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Anti-microbial Resistance Patterns in *Escherichia coli* and *Klebsiella Pneumoniae*: A Comparison between Nosocomial and Community Environments

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The increasing bacterial resistance to anti-microbial drugs is a major public health issue. A number of studies reported different bacterial agents of human infections carrying anti-microbial multiple resistance, not only in the nosocomial environment but also in the community as a whole (references (Canton *et. al.*; Shetty & Barnes, 2003).

Bacterial multiple resistance is determined by a great variety of genes that are generally associated in genetic elements (plasmids) and transferred by bacterial conjugation (Dale, 1998; Broda, 1979; Mitsuashi *et. al.*, 1977). The molecular basis for bacterial multiple resistance evolution includes both homologous DNA recombination, transposition events and gene integration through "replicons" as plasmids, promoted by transposons (Jacoby & Archer, 1991; Hughes *et. al.*, 1981; Richmond *et. al.*, 1975). Regardless of antimicrobials pressures, transposition events occur in low frequencies per cell per generation. However, under antibiotic pressures, bacteria containing transposons with genes codifying for antimicrobials resistance are mainly selected, hence resulting in greater prevalence of the genetic element (Lewin, 2001).

A systemic approach to study this phenomenon is proposed. It consists of a statistical data analysis based on a thorough survey of exam results for bacterial anti-microbial sensitivity extracted from hospital files over three years. These exams are routinely carried out in hospital laboratories in order to orient anti-microbial therapeutics. To make the study more consistent the analysis is restricted to *Escherichia coli* and *Klebsiella pneumoniae*, major nosocomial and community urinary infection agents in our (Pereira, 1990; Pereira & Suassuna, 1986) and in several others hospitals (Mayhall, 1996).

More specifically, the study purpose is to investigate whether the occurrence of resistance markers is more unsystematic randomic in the community environment than in the nosocomial one. This would show evidence of a hidden systematic mechanism acting in the nosocomial environment. The study variable is the observed number of resistance markers for a given set of eight antimicrobial. Comparisons between the patterns and numbers of resistance markers observed in each environment, are carried out. In the proposed methodology, we applied a general procedure for fitting count data, distributed in frequency tables, proposed by Platt (1987). It consists of fitting both the classic and the truncated Poisson models to the distribution of the numbers of resistance markers under each environment of different species and years,. In addition, the Zero-Inflated Poisson Model ZIP (see D Böhning, 1998) is also fitted to the data.

In this paper we analyse patterns of occurrence of resistance markers in two distinct environments. If this pattern is more unsystematic in the community environment than in the nosocomial one, it can be argued that a Poisson model would provide a better fit for the number of resistance markers observed in *Enterobacteriaceae* under the community environment than under the nosocomial environment. (Henceforth, occurrence of a resistance marker in *Enterobacteriaceae* will be referred as an event). Therefore, the emphasis here is to compare environments concerning the absence of systematic mechanisms rather than to test how well the Poisson model fits each data set.

Key words: nosocomial infection; anti-microbial resistance; Poisson distribution; Negative Binomial distribution.