INTRODUCTION

Nosocomial infection due to antibiotic-resistant, enteric Gram-negative bacilli have increased at an alarming rate in intensive care facilities, and are frequently associated with immuno-compromised hosts, for whom they may be particularly devastating ***(Pfaller, 2002).***

Multi drug- resistant *Escherichia coli* and *klebsiella* (as well as other Enterobacteriaceae) carrying plasmid-borne extended-spectrum β-lactamases (ESBLS) have attracted much interest among clinical microbiologists, infectious disease specialists and infection control practitioners ***(Bradford, 2001)***. In contrast, the carriage of chromosomally encoded AmpC β-lactamases by these organisms has been deemed a commensal trail of uncertain consequence and thus the management of such infection has not benefied from the multidisciplinary approach taken with ESBL infection ***(Siebert et a., 1993).***

Standard Kirby-Bauer Disc Diffusion (KBDD) methods and automated Minimal Inhibitory Concentration (MIC) instruments used in most clinical laboratories do not readily detect AmpC-type of inducible resistance. Therapy based on such susceptibility reports may result in induction of resistance in vivo ***(Fish et al., 1995).***

Few instructive studies have reported that the observation of increased MICs to third- generation cephalosporins using high-density inocula (increased sensitivity for detection of derepressed mutant subpopulation) may be used to predict clinical failures ***(Thomson & Moland, 2001).***

To address this gap, the National Committee for Clinical Laboratory Standard (NCCLS) has included warning such as ″Enterobacter, Citrobacter, and Serratia spp. May develop resistance during prolonged therapy with third generation cephalosporins″ in its interpretive guideline publication. However, methods that can be used to detect such resistance have not been suggested.