Full description of the clinical case

A 71 year-old man was diagnosed with a muscle-invasive transitional cell carcinoma of the urinary bladder. Radical cystectomy and ileal conduit construction were performed. Peri- and post-operatively, he was given amoxicillin- clavulanate, metronidazole and tobramycin for 48 hours; then amoxicillin-clavulanate was continued for another 9 days. On day 6 after surgery, the patient developed a superficial surgical site infection. Clips were removed and the superficial incision reopened. On day 14, a wound swab was obtained and showed growth of an *Ec* (*Ec*-1) resistant to ceftriaxone but cefepime-susceptible; this isolate was clinically considered as a colonizer.

One week later, the patient developed pneumonia (1^{st} pulmonary infection episode, PIE). Empirical treatment was started with ceftriaxone (2g q24h). After 2 days, therapy was switched to cefepime (1g q12h) and continued for 6 days, resulting in a favorable clinical response. Two weeks after cessation of cefepime, bronchoalveolar lavage was performed because of purulent bronchitis (2^{nd} PIE). An *Ec* (*Ec*-2) resistant to both ceftriaxone and cefepime was detected, though antibiotic treatment was not administered. Two months later, the patient developed a 3^{rd} PIE and responded to piperacillin/tazobactam (4.5 g q8h) treatment for 12 days. Another two weeks later, he presented with a relapse (4^{th} PIE), again responding to piperacillin/tazobactam (4.5 g q8h) for 8 days. No causative pathogen was isolated during 3^{rd} and 4^{th} episodes. A 5^{th} PIE occurred 7 days after cessation of piperacillin/tazobactam, revealing both *Ec*-1 and *Ec*-2 in a purulent sputum sample. Finally, a scintigraphy detected leucocyte enhancement consistent with a pulmonary abscess. Currently, the patient is treated with ciprofloxacin (500 mg q12h) and has been relapse-free in the 4-week follow-up investigation.

Notably, during the 4^{th} PIE a rectal swab revealed that the patient was colonized with 3GC-R *Ec* isolates: five colonies (strains named from *Ec*-A to *Ec*-E) were randomly chosen from the selective plates for further investigations.

Figure S1. Plasmid restriction analysis with EcoRV and PstI enzymes for the *E. coli* transconjugants producing CMY-2 or CMY-33. The IncI1 plasmid carrying both bla_{CMY} genes has identical restriction pattern.

