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 nomenspecies 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 and urinary tracts (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). While *C. amalonaticus* sensu stricto still appears to be the *Enterobacteriaceae* within the family *Enterobacteriaceae* and defined a new biogroup (*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>
</tr>
</thead>
<tbody>
<tr>
<td>C. amalonaticus</td>
<td>C. farmeri</td>
</tr>
<tr>
<td>Sucrose</td>
<td>–</td>
</tr>
<tr>
<td>Raffinose</td>
<td>–</td>
</tr>
<tr>
<td>Melibiose</td>
<td>–</td>
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</tbody>
</table>

* 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).