DOES LOW MAGNESIUM IN CYSTIC FIBROSIS CONTRIBUTE TO BACTERIAL PATHOGENICITY?

Barbara M. Coffey (Gregory G. Anderson), Department of Biology, Purdue School of Science, Indiana University–Purdue University Indianapolis, Indianapolis, Indiana 46202

Cystic fibrosis (CF) is a genetic disease for which there is currently no cure. Individuals with CF are plaqued by myriad symptoms, including chronic pneumonia, which diminishes quality of life and reduces life expectancy to 40 years. The most common bacterium in CF patients' lungs is Pseudomonas aeruginosa, a highly adaptable organism capable of surviving robust antibiotic treatment. At the heart of developing improved treatments for CF patients is the need to better understand *P. aeruginosa* pathogenicity. To this end, we have been studying the role of magnesium, which is often found at below normal levels in CF patients. Magnesium is an essential element in numerous cellular functions in both bacteria and humans. In previous research, we developed a P. aeruginosa strain with a deletion of the magnesium transport protein MgtE, as well as 16 plasmids carrying different mutations of the matE gene. Experiments with these constructs demonstrated a relationship between magnesium transport and bacterial toxin production. In the research presented here, we hypothesize that lower levels of magnesium may trigger a bacterial response, causing a change in P. aeruginosa pathogenicity. Changes may include differential growth, toxin release, and formation of biofilms, which are surface-adhered, antibiotic tolerant bacterial communities in a protective polysaccharide matrix. Using various magnesium levels, we have measured *P. aeruginosa* growth rates, motility, biofilm formation, and cytotoxicity toward cultured cells derived from the CF bronchial epithelium. Preliminary results suggest that lower magnesium contributes to changes in the bacterium that favor persistence in the CF lung. Ongoing studies include the effect of long-term growth of *P. aeruginosa* in low magnesium and how this impacts a number of virulence factors. We anticipate that our research will elucidate the relationship between magnesium and *P. aeruginosa* pathogenicity and potentially lead to improved treatments for CF patients.

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