Curriculum Vitae

Name:	Ignácz Attila
Nationality:	Hungarian
Birth:	1993. 06. 22.
Address:	Budapest, 1032 Ágoston u. 8. V/27
E-mail:	attila.ign@gmail.com

Education:

Veres Péter High School, Budapest, 2012.

ELTE Faculty of Science BSc in Biology (2012-2016)

ELTE Faculty of Science MSc in Biology (2016-)

Language:

English intermediate (B2)

French intermediate (B2)

Research experience

Research institute:	ELTE Faculty of Science – Institute of Biology – MTA-ELTE NAP-B Neural Cell Biology Group
Group leader:	Dr. Katalin Schlett
Field of research:	neuronal cell biology

Student Conferences

ELTE Biology Scientific Students' Associations Conference ('BTDK' Budapest, 2016) – Neurobiology and behavioural physiology section: **II. place**

XXXIII. National Scientific Students' Associations Conference ('OTDK' Debrecen, 2017) – Biology section – Neurophysiology division: **I. place**

Oral presentations

1st Hungarian Neuroscience Doctoral Conference (1st HuNDoC, Budapest, 2016)

Second Elevator Speech Festival in Life Sciences (Budapest, 2016): **Special award**

Publication

Szíber Zsófia, Liliom Hanna, Carlos O. Oueslati Morales, Ignácz Attila, Rátkai Erika Anikó, Kornelia Ellwanger, Gisela Link, Szűcs Attila, Angelika Hausser és Schlett Katalin (2017) **Ras and Rab interactor 1 controls neuronal plasticity by coordinating dendritic filopodial motility and AMPA receptor turnover.** *Molecular Biology of the Cell*, 28(2), 285295. http://doi.org/10.1091/mbc.E16-07-0526

Posters

Ignácz Attila, Sven Beyes, Bencsik Norbert, Iris Ranz, Arian Badamdeh, Tárnok Krisztián, Angelika Hausser, és Schlett Katalin (2016) **Ras and Rab interactor 1 (RIN1) controls dendritic arborisation and filopodial motility in a protein kinase D-dependent manner.** *IBRO workshop Budapest*

Rátkai Anikó, Tárnok Krisztián, Ignácz Attila, Hernáth Ferenc, Szűcs Attila és Schlett Katalin (2016) **Protein kinase D regulates homeostatic plasticity in hippocampal cell cultures.** *IBRO workshop Budapest*

Szíber Zsófia, Ignácz Attila, Rátkai Anikó, Liliom Hanna, Kornelia Ellwanger, Carlos O. Oueslati Morales, Gisela Link, Szűcs Attila, Angelika Hausser és Schlett Katalin (2017) **RIN1 – a novel molecular element inhibiting post-traimatic stress disorder (PTSD)?** *Brain awareness week, Budapest*

Scientific awards and scholarships

Stephen W. Kuffler Research Scholarship (2017)

ELTE TTK Excellence of the Faculty Award (Budapest, 2017)

ELTE TTK Scientific Students' Associations Conference Award (Budapest, 2017)

University of Stuttgart, Institute of Cell Biology and Immunology – TKA-DAAD exchange program (Stuttgart, 2016, 2017)

Research interest

In the Neural Cell Biology Group I investigate the effects of RIN1 protein on synaptic plasticity. Ras and rab interactor 1 (RIN1) is a protein expressed mostly in the central nervous system. It is known that RIN1 has a role in fear dependent learning and memory extinction or modification. According to our preliminary data, RIN1 exerts its effects in the dendritic spines where it regulates actin dynamics through Abl kinases and AMPA receptor endocytosis through Rab5 GTP-ases. During our research we aim to better understand these molecular pathways.

During our work we prepare embryonic hippocampal cell cultures from RIN1 knock-out (*RIN1ko*) mice and transfect them with fluorescently labelled wild type and point mutant RIN1 constructs. We try to determine the effects of RIN1 on actin cytoskeleton recording time-lapse image series of living neurons expressing an actin labeling protein *actin-chromobody.* After bleaching the actin chromobody signal we measure the fluorescence recovery which originates from the actin cytoskeletal remodeling in the filopodia.

By quantitative confocal microscopy in transfected RIN1ko neurons we show that re-introducing RIN1 decreases the amount of GluA1 AMPA receptor (AMPAR) subunits within the plasma membrane regions of Shank2 immunopositive postsynaptic areas. In RIN1ko neurons, overexpression of wild type RIN1 exerts its effects on GluA1 endocytosis via Rab5 activation. As the decrease of the synaptic efficacy (LTD – *long term depression*) is accompanied by endocytosis of AMPA receptors we plan to investigate the effects of RIN1 on AMPA receptor endocytosis during chemically induced LTD.

Our results indicate that RIN1 destabilizes synapses, reduces synaptic efficacy and prevents the formation of new connections, highlighting RIN1 as a key regulator of extincting fear and aversive memories.