GENOTYPIC AND PHENOTYPIC RELATIONSHIP IN *BURKHOLDERIA PSEUDOMALLEI* INDICATES COLONIZATION WITH CLOSELY RELATED ISOLATES

Son Radu[1], Samuel Lihan[1], Azhar Idris[3], Oii Wai Ling[1], Muthafar Husein Al-Haddawi[4] and Gulam Rusul[2]

[1]Department of Biotechnology; [2]Department of Food Science, Faculty of Food Science and Biotechnology, University Putra Malaysia, Serdang, Selangor, Malaysia; [3]Regional Veterinary Laboratory, Kuantan, Pahang, Malaysia; [4]Faculty of Veterinary Medicine, University Putra Malaysia, Serdang, Selangor, Malaysia

Abstract. Seven isolates of *Burkholderia pseudomallei* from cases of melioidosis in human (2 isolates) and animal (2 isolates), cat (one isolate) and from soil samples (2 isolates) were examined for *in vitro* sensitivity to 14 antimicrobial agents and for presence of plasmid DNA. Randomly amplified polymorphic DNA (RAPD) analysis was used to type the isolates, using two arbitrary primers. All isolates were sensitive to chloramphenicol, kanamycin, carbenicillin, rifampicin, enrofloxacin, tetracycline and sulfamethoxazole-trimethoprim. No plasmid was detected in all the isolates tested. RADP fingerprinting demonstrated genomic relationship between isolates, which provides an effective method to study the epidemiology of the isolates examined.

INTRODUCTION

Burkholderia pseudomallei (previously known as *Pseudomonas pseudomallei*), is the causative agent of melioidosis in humans and a large variety of animals (Supputtamongkol *et al*, 1994). The major areas where melioidosis is endemic are Southeast Asia and Northern Australia (Dance, 1991). In Malaysia the disease has been reported in humans and animals (Embi *et al*, 1992; Idris *et al*, 1998; Heng *et al*, 1998). The spectrum of melioidosis ranges from subclinical disease to chronic pulmonary infection to fulminant septicemia with metastatic abscesses. Improve therapy has decrease the mortality in severe meliodosis from 80% to 40-50% (Supputtamongkol *et al*, 1994). The epidemiology and pathogenesis of melioidosis remain unclear. Because of the lack of a sensitive enough method that allows two isolates to be defined as identical strains, no correlation between isolates of soil, human and animal sources has been proven (Lew and Desmarchelier, 1993). Randomly amplified polymorphic DNA (RAPD) analysis is a PCR-based method using a single short random primer. This method is now widely used for the study of population genetics in a large variety of species (Kersulyte *et al*, 1992; Haase *et al*, 1995). In the work reported here, *B. pseudomallei* isolated from human, animals and soil were examined for their antibiotic resistance, plasmid profiles and random amplification of polymorphic DNA analysis.

Corresponding: Dr Son Radu, Department of Biotechnology, Faculty of Food Science and Biotechnology, University Putra Malaysia, 43400 UPM Serdang, Selangor, Malaysia.
Tel: +6 03 9486101 ext 3446; Fax: +6 03 9423552; E-mail: son@fsb.upm.edu.my

MATERIALS AND METHODS

Bacterial strains

The seven strains of *B. pseudomallei* had been identified by the forwarding laboratory (Regional Veterinary Laboratory, Kuantan, Pahang) by standard tests. In this case study, two isolates each were isolated from a child admitted to the hospital with melioidosis, a dead goat reared by the child's family and soil samples from the surrounding compound of the family's house in a rural village. A single unrelated isolate from a cat was also included.

Sensitivity to antimicrobial agents

Antimicrobial sensitivity tests were performed by the disc diffusion method according to National Committee for Clinical Laboratory Standard (1997). The *B. pseudomallei* strains were tested against the following antibiotics discs (BBL, Becton Dickinson Microbiology Systems, Cockeysville, Maryland, USA) on Mueller Hinton agar; ampicillin (10 μg), carbenicillin (100 μg), cephalexin (30 μg), chloramphenicol (30 μg), clindamycin (2 μg), enrofloxacin (5 μg), kanamycin (30 μg), latamoxef (30 μg), furazolidone (100 μg), penicillin (10 μg), rifampicin (30 μg), streptomycin (15 μg), sulfamethoxazole-trimethoprim