Curriculum Vitae

Agata Nowacka

Date of birth: 07.02.1996

Place of birth: Warsaw, Poland

Address: Staszica 20/2 Łomianki, Poland

E-mail: agata.m.nowacka@student.uw.edu.pl

Education

2017- University of Warsaw, Faculty of Biology, Poland
Biotechnology, MSc

2014-2017 University of Warsaw, Faculty of Biology, Poland
Biotechnology, BSc

2011-2014 LXIV St. I. Witkiewicz High School, Warsaw, Poland

Language

English (advanced, C2)

French (intermediate, B1)

Research Experience

2015- Laboratory of Molecular Basis of Behavior, Nencki Institute of Experimental Biology PAS, Warsaw, Poland
Supervised by Katarzyna Radwańska, PhD

06/2018-08/2018 Laboratory of Molecular Neurophysiology and Plasticity, Department of Biomedicine, Aarhus University, Denmark
Supervised by Professor Mai Marie Holm

07/2017-08/2017 Laboratory of Molecular Basis of Synaptic Plasticity, Centre of New Technologies, University of Warsaw, Poland
Supervised by Magdalena Dziembowska, PhD

Scholarships/awards

2018 Stephan W. Kuffler Research Scholarship

2018 Award for outstanding oral presentation „Activity-dependent trafficking of PSD-95 after LTP and LTD" at Neuronus IBRO Neuroscience Forum, Cracow, Poland

2017/2018 University of Warsaw Rector's scholarship for Academic Excellence

2016/2017 University of Warsaw Rector's scholarship for Academic Excellence

2015/2016 University of Warsaw Rector's scholarship for Academic Excellence

Conference Attendance

2018 Neurons in Action 3rd Nencki Symposium on the Brain
Poster presentation: Nowacka Agata, Borczyk Małgorzata, Radwańska Katarzyna „Activity-dependent trafficking of PSD-95 after LTP and LTD"

2018 Neuronus IBRO Neuroscience Forum, Cracow, Poland
Oral presentation: Nowacka Agata, Borczyk Małgorzata, Radwańska Katarzyna „Activity-dependent trafficking of PSD-95 after LTP and LTD" (awarded)

2017 Aspects of Neuroscience, Warsaw, Poland

2017 Neurons in Action 2nd Nencki Symposium on the Brain, Warsaw, Poland

2016 Aspects of Neuroscience, Warsaw, Poland
Poster presentation: Nowacka Agata, Borczyk Małgorzata, Radwańska Katarzyna „Degradation resistant PSD-95 enlarges dendritic spines"

2016 Neuronus IBRO Neuroscience Forum, Cracow, Poland

2015 Aspects of Neuroscience, Warsaw, Poland.
Poster presentation: Nowacka Agata, Łukasiewicz Kacper, Ziółkowska Magdalena, Cały Anna, Radwańska Kasia „The role of actin in ethanol-induced amnesia"

Publications

Łukasiewicz, K., Borczyk, M., Cysewski, D., Ziółkowska, M., Lipiński, M., Nowacka, A., Matuszek, Ż., Dziembowski, A. & Radwańska, K. αCAMKII dysfunction enhances ethanol-induced amnesia and redox-mediated depolymerization of hippocampal actin *(in submission)*

Szczałuba, K., Chmielewska, J., Sokołowska, O., Rydzanicz, M., Szymańska, K., Feleszko, W., Włodarski, P., Biernacka, A., Pienkowski, V.M., Walczak, A., Nowacka, A., Stawiński, P., Nowis, D., Dziembowska, M., Płoski, R. Neurodevelopmental phenotype caused by a de novo PTPN4 point mutation disrupting protein localization in neuronal dendritic spines *(in submission)*

Research Interest

The ability of the nervous system to learn and form new memories, hence adapt, is believed to be based on activity-dependent modifications of synaptic connections, a process known as synaptic plasticity. These are accompanied by their morphological alterations. It is not yet known what exact molecular mechanisms underlie these morphological changes. PSD-95, a

major scaffolding protein of the postsynaptic density (PSD) is known to be involved in the regulation of LTP (long-term potentiation) (Ehrlich & Malinow 2004) and LTD (long-term depression) (Sturgill et al. 2009), two major forms of synaptic plasticity. Activity-dependent trafficking of PSD-95 out of the dendritic spine in LTP is regulated by phosphorylation of serine 73 (S73) via αCaMKII (Steiner et al. 2008). My research is focused on delineating the role of PSD-95 and αCaMKII interaction in the context of molecular basis of memory formation and remodeling.

To this point using the model of organotypic hippocampal slice cultures from rats and immunofluorescence techniques I showed that after both chemically induced LTP and LTD PSD-95 is removed from the stratum radiatum of CA1 pyramidal neurons. With AAV approach and overexpression of mutated forms of PSD-95 I have shown that αCaMKII phosphorylation of serine 73 in PSD-95 is a common mechanism regulating postsynaptic scaffolding disassembly and PSD-95 removal after both LTP and LTD. Experiments I performed on dissociated hippocampal cultures have additionally shown that this interaction also regulates dendritic spine morphology thus, removal of PSD-95 might be a crucial step promoting activity-dependent changes of dendritic spine structure. Behavioral experiments have proven that overexpression of unphosphorylable PSD-95 S73A in mice results in impaired fear memory extinction. These results prove the crucial role of PSD-95 removal and its regulation by αCaMKII phosphorylation in memory remodeling.

The next step is to use the CLEM (correlative light electron microscopy) technique which combines confocal and electron microscopy to examine the changes that occur in dendritic spine morphology and ultrastructure during LTP and LTD. Combining this with an AAV approach will enable me to observe morphological and ultrastructural effects of αCaMKII-PSD-95 interaction in dendritic spines. Overall these studies contribute novel finding towards better understanding of molecular mechanisms of memory. Hopefully they will also bring us closer to finding solutions for disease related to impaired memory and memory remodeling such as memory deficits, posttraumatic stress disorder or addiction.