ESRF	**Experiment title:** InlB / Met complex	**Experiment number**: MX 521
Beamline: ID23-2	**Date of experiment**: from: 19.05.2006 to: 20.05.2006	**Date of report**: 01.08.2007
Shifts: 3	**Local contact(s)**: David Flot	*Received at ESRF:*

Names and affiliations of applicants (* indicates experimentalists):

Hartmut Niemann * 1), Dirk Heinz, Gregor Hagelüken *

Helmholtz Centre for Infection Research, Division of Structural Biology

Inhoffenstraße 7, D-38124 Braunschweig, Germany

1) present address: Universität Bielefeld, Fakultät für Chemie
Universitätsstraße 25 , D-33615 Bielefeld, Germany

Report:

Niemann HH, Jäger V, Butler PJ, van den Heuvel J, Schmidt S, Ferraris D, Gherardi E, Heinz DW.

Structure of the Human Receptor Tyrosine Kinase Met in Complex with the *Listeria* Invasion Protein InlB.

The tyrosine kinase Met, the product of the c-met proto-oncogene and the receptor for hepatocyte growth factor/scatter factor (HGF/SF), mediates signals critical for cell survival and migration. The human pathogen *Listeria monocytogenes* exploits Met signaling for invasion of host cells via its surface protein InlB. We present the crystal structure of the complex between a large fragment of the human Met ectodomain and the Met-binding domain of InlB. The concave face of the InlB leucine-rich repeat region interacts tightly with the first immunoglobulin-like domain of the Met stalk, a domain which does not bind HGF/SF. A second contact between InlB and the Met Sema domain locks the otherwise flexible receptor in a rigid, signaling competent conformation. Full Met activation requires the additional C-terminal domains of InlB which induce heparin-mediated receptor clustering and potent signaling. Thus, although it elicits a similar cellular response, InlB is not a structural mimic of HGF/SF.

Cell. 2007 Jul 27;130(2):235-246.