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Phenotypic and genomic characteristics of members of the "Burkholderia cepacia complex"

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Abstract

The aim of my project was to compare representatives of each of seven genomovars/species comprising the "*Burkholderia cepacia* complex" with respect to overall genome size and chromosome number and also to examine their capacity to form *N*-acyl homoserine lactones (AHLs). A major goal was to determine whether there were significant differences between clinical and non-clinical isolates of these bacteria. My working hypothesis was that clinical isolates (primarily members of genomovar III) might have reduced catabolic and biosynthetic potential and correspondingly smaller genomes than non-clinical isolates. It also seemed reasonable that AHL-dependent quorum sensing, a mechanism that commonly governs expression of genes important for host colonization, would be more prominent in clinical isolates. ^ A survey of 34 *B. cepacia* complex isolates including representatives of all seven genomovars indicated that all had large multichromosomal genomes. Randomly linearized replicons from preparations of intact chromosomal DNA were resolved by pulsed-field gel electrophoresis and their sizes estimated by comparison of their electrophoretic mobilities with yeast DNA size markers. Overall genome size was confirmed by determining the sums of the molecular weights of macro restrict ion fragments obtained by digestion of chromosomal DNA with enzymes such as *CeuI* and *SwaI*. Analysis of a larger group of 62 isolates indicated that all of the strains were prototrophs which exhibited a high degree of nutritional versatility. Thus the notion that clinical isolates had lost DNA and functions important for survival as free-living bacteria proved incorrect. ^ A survey for ability to produce AHLs indicated that members of genomovars I–IV formed low levels of these compounds compared to members of genomovars V–VII. I subsequently isolated mutant derivatives of representatives of genomovars II and III which produced extremely high levels of AHL. I

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characterized a genetic locus, *bmuIR* from strain 17616 which contained genes encoding AHL synthase and AHL-binding transcriptional activator. The mutations leading to increased AHL formation were located outside of the *bmuIR* locus, indicating that other regulatory genes influence AHL formation. ^ An important result of my studies of AHL formation was the development of a rapid procedure for concentration of AHLs from culture supernatants, which involved their adsorption to a copolymer of divinylbenzene and *N*-vinylpyrrolidone. ^

Subject Area

Biology, Microbiology

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