *Histoplasma* antigen test was positive, and the patient started treatment with amphotericin B. Culture of bone marrow yielded *Mycobacterium avium* complex. The patient’s clinical condition gradually improved with appropriate antibiotic therapy, and he began receiving chemotherapy for Kaposi’s sarcoma.

On 9 August 1995, the patient was transferred to a chronic care facility. On arrival, he was febrile (temperature to 104°F) and hypotensive. Blood cultures were performed, and he started receiving treatment with intravenous vancomycin for presumed intravascular catheter-related sepsis. His WBC count was 12,600/mm$^3$. After 48–72 hours, the patient defervesced, his blood pressure...
stabilized, and his WBC count returned to baseline (1,600/mm³). One of two blood cultures yielded an anaerobic gram-positive bacillus with terminal spores that was later identified as *C. paraputrificum* (RAPID ANA II System, Innovative Diagnostic Systems, Norcross, GA). On identification of the organism (its susceptibility profile was not determined), antibiotic therapy was changed to intravenous metronidazole. Two subsequent sets of blood cultures were negative. The patient again received chemotherapy; however, on 27 August he died of sepsis due to *Staphylococcus epidermidis*.

*C. paraputrificum* is a gram-positive bacillus that forms terminal spores, is not hemolytic when grown on blood agar plates, and does not produce toxins. *C. paraputrificum* is found in the soil, animal and human feces, and clinical specimens—most notably blood, wounds, peritoneal fluid, and intraabdominal sources [1]. Various dietary factors have been associated with decreased levels of *C. paraputrificum* in fecal microflora (e.g., a high-fiber diet) [2].

Clostridia account for <1%–3% of all blood culture isolates [3, 4] and 4%–11% of all anaerobic blood culture isolates [4, 5]. *C. paraputrificum* accounted for ≤2% of clostridial isolates from all clinical specimens recovered at Indiana University Medical Center from 1979 to 1988 [6]. In reports of clostridial bacteremia, *C. paraputrificum* was the sole blood isolate in two of 12 adults [4] and one of 10 children [7] and in no patients from other series [3, 5]. Fourteen cases of in which *C. paraputrificum* was isolated in blood cultures have been described in the literature. The underlying conditions associated with these cases included gastrointestinal pathology, alcohol abuse, aspiration pneumonia, diabetes mellitus, sickle cell anemia, cyclic neutropenia, and poor nutrition [4, 7–9]. The mortality among patients with sepsis due to this organism is high but may reflect the severity of underlying disease and loss of host defense mechanisms.

Of 39 strains of *C. paraputrificum* reported in *Bergey’s Manual of Systemic Bacteriology* [1], 35 were resistant to clindamycin, 13 to erythromycin, 3 to tetracycline, 3 to penicillin, and 1 to chloramphenicol. Six strains were tested by Brazier et al. [10], who found that 4 were resistant to clindamycin and 1 to penicillin, and all 6 were susceptible to erythromycin, tetracycline, chloramphenicol, ampicillin/sulbactam, and metronidazole. No isolates of *C. paraputrificum* in any report were tested against vancomycin.

To our knowledge, this is the first case of *C. paraputrificum* bacteremia reported in a patient with AIDS. The presence of predisposing factors for *C. paraputrificum* bacteremia (gastrointestinal malignancy, neutropenia, and malnutrition), the clinical signs of sepsis temporally related to positive blood cultures, and the clinical improvement with intravenous vancomycin and metronidazole therapy suggest that the organism was a pathogen. Although susceptibility of *C. paraputrificum* to vancomycin has not been reported in the literature, our patient’s bacteremia did resolve while he was receiving treatment with this drug.

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References


**Generalized Infection with Bartonella henselae Following Infection Due to Epstein-Barr Virus**

Regional lymphadenopathy is the predominant clinical feature of cat-scratch disease (CSD), which is usually preceded by an erythematous papule at the site of inoculation [1]. *Bartonella henselae* has been isolated from lymph nodes of patients with CSD and has been detected by PCR of specimens from these nodes [1, 2]. We describe a patient with general lymphadenopathy who underwent seroconversion to *B. henselae* following an Epstein-Barr virus (EBV) infection.

A 19-year-old man was referred to Cantonal Hospital in Winterthur, Switzerland, on 27 October 1994 with a 4-week history of intermittent high fever, fatigue, nausea, night sweats, and cough as well as a weight loss of 8 kg. His primary care physician had tentatively diagnosed his condition as lymphoma because of a newly detected supraclavicular swelling after an episode of acute mononucleosis in mid-August 1994. On admission to the hospital, the patient looked ill; his temperature was 37.3°C. A left-sided supracla-