

Plasmids-Mediated Antibiotic Resistance in *E. coli*

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ABSTRACT

In this study, E. coli strains were isolated from thirty hospitalized patients receiving antimicrobials, thirty not on antimicrobials and thirty healthy persons were used as controls. Antimicrobial susceptibility test was done to thirteen of the commonly used antimicrobials in hospitals. There was a significant increase in the percentage of E. coli resistant to most of the antimicrobials on comparing the patients receiving antimicrobials and control group. Among the 14 E. coli strains tested, plasmid profile analysis of resistant strains was done and plasmid numbers ranged from 0 to 8 indigenous plasmids with variable sizes. Curing and transformation experiments revealed the existence of CTX^r, CRO^r, CFB^r and OFX^r genes on plasmids. Among these plasmids, the OFX^r plasmids of strains 1 and 14 were found to be conjugative.

Key words: Cephalosporins, Antibiotic resistance genes, Conjugative plasmids.

INTRODUCTION

Bacterial resistance to antimicrobial agents is a threat to public health throughout the world. During the last years, outbreaks of diseases due to multiresistant strains of enteric bacteria pathogens have occurred. The resistance patterns exhibited by these organisms have included those antibiotics used most heavily at the time of the outbreak, as well as older agents. The consequences of resistance affect not only the ability to treat the infection, but also the cost and duration of treatment (Ismaeel, 1993).

Cephalosporin C is an important antibiotic which attacks the cell wall by inhibition of peptidoglycan synthesis. It possesses a β -lactam ring structure similar to

the penicillins. Both side chains of cephalosporin C have been changed by chemical treatment to yield successive generations of cephalosporins with vastly improved properties, including greater antimicrobial activity, broader range of organisms inhibited, improved β -lactamase resistance and different pharmacological properties.

Extended spectrum β -lactamases (ESBLs) exhibit an enhanced ability to hydrolyze the expanded-spectrum β -lactams. The rapid spread of ESBLs caused significant threats to the therapy for infections and usage of the expanded-spectrum β -lactams. Thus, the challenge to clinicians and microbiologists to recognize susceptibility patterns indicative of the presence of specific β -lactamases, such as the extended-spectrum β -lactamases, will