gastric cell. Interestingly, sLex/a was previously identified as a tumour antigen and a marker of gastric dysplasia. Boren's group showed that *H. pylori* infection causes formation of sLex/a antigens on the surface of human and monkey gastric epithelial cells.

SabA is not the only binding molecule that *H. pylori* possesses. Borén and colleagues previously showed that another bacterial adhesin protein, BabA, promotes attachment to the Lewis B (Leb) molecule on the gastric-cell surface. But bacteria could still adhere to these cells after the *babA* gene was deleted, so the sLex–SabA interaction is likely to be an important component of tumorigenesis.

So, what are the advantages of these adhesin proteins? They allow *H. pylori* strains to bind to gastric epithelial cells and they also protect the bacteria from being shed into the gastric lumen. The ability of these bacteria to cling tightly to gastric mucosal cells also ensures their access to the nourishing exudate from gastric epithelium that is damaged by the infection. Their persistence in the gastric mucosa causes the host inflammatory response to be maintained.

This study not only sheds new light on the mechanism that allows *H. pylori* to take advantage of the host inflammatory response, but also opens the possibility of more effective treatment for *H. pylori* infection. These bacteria have developed increasing resistance to antibiotics, so the BabA and SabA proteins might be useful components of a gastric cancer vaccine.

Kristine Novak

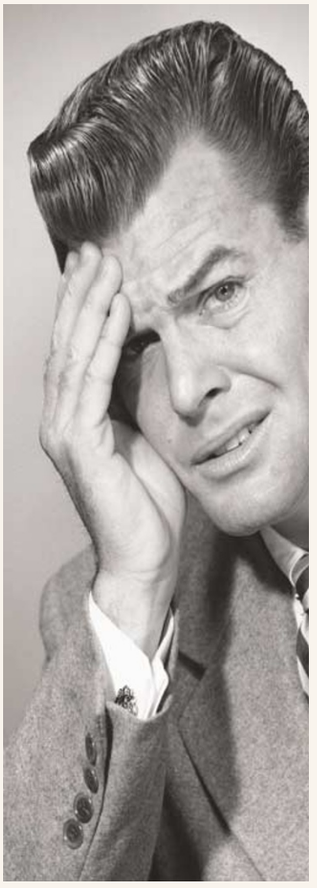
References and links

ORIGINAL RESEARCH PAPER Mahdavi, J. *et al*. *Helicobacter pylori* SabA adhesin in persistent infection and chronic inflammation. *Science* **297**, 573–578 (2002)
FURTHER READING Peek, R. M. & Blaser, M. *Helicobacter pylori* and gastrointestinal tract adenocarcinomas. *Nature Rev. Cancer* **2**, 28–37 (2002)
WEB SITES
An animated movie of *H. pylori* adherence: http://www.eurekalert.org/pub_releases/science/2002-07/mahdavi-7-26-02-public.html
Thomas Boren's web site: http://www.odont.umu.se/forskning/0boren_proj_eng.html

The other recurrent alterations were mapped onto the human genome, and literature and database searches revealed that many of the homologous regions were also altered in human carcinomas. This type of analysis could therefore facilitate the identification of new genes that are involved in human cancer.

So, it seems that telomere dysfunction can drive tumorigenesis in mice, as it is thought to in aged humans. Among the remaining questions are the frequency with which this type of genomic instability causes tumour formation, the genes that are involved and how this determines the tumour types that develop.

Emma Greenwood



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WEB SITE
Ron DePinho's lab: http://www.hms.harvard.edu/dms/bbs/fac/depinho.html

IN BRIEF

GLIOBLASTOMA

Blockage of Ca^{2+}-permeable AMPA receptors suppresses migration and induces apoptosis in human glioblastoma cells.

Ishiuchi, S. *et al. Nature Med.* 12 Aug 2002 (doi:10.1038/nm746)

Glioblastoma multiforme is highly invasive and difficult to treat. Glioblastoma cells express Ca^{2+}-permeable AMPA-type glutamate receptors that are involved in the migration response. Their overexpression resulted in migration and proliferation, and their conversion to Ca^{2+}-impermeable receptors inhibited cell migration and induced apoptosis. So, inhibition of these receptors might prevent glioblastoma invasion.

CELL MIGRATION

Src-induced de-regulation of E-cadherin in colon cancer cells requires integrin signalling.

Avizienyte, E. *et al. Nature Cell Biol.* **4**, 632–638 (2002)

Src activity is raised in colon cancer, but what is its effect? Increased Src activity results in the redistribution of components of adherens junctions to integrin–adhesion complexes in the presence of high calcium levels. E-cadherin remains internalized, so cannot assemble the correct intercellular contacts. A Src mutant that lacks the kinase domain does not deregulate E-cadherin. So, expression of catalytically active Src might facilitate cancer-cell migration.

EPIDEMIOLOGY

Risk of cancer in patients with human pituitary growth hormone in the UK, 1959-85: a cohort study.

Swerdlow, A. J. *et al*. *Lancet* **360**, 273–277 (2002)

Growth hormone raises serum concentrations of insulin-like growth factor 1 (IGF-1), and raised levels of growth hormone and IGF-1 might be associated with an increased risk of certain solid cancers. Follow-up of nearly 2,000 patients who were treated during childhood with human pituitary growth hormone showed a significant increase in the risk of developing colorectal cancer and Hodgkin's disease. This was based on very small numbers of cancer cases, but the findings are nevertheless of concern and warrant further investigation.

THERAPEUTICS

Inhibition of glioma growth by tumor-specific activation of double-stranded RNA-dependent protein kinase PKR.

Shir, A. & Levitzki, A. *Nature Biotechnol*. 19 Aug 2002 (doi:10.1038/nbt730)

A new antisense RNA strategy selectively targets tumour cells. Antisense RNAs that are complementary to sequences that flank a deletion or translocation bind the mutant RNAs to produce a double-stranded molecule. These molecules activate a double-stranded RNA-dependent protein kinase (PKR), which signals cells to undergo apoptosis. A lentiviral vector expressing a 39-nucleotide antisense RNA that is complementary to the flanking regions of an epidermal-growth-factor receptor (*EGFR*) gene deletion was shown to activate PKR, causing glioblastoma cells to undergo apoptosis.