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Mutator strains of *Pseudomonas*commonly found in CF lungs

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Keywords

Bronchiectasis, cystic fibrosis, *Pseudomonas*

Introduction

Pseudomonas is a chronic colonizing/infecting organism of the respiratory tract and is especially prevalent in patients with CF. *Pseudomonas* is widely distributed through the environment, commonly isolated from wet surfaces, including sinks, taps and drains. Although the initial colonizing organism in the lung is generally thought to be a rough strain, naturally occurring mutations result in the production of a protective alginate coat, so that the organism appears smooth or mucoid in culture. Mucoid strains are commonly the organism responsible for long-term colonisation of the CF respiratory tract.

Recent investigations into the adaptive strategies of bacteria to a rapidly changing environment show that the bacteria with hypermutable (mutator) strains are selected on a long term basis. These mutator strains are produced mainly by alterations in the DNA repair and correction genes. Once adapted, the mutator is thought to spontaneously revert to the original non-mutator state if the selection pressure is removed.

Comments

This paper demonstrates that certain mutator strains of *Pseudomonas* are commonly found in the sputum of cystic fibrosis (CF) patients, although they are not found in *Pseudomonas* in other situations, eg blood cultures from patients with febrile neutropenia. Whilst it is clear that there is a difference in the *Pseudomonas* organisms isolated from the CF lung, it is not clear why this difference exists - is the important difference related to the host, or the organism? For instance, do mutator strains better colonise CF patients, or do they colonise the respiratory tract better than non-mutator strains? Does the CF lung increase the expression of the *Pseudomonas* mutator strains? Does treatment of CF lung disease with

antibiotics select out those mutator organisms? Further assessment of mutator strains in early CF colonisation and chronic colonisation in non-CF disease will be necessary to answer these questions.

Methods

Mutator strains were assessed using rifampicin or streptomycin mutation frequencies

Genotype was measured using the random amplification of polymorphic DNA (RAPD) technique

Results

Mutator strains were obtained from 11 of 30 patients (37%); in total 19.5% of 128 isolates were of the mutator type. Although these isolates show different phenotypes, genotype analysis showed only a single RAPD type in the vast majority of patients. This suggests that a single colonising organism can evolve with the development of different mutator strains. Associated with the mutator phenotype were isolates, which were highly resistant to a wide variety of standard antibiotics, including Ticarcillin, Ceftazidime, Gentamicin, Tobramycin and Amikacin. In contrast, none of the 75 *Pseudomonas* isolates from non-CF patients (predominantly from blood cultures) showed the mutator phenotype.

Discussion

The question that is not yet answered is whether the *Pseudomonas* isolates that can mutate are the ones that then colonise the human respiratory tract, both in CF and non-CF. Alternatively, something inherent in the CF milieu may induce the *Pseudomonas* isolates to develop the capacity to have mutator strains. Finally, the possibility remains that the mutator strains are simply the reaction of the *Pseudomonas* to the recurrent episodes of 'noxious insults' namely courses of antibiotics aimed at eradicating the *Pseudomonas*.

Additional information

Pseudomonas interaction. Recent papers have explored the possibility that the CF transmembrane conductance regulator is involved in bacterial adherence and entry into respiratory epithelial cells. By combining the two approaches, it is likely that better understanding of the interactions of the respiratory tract with *Pseudomonas* will be determined, hopefully with therapeutic benefit to the patient.

References

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