Sodeik, Beate

D.o.B. 10-11-1963

Current Position

Since 2007 Full Professor (W2) at the Institute of Virology, MHH

Undergraduate and Postgraduate Training

1982 - 1988 Study of Biology in Bonn, Germany
1984 Pre-Diploma in Biology, University Bonn, Germany
(Genetics, Microbiology, Zoology, Botany, Chemistry, Physics)
1988 Diploma in Cell Biology, Zoology, Physics, University Bonn, Germany
1993 PhD in Cell Biology, Virology and Botany; University Heidelberg, Germany
2002 *Habilitation* and *Venia legendi* in Biochemistry, MHH

Academic and Research Posts

1986 - 1988 Research Assistant, Institute of Cell Biology, University Bonn, Germany
1988 - 1989 DAAD Fellow, Department of Physiology & Cellular Biophysics,
College of Physicians & Surgeons, Columbia University, New York, NY, USA
1989 - 1993 EMBL Predoctoral Fellow, Cell Biology, EMBL, Heidelberg, Germany
1993 - 1995 EMBO Postdoctoral Fellow, Dept. Cell Biology, School of Medicine,
Yale University, New Haven, CT, USA
1995 - 1997 Research Associate, Dept. Cell Biology, School of Medicine,
Yale University, New Haven, CT, USA
1997 - 2002 Assistant Professor (C1), Institute of Physiological Chemistry, MM
2002 – 2007 Associate Professor (C2), Institute of Virology, MHH
Since 2007 Full Professor (W2), Institute of Virology, MHH

Other Scientific Roles

1998 - 2004 Founding & Steering Committee DFG Graduate School GRK 745 (TiHo & MHH)
2001 - 2004 Founder and Chair, Study Group Cell Biology, *Gesellschaft für Virologie*
2001, 2004 Chair, EMBO Workshop on Cell Biology of Viral Infections
2007 - 2013 Member, MHH Senate Commission, MSc Program Biomedicine
2007 - 2019 Member, MHH Senate Commission on Research (*Forschungskommission*)
2009 - 2017 Steering Committee DFG-SFB900, Chronic Infections
since 2011 Scientific Advisory Committee, International Herpesvirus Workshop
2016 - 2020 Elected Member, Study Section, German Research Foundation, Bonn
(DFG Fachkollegium 204: Mikrobiologie, Virologie und Immunologie)
2017 - 2020 Vice President, *Gesellschaft für Virologie*
since 2002 Person in Charge, Module Virology, MSc Biomedicine & MSc Biochemistry, MHH
since 2015 Member, *SAW (Senatsausschuss Wettbewerb),* Leibniz-Gemeinschaft, Berlin

Awards and Prizes

1988 - 1989 DAAD Undergraduate Student Fellowship, Columbia University, NY, NY, USA
1989 EMBL Predoctoral Fellowship, EMBL, Heidelberg, Germany
1993 EMBO Postdoctoral Short-term Fellowship, EMBL, Heidelberg, Germany
1993 - 1995 EMBO Postdoctoral Long-term Fellowship, Yale University, New Haven, CT, USA

10 Selected Publications (h index 36, Google Scholar)

Turan A, Grosche L, Krawczyk A, Mühl-Zürbes P, Drassner C, Düthorn A, Kummer M, Hasenberg M, Voortmann S, Jastrow H, Dörrie J, Schaft N, Kraner M, Döhner K, Sodeik B, Steinkasserer A, Heilingloh CS. Autophagic degradation of lamins facilitates the nuclear egress of herpes simplex virus type 1. *J Cell Biology* 2019; 218:508-523.

Döhner K*, Ramos-Nascimento A*, Bialy D*, Anderson F, Hickford-Martinez A, Rother F, Koithan T, Rudolph K, Buch A, Prank U, Binz A, Hügel S, Lebbink RJ, Hoeben RC, Hartmann E, Bader M, Bauerfeind R, Sodeik B. Importin α1 is required for nuclear import of Herpes Simplex Virus proteins and capsid assembly in fibroblasts and neurons. *PLoS Pathogens* 2018; 14(1):e1006823.

Buch A, Müller O, Ivanova L, Döhner K, Bialy D, Bosse JB, Pohlmann A, Binz A, Hegemann M, Nagel CH, Koltzenburg M, Viejo-Borbolla A, Rosenhahn B, Bauerfeind R, Sodeik B. Inner tegument proteins of Herpes Simplex Virus are sufficient for intracellular capsid motility but not for axonal targeting. *PLoS Pathogens* 2017; 13(12):e1006813.

Snijder J, Radtke K, Anderson F, Scholtes L, Corradini E, Baines J, Heck AJR, Wuite GJL, Sodeik B (corresponding), Roos WH (corresponding). Vertex-Specific Proteins pUL17 and pUL25 Mechanically Reinforce Herpes Simplex Virus Capsids. *J Virology* 2017; doi: 10.1128/JVI.00123-17.

Ivanova L, Buch A, Döhner K, Pohlmann A, Binz A, Prank U, Sandbaumhüter M, Bauerfeind R (corresponding), Sodeik B (corresponding). Conserved tryptophan motifs in the large tegument protein pUL36 are required for efficient secondary envelopment of herpes simplex virus capsids. *J Virology* 2016; 90: 5368-5383, Cover Image.

