promised patients [7, 8]. In addition, *Lactobacillus* spp. have an elevated capacity for translocation [9]. A viral gastrointestinal infection may have damaged the mucosa and facilitated the passage of bacteria in three of the reported cases (3/11, 27%) [2].

Lengthy or high-dose vancomycin administration prior to an infectious episode may alter the gastrointestinal flora, resulting in the selection of a vancomycin-resistant strain of *Lactobacillus* that may overgrow other enteric bacteria, promoting bacterial transloca-tion [7, 9]. Previous vancomycin therapy was adminis-tered in our case as well as in two-thirds of the cases that we reviewed [4–6].

Lactobacillus spp. can be difficult to eradicate in deep-seated infections [1, 2]: the mortality rate of the infec-tion is between 10 and 25%, according to the different publications [1–6]. Penicillin-aminoglycoside combina-tions are effective both in vitro [1, 10] and in the clinical setting [1], as was the case in our patient. Our observa-tion strengthens the hypothesis that *Lactobacillus* spp. should be considered opportunistic pathogens in immu-nocompromised patients and emphasizes the role of the selective pressure of vancomycin therapy in the patho-genesis of such infections.

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Emergence of Cholera in the Central African Republic

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The Central African Republic (CAR) was cholera-free until June 1997 [1], when cases were reported in several regions during the rainy season. The first of these cases occurred in the south, along the Oubangui river, near the border with the Democratic Republic of Congo. Further cases were reported the same month in northern CAR and in the region of Bangui, the capital city. Toxigenic *Vibrio cholerae* O1, serotype Inaba, biotype El Tor isolated from stool specimens was iden-tified using standard bacteriological methods and the polymerase chain reaction. Records of cases in which patients presented symptoms of cholera and required intravenous rehydration are shown in Table 1.

The areas affected in southern CAR are along the Oubangui river from Mobaye to Mongoumba. Cases of cholera occurred in Kouango and Mobaye, two villages located upstream, between July and October 1997, shortly after refugees arrived from Zaïre and Rwanda. This recent immigration of refugees into southern CAR may have played a role in the emergence of the cholera outbreak.

In northern CAR, cases were investigated by Médecins Sans Frontières (Dr. Javier Barabdiaran). The epidem-iological survey implicated contaminated food sold to a Central African woman on 28 June 1997 at the market in Mini, a village on the CAR Chad border where cases of cholera were also identified. Contaminated food bought at the market in Mini was carried by this woman to Bang, a village 5 km southeast of Mini. On 30 June, a few hours after lunch, she began to experi-ence symptoms of cholera. Later that day she was taken by her brother-in-law to the Hospital of Ngoundaye, 7 km to the east of Bang, where she died. The brother-in-law died of cholera on 2 July. Cholera spread through the region from the hospital of Ngoundaye via persons visiting patients. Funeral practices (cleaning of the bodies by the families) contributed to transmission in the villages around Ngoundaye.

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Table 1 Places of isolation, number of cholera cases, and antimicrobial resistance patterns of *Vibrio cholerae* O1 strains isolated in the Central African Republic in 1997

Outbreak period[a]	Location[a]	Geographic area	No. of cases[a]	No. of deaths[a]	No. of strains studied	Antibiogram	Ribo-type
June	Mongoumba (Lobaye river)	southwest 3°42′N/18°35′E	48	23	1	PB	I
June–Sept.	Kouango (Gbamoko)	southeast 4°55′N/20°10′E	44	8	1	A, Cp, S, TSX, C, PB, O/129	I
June–Sept.	Kouango	southeast 4°55′N/20°9′E	141	14	2	PB	I
July–Sept.	Mobaye	southeast 4°23′N/21°11′E	134	40	2	PB	I
Sept.—Oct.	Damara	south (40 km north of Bangui) 4°58′N/18°35′E	76	3	3	PB	I
June–Sept.	Bang	northwest 7°19′N/15°36′E	13	3	1	S, TSX, C, F, PB, O/129	II
July–Oct.	small villages around Ngoundaye	northwest 7°19′N/15°37′E	91	16	2	S, TSX, C, F, PB, O/129	II
Aug.	Ngoundaye	northwest 7°19′N/15°37′E	9	0	3	S, TSX, C, F, PB, O/129	II

