proteome of *V. cholerae* in a non-adhering mutant strain CD11 (Jacob et al., 1993); however, no data have been presented by Sharma et al. (2011) to demonstrate that VC1929 is the protein missing in this strain. In their recent report, they identify a frameshift in VC1929 which would account for the loss of a functional VC1929 protein in this strain, yet in their original publication (Jacob et al., 1993), the full-length 33 kDa protein band can be seen clearly in CD11 extracts when the protease inhibitor PMSF is added to their sample, suggesting that in this strain, VC1929 is less stable than in the wild-type, rather than it being truncated by a frameshift. Together, these data actually suggest that VC1929 is not the 33 kDa protein identified in their original study and if we apply Occam’s razor, it is most likely to be a simple C4-dicarboxylate binding protein from a TRAP transporter. In conclusion, all our work points to the VPI-2-encoded genes as being those involved in sialic acid transport and utilization and our experience with the functional characterization of diverse TRAP transporters (Thomas et al., 2006; Mulligan et al., 2011) suggests that VC1929 is the SBP component of a C4-dicarboxylate transporter in *V. cholerae*.

Gavin H. Thomas1 and E. Fidelma Boyd2

1Department of Biology, University of York, York YO10 5YW, UK
2Department of Biological Sciences, Wolf Hall, University of Delaware, Newark, DE 19716, USA

Gavin H. Thomas (gavin.thomas@york.ac.uk) E. Fidelma Boyd (fboyd@udel.edu)


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**Authors’ response: on sialic acid transport and utilization by *Vibrio cholerae***

At the outset we agree and concur with data generated by Boyd and colleagues that a cluster of genes situated on vibrio pathogenicity island 2 (VPI-2) may be involved in transport and catabolism of sialic acid in *Vibrio cholerae*. We have not contradicted but acknowledged their contribution in our recent publication (Sharma et al., 2011). While we were analysing the data published by the authors, we were excited about the quality of the work and the significance of their findings. We believe that sialic acid, being a carbohydrate, plays a crucial role in the pathogenesis of *Vibrio cholerae*.

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CD11 is a non-colonizing mutant and, since its isolation, has never caused mortality with signs of diarrhoea in infected infant mice (Srivastava et al., 1980; Jacob et al., 1993). The protection against cholera given by DctP to rabbits should be considered interesting because if neuraminidase is upregulated and Vibrio species utilize sialic acid as an energy source within gut, then antibodies against DctP should prevent bacterial multiplication in the intestine, and hence induce immunity.

S. K. Sharma, T. S. Moe, R. Srivastava, D. Chandra and B. S. Srivastava
Central Drug Research Institute, CSIR, Lucknow 226001, India
Correspondence: B. S. Srivastava (drbrahm@gmail.com)


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