Over the past several years the genus *Citrobacter* has undergone major taxonomic revisions. A 1993 study by Brenner and colleagues (2) proposed sweeping changes within the *C. freundii* complex, previously known to be genetically heterogeneous at the DNA level. Based upon DNA relatedness, four new species were described in this investigation: *C. youngae*, *C. braakii*, *C. werkmanii*, and *C. sedlakii*. Three other species (genomospecies 9 to 11) were left unnamed due to the lack of a sufficient number of strains for definitive analysis. In 1995, genomospecies 9 was named *C. rodentium* by Schauer and others (8). Brenner et al. (2) also proposed that a former biogroup of *C. amalonaticus* (biogroup 1) be elevated to species status (*C. farmeri*) based upon the results of DNA-DNA reassociation kinetics. At present the genus *Citrobacter* is now composed of nine nomenclature species and two unnamed genomospecies.

Since these taxonomic revisions, few studies have addressed the clinical significance or frequency of the newer citrobacteria (1, 4, 6). While *C. freundii* sensu stricto still appears to be the most commonly isolated species in the laboratory, many of the newer species have been frequently recovered from anatomic sites often containing polymicrobial flora, such as the gastrointestinal tract (4). In many of these instances their role in the disease process is unclear. *C. youngae* has been isolated from blood on three separate occasions, *C. farmeri* has been so isolated twice, and *C. sedlakii* and *C. werkmanii* have been so isolated once each, suggesting that these groups are pathogenic for humans (2, 4, 6). However, a detailed case history was not available for any of these blood isolates. In this paper we describe the detailed case report of *C. farmeri* sepsis in a pediatric patient with short-bowel syndrome (SBS).

A 5-year-old male at the UCLA Medical Center developed high fever and chills 13 min after commencement of a total parenteral nutrition (TPN) infusion. His past medical history was significant for SBS secondary to bowel resection for gastroschisis; he was TPN dependent. Physical examination of the child revealed no symptoms or abnormalities other than the fever and chills. The line site was without erythema or pus. A tentative diagnosis of line sepsis was entertained, two sets of blood cultures (central line) were drawn, and the patient was admitted to the pediatric intensive care unit. He was empirically placed on vancomycin and ceftazidime. In less than 12 h, three of four blood culture bottles were positive for gram-negative rods (BacT/Alert; Organon Teknika, Oklahoma City, Okla.). Gentamicin was added to his therapeutic regimen based upon the Gram stain result. Within 24 h, his condition had improved and his fever had decreased. Four days after admission, vancomycin and ceftazidime were discontinued and the patient was placed on 2 g of ceftriaxone (four times daily) and 100 mg of gentamicin (twice daily) based upon the susceptibility report for the blood isolate. The infection resolved without further sequelae and without the removal of the line. The TPN solution was not cultured.

Blood isolates were initially identified as *Pantoea* ("Enterobacter") agglomerans (very good identification) by the API 20E system (bioMérieux Vitek, Inc., Hazelwood, Mo.). As part of a research protocol the blood isolate was additionally tested by two other systems. The Vitek system (software version R03.01; bioMérieux Vitek, Inc.) identified this isolate as *Enterobacter cloacae* (72%, low probability). The MicroScan W/A system (Dade MicroScan Inc., West Sacramento, Calif.) identified this strain as *Leclercia aderovarosae* (56%, low probability). None of these systems currently have updated databases including the new *Citrobacter* species. Because of these discrepancies, the strain was forwarded to the Microbial Diseases Laboratory for further evaluation. The strain (96A-03859) was an oxidase-negative, catalase-positive, gram-negative, facultatively anaerobic bacillus. On triple sugar iron, 96A-03859 produced an acid/acid with gas reaction (H₂S negative) and was indole, urease, citrate, and KCN positive and lysine decarboxylase and Voges-Proskauer negative. These reactions placed this strain in the genus *Citrobacter*. Failure to produce acid from adonitol or α-arabitol and to utilize malonate as a source of energy indicated that it belonged in the *C. amalonaticus* group. Subsequent testing identified the strain as *C. farmeri* ("C. amalonaticus" biogroup 1) based upon the fermentation of key carbohydrates (Table 1).

In 1985, Farmer and colleagues (3) described new groups within the family *Enterobacteriaceae* and defined a new biogroup of *C. amalonaticus* (biogroup 1) based upon the ability of these strains to ferment sucrose, raffinose, α-methyl-d-glucoside, and melibiose and not utilize citrate. *C. amalonaticus* biogroup 1 ("C. farmeri") was described as a rarely isolated enteric bacterium from feces, urine, and wounds in that study. It was listed as possibly clinically significant at some sites but not at others. Further information was not available. In the subsequent systematic investigation of Brenner et al. (2), the clinical distribution of *C. farmeri* was expanded to include blood although no clinical information on these strains was provided. Most *C. farmeri* strains are citrate negative (93%) at 48 h; however, 86% are reported to be citrate positive (Simmons) at 7 days (2).
In the present case report we provide the first clinical evidence indicating that *C. farmeri* is indeed a human pathogen. The fact that *C. farmeri* was isolated in pure culture twice from the blood of a child with systemic signs of sepsis (fever and chills) supports its role in the illness. The most likely factor triggering *C. farmeri* septicemia was the SBS condition causing TPN dependency. SBS is a serious medical complication resulting from major resection of the small intestine and in some instances the colon (9). The remaining abbreviated intestine results in a malabsorption and malnutrition condition requiring TPN (5, 9). A consequence of this required catheterization is sepsis, often involving gram-negative bacilli such as *Escherichia coli*, *Klebsiella* species, and *Enterobacter cloacae* (5, 7, 10). Sepsis may result from the gastrointestinal translocation of gram-negative and gram-positive bacteria into the mesenteric lymph nodes, where they eventually spread hematogenously, seeding central venous catheters (5). Alternatively, infection may result from the frequent bowel movements that SBS patients have, thereby increasing the chance of rectal contamination of central venous catheters (7). In the present case, the short time span (13 min) that elapsed between the commencement of TPN and signs of sepsis suggests that the infection may have resulted from contaminated parenteral nutrition. Regardless of which mechanisms are operative, SBS patients run a much higher risk of developing sepsis, and *C. farmeri* should now be added to the list of agents documented to cause such infections.

### REFERENCES


### TABLE 1. Distinguishing features of the *C. amalonaticus* group

<table>
<thead>
<tr>
<th>Substrate for acid production</th>
<th>Result* for:</th>
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<tbody>
<tr>
<td></td>
<td><em>C. amalonaticus</em></td>
</tr>
<tr>
<td>Sucrose</td>
<td>–</td>
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<tr>
<td>Raffinose</td>
<td>–</td>
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<tr>
<td>Melibiose</td>
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* Abbreviations: +, ≥85% of strains positive for acid production from the indicated substrate (48 h); –, ≤15% of strains positive for acid production from the indicated substrates (48 h). Reactions for *C. amalonaticus* and *C. farmeri* are from the work of Farmer et al. (3).