[a] Data were collected from the Ministry of Health of the Central African Republic and reports from Médecins Sans Frontières

A, ampicillin; Cp, cephalothin; C, chloramphenicol; F, furazolidone; PB, polymyxin B; S, sulfamides; TSX, trimethoprim-sulfamethoxazole; O/129, vibriostatic agent

Vibrio cholerae O1 strains isolated in CAR were characterized by ribotyping [2] and antimicrobial susceptibility testing. The isolates were tested by a disk diffusion method for susceptibility to trimethoprim-sulfamethoxazole, sulfamides, chloramphenicol, ampicillin, tetracycline, erythromycin, furazolidone, polymixin B, pefloxacin, cephalothin, cefotaxime, and the vibriostatic agent O/129.

On the basis of ribotyping with restriction enzyme *Bgl*I, *Vibrio cholerae* O1 strains isolated in southern CAR, along the Oubangui river, and near Bangui and Damara all had a single common DNA restriction pattern that contains DNA fragments from 2 to 12 kb (pattern I, Figure 1, lanes 1, 2, 3, 4, 5). This pattern is similar to the B5a pattern, described as the predominant ribotype of the seventh pandemic in Africa and Asia [3]. Pattern I contains an additional fragment of 5.6 kb, which is absent from the B5a pattern. These strains showed two patterns of antimicrobial susceptibility (Table 1), indicating that two clonal groups of strains were circulating in the southern region of CAR. None of the studied strains harbored plasmid DNA (data not shown). This suggests that the antibiotic resistance is encoded by the chromosome, as in other *Vibrio cholerae* strains involved in the seventh cholera pandemic, particularly those isolates resistant to trimethoprim-sulfamethoxazole, polymyxin B, and the vibriostatic agent O/129 [4, 5].

All strains isolated in the northwestern area of CAR had a single common DNA restriction pattern (pattern II, Figure 1, lanes 6, 7, 8) that was different from pattern I (strains isolated in the south). Pattern I and pattern II contain common fragments of 2.2, 4.1, 5.9, 6.1, 6.2, 6.7, and 10.1 kb, but strains belonging to pattern II ribotype showed two additional fragments of 9.3 and 11.5 kb. Pattern II is dissimilar from all ribotype patterns described previously [6, 7], other than that recently described for strains isolated in Guinea-Bissau in 1994 and 1995 [4]. A single clone of *Vibrio cholerae* O1 seems to be involved in all cases from the northern area. This is supported by the results of antimicrobial susceptibility tests, which showed that all isolated strains were resistant to sulfamides, trimethoprim-sulfamethoxazole, furazolidone, polymyxin B, and the vibriostatic agent O/129.

