
CURRICULUM VITAE



Christiane Ott

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PROFESSIONAL CAREER/EDUCATION

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| since 2017 | Postdoc at the German Institute of Human Nutrition Potsdam-Rehbruecke, Department of Molecular Toxicology, Nuthetal, Germany |
| 2015 | 3-month research stay in Montevideo, Uruguay (Departamento de Bioquímica. Facultad de Medicina, Universidad de la República) |
| 2012 - 2016 | PhD student at the German Institute of Human Nutrition Potsdam-Rehbruecke, Department of Molecular Toxicology, Nuthetal, Germany (2014-2016) and at the Institute of Nutrition, Department of Nutritional Toxicology, Friedrich-Schiller-University Jena, Germany (2012-2014)

PhD-thesis: The interactions of the intracellular proteolytic systems during aging: Impact of protein nitration |
| 2010 - 2011 | Diploma student at the Institute of Nutrition, Department of Nutritional Toxicology, Friedrich-Schiller-University Jena, Germany

Diploma-thesis: Induction of Immunoproteasome by Advanced Glycation End Products: Impact of resveratrol |
| 2006 - 2011 | Diploma studies of Nutrition Science, Friedrich-Schiller-University of Jena, Germany |

MEMBERSHIPS

German Centre for Cardiovascular Research (since 2014)

German Nutrition Society (since 2007)

Society for Free Radical Research Europe (SFRR-E) (since 2015)

ACHIEVEMENTS

Young Investigator Award 2015 of the SFRR-E, Stuttgart, Germany

Landesgraduiertenstipendium der Friedrich-Schiller-University Jena, Germany
(2012-2014)

RESEARCH PROFILE

My research focus lies on the biological phenomenon of autophagy and aging in the cardiovascular system. In particular I am interested in the impact of modified proteins and autophagy on heart and cardiomyocyte function during aging. I work with *in vitro* and *in vivo* aged primary cells, cell lines as well as with purified human proteins to investigate processes of protein turnover in aging, protein oxidation and nitration. Currently I am investigating the impact of modified proteins and autophagy on heart function and cardiomyocyte contractility, using isolated primary cardiomyocytes from young (4mo) and old mice (25mo). Additionally, to mimic the effects of age-related protein aggregates on cardiomyocyte function we are using artificial lipofuscin. Since autophagy and cardiac function decline with age, we are aiming to investigate how declined autophagy and in consequence increased protein aggregates can impact cardiomyocyte contractility, to clarify molecular processes involved in impaired cardiomyocyte functionality and to develop novel therapeutic strategies, using nutritional interventions.