Microbiology Comment provides a platform for readers of *Microbiology* to communicate their personal observations and opinions in a more informal way than through the submission of papers.

Most of us feel, from time to time, that other authors have not acknowledged the work of our own or other groups or have omitted to interpret important aspects of their own data. Perhaps we have observations that, although not sufficient to merit a full paper, add a further dimension to one published by others. In other instances we may have a useful piece of methodology that we would like to share.

The Editors hope that readers will take full advantage of this new section and use it to raise matters that hitherto have been confined to a limited audience.

Jon Saunders
Editor-in-Chief

Lipid profiles of bacteria grown at low temperature

In the context of the paper by Nichols & Russell (1) on fatty acid adaptation in Antarctic bacteria, we consider it worthwhile to mention a recent report by Tsuchido *et al.* (2). Previously, these authors had shown that autolysis in *Bacillus subtilis* was induced in response to cold shock (3). In their recent investigation (2), they analysed lipid profiles of *B. subtilis* grown at 37 and 25 °C. The proportions of short-chain acids (C₁₂–C₁₅) to long-chain acids (C₁₆–C₁₈) and that of *anteiso* acids to *iso* acids were higher in cells grown at 25 °C. The resultant increase in the membrane fluidity in this batch was associated with significantly higher resistance of the cells to cold-shock-induced autolysis compared to their counterparts grown at 37 °C.

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1. Nichols, D.S. & Russell, N.J. (1996). Fatty acid adaptation in an Antarctic bacterium – changes in primer utilization. *Microbiology* **142**, 747–754.
2. Tsuchido, T., Nishino, T., Kato, Y. & Takano, M. (1995). Involvement of membrane lipids in cold shock induced

autolysis of *Bacillus subtilis* cells. *Biosci Biotechnol Biochem* **59**, 1636–1640.

3. Svarachorn, A., Tsuchido, T., Shinmyo, A. & Takano, M. (1991). Autolysis of *Bacillus subtilis* induced by low temperature. *J Ferment Bioeng* **71**, 281–283.

Author's reply

Response to cold shock: lipid profiles and autolysis of bacteria

With reference to the comments of Ghanekar & Chattopadhyay on the possible link between membrane fluidity and cold-shock-induced autolysis of *Bacillus subtilis*, in the context of the paper by Nichols & Russell (1), it is doubtful if the kind of temperature-dependent selection of primers by fatty acid synthase is involved in the sensitivity to autolysis directly. The enzyme does, of course, contribute indirectly by providing the available pool of fatty acids from which the membrane lipid acyl chains are selected. The lysis of cells after cold shock will involve membrane damage, so conversely recovery from (and probably resistance to) cold shock is likely to involve membrane repair and therefore lipid synthesis. Tsuchido *et al.* (2) also found that cells grown in the presence of cerulenin, an inhibitor of fatty acid biosynthesis, were lysed more rapidly after cold shock than were control cells. The mechanistic basis of their proposal for the involvement of lipids in cold shock could well stem from the generation (by loss of a fatty acid) of lysophospholipids, which are well known (indeed named!) for their ability to lyse cells. Thus, it is not surprising that they also found that lysophospholipids are less effective than phospholipids in suppressing cold-shock-induced autolysis.

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1. Nichols, D.S. & Russell, N.J. (1996). Fatty acid adaptation in an Antarctic bacterium – changes in primer utilization. *Microbiology* **142**, 747–754.
2. Tsuchido, T., Nishino, T., Kato, Y. & Takano, M. (1995). Involvement of membrane lipids in cold shock induced autolysis of *Bacillus subtilis* cells. *Biosci Biotechnol Biochem* **59**, 1636–1640.

Response to cold shock: transcriptional and translational control?

Studies of the cold-stress response are clearly divided into two periods: before and after the work of Jones *et al.* (3). From the publication of this paper to date, numerous researchers have focused their work in order to find a sigma factor(s) that could control the cold-shock response as σ^{32} and σ^{24} do in response to heat shock in *Escherichia coli* (1). Whilst this 'cold sigma' has never been found, studies of CspA (alias CS7.4), a protein known to be the major cold-shock protein (CSP) in *E. coli*, and its homologue in *Bacillus subtilis*, CspB, have led to major discoveries (2, 10). Significantly, these proteins contain motifs similar to the RNP1 and RNP2 RNA-binding domains, and CspA is involved in the transcriptional regulation of at least two genes, *hns* and *gyrA* (4, 6). Nevertheless, the huge quantity of work developed around both CspB and CspA, particularly in the field of structural biochemistry, is not in accord with the level of understanding of cold-shock response regulation (8, 13–15).

Parallel to this work, mostly oriented towards a transcriptional regulation, other authors have intermittently tackled the problem from a translational perspective. Initially, VanBogelen & Neidhardt (17) showed that ribosomes could

► GUIDELINES

Communications should be in the form of letters and should be brief and to the point. A single small Table or Figure may be included, as may a limited number of references (cited in the text by numbers, and listed in alphabetical order at the end of the letter). A short title (fewer than 50 characters) should be provided.

Approval for publication rests with the Editor-in-Chief, who reserves the right to edit letters and/or to make a brief reply. Other interested persons may also be invited to reply. The Editors of *Microbiology* do not necessarily agree with the views expressed in *Microbiology Comment*.

Contributions should be addressed to the Editor-in-Chief via the Editorial Office.