european journal of histochemistry
a journal of functional cytology

ISSN 1121-760X
volume 63/ supplement 2
2019

Proceedings of the

65th Congress of the GEI-Italian Society of Development and Cell Biology (GEI-SIBSC)

38th Congress of the Italian Society of Histochemistry (SII)

24-27 June 2019

Polo Villarey, Ancona
Università Politecnica delle Marche

Scientific Committee

GEI-SIBSC

Fiorenza De Bernardi - Oliana Carnevali
Giorgia Gioacchini - Francesca Maradonna
Ike Olivotto - Mario Pestarino

SII

Roberto Di Primio - Elisabetta Falcieri
Saverio Marchi - Monica Mattioli Belmonte
Monia Orciani - Carlo Pellicciari

Local organizing Committee

GEI-SIBSC

Adriana Canapa - Vincenzo Caputo Barucchi
Oliana Carnevali - Giorgia Gioacchini
Francesca Maradonna - Ike Olivotto

SII

Roberto Di Primio - Raffaella Lazzarini
Guendalina Lucarini - Saverio Marchi
Monica Mattioli Belmonte - Monia Orciani

Organizing Secretariat

Congressare - Promisegroup

Guest Editors

Oliana Carnevali - Roberto Di Primio

ejh

under the auspices of
the University of Pavia, Italy

Published by PAGEPress, Pavia, Italy

Editorial Office:
Via A. Cavagna Sangiuliani 5, 27100 Pavia, Italy
Phone: +39.0382.464340 - Fax: +39.0382.34872
E-mail: info@pagepress.org

Printed quarterly by:
Press Up s.r.l.
via E.Q. Visconti, 90, 00193 Roma, Italy
Tel. +39.0761.527351 – Fax +39.0761.527254.

Annual Subscriptions
Europe: Euro 250
All other Countries: Euro 300

Subscriptions, cancellations, business correspondence and any enquiries must be sent to PAGEPress Publications, Pavia, Italy.
Cancellations must be received before the end of September to take effect at the end of the same year.

Reg. Tribunale di Pavia n. 289/23.2.1984.

Recognized by the Ministero per i Beni e le Attività Culturali, Italy as a publication of high cultural value.

Associato all'USPI
Unione Stampa Periodica Italiana

european journal of histochemistry
a journal of functional cytology

ISSN 1121-760X
volume 63/ supplement 2
2019

ejh

under the auspices of
the University of Pavia, Italy

European Journal of Histochemistry
a journal of functional cytology

The *European Journal of Histochemistry* was founded in 1954 by Maffo Vialli and published till 1979 under the title of *Rivista di Istochimica Normale e Patologica*, from 1980 to 1990 as *Basic and Applied Histochemistry* and in 1991 as *European Journal of Basic and Applied Histochemistry*. It is now published under the auspices of the University of Pavia, Italy.
The *European Journal of Histochemistry* is the official organ of the Italian Society of Histochemistry and a member of the journal subcommittee of the International Federation of Societies for Histochemistry and Cytochemistry (IFSHC), and has been an influential cytology journal for over 60 years, publishing research articles on functional cytology and histology in animals and plants.

The Journal publishes Original Papers, Technical Reports, Reviews, Brief Reports, Letters to the Editor, Views and Comments, and Book Reviews concerning investigations by histochemical and immunohistochemical methods, and performed with the aid of light, super-resolution and electron microscopy, cytometry and imaging techniques; attention is also given to articles on newly developed or originally applied histochemical and microscopical techniques.

Coverage extends to:
- functional cell and tissue biology in animals and plants;
- cell differentiation and death;
- cell-cell interaction and molecular trafficking;
- biology of cell development and senescence;
- nerve and muscle cell biology;
- cellular basis of diseases.

2017 Impact factor: 2.217. ®JCR Clarivate Analytics

table of contents

Proceedings of the 65th Congress of the GEI-Italian Society of Development and Cell Biology (GEI-SIBSC) 38th Congress of the Italian Society of Histochemistry (SII)

european journal
of histochemistry
ISSN 1121-760X
volume 63/supplement 2
2019

EFFECTS MEDIATED BY 7 NICOTINIC RECEPTOR IN RAT SCHWANN CELLS: IMPLICATIONS IN PERIPHERAL NERVE REGENERATION

A. Matera[1], R. Piovesana[1], I.M. Salazar [1], M. Taggi[2], R. Canipari[2], C. Fabrizi [2], C. Dallanoce[3], A.M. Tata[1]

[1]*Dip. Biologia e Biotecnologie Charles Darwin, Sapienza, Università di Roma, Italy;* [2]*Dip. SAIMLAL, Sapienza, Università di Roma, Italy;* [3]*Dip. Scienze Farmaceutiche, Sezione di Chimica Farmaceutica, Pietro Pratesi, Università di Milano, Italy*

