IN BRIEF

BIOFILMS

Proteomic analysis of *Campylobacter jejuni* 11168 biofilms reveals a role for the motility complex in biofilm formation

Kalmokoff, M. *et al. J. Bacteriol.* **188**, 4312–4320 (2006)

Campylobacter jejuni has previously been shown to attach as single cells to solid matrices and to associate with biofilms containing other bacterial species. Now, a Canadian research group has discovered that *C. jejuni* 11168 can form multicellular biofilms on various surfaces, including steel, nitrocellulose and glass fibres, and they report their results in a recent issue of the *Journal of Bacteriology*. Intriguingly, proteomic analysis revealed that proteins belonging to the *C. jejuni* motility complex, including the flagellins, the basal body proteins and the chemotactic proteins, are expressed at higher levels in biofilm-grown cells than in planktonic cells. Additionally, in liquid culture, an aflagellate mutant was unable to form a pellicle at the air–water interface and insertional inactivation of flagellar filament genes delayed pellicle formation, indicating that the motility complex is important for cell–cell interactions in mature *C. jejuni* biofilms.

INNATE IMMUNITY

Role for erbin in bacterial activation of Nod2

Kufer, T. A. *et al. Infect. Immun.* **74**, 3115–3124 (2006)

NOD-like receptors are a family of cytosolic pattern-recognition receptors that are an important component of the host innate response to infection. Nod2 is known to respond to intracellular muramyl dipeptide, a subunit of bacterial peptidoglycan, triggering the nuclear factor-κB pathway. To gain further insights into the Nod2 signalling pathways, Kufer *et al.* used a yeast two-hybrid screen to search for interaction partners. In a recent issue of *Infection and Immunity*, they outline the characterization of a new binding partner for Nod2, the human protein erbin. Erbin is a member of the LAP (leucine-rich-repeat and PDZ-containing) protein family, and Kufer *et al.* postulate that it is a negative regulator of Nod2.

VIROLOGY

Ultrastructure and origin of membrane vesicles associated with the severe acute respiratory syndrome coronavirus replication complex

Snijder, E. J. *et al. J. Virol.* **80**, 5927–5940 (2006)

In members of the *Nidoviridae* family, replication complexes are thought to localize to virus-induced double-membrane vesicles (DMVs), and this has been studied in most detail for the coronavirus mouse hepatitis virus (MHV) and the arterivirus equine arteritis virus (EAV). Now, Eric Snijder and colleagues have analysed in detail the early stages of infection of Vero E6 cells with the SARS coronavirus (SARS-CoV), focusing on the formation of the membrane structures involved in viral RNA synthesis. They find evidence for the early formation of DMVs, although they note that these structures are more fragile than the DMVs seen in MHV and EAV infection. In contrast to results reported for MHV, the SARS-CoV replication complexes seem to remain separated from the site of virus assembly. Moreover, their results indicate that the vesicles are not derived from the autophagic pathway but instead are derived from the endoplasmic reticulum.