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DOI 10.1099/mic.0.024752-0

Sugar sweet and deadly: author's response

Drs Reid and Bell provide thoughtful and informative comments on our recent report (Bartholdson *et al.*, 2008), which describes the production of exopolysaccharide (EPS) in the presence of mannitol, and other sugar alcohols, by the *Burkholderia cepacia* complex (Bcc). My co-authors and I are grateful for these insights. We would also point out that the

role of EPS in the pathogenesis of Bcc may demonstrate additional idiosyncrasy and subtlety in these organisms relating to a suggested similarity between the Bcc and the characteristic conversion to a mucoid, alginate-producing phenotype observed in *Pseudomonas aeruginosa* during the chronic stages of infection in individuals with cystic fibrosis (CF). In our study, we were initially surprised to observe a lack of EPS biosynthesis in the highly virulent *Burkholderia cenocepacia* ET12 lineage. However, as we suggested, loss of mucoidy in *B. cenocepacia* isolates could provide a striking contrast with the pathogenesis of *P. aeruginosa*. Zlosnik *et al.* (2008) recently observed that isolates of *B. cenocepacia* are frequently non-mucoid, and also a surprising mucoid to non-mucoid conversion in sequential isolates from chronically infected patients. As they suggest, Bcc EPS could be responsible for the persistence of Bcc in CF airways, whilst loss of EPS could lead to enhanced

virulence. The influence of this change in EPS phenotype on mannitol therapy in individuals with CF, including CF-related diabetes, is unclear.

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