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M2 MUSCARINIC RECEPTOR ACTIVATION HELPS THE MAINTENANCE OF HUMAN SCHWANN-LIKE ADIPOSE-DERIVED STEM CELL PHENOTYPE: IMPLICATION IN PERIPHERAL NERVE REGENERATION

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Schwann cells (SCs) play a central role in the response to axon injury but there are several restrictions hindering their clinical application[1]. Adipose-derived stem cells (ASCs) present good properties for peripheral nerve regeneration and when exposed to specific growth factors *in vitro*, they can acquire a SC-like phenotype (dASCs)[2]. Unfortunately, ASC differentiation protocol is a constant chemical stimulation and after growth factor withdrawal, dASCs revert their morphology and gene expression towards ASC phenotype. M2 muscarinic receptors are potential pharmacological targets and are expressed in rat and human SCs[3,4] and dASCs[5,6], with roles in the regulation of cell growth, neurotrophic properties and differentiation. Here we present the role of M2 receptor in controlling human dASC differentiation. M2 stimulation, using the preferential agonist Arecaidine Propargyl Ester (APE), is able to decrease dASC cell growth, enhancing the differentiation phenotype. Moreover, in absence of growth factors but with M2 receptor selective stimulation, human dASCs do not revert towards undifferentiated ASCs but maintain a spindle-shaped morphology and SC-like marker expression. These data are the first evidence that human dASCs are cholinoceptive and M2 selective activation contributes to dASC terminal differentiation.

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BUTYRATE EFFECTS ON LIVER MITOCHONDRIAL COMPARTMENT IN INSULIN-RESISTANT OBESE MICE: AN ULTRASTRUCTURAL AND STEREOLOGICAL STUDY

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Fatty liver, mitochondrial dysfunction and oxidative stress represent pathophysiological features of insulin resistance (IR) and obesity. Butyrate, a short-chain fatty acid product in the large intestine by gut microbiota fermentation and its synthetic more palatable derivative, the N-(1-carbamoyl-2-phenyl-ethyl) butyramide (FBA) have been demonstrated to be protective against diet-induced insulin resistance and fatty liver[1]. Mitochondria were identified as the main target of the beneficial effect of both compounds[2]. We comparatively evaluated the effects of sodium butyrate and FBA on liver lipid content and mitochondrial compartment in a mice model of obesity and IR, by using transmission electron microscopy and the point-sampling technique of classic stereology to measure mitochondrial density and lipid volume density. Four experimental groups were considered: standard diet (STD)-fed, high-fat diet (HFD)-fed, HFD-fed treated with butyrate or FBA animals. In HFD-fed mice, the lipids are more abundant and larger than in STD-, butyrate- and FBA-treated mice; stereology investigations revealed that lipid density was significantly decreased in the butyrate and even more in FBA groups compared with HFD mice. In the liver of the HFD mice, mitochondrial dumbbell-shaped and fission pictures are evident, while fusion events are recognizable in butyrate-treated mice; giant and elongated mitochondria, resulting from fusion, are recognizable in the FBA group. Mitochondrial area and volume density were significantly lower in the HFD group compared with the other groups, probably associated to the increased lipid compartment. Our results confirm the association between HFD-induced hepatocellular lipids storage and alterations in the mitochondrial compartment[3], furthermore demonstrating a restoring activity of butyrate and FBA.

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GEBR-7B COUNTERACTS THE EPITHELIAL-TO-MESENCHYMAL TRANSITION BY MODULATING TRANSCRIPTION OF THE IGF2/H19 CLUSTER IN HCC CELL LINES

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The epithelial-to-mesenchymal transition (EMT) is associated with metastasis and chemoresistance in many types of cancers including hepatocellular carcinoma (HCC). In a few tumours, a connection between EMT activation and disruption of the cAMP pathway has been found. Moreover, overexpression of phosphodiesterase 4D (PDE4D) seems to be involved in this process. Our previous study[1] showed that PDE4D is overexpressed in HCC cell lines and tissues, and its depletion/inhibition reduced the growth of HCC cells by causing apoptosis and interfering with the expression of various cell cycle effectors and other pro-oncogenic genes, such as the insulin growth factor 2 (IGF2) gene. However, regulation of the PDE4D-IGF2 network and its role in the EMT remain to be explored. IGF2 is an imprinted gene whose transcription is regulated in a cluster with the H19 gene, which produces a non-coding RNA. After PDE4D silencing/inhibition we found a significant downregulation of IGF2 gene and protein expression in HCC, and in this tumour IGF2 protein expression positively correlated with cell proliferation, migration and invasion. Here, we also show that selective pharmacological inhibition of PDE4D, using Gebr-7b, induces cell-cell adhesion proteins, such as E-cadherin, and decreases mesenchymal markers, including Snail and Twist, in Western Blot experiments. Gebr-7b

