

Dr. Annett Halle, MD

center of advanced european studies and research (caesar)



center of advanced european studies and research (caesar), Max Planck Research Group „Neuroimmunology“

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Research Expertise

Dr. Halle's group studies innate immune mechanisms and microglial function in Alzheimer's disease using cell culture techniques and mouse models of Alzheimer's disease.

Education / Training

Charité – University Medicine Berlin,

Medical Neuroscience, MD thesis, 2005

Free University Berlin, Humboldt University Berlin,
Clinical Medicine, MD, 2003

Appointments / Positions Held

2011 - present

Max-Planck Research Group leader

center of advanced european studies and research (caesar),
Bonn, Germany

2009 - 2011

Research fellow and resident in Neuropathology

Department of Neuropathology, Charité – University Medicine
Berlin, Germany

2005 - 2008

Postdoctoral fellow and instructor in Internal Medicine,

Department of Infectious Diseases, University of
Massachusetts, Worcester, USA

2003 - 2005

Medical dissertation and resident in Neurology, Department of
Experimental Neurology, Charité – University Medicine Berlin,
Germany

2000

Research internship, Department of Cell Biology, Harvard
University, Boston, USA

Honors / Awards

2010

Ernst Jung-Career Award for Medical Research

2008

Lydia Rabinowitsch Fellowship for young scientists, Charité
Berlin

2005 - 2007

Postdoctoral Fellowship, German Academic Exchange
Foundation (DAAD)

2006

Young Scientist Award, Science Foundation Berlin, Germany

2006

Award for the best medical dissertation of 2005, Berlin Society
for Psychiatry and Neurology

2005

Humboldt Prize (Prize for best dissertation of the year,
Humboldt University Berlin)

10 Most Relevant Publications for Dr. Annett Halle

1. Schnaars M, Beckert H, **Halle A.** Assessing β -amyloid-induced NLRP3 inflammasome activation in primary microglia. *Methods Mol Biol.* 2013;1040:1-8.
2. Krabbe, G.*, **Halle, A.***, Matyash, V., Rinnenthal, J. L., Eom, G. D., Bernhardt, U., Miller, K. R., Prokop, S., Kettenmann, H. and Heppner, F. L., Functional impairment of microglia coincides with Beta-amyloid deposition in mice with Alzheimer-like pathology. *PLoS One* 2013. 8: e60921.
3. Heneka, M. T., Kummer, M. P., Stutz, A., Delekate, A., Schwartz, S., Vieira-Saecker, A., Griep, A., Axt, D., Remus, A., Tzeng, T. C., Gelpi, E., **Halle, A.**, Korte, M., Latz, E. and Golenbock, D. T., NLRP3 is activated in Alzheimer's disease and contributes to pathology in APP/PS1 mice. *Nature* 2013. 493: 674-678.
4. Stewart, C. R., Stuart, L. M., Wilkinson, K., van Gils, J. M., Deng, J., **Halle, A.**, Rayner, K. J., Boyer, L., Zhong, R., Frazier, W. A., Lacy-Hulbert, A., El Khoury, J., Golenbock, D. T. and Moore, K. J., CD36 ligands promote sterile inflammation through assembly of a Toll-like receptor 4 and 6 heterodimer. *Nat Immunol* 2010. 11: 155-161.
5. Siednienko, J., **Halle, A.**, Nagpal, K., Golenbock, D. T. and Miggin, S. M., TLR3-mediated IFN-beta gene induction is negatively regulated by the TLR adaptor MyD88 adaptor-like. *Eur J Immunol* 2010. 40: 3150-3160.
6. **Halle, A.**, Hornung, V., Petzold, G. C., Stewart, C. R., Monks, B. G., Reinheckel, T., Fitzgerald, K. A., Latz, E., Moore, K. J. and Golenbock, D. T., The NALP3 inflammasome is involved in the innate immune response to amyloid-beta. *Nat Immunol* 2008. 9: 857-865.
7. **Halle, A.***, Zhou, S*., Kurt-Jones, E. A., Cerny, A. M., Porpiglia, E., Rogers, M., Golenbock, D. T. and Finberg, R. W., Lymphocytic choriomeningitis virus (LCMV) infection of CNS glial cells results in TLR2-MyD88/Mal-dependent inflammatory responses. *J Neuroimmunol* 2008. 194: 70-82.
8. Hornung, V., Bauernfeind, F., **Halle, A.**, Samstad, E. O., Kono, H., Rock, K. L., Fitzgerald, K. A. and Latz, E., Silica crystals and aluminum salts activate the NALP3 inflammasome through phagosomal destabilization. *Nat Immunol* 2008. 9: 847-856.
9. Jain, V., **Halle, A.**, Halmen, K. A., Lien, E., Charrel-Dennis, M., Ram, S., Golenbock, D. T. and Visintin, A., Phagocytosis and intracellular killing of MD-2 opsonized gram-negative bacteria depend on TLR4 signaling. *Blood* 2008. 111: 4637-4645.
10. **Halle, A.***, Bermpohl, D.*, Freyer, D., Dagand, E., Braun, J. S., Bechmann, I., Schroder, N. W. and Weber, J. R., Bacterial programmed cell death of cerebral endothelial cells involves dual death pathways. *J Clin Invest* 2005. 115: 1607-1615.

*These authors contributed equally