It is possible, of course, that more than one enterotoxigenic organism may be involved in an episode or outbreak of diarrhea, particularly if those organisms are present in sufficiently high concentrations and produce enterotoxins that act at different sites in the intestinal tract. A number of patients had very rapid onset of diarrhea following the implicated meal; this is not a feature of *C. perfringens* food poisoning but would not be inconsistent with *C. perfringens* and *K. pneumoniae* as coexisting pathogens. Further in vitro studies are necessary to test this hypothesis.

Phenotypic Conversion of *Pseudomonas aeruginosa*

Two recent articles discussed the phenotypic conversion of the mucoid *Pseudomonas aeruginosa* which frequently colonizes the lungs of patients with cystic fibrosis (5, 11). These articles contained several hypotheses which have been offered to explain the phenotypic conversion, including: (i) antimicrobial pressure (6), (ii) phage induction (9), and (iii) nosocomial transmission (2).

Speert et al. proposed that phenotypic conversion is a consequence of nutritional limitations within the cystic fibrosis respiratory tract rather than features unique to these strains of bacteria. The data in their article seem to support this view (11).

Fegan et al. suggested that *P. aeruginosa* undergoes phenotypic conversion with respect to exoenzyme secretion, serum sensitivity, and colony form as the clinical condition of patients with cystic fibrosis worsens (5).

Any hypothesis which is accepted to explain phenotypic conversion needs to explain some additional factors, such as the following:

(i) Mucoid strains of *P. aeruginosa* have been isolated from bronchial secretions of patients with a chronic pulmonary disease other than cystic fibrosis. At one time, the isolation of mucoid *P. aeruginosa* was considered diagnostic of cystic fibrosis (10).

(ii) Mucoid strains of *P. aeruginosa* have been isolated from chronic urinary tract infections (3).

(iii) Mucoid strains of gram-negative bacilli other than *P. aeruginosa* have been isolated from sputum of patients with cystic fibrosis (7).

(iv) The compositions of the capsules of *P. aeruginosa* and some of the other gram-negative bacilli isolated from sputum of patients with cystic fibrosis are similar but not identical (4, 8).

There seems to be some agreement in the articles of Speert et al. and Fegan et al. that it is the uniqueness of the environment of the lungs of patients with cystic fibrosis or chronic pulmonary disease which contributes to the phenotypic conversion of *P. aeruginosa* and other gram-negative bacilli.

This uniqueness may be the ionic composition of the sputum, with two ions receiving special attention. Calcium concentrations have been found to be altered in sputum from patients with cystic fibrosis. There also appears to be an alteration in the function of calmodulin and/or calcitonin, which control calcium concentration (12). Iron has been found to markedly enhance the accumulation of nonmucoid revertants of *P. aeruginosa* in culture (1).

Although an explanation for phenotypic conversion of *P. aeruginosa* is not yet available, there is one thing which might be done to help in the treatment of patients with cystic fibrosis. It is suggested that patients with cystic fibrosis (or their parents) be encouraged to monitor the bacterial flora in the cystic fibrosis sputum themselves. This monitoring could be done using a specially designed five- or three-well Unibac Plate (Endotech Clinical Products, Indianapolis, Ind.).

Patients with cystic fibrosis are trained to collect sputum by using various physical therapy techniques, and because of the ease of inoculation of the five- and three-well Unibac Plates, they could be trained to culture this sputum once it is collected. This would shorten the time between the colonization of *P. aeruginosa* in the lungs of patients with cystic fibrosis and the first detection of *P. aeruginosa* on culture.

If a patient is already colonized with *P. aeruginosa*, having the patient monitor his sputum bacterial flora would shorten the time between the occurrence and detection of phenotypic conversion of *P. aeruginosa*. Quicker detection of phenotypic conversion would allow a faster antimicrobial change, which might result in a slowing of the worsening of the patient’s clinical condition due to mucoid *P. aeruginosa*.

REFERENCES


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Author's Reply

An explanation for the phenotypic conversion that appears to occur within the respiratory tract of patients with cystic fibrosis (CF) remains elusive. Comments made in Dr. Kilbourn's letter reiterate some of the points in our paper (2). It is possible that *Pseudomonas aeruginosa* strains from patients with CF undergo a switch to the characteristic phenotype (mucoid and lipopolysaccharide-rough) under environmental pressures within the endobronchial space. There is not yet sufficient information, however, to determine whether restriction of any specific nutrient is necessary or sufficient for this phenotypic conversion to occur. Further investigations to delineate the specific environmental and/or nutritional factors needed to mediate such a switch will be of great interest to microbiologists and to clinicians responsible for the care of these patients.

The concept of home collection and culture of CF sputum specimens is new but in my opinion will not alter the natural course of pulmonary infections in these patients. Although it is useful for physicians to know when patients with CF become colonized with *P. aeruginosa*, therapy is not instituted generally until symptomatic pulmonary exacerbations develop. The advent of home-based culturing would simply burden these patients and their parents with another chore, thereby raising their anxiety and detracting from other more productive aspects of their care. The stated aim of home culturing (more rapid detection of carriage) would probably not benefit the patients; early stages of colonization are not associated with a notable change in clinical condition or respiratory status (1). For the same reasons, more rapid detection of phenotypic conversion would likely be of no direct diagnostic benefit to physicians caring for patients with CF.

REFERENCES


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