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How bacteria induce stomach ulcers

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Helicobacter pylori is a Gram-positive bacterium that colonizes the gut mucosa and can induce gastric ulcers. In an Advanced Online Publication in Nature Genetics Fujikawa *et al.* report how the *H. pylori* cytotoxin VacA causes ulcer formation (*Nature Genetics*, 24 February 2003, doi:10.1038/ng1112). Mice lacking the *Prptz* gene, which encodes a protein tyrosine phosphatase receptor type 2 that is expressed in gastric glands, are resistant to VacA-induced ulcers. VacA is taken up equally well by epithelial cells, in culture or *in vivo*, in the absence of *Prptz*, with no differences in vacuole development or cell survival. But, VacA binding to Ptprz initiates a signalling cascade that leads to cell detachment from the basement membrane. Furthermore, this can be mimicked with an endogenous Ptprz ligand that was also found to induce severe gastritis. These findings indicate potential novel clinical strategies based on inhibiting the VacA-Ptprz pathway or downstream events that lead to cell detachment and subsequent gastritis.

References

1. *Nature Genetics*, [http://www.nature.com/naturegenetics]

2. Gene structure of the *Helicobacter pylori* cytotoxin and evidence of its key role in gastric disease.