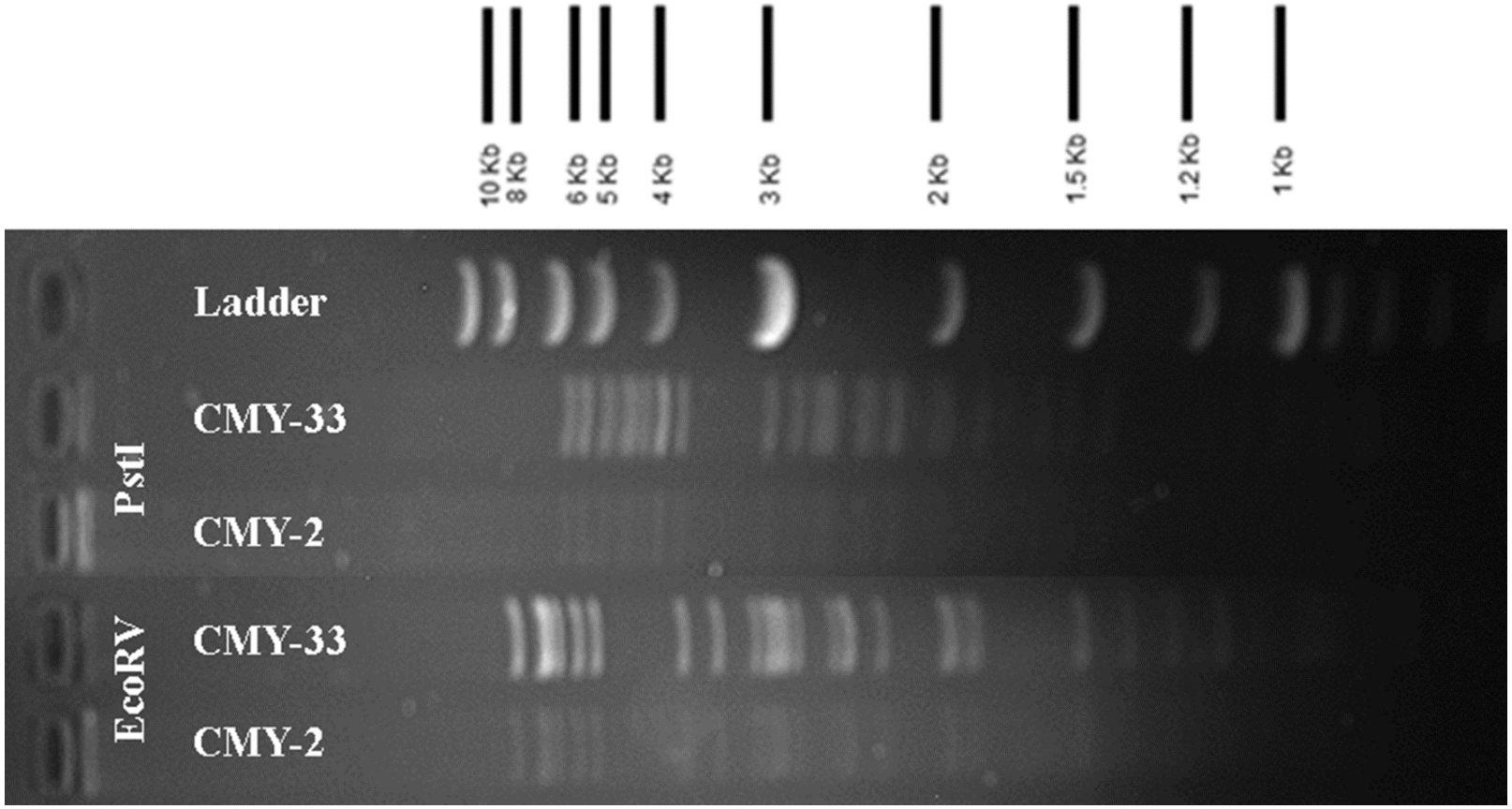

Figure S2. Rep-PCR analysis for the CMY-producing *E. coli* isolates detected from the patient. *Ec*-1, bla_{CMY-2}-positive (from wound swab); *Ec*-2, bla_{CMY-33}-positive (from respiratory sample); *Ec*-A to *Ec*-E (all bla_{CMY-2}-positive) from rectal swab; 5-1, 8-1, and 9-1 are control *E. coli* isolates detected in other patients. Clinical isolates *Ec*-1, *Ec*-2, and *Ec*-A to *Ec*-*E* are identical. The seven strains detected in the patient are also of ST131 and *fimH*22/*fimA*7.

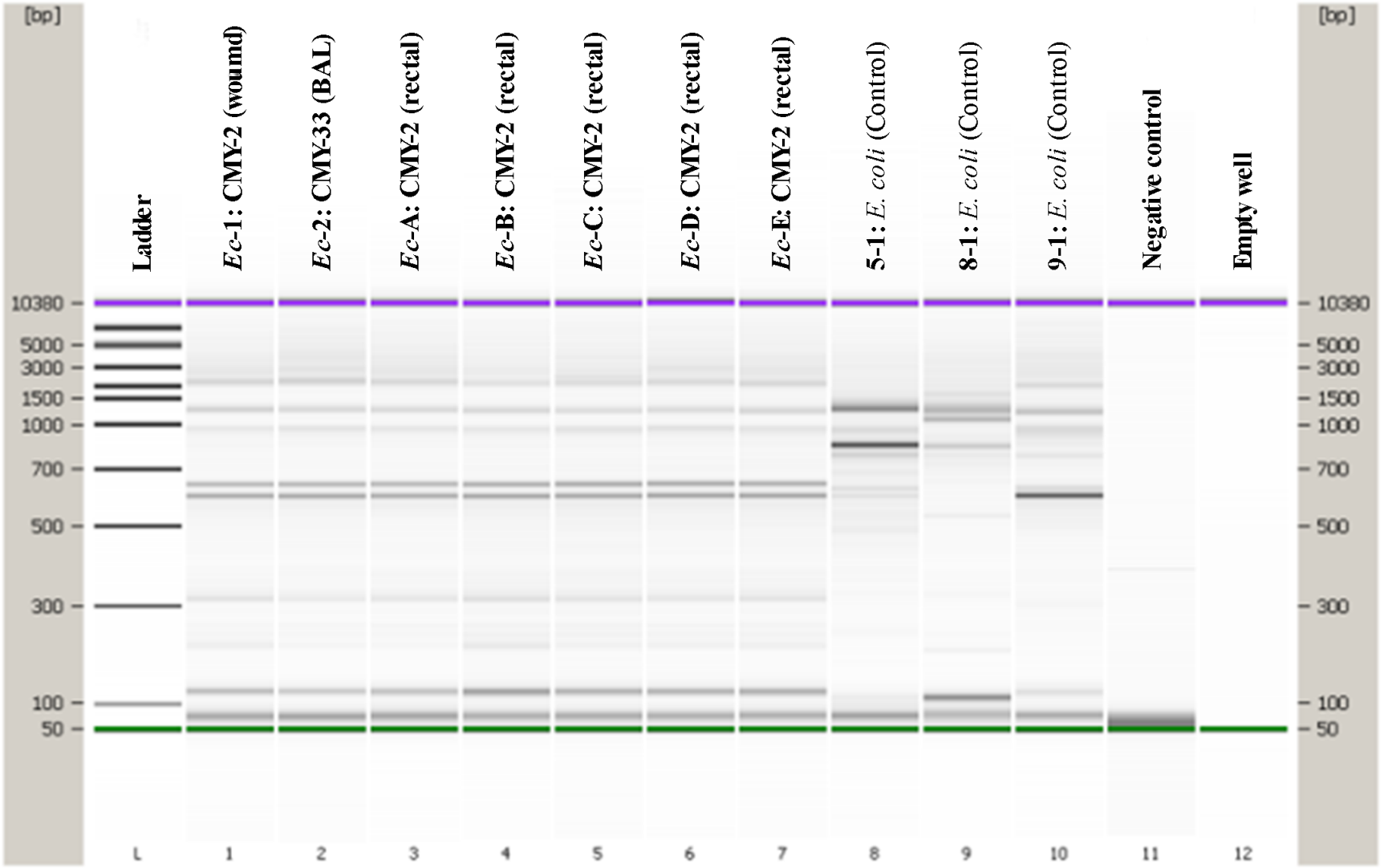

Figure S3. ESI MS of CMY-2 (A) and CMY-33 (B) after purification (>90%). Molecular weight of CMY-33 is less than CMY-2 due to the Leu293-Ala294 deletion.