How E. coli passes safely through stomach acid

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In some parts of the world, many small children become infected with severe diarrhea which often proves fatal. The condition is usually caused by strains of Escherichia coli (commonly known as E. coli) bacteria, and bacteria of the genus Yersinia. These bacteria attach themselves to the wall of the small intestine and use a needle-like apparatus to inject toxins into the tissue. Yet these bacteria usually enter the human body via the mouth – and you would expect them to be killed off by the strong acid in the stomach, which provides a barrier against infection.

Members of the collaborative research center "The Bacterial Cell Envelope" at the University of Tübingen including researchers from the Tübingen University Hospitals as well as Jack C. Leo and Professor Dirk Linke of the Max Planck Institute for Developmental Biology investigated this phenomenon and discovered how these bacteria can protect themselves from acid and mechanical stress as they pass through the stomach. The results were published in the latest issue of *Molecular Microbiology*.

E. coli and Yersinia bacteria attack cells in the small intestine which absorb nutrients. They use adhesins such as intimin (a protein; the name comes from "intimate adherence") to stick to intestinal epithelial cells and to subsequently form tiny channels between the bacteria and the intestinal cells. In this way they are able to introduce diarrhea-causing toxins into the intestine.

The intimin is inserted into the bacterial cell envelope, where it binds

with the bacteria´s stabilizing structure, peptidoglycan, a mesh-like molecule consisting of sugars and amino acids. "But the binding of intimin with peptidoglycan only works under acid conditions," says Dirk Linke. "We assume that this mechanism protects against acidic and mechanical stress and that E. coli bacteria can pass through the stomach unharmed." Intimin therefore supports the infection process by bacteria which would otherwise have difficulty reaching the small intestine. The researchers suspect that intimin boosts the bacteria's virulence.

More information: Jack C. Leo, Philipp Oberhettinger, Manish Chaubey, Monika Schütz, Daniel Kühner, Ute Bertsche, Heinz Schwarz, Friedrich Götz, Ingo B. Autenrieth, Murray Coles, Dirk Linke: "The Intimin periplasmic domain mediates dimerisation and binding to peptidoglycan." *Molecular Microbiology*, DOI: 10.1111/mmi.12840

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