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

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higher amount of associated cytokines, inducible nitric oxide synthase (iNOS) and vascular endothelial growth factor (VEGF)[2]. The purpose of our study was to evaluate the effects of Apremilast drug, an oral phosphodiesterase 4 inhibitor, on the expression of VEGF, iNOS and the tryptophan metabolism enzyme indoleamine 2,3-Dioxygenase (IDO)[3] by MSCs isolated from skin of healthy subjects and psoriatic- patients. MSCs from skin of control (C-MSC) and psoriatic (PsO-MSCs) subjects were isolated at baseline (T_0) and after 12 weeks of treatment (T_{12}) with Apremilast. MSCs were characterized according to the Dominici's criteria, and the expression of VEGF, iNOS and IDO was analized by immunocytochemistry and immunohistochemistry. Our results show that both PsO-MSCs and healthy MSCs attain the minimum criteria for MSCs definition; PsO-MSCs at T_0 express higher level of iNOS and VEGF than C- and Pso-MSCs at T_{12}, whereas IDO expression was lower. In conclusion, Apremilast may affect the physiopathological pathway of psoriasis; after 12 weeks of treatment, PsO-MSCs display properties nearer to the phisyological profile of C-MSCs. The drug is therefore able to drive psoriatic cells towards controls.

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IDENTIFICATION AND EXPRESSION STUDIES OF PUTATIVE STEM/PROGENITOR CELL MARKERS IN THE UROCHORDATE *Botryllus schlosseri*

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In the colonial ascidian *Botryllus schlosseri*, a cyclical generation change guarantees the recurrent (weekly at 20°C) renewal of the zooids. During the blastogenetic cycle (*i.e.*, the interval of time between a generation change and the following one), buds progressively grow to the adult size before replacing the old zooids. With the aim of better elucidating the process stem cell differentiation, with particular reference to the genesis of haemocytes during the of the colonial ascidian, we screened the *B. schlosseri* genome and transcriptome, looking for transcripts/genes showing similarity to vertebrate molecular markers of haematopoietic stem/progenitor cells. On these sequences, after an in silico translation, we performed the phylogenetic reconstruction that, always, returned us the tunicate relevant position, within the protochordates cluster, of vertebrate sister group. The four mammalian orthologous genes, used as markers for the recognition of haematopoietic stem/progenitor cells, identified in *B. schlosseri*, are *bsabcg2*, *bscd133*, *bsgata1/2/3* and *bsgata4/5/6*. The ISH assay, performed by antisense specific riboprobes, on haemocyte monolayers and colony sections, resulted in a labelling of the sub-endostylar haemolymph lacunae. This results matches previously morphological data that identified the endostyle as a stem cell niche, strengthening our idea to use *bsabcg2, bscd133, bsgata1/2/3 and bsgata4/5/6* genes for the identification of haematopoietic stem/progenitor cells in *B. schlosseri*. Quantitative real time PCR (qRT-PCR) highlighted the overexpression of the considered genes in the mid-cycle phase of the blastogenetic cycle. During this phase, there is the formation of new secondary buds emerging from the primary buds. The higher transcription levels of *bsabcg2*, *bscd133*, *bsgata1/2/3* and *bsgata4/5/6* in the mid-cycle phase reflect the presence of undifferentiated cells involved in proliferative and differentiation events required for the formation of the new blastogenetic generation.

FAS/FASL PATHWAY IN IMMUNO-ESCAPE AND PROMOTION OF CHONDROGENIC DIFFERENTIATION OF HUMAN DENTAL PULP STEM CELLS

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Human dental pulp stem cells (hDPSCs) represent a suitable source of stem cells due to high proliferation rate, pluri/multipotency and ability to modulate inflammatory processes through different pathways. As previously demonstrated, after direct coculture with PBMCs, hDPSCs induced apoptosis in $CD4^+$ and $CD8^+$ T-cells through Fas/FasL pathway. However, cell death did not occur in Fas bearing hDPSCs[1,2]. This study aimed 1) to evaluate how Fas and FasL are modulated following the activation of extrinsic apoptotic pathway and 2) to investigate the role of Fas and FasL in inducing chondrogenic differentiation. c-Kit^+/$STRO\text{-}1^+$ hDPSCs were co-cultured with activated PBMCs and exposed to different concentrations of human recombinant FasL (rc FasL) protein. The expression of extrinsic apoptosis-related markers (c-FLIP, FADD, caspase 8 and 3) and chondrogenic markers (Sox9, Collagen I and Collagen II) was evaluated. After rc FasL-stimulation hDPSCs displayed a higher proliferation rate and no expression of cleaved caspases 8 and 3. In addition, increased FasL and c-FLIP in hDPSCs were detected after stimulation. Conversely, the Fas expression was reduced after rc FasL treatment. These effects were partially reverted by the use of FasL-inhibitor. On the other hand, as long as chondrogenic induction occurred, hDPSCs exhibited an increase of both Fas and FasL at day 7, besides an increased expression of Sox9, which was even up-regulated after rc FasL stimulation. The chondrogenic commitment of hDPSCs was then confirmed by augmented expression of Collagen I and Collagen II. Our data indicate that hDPSCs are able to modulate the activation of extrinsic apoptotic pathways through c-FLIP and that Fas/FasL pathway plays a key role both in immuno-escape and in favoring chondrogenic induction.

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REGENERATIVE POTENTIAL OF HUMAN DENTAL PULP STEM CELLS IN AN ANIMAL MODEL OF STRESS URINARY INCONTINENCE

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Stress urinary incontinence (SUI), the most common type of urinary incontinence, is defined as an involuntary leakage of urine due to physical stress involving an increase in bladder pressure. It is associated with life quality issues, depressive symptoms and social discomfort. The pathophysiology consists in a damage of the external urethral sphincter affecting both muscle and nerve tissue components. The current conventional therapies are mainly represented by rehabilitating methods, pharmacological and/or surgical treatments. However, these therapies are not able to revert the primary cause of incontinence, in fact only symptoms can take relief by those treatments[1]. Regenerative medicine with