treatment also significantly reduced HCC cell migration, as revealed by Incucyte® Scratch Wound Assays. In addition, Gebr-7b treatment induces deregulation of the IGF2/H19 cluster. It is conceivable that this PDE4D-dependent modulation of the IGF2/H19 cluster could be crucial in control of the EMT in HCC. These preliminary data suggest that targeting of PDE4D may reverse the EMT, thus preventing metastatic dissemination of HCC by acting on the IGF2/H19 cluster.

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CELL PROLIFERATION INCREASE INDUCED BY PROTEIN SYNTHESIS UPREGULATION ARRESTS NEURONAL *DIFFERENTIATION IN DROSOPHILA* MELANOGASTER NERVOUS SYSTEM

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The tissue homeostasis in the development and adult organs is fine maintained by the balance between cell proliferation and cell differentiation[1]. In *Drosophila melanogaster*, an aberrant overground of a specific tissue due to genetic mutations or diseases, such tumors, reduces the growth of others organs[2]. Our preliminary results report that the up-regulation of global translation in eye imaginal discs increases the proliferation of neuronal undifferentiated cells to reduce or set back the differentiation in photoreceptors, the cells organized in ommatidia which compose the adult eyes. By the *ey-gal4>uas* system, we up-regulated the ribosomal scaffold protein, RACK1[3], in eye imaginal discs and observed by immunofluorescence studies an increase of global protein synthesis and the phosphorylation of H3 histone, used as proliferation index. Moreover, the cell morphology, visualized by phalloidine-staining was also altered by the RACK1 up-regulation. These larval defects reduced the size of eye adult when compared to control animals. The up-regulation of RACK1 by *ppk-gal4>uas* method in C4da neurons localized in the peripheric nervous system reduced the dendritic arborization and the translation of specific mRNA, Mical, required for neuronal differentiation[4]. Thus, these results indicate that the modulation of global translation and the translation of specific mRNA hold the balance of power in cell proliferation and differentiation.

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EFFECTS OF FATTY ACID AMIDE HYDROLASE INHIBITION ON THE PROLIFERATION OF NEURAL STEM CULTURES DERIVED FROM THE MURINE DEVELOPING CORTEX

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Fatty acid amide hydrolase (FAAH) is an integral membrane serine hydrolase, highly expressed in the brain and upregulated in several neurological conditions[1]. FAAH catalyzes the degradation of acylethanolamides (NAEs), like palmitoylethanolamide (PEA), Oleoylethanolamide (OEA) and Anandamide (AEA), one of the most characterized endocannabinoids. In neurons, endocannabinoids inhibit neurotransmitter release from presynaptic elements. Furthermore, a functional endocannabinoid system is present in neural stem/progenitor cells (NSPCs) in the embryonic cortex[2], suggesting a role in neurogenesis. We studied the effects of FAAH inhibition on NSPC cultures derived from the murine cerebral cortex at embryonic day 13.5, when the peak in neurogenesis occurs[3]. We employed this *in vitro* system to dissect the mechanisms of FAAH function in NSPCs, which is difficult to do in the complex *in vivo* brain environment, using the previously characterized irreversible FAAH inhibitor PF3845. Four days after seeding with different doses of PF3845, the culture growth of NSPCs was significantly reduced compared to controls in a dose-dependent manner. A time-course of three days treatment showed a dose-dependent increase in trypan blue-positive cells in the treated cultures with a peak at 24h. The cell cycle analysis of cultures treated with PF3845 1 M by flow cytometry showed an increase of 55% in the sub-G1 fraction (apoptotic cell fraction) and an increase of 5% in G0/G1 fraction at the expense of S and G2/M fractions, as confirmed by Ki67 immunostaining. Gene expression analysis revealed an increase in GADD45, p21 and BAX and a decrease in cdk4 and ARPC5 consistently with the reduced growth phenotype.

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STUDY OF BONE DEVELOPMENT MODULATION BY TWO PROBIOTIC SPECIES USING SP7: GFP AND COL10A1A: COL10A1A- GFP ZEBRAFISH TG LINES

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Many probiotic bacterial species, including *Bacillus subtilis*[1] and *Lactococcus lactis*[2], are documented producers of various menaquinone (vitamin K2) forms. Menaquinones are considered to have an important role in bone health since vitamin K is the enzyme co-factor for catalysing the carboxylation of glutamate