Until 1997, there had been no cases of cholera in CAR since the emergence of the seventh pandemic in 1961 and its entry into Africa in 1970. The epidemiological data available and the results obtained by ribotyping and antimicrobial susceptibility testing indicate that the emergence of cholera in CAR was due to more than one different strain of *Vibrio cholerae* O1 coexisting simultaneously, and that the seventh cholera pandemic caused by biotype El Tor was involved. The Central African ecosystem might not be suitable for long-term survival of *Vibrionaceae*, since no strain of *Vibrio*, *Plesiomonas*, or *Aeromonas* had been isolated within the 3 years prior to 1997 during several studies on diarrhea in CAR. Since October 1997, only isolated cases of cholera in the southern area of CAR have been reported, suggesting that the country is in an interepidemic or endemic period.
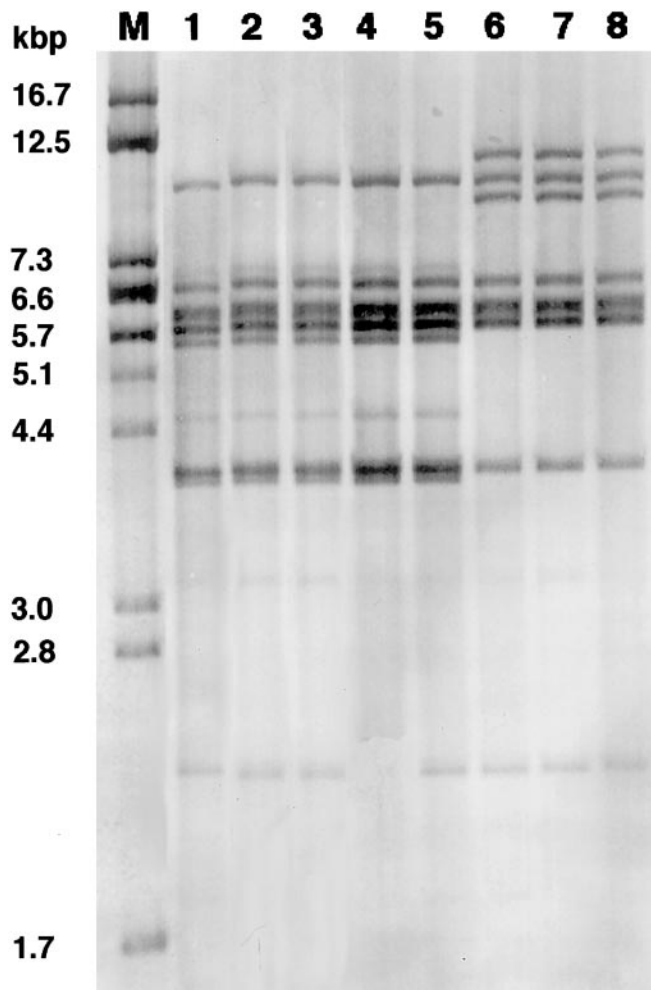
Figure 1 rRNA gene restriction patterns of *Vibrio cholerae* O1 chromosomal DNA digested by *Bgl*I and hybridized with acetyl-aminofluorene-labeled *Escherichia coli* 16+23 S rRNA. Lane numbers refer to strains designated by their place of isolation. *M* molecular weight standards in kilobase pairs; *lane 1* Mongoumba; *lane 2* Kouango (Gbamoko); *lane 3* Kouango; *lane 4* Mobaye; *lane 5* Damara; *lane 6* Bang; *lane 7* villages around Ngoundaye; *lane 8*, Ngoundaye

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In Vitro Activity of Ciprofloxacin, Ofloxacin, and Levofloxacin against *Micrococcus* Species and *Stomatococcus mucilaginosus* Isolated from Healthy Subjects and Neutropenic Patients

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The list of gram-positive pathogens causing bacteremia, especially in neutropenic patients, includes a growing number of commensal bacteria: coagulase-negative staphylococci, viridans streptococci, and, most recently, *Stomatococcus mucilaginosus* [1–3] and *Micrococcus* spp. [1, 4, 5]. Although these organisms appear to be of low virulence, the increase in reported cases of infection due to *Stomatococcus mucilaginosus* and *Micrococcus* spp. has established their pathogenic potential, particularly in neutropenic patients [1–5].

Previous studies examined the susceptibility either of single isolates or of a strain collection of *Micrococcus* spp. or *Stomatococcus mucilaginosus* only against compounds other than the fluoroquinolones [6, 7], irrespective of the wide-scale use of these compounds against a broad range of infections, including infections in neutropenic patients [8]. Therefore, we compared the in vitro activity of ofloxacin, ciprofloxacin, and levofloxacin against 191 isolates of micrococci and 65 isolates of *Stomatococcus mucilaginosus* isolated from healthy subjects and neutropenic patients.

Stomatococcus mucilaginosus was isolated from mucous membranes of the cheek and gingiva, *Micrococcus* spp. additionally from the skin. Thirty-eight isolates of *Stomatococcus mucilaginosus* and 108 isolates of micrococci were obtained from swabs of 50

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