

Hanschmann, Eva-Maria



General Information

Name: Hanschmann, Eva-Maria
Academic title: Dr. rer. nat.
Gender: Female
Institute address: Department of Neurology, Heinrich-Heine Universität, Life Science Center, Merowingerplatz 1a, 40225 Düsseldorf
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E-Mail address: Eva-Maria.Hanschmann@med.uni-duesseldorf.de
Current position/status: Principle Investigator

Academic Education and Qualification

1993-2002 BMV Schule in Essen, Gymnasium, degree: Abitur
11/2002-03/2007 Studies of biology, Ruhr-Universität Bochum, Diploma thesis: "The role of human Grx2", Karolinska Institutet, Medical Nobel Institute for Biochemistry, Stockholm, Sweden, supervision: Dr. C. Berndt, Prof. A. Holmgren
03/2009-09/2011 PhD student: „Trx family proteins in physiology and disease“, Philipps-Universität Marburg, Clinical Cytobiology and Cytopathology, supervision: PD Dr. C.H. Lillig, degree: magna cum laude (1.0)

Professional Career

05/2007-05/2008 Research assistant in an industry-financed project complementing ongoing clinical trials (ThyoGen), Karolinska Institutet, The Medical Nobel Institute for Biochemistry, Department of Medical Biochemistry and Biophysics, Stockholm, supervision: Prof. Dr. Arne Holmgren
06/2008-02/2009 Project student in an ongoing clinical trial in Tanzania/Zanzibar, „Oxidative stress in malaria: genetic susceptibilities to malaria infections“ Karolinska Sjukhuset, Department of Medicine, Stockholm, Sweden, supervision: Dr. J.P. Gil
11/2011-03/2016 Postdoctoral position: Redox regulation of cellular functions, Ernst-Moritz-Arndt Universität Greifswald, Universitätsmedizin, Department of Medical Biochemistry and Molecularbiology
08/2013-10/2013 Guest researcher at the Universidad de Buenos Aires in Argentina, Instituto de Investigaciones Cardiológicas (ININCA), Medical faculty, (funded by DAAD)
10/2015-11/2015 Guest researcher at the Trafford Medical Center, Brighton, UK (funded by FEBS)

since 05/2016

Postdoctoral position: Extracellular Thioredoxin proteins and regulatory thiolswitches (in neuroinflammation), Heinrich-Heine Universität, Department for Neurology, Life Science Center, Düsseldorf, Germany

Professional Awards and Funding

2007	Fellowship, The Medical Nobel Institute for Biochemistry, Medical Biochemistry and Biophysics
2013	Grant, Department für Experimentelle Therapie, Universitätsmedizin Greifswald
2015	Short-term fellowship, FEBS (Federation of European Biochemical Societies)
2016	Youth Travel Fund, FEBS (Federation of European Biochemical Societies)
2016	Initial funding for grant application preparation for second funding period, DFG Priority Program 1710
2017	Grant, DFG Priority Program 1710

Publications (5 most important publications)

2015	Mullen L., Hanschmann E.M. , Lillig C.H., Herzenberg L.A. and Ghezzi P. <i>Cysteine Oxidation Targets Peroxiredoxins 1 and 2 for Exosomal Release through a novel mechanism of redox-dependent secretion</i> . Mol Med. 21:98-108
2014	Salzano S.*, Checconi P.*, Hanschmann E.M. , Lillig C.H., Bowler L., Chan P., Vaudry D., Mullen L., Coppo L., Sacre S., Atkuri K.R., Sahaf B. Herzenberg L.A., Herzenberg L.A., Mengozzi M., Ghezzi P., <i>Glutathionylated Peroxiredoxin 2 is released in inflammation and act as an inflammatory danger signal</i> . Proc Natl Acad Sci USA. 111(33):12157-62
2013	Haunhorst P.*, Hanschmann E.M.* , Bräutigam L., Stehling O., Hoffmann B., Mühlhoff U., Lill R., Berndt C. and Lillig C.H. <i>Crucial function of vertebrate glutaredoxin 3 (PICOT) in iron homeostasis and hemoglobin maturation</i> . Mol Biol Cell, 24(12):1895-1903
2013	Hanschmann E.M. , Godoy J.R., Berndt C., Hudemann C. and Lillig C.H. <i>Thioredoxins, glutaredoxins, and peroxiredoxins - molecular mechanisms and health significance: from cofactors to antioxidants to redox signaling</i> . Antioxid Redox Signal., 19(13):1539-605
2010	Hanschmann E.M. , Lönn M.E., Schuette L.D., Funke M., Godoy J.R., Eitner S., Hudemann C. and Lillig C.H. <i>Both thioredoxin 2 and glutaredoxin 2 contribute to the reduction of the mitochondrial 2-Cys peroxiredoxin Prx3</i> . J. Biol. Chem. 285(52):40699-705

* contributed equally to this work