Wnek M, Ressel L, Ricci E, Rodriguez-Martinez C, Villalvazo Guerrero JC, Ismail Z, Smith C, Kipar A, Sodeik B, Chinnery PF, Solomon T, Griffiths MJ. Herpes simplex encephalitis is linked with selective mitochondrial damage; a post-mortem and in vitro study. *Acta Neuropathol* 2016; 132: 433-451.

Radtke K, Kieneke D, Wolfstein A, Michael K, Steffen W, Scholz T, Karger A, Sodeik B. Plus- and minus-end directed microtubule motors bind simultaneously to herpes simplex virus capsids using different inner tegument structures. *PLoS Pathogens* 2010; 6: e1000991.

Roos WH (joint first), Radtke R (joint first), Kniesmeijer E, Geertsema H, Sodeik B (corresponding) & Wuite G (corresponding author). Scaffold expulsion and genome packaging trigger stabilization of Herpes Simplex Virus capsids. *Proc Natl Acad Sci USA* 2009; 106:9673-9678.

Sodeik B (corresponding), Ebersold MW & Helenius A. Microtubule-mediated transport of incoming herpes simplex virus 1 capsids to the nucleus. *J Cell Biology* 1997; 136: 1007-1021.

Sodeik B, Doms RW, Ericsson M, Hiller G, Machamer CE, van't Hof W, van Meer G, Moss B, Griffiths G. Assembly of vaccinia virus: role of the intermediate compartment between the endoplasmic reticulum and the Golgi stacks. *J Cell Biology* 1993; 121: 521-541.

Research Interest in RESIST

Our research focus is on the cell biology of alphaherpesviruses. We analyse the interactions of viral proteins with host proteins and their relevance for pathogenesis in epithelial cells and fibroblasts of the skin, immune cells and neurons. For RESIST, we investigate capsid assembly and early tegumentation of alphaherpesviruses (area D2). In a phenotypic screen, we have identified several small chemical compounds that block assembly of herpes simplex virus (HSV) in the nucleus or the cytoplasm. We now characterize the viral targets of these compounds and evaluate their potential for the development of novel antiviral therapies. The most promising candidates will be validated in primary human keratinocytes and iPSC derived neurons, and in our recently developed ex vivo and in vivo murine skin HSV-1 infections models. Furthermore, we will support the characterization of the function of potential susceptibility host factors which might contribute to the development of severe HSV and VZV diseases (area A1), and which are identified by the RESIST AD cohort (Atopic dermatitis and disseminated HSV infections) and the Zoster cohort (Severe manifestations of Herpes Zoster).

For our projects, we use HSV mutants and state-of-the-art biochemical, life-cell imaging, and electron microscopy studies as well as murine infection models. We have generated fluorescently tagged herpes simplex virus (HSV) strains to chase the viral envelope, the tegument, and the capsids during cell entry, nuclear targeting of the incoming capsids, viral gene expression, virus assembly, egress and spread within the skin and the nervous system. We want to elucidate the molecular mechanisms how HSV overcomes intrinsic resistance factors and innate immunity to utilize the intracellular microtubule highways, the nuclear pores, and the intracellular membrane systems during cell entry as well as assembly.

Forschungsinteresse in RESIST

Unser Forschungsschwerpunkt liegt auf der Zellbiologie von Alphaherpesviren. Wir analysieren die Wechselwirkungen von viralen Proteinen mit Wirtsproteinen und deren Relevanz für die Pathogenese in Epithelzellen und Fibroblasten der Haut, in Immunzellen und in Nervenzellen. Für RESIST untersuchen wir die Assemblierung und die frühe Tegumentierung der viralen Kapside von Alphaherpesviren (Bereich D2). In einem phänotypischen Screen haben wir mehrere kleine chemische Verbindungen identifiziert, die den Zusammenbau von Herpes Simplex Virionen (HSV) im Zellkern oder im Zytoplasma blockieren. Wir charakterisieren nun die viralen Ziele dieser Verbindungen und untersuchen ihr Potenzial für die Entwicklung neuer antiviraler Therapien. Die vielversprechendsten Kandidaten werden wir in primären humanen Keratinozyten und iPSC-basierten Nervenzelle sowie in unseren kürzlich entwickelten murinen ex vivo und in vivo HSV-1-Infektionsmodellen validieren. Darüber hinaus werden wir die Charakterisierung der Funktion potenzieller Suszeptibilitätsfaktoren, die zur Entwicklung schwerer HSV- und VZV-Erkrankungen (Bereich A1) beitragen könnten, und die mittels der Kohorte RESIST AD (Atopische Dermatitis und disseminierte HSV-Infektionen) und der Kohorte Zoster (schwere Erscheinungsformen von Herpes Zoster) identifiziert werden, unterstützen.

Für unsere Projekte verwenden wir HSV-Mutanten und modernste biochemische, zellbiologische, und elektronenmikroskopische Untersuchungen sowie murine Infektionsmodelle. Wir haben fluoreszierend markierte Herpes Simplex Virus (HSV)-Stämme hergestellt, um die virale Hülle, das Tegument und die Kapside während des Zelleintritts, der Ansteuerung der Kernporen durch die eintretenden Kapside, der viralen Genexpression, der Virusassemblierung, des Virusaustritts und der Virusausbreitung in der Haut und des Nervensystems zu untersuchen. Wir wollen die molekularen Mechanismen aufklären, wie HSV intrinsische Resistenzfaktoren und doe angeborene Immunität überwindet, um die intrazellulären Mikrotubuli-Autobahnen, die Kernporen und die intrazellulären Membransysteme während des Zelleintritts und der Virusassemblierung zu nutzen.