signals through both Gαq and Gβγ protein subunits and two different MAPKs (mitogen activated protein kinases) to stimulate downstream transcription factors, which ultimately lead to *VEGF* promoter activation. Consistent with this, NIH-3T3 cells that expressed US28 formed highly vascularized, VEGF-expressing tumours 2 weeks after inoculation into nude mice. This indicates that VEGF-mediated angiogenesis is responsible for at least some of the oncogenic properties of US28.

To verify these findings the authors used an HCMV strain that does not express US28 to infect a glioblastoma cell line. This strain failed to induce *VEGF* promoter activation, unlike the wild-type virus.

Interestingly, the expression of US28 in non-tumorigenic cells can also induce apoptosis. So, it seems that the cellular context determines whether *US28* functions as an oncogene and a pro-angiogenic factor. The authors conclude that US28 might be a potential target for the treatment of early-stage HCMV-related proliferative diseases.

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ORIGINAL RESEARCH PAPER Maussang, D. *et al.* Human cytomegalovirus-encoded chemokine receptor US28 promotes tumorigenesis. *Proc. Natl Acad. Sci. USA* **103**, 13068–13073 (2006)
FURTHER READING Damania, B. Oncogenic γ-herpesviruses: comparison of viral proteins involved in tumorigenesis. *Nature Rev. Microbiol.* **2**, 656–668 (2004).

O157:H7. Interestingly, the length of the induced closure differed between these two pathogens — with *E. coli* O157:H7, closure persisted for the 8-hour duration of the experiment, whereas *Pst* DC300-induced closure was reversed after ~3 hours. This suggested that *Pst* DC300 might have evolved a mechanism to reopen the stomata. *Pst* DC300 has two main virulence factors, a type III secretion system (T3SS) and the phytotoxin coronatine. Analysis of the response to *Pst* DC300 mutants deficient in coronatine or with a defective T3SS demonstrated that coronatine is the virulence factor involved in suppressing stomatal closure, and it was shown to function downstream of ABA.

So, in addition to their key role in gaseous exchange and transpiration, plant stomata also function as 'innate immune gates'. Rather than being able to freely enter plant tissues through the stomata, *Pst* DC300 triggers initial stomatal closure through the detection of PAMPs and the ABA signalling pathway. The bacteria then counteract this defence response by secreting coronatine, which causes the stomata to reopen. Given that stomata are present in all vascular plants, the authors speculate that PAMP-induced stomatal closure could be a widespread phenomenon and that the inhibition of this defence response might have been a key adaptation in the evolution of plant pathogens.

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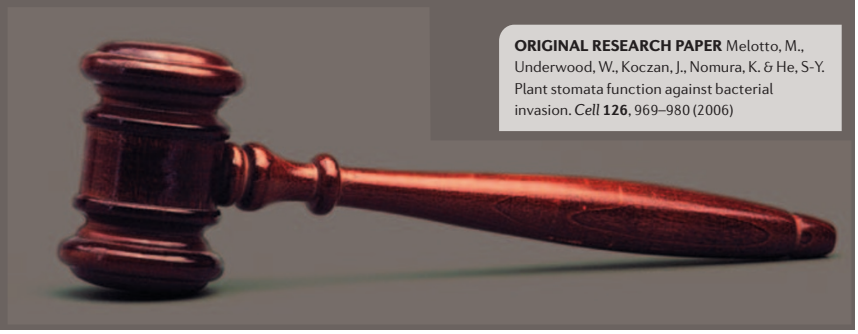

ORIGINAL RESEARCH PAPER Melotto, M., Underwood, W., Koczan, J., Nomura, K. & He, S-Y. Plant stomata function against bacterial invasion. *Cell* **126**, 969–980 (2006)

IN BRIEF

SYMBIOSIS

Symbiosis insights through metagenomic analysis of a microbial consortium

Woyke, T. *et al. Nature* 17 September 2006 (doi:10.1038/nature05192)

Nicole Dubilier, Edward Rubin and colleagues report in a recent issue of *Nature* on their metagenomic analysis of the bacterial endosymbionts present under the cuticle of the marine oligochaete worm *Olavius algarvensis*. Using shotgun sequencing and metabolic pathway reconstruction, Woyke *et al.* were able to characterize four bacterial cosymbionts. The bacteria are either γ- or δ-proteobacteria, and can generate energy by carbon fixation and either oxidization of sulphides or reduction of sulphates. Other detailed metabolic information obtained allowed the authors to reconstruct the physiology of two of the symbionts and their interactions with the worm.

BACTERIAL PATHOGENICITY

Virulence factors of *Yersinia pestis* are overcome by a strong lipopolysaccharide response

Montminy, S. W. *et al. Nature Immunol.* **7**, 1066–1073 (2006)

Yersinia pestis can undergo temperature-dependent alterations in its lipopolysaccharide (LPS). At 21–27°C, the average temperature of the flea vector, the lipid A component of LPS is hexa-acylated whereas at 37°C, mammalian body temperature, it is tetra-acylated. It had previously been suggested that this temperature-dependent switch could be involved in immune evasion, as tetra-acylated LPS is a poor stimulator of Toll-like receptor 4 (TLR4). Montminy *et al.* expressed the gene encoding the *Escherichia coli* LpxL acyltransferase in *Y. pestis*. The presence of this gene caused *Y. pestis* to synthesize hexa-acylated LPS, which was a potent TLR4 stimulator, but this strain of *Y. pestis* (KIM1001-pLpxL) was avirulent in mice. Resistance to KIM1001-pLpxL was found to require the presence of TLR4 and the TLR4 adaptor MyD88 and co-receptor MD-2. These results indicate that the virulence of *Y. pestis* is strongly dependent on the evasion of the LPS–TLR4 signalling pathway.

QUORUM SENSING

Ligand-induced asymmetry in histidine sensor kinase complex regulates quorum sensing

Neiditch, M. B. *et al. Cell* **126**, 1095–1108 (2006)

The quorum-sensing signal autoinducer 2 (AI-2) is produced and detected by both Gram-negative and Gram-positive species. In *Vibrio harveyi*, the AI-2 receptor comprises LuxP and LuxQ, a periplasmic protein and membrane sensor histidine kinase, respectively. Neiditch and colleagues present the crystal structures of the periplasmic domain of LuxQ ($LuxQ_p$) and of $LuxPQ_p$ bound to AI-2. The most notable ligand-induced structural change is a major conformation change in LuxP — AI-2 binding promotes the dimerization of the LuxP periplasmic regions, generating asymmetric $LuxPQ_p$ dimers. The authors propose a model in which, in the absence of AI-2, the interaction between LuxP and LuxQ forms a 'clasp' between the proteins, and in the presence of AI-2, this clasp is released.