Acta Microbiologica et Immunologica Hungarica, 56 (4), pp. 399–402 (2009)
DOI: 10.1556/AMicr.56.2009.4.9

VIRULENCE AND ANTIMICROBIAL RESISTANCE DETERMINANTS OF HUMAN PATHOGENIC AND COMMENSAL STRAINS OF *PSEUDOMONAS AERUGINOSA**

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(Received: 21 October 2009; accepted: 26 October 2009)

Keywords: *Pseudomonas,* virulence, antimicrobial resistance, genetic determinants, PCR, mouse-lung

Introduction

Pseudomonas aeruginosa (Pa) is an opportunistic pathogen, frequently causing local infections in tissues with decreased innate immunity in animals and man, which are difficult to treat due to antimicrobial resistance. In order to survive in host tissues, the *Pa* strains are known to utilize a wide range of virulence-, and fitness factors. Many of these virulence-, and drug resistance factors have their genetic determinants on the chromosome, but some others reside on mobile genetic elements [1]. One of the most remarkable and concerning characteristics of *Pa* is the low antibiotic susceptibility, which under certain conditions can be enhanced by the uptake of mobile elements that may help commensal strains of *Pa* to develop into multidrug resistant human pathogens [2]. Phenotypic resistance can be further increased by appropriate changes in colony formation such as biofilms, or small colony variation [3].

* Extended abstract of lecture BL-3, at CEFORM-2, 7–9 October 2009, Keszthely, Hungary.
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The goal of the present studies was to assess and compare the genetic determinants of virulence-, and antimicrobial resistance of environmental, animal (bovine) non-clinical, and human (clinical) isolates of *Pa*. Furthermore, it was aimed to compare pathogenicity of the representatives of the three major sources (environment, bovine and human) using a mouse lung model. It was assumed that such studies may help the environmental and food safety risk assessment of *Pa* derived from different sources.

Materials and Methods

Bacteria included *Pa* strains from ground water (3), from bovine colonic mucosa, from bovine faeces and from milk samples (30) with clinically negative background, and strains from human clinical cases (13). Comparative studies were made on antimicrobial resistance phenotype using disk diffusion tests for 16 antibiotics used in human and veterinary medicine. Phenotyping studies also included O-typing, pyocin-, and phage typing [4]. Genetic analysis included PFGE, and PCR for the presence of integrons [5]. For virulence determinants a PCR mapping was performed for genes *toxA* (representing T2SS), and *exoS, exoT, exoY, exoU* (representing T3SS) and for *algD* (representing biofilm formation cluster), all known to contribute to virulence and fitness of *Pa* in animals and man [1]. Virulent phenotype was tested by using a mouse lung infection model [6].

Results and Discussion

The studies described here have indicated that the frequency of carriage of the most important genetic determinants of virulence of the *Pa* strains from the three major sources was quite comparable: all were equally present, except *exoU* only present in one human and one environmental strain. O-typing and pyocin typing did not reveal tendencies of differences. The PFGE profile showed a great genetic versatility of human, bovine and environmental strains with some clonal clusters of the bovine strains.

Antimicrobial resistance of the human strains show a much wider scale, and only the human strains were characterized by the presence of integrons. Human strains were characterized by resistance to aminoglycosides (especially gentamicin and amikacin), fluoroquinolones, and cephalosporines, while bovine and environmental strains were overwhelmingly sensitive to these drugs.