E-mail: adamaria.tata@uniroma1.it

Peripheral nerve fibers are able to regenerate. During nerve regeneration, Schwann cells (SCs) assume a phenotype known as *Repair Schwann Cells*, relevant for promoting an anti-inflammatory environment and axonal regeneration. SCs are cholinoceptive; in fact they express functional muscarinic cholinergic receptors favoring SCs differentiation towards myelinating phenotype[1-3]. Recently we have also characterized the expression of 7 nicotinic receptor. This receptor is faintly expressed in sciatic nerve fibers and in SCs *in vitro*. Its expression significantly increases both *in vivo* and *in vitro*, after nerve injury or in presence of Bradykinin (Bk), a neuropeptide known for its pro-inflammatory effects. In fact we observed that sciatic nerve dissected and maintained *in vitro* for 24 h both in absence and in presence of BK, showed a significant increase of 7 receptors. Moreover the selective activation of this receptor with (R)-ICH3 caused a modulation of uPA and MMPs responsible of the microenvironment modifications favoring Wallerian degeneration and promoting nerve regeneration. Similarly, the treatment of cultured SCs with BK appears to promote the *repair Schwann cells* phenotype, favoring the changing in cell morphology and up-regulating the expression of GFAP and c-jun. The activation of 7 receptor by selective agonist (R)-ICH3 after BK treatment, appears further promote this phenotype modulating inflammatory environment in terms of cytokines, growth factors and proteases production. These results suggest that 7 nicotinic receptor may be a cholinergic receptor expressed only by repair Schwann cells. Considering its anti-inflammatory role, 7 receptors may contribute to generate a microenvironment improving peripheral nerve regeneration.

References

1. Loreti S, et al. Neuron Glia Biol 2007; 3:269-79.
2. Uggenti C, et al. Dev Neurobiol 2014; 74:676-91.
3. Piovesana R, et al. J Cell Physiol 2018;233:5348-60.

PROTECTIVE AND REPARATIVE EFFECTS OF THE ANTI-FUNGAL DRUG FLUCONAZOLE ON SEROTONIN-INDUCED ALTERED NEURONAL DIFFERENTIATION: RESULTS ON MIDBRAIN MICROMASS CULTURES

E. Menegola[1], M. Battistoni[2], F. Metruccio[2], F. Di Renzo[1]

[1]*Department of Environmental Science and Policy; and* [2]*Department of Biomedical and Clinical Sciences, University of Milan, Italy.* [3]*ASST Fatebenefratelli Sacco, ICPS, Milano, Italy*

Multiple sclerosis (the second cause of neurological disability in European young adults) is an inflammatory autoimmune disease characterized by disruption of myelin ensheathing axons and axonal damage. Recent data showed the antifungal azole miconazole to induce prodifferentiating effect in oligodendrocyte precursors through the inhibition of retinoic acid (RA) catabolic enzymes. The aim is to evaluate the protective and reparative effects of azoles on serotonin (5HT)-induced alterations of neuronal differentiation, using the micromass test. Rat embryo (E13) midbrain cells cultured at high density normally move together and form distinct foci (micromasses, tridimensional aggregation of cell bodies) interconnected by bundles (aggregation of neuronal processes). Micromasses were incubated during the whole culture period with a known promoter of differentiation (RA), with a known differentiation inhibitor (5HT) or with azoles in clinical use (fluconazole, miconazole and itraconazole) alone or in mixture. 5HT inhibitory effects at 50-100 μM was confirmed. Among RA and the selected azoles, the most promising molecule in our model was fluconazole, tested at 5-100 μM. In order to test protective effects of fluconazole on 5HT inhibition, we co-exposed micromasses to both molecules during the whole culture period. Results show that co-exposed groups displayed parameters comparable to controls, suggesting a protective effect of fluconazole. A second set of experiments were devoted to the evaluation of a fluconazole-related reparative effect. Cultures were exposed during the first day to 5HT alone and during the remaining culture days to fluconazole alone. The one-day 5HT exposure affected development while after the post-exposure to fluconazole a reparative effect was evident. The data of the present work suggest both a protective and reparative effect of fluconazole in a micromass model of neurodegeneration, suggesting this drug as a good candidate for pharmacological repurposing.

CDK5 INVOLVEMENT IN ASCIDIAN NEUROGENESIS

S. Mercurio[1], S. Messinetti[1], M. Venturin[2], L. G. Folci[3], L. Manni[3], R. Pennati[1]

[1]*Department of Environmental Science and Policy; and* [2]*Department of Medical Biotechnology and Translational Medicine, University of Milan;* [3]*Department of Biology, University of Padua, Italy*

E-mail: sil.mercurio@gmail.com

Cyclin-dependent kinases (CDKs) are a family of serine-threonine kinases whose activity requires interaction with cyclins[1]. Although the majority of CDKs plays a key role in controlling cell division cycle, there is an important exception: Cyclin-dependent kinase 5 (CDK5). CDK5 gets activated by its neural specific activators, CDK5R1 and CDK5R2[2], and its proper activity is critical during vertebrate neurogenesis. During brain development, CDK5 complex is implicated in neural survival, migration as well as dendritic outgrowth and synapse formation[3]. Most research on CDK5 has focused on vertebrates, while only few studies have been performed in other animal groups[4]. In the genome of the ascidian *Ciona robusta*, homologs of CDK5 and its regulators are present and, based on our analysis, their expression patterns are comparable with those reported in vertebrates. We started exploring CDK5 involvement in neural development of *C. intestinalis*, specifically inhibiting CDK5 activity by drug treatments. Even if the overall morphology was not affected, the central nervous system of larvae exposed to low doses of the inhibitor showed specific malformations. Overall, our results suggested that CDK5 functions are highly conserved between ascidians and vertebrates, setting the stage for further research about its regulation during ascidian development.

References

1. Tarricone C, et al. Mol Cell 2001; 8:657-669.
2. Shah K, Lahiri DK. J Cell Sci 2014;127:2391-400.
3. Jessberger S, et al. Trends Neurosci 2009;32(11):575-82.
4. Zhao G.et al. Cell Stress Chaperones 2018;23 13-28.