

**Name**

Prof. Dr. med. Georg Häcker

**Institution:** Institute of Medical Microbiology and Hygiene, University Medical Center, Faculty of Medicine, Freiburg University

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**Position:** Head of Institute

**Academic education including academic degrees**

1984 – 1990 Human Medicine, University Ulm (state examination)

2004 – 2008 Bachelor of Law (LLB), 2004-2008, University of London, England (distance)

**Scientific graduation**

1991 Dr. med. (MD), University of Ulm

**Employment**

Since 7/2009 Director, Institute of Medical Microbiology and Hygiene, University Medical Center Freiburg W3-Professor, University of Freiburg, Germany

2000 - 6/2009 C3-Professor, Institute for Medical Microbiology, Immunology and Hygiene, TU München, Munich, Germany

2002-2009 Consultant, Institute f. Microbiology/Klinikum rechts der Isar, Munich

1998 – 2000 Privatdozent (Lecturer), Institute for Medical Mikrobiologie, TU München

1996 – 2000 Group leader, Institute for Medical Microbiology, TUM

1995 Visiting Scientist, Walter and Eliza Hall Institute for Medical Research (WEHI), Melbourne, Australia.

1993 - 1995 DFG-scholarship, WEHI

1991 - 1993 Arzt im Praktikum/assistant doctor, Institut for Medic Microbiology, TUM

**Other activities, awards and honours**

Since 2024 Member of the grants committee CRC (DFG)

Since 10/2023 Vice speaker, SFB-TRR353 (since 10/2023)

2018 – 2022 President, Deutsche Gesellschaft für Hygiene und Mikrobiologie (DGHM)

2010 – 2023 Elected member, Fakultätsrat Medical Faculty Freiburg (2010-2023)

Since 2014 Member, Standing Committee for Habilitationen/apl. Prof., Medical Faculty Freiburg (since 2014)

2002 Medical Specialist for Microbiology and Infection Epidemiology, Bayerische Landesärztekammer

1998 Habilitation, Medical Microbiology and Immunology (Faculty of Medicine, TUM)

1992 Approbation/License to practise medicine

**Ten selected publications****The Caspase-Activated DNase promotes cellular senescence.**

Haimovici, A., Rupp, V., Amer, T., Moeed, A., Weber A., Häcker, G.  
EMBO J. (2024) doi: 10.1038/s44318-024-00163-9.

**The Caspase-Activated DNase drives inflammation and contributes to defense against viral infection.**

Moeed A, Thilmann N, Beck F, Puthussery BK, Ortman N, Haimovici A, Badr MT, Haghighi EB, Boerries M, Öllinger R, Rad R, Kirschnek S, Gentle IE, Donakonda S, Petric PP, Hummel JF, Pfaffendorf E, Zanetta P, Schell C, Schwemmler M, Weber A, Häcker G.  
Cell Death Differ. (2024) doi: 10.1038/s41418-024-01320-7.

**Mitochondria supply sub-lethal signals for cytokine secretion and DNA-damage in *H. pylori* infection.**

Dörflinger, B., Badr, M.T., Haimovici, A., Fischer, L., Vier, J., Metz, A., Eisele, B., Bronsert, P., Aumann, K., Höppner, J., Waguia Kontchou, C., Parui, I., Weber, A., Kirschnek, S. and Häcker, G.

Cell Death Diff. (2022) 29 (11): 2218-2232 doi: 10.1038/s41418-022-01009-9.

**A non-death function of the mitochondrial apoptosis apparatus in immunity.**

Brokatzky, D., Dörflinger, B., Haimovici, A., Weber, A., Kirschnek, S., Vier, J., Metz, A., Henschel, J., Steinfeldt, T., Gentle, I.E. and Häcker, G.

EMBO J. (2019) pii: e2018100907. doi: 10.15252/embj.2018100907.

**Dynein light chain 1 induces assembly of large Bim complexes on mitochondria that stabilise Mcl-1 and regulate apoptosis.**

Singh, P.K., Roukounakis, A., Frank, D.O., Kirschnek, S., Das, K.K., Neumann, S., Madl, J., Römer, W., Zorzini, C., Borner, C., Haimovici, A., Garcia-Saez, A., Weber, A.\* and Häcker, G.\*

Genes & Development (2017) 31(17):1754-1769.doi:10.1101/gad.302497.117.

**Immunization against poly-*N*-acetylglucosamine reduces neutrophil activation and GVHD while sparing microbial diversity.**

Hülsdünker, J., Thomas, O.S., Haring, E., Unger, S., Gonzalo Núñez, N., Tugues, S., Gao, Z., Duquesne, S., Cywes-Bentley, C., Oyardi, O., Kirschnek, S., Schmitt-Graeff, A., Pabst, O., Koenecke, C., Duyster, J., Apostolova, P., Blaser, M.J., Becher, B.\*, Pier, G.B.\*, Häcker, G.\*, Zeiser, R\*.

Proc Natl Acad Sci U S A. (2019) pii: 201908549. doi: 10.1073/pnas.1908549116.

**Neutrophil granulocytes recruited upon translocation of intestinal commensal bacteria enhance graft- versus-host disease via local tissue damage.**

Schwab, L., Goroncy, L., Palaniyandi, S., Gautam, S., Triantafyllopoulou, A., Mocsai, A., Reichardt, W., Karlsson, F. J., Radhakrishnan, S. V., Hanke, K., Schmitt-Graeff, A., Freudenberg, M., von Loewenich, F. D., Wolf, P., Leonhardt, F., Baxan, N., Pfeifer, D., Schmah, O., Schönle, A., Martin, S. F., Mertelsmann, R., Duyster, J., Finke, J., Prinz, M., Henneke, P., Häcker, H., Hildebrandt, G. C.\*, Häcker, G.\*, Zeiser, R.\* Nat. Med. (2014) 20(6):648-54. doi: 10.1038/nm.3517.

**clAPs block Ripoptosome formation, a RIP1/caspase 8 containing intracellular cell death complex differentially regulated by cFLIP isoforms.**

Feoktistova, S., Geserick, P., Kellert, B., Panayotova Dimitrova, D., Langlais, C., Hupe, M., Cain, K., MacFarlane, M., Häcker, G. and Leverkus, M.

Mol. Cell (2011) 43(3):449-63. doi: 10.1016/j.molcel.2011.06.011

**Cytopathicity of Chlamydia infection can be largely reproduced by expressing a single chlamydial gene, Chlamydial Protease-like Activity Factor.**

Paschen, S., Christian, J. G., Vier, J., Schmidt, F., Walch, A., Ojcius, D. M. and Häcker, G.

J Cell Biol. (2008) 182(1):117-27 doi: 10.1083/jcb.200804023.

**BimS Induced Apoptosis Requires Mitochondrial Localization but not Interaction with Anti-Apoptotic Bcl-2 Proteins.**

Weber, A., Paschen, S. A., Heger, K., Wilfling, F., Frankenberg, T., Bauerschmitt, H., Seiffert, B. M., Kirschnek, S., Wagner, H. and Häcker, G.

J Cell Biol. (2007) 177(4):625-36. doi: 10.1083/jcb.200610148.

\* Co-senior author

## **Research**

We are particularly interested in the reaction of mammalian cells to challenges such as during infection with bacteria, viruses or parasites. In recent years, we have been focusing on the role of the mitochondrial apoptosis pathway in such infections. Apoptosis is rarely induced, but the cells activate the pathway nevertheless to a sub-lethal level, which has numerous consequences, such as inflammation and senescence. We are trying to understand molecular signals at and around mitochondria, as well as the biomedical consequences of this activity.