

Letter to editor

Plasmid mediated multidrug resistance of clinical *Escherichia coli* isolates

Sir,

The report by Kalantar *et al.* [1] in their work “**frequency, antimicrobial susceptibility and plasmid profiles of *Escherichia coli* pathotypes obtained from children with acute diarrhea**” from Iran is important and timely, since the diarrheagenic *E. coli* infection is an important cause of morbidity and mortality in the pediatric population of developing countries; moreover the emergence of plasmid mediated multi-drug resistance among the strains causing occasional outbreaks is a great cause of concern [2].

Plasmid profile analysis is an important tool in epidemiological investigation [3,4]. Kalantar *et al.* [1] studied the presence of plasmids of molecular sizes ranging from 1.4kb to 4.5kb among the acute diarrhea causing *E. coli* isolates showing resistance to ampicillin (A), chloramphenicol (C) and tetracycline (T), and stated that these resistances are plasmid mediated. The authors did not carry out conjugation or plasmid curing experiments in order to establish the involvement of the plasmid in carrying ACT-resistance among the isolates studied. Rather, the current authors have taken support of the findings of Uma *et al.* [3], who reported from Annamalainagar, India that ‘A-imipenem (IP)-cotrimoxazole (Co)’ resistance among *E. coli* isolates, obtained from patients below five years of age with diarrhea, was encoded by 4.8kb plasmid, based upon the fact that same plasmid was found in the transconjugants, conferring similar antibiotic resistance pattern.

Mandal *et al.* [4] also, reported from Kolkata, India, the transfer of approximately 55kb plasmid from the urinary tract infection causing *E. coli*

isolates (resistance pattern ACCoT) to the plasmid less antibiotic sensitive *Salmonella enterica* serovar Typhi that on acquisition of the plasmid expressed ACCoT-resistance. Thus, expression of acquired drug resistance, following conjugation experiments, in the transconjugants [3], and/or concomitant loss of drug resistance due to the loss of plasmid, following curing experiments [5], might be the basis to assess the involvement of plasmid in mediating bacterial resistance to antibiotics.

The plasmid DNAs from the *E. coli* V517 strain has been used widely as the molecular marker [4], but Kalantar *et al.* [1] used linear DNAs (not clearly mentioned in the article, just shown in the figure 1) in order to determine the molecular size of the isolated plasmids from the *E. coli* isolates, and in the study this is also not clear which of the isolated plasmid DNAs is responsible for ACT-resistance. This fact has been partially supported by the use of λ DNA digested with Hind III and Eco RI as the DNA standard marker in an earlier study [3]; however, in this study, the role of 4.8kb plasmid in mediating AIPCo-resistance among test isolates has been documented well.

However, since the widespread occurrence of antibiotic resistant *E. coli* in developing countries, including Iran, necessitated the continuous monitoring of *E. coli* pathotypes, the study will be of help in the vigilance of the R-plasmid, and in taking decision to make the proper regional treatment regimen in order to combat multidrug resistance of *E. coli*, at least in that part (Iran) of the globe.

References

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