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WORLD BIOCHEM 2019 & REGENERATIVE MEDICINE 2019

POSTERS

Daniella Feher et al., J Genet Mol Biol 2019, Volume 3

TISSUE ENGINEERED POLY (VINYL ALCOHOL) MESH FOR THE TREATMENT OF ABDOMINAL HERNIA

Daniella Feher and **Kristóf Molnár**
Semmelweis University, Hungary

Hernia is the weakness or defect in the abdominal wall or inguinal area. One of the solutions can be the usage of surgical mesh. To fend off the effects of intraperitoneal positioned non-degradable mesh our research group created absorbable scaffolds by electrospinning. For the biocompatibility experiments *In vitro* studies were performed on Human lung epithelial (A549) cell line and the *In vivo* evaluations were observed on Wistar rats (n=45, 200-250g). In this animal model to determine the biological behavior abdominal wall defect was performed than was covered with the nanofiber mesh. Adhesion formations were measured by a modified Diamond score. From the samples macroscopically and histological responses were graded. *In vitro* examination showed that the monomers of the nanofiber are biocompatible for the cells. According to the histological examinations all samples were integrated to the surrounding tissue and there were no foreign body reaction. Significantly more adhesion formation were found on the non-absorbable suture line (n=19) than were attached to the surface of the mesh.

The biocompatibility of the nanofiber surgical mesh was demonstrated by our studies. This nanofiber mesh could be a promising scaffold for the tissue engineering.

BIOGRAPHY

Daniella Feher is a PhD student from Semmelweis University, Hungary. Her research is about regenerative medicine tissue engineering and molecular biology which deals with the process of replacing, engineering or regenerating cells, tissues and nanofibers to restore and establish normal function.

daniella.feher@gmail.com

Note:

Veronica Hidalgo-Alvarez, J Genet Mol Biol 2019, Volume 3

DEVELOPMENT OF INTRAOCULAR DELIVERY SYSTEM FOR CONTROLLED RELEASE OF THERAPEUTIC AGENTS USED IN THE TREATMENT OF PCO

Veronica Hidalgo-Alvarez
University of East Anglia, UK

Cataract is the primary cause of blindness worldwide. Currently, the most effective treatment is surgery with implantation of an intraocular lens (IOL). Even though this procedure has proven effective in restoring vision, cataract shows a very high recurrence rate. This is due to the wound-healing response triggered by the lens epithelial cells that remain in the portion of the natural lens that is left after surgery. As a result, these cells undergo a transdifferentiation process, they encroach onto the posterior side of the IOL and deposit aberrant extracellular matrix proteins. Consequently, a secondary cataract forms. This pathology is also known as posterior capsule opacification (PCO). Due to the difficulties encountered when attempting to treat it, prevention of this disease is preferable. This can be achieved by formulating IOLs with biomaterials that can modulate cell adhesion, by including elements of design that control cell migration or by administering chemical agents that block the signalling pathways that lead to the development of PCO. Drug delivery systems using intraocular implants as platforms for their assembly have been developed to dispense drugs during surgery. While research on this area has shown promising results, there are still some issues that need to be overcome, such as the premature release of the drug. The present project aims to address these limitations by creating a hydrogel-based delivery system formulated to release a therapeutic agent for reduction of PCO only when this disorder starts to develop. This delivery system uses an IOL as a vehicle, thus making it minimally invasive for the patient. The drug that has been used in this study is an anti-VEGF molecule that has been shown to reduce PCO in recently published studies. The efficiency of this system has been evaluated through *in vitro* studies of release in a 2D cell culture system, and its effects on the cellular responses have been assessed.

BIOGRAPHY

Veronica Hidalgo-Alvarez is undertaking her PhD in Bioengineering at the University of East Anglia (UK) under the supervision of Aram Saeed and Michael wormstone. She graduated in 2013 with a MSc in Biotechnology from the University of Leon (Spain). After that, she undertook a placement at the Cancer Research Centre, affiliated with the University of Liverpool, where she worked on the detection of biomarkers for lung cancer. This was followed by a second placement at the Institute of Ageing and Chronic Disease, in the same University, working on the characterization of the limbal stem cell niche in the eye.

v.hidalgo-alvarez@uea.ac.uk

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ACCEPTED ABSTRACT

J Genet Mol Biol 2019, Volume 3

POTENTIAL USE OF DIFFERENT FORMS OF COLLAGEN FOR REGENERATIVE THERAPEUTICS

Toshihiko Hayashi
China-Japan Research Institute of Medical and Pharmaceutical Sciences, Japan

Understanding of collagen as environmental factor for cell fate is essential for construction of therapeutic strategy in regenerative medicine. Yet it is generally difficult to point out what is a direct signal to the cells that are in contact with collagen. The present study reports an interpretation for characteristic effects of collagen on the cells based on the findings of cell behavior *in vitro* with collagen in three different forms as to conformation; molecular, aggregates and unfolded. Differentiation of 3T3-L1 cells into myofibroblasts, is promoted with all the three forms of collagen, even though earlier signaling mechanisms are different or even opposite. However, migration or proliferation of 3T3-L1 cells is enhanced or repressed depending on the collagen forms; enhanced migration on molecular collagen, but repressed migration on fibrillar collagen. Cell migration is evaluated by cell appearance in the sites where cells did not exist. The total number of the cells do not change during the migration, suggesting that increased cells are not due to cell proliferation. Furthermore, the post-confluent cells that have been continued in cell culture after confluence on the dish coated with molecular collagen, showing no cell proliferative markers, have acquired higher migration activity. The post-confluent 3T3-L1 cells are found to have enhanced nuclear YAP expression that leads to elongated primary cilia, since inhibition of YAP does not cause primary cilia elongation. The finding that cell migration activity depends on primary cilia is confirmed by the experiment in which inhibition of primary cilia elongation by siRNA to the component(s) of primary cilia lowered cell migration activity. Migration enhancement is to our knowledge a novel function of primary cilia. In the case of PMA-treated U937 cells, which are often used as human macrophage-like cells, both molecular collagen and gelatin promote cell aggregation, whereas induction of autophagy is repressed and enhanced on molecular collagen and gelatin, respectively. Enhanced autophagy is correlated with phagocytosis activity, one of the most important functions of macrophage, suggesting forms of collagen, but not the gene product is important for macrophage activity. We have noticed that differential events including FAK, ROS production, levels of ROS scavengers, YAP nuclear translocation, and activation of NF-kappaB are observed prior to the total manifestations of cell behaviors such as differentiation, migration, proliferation etc. for the cells cultured on the different forms of collagen. Thus expression levels of collagen gene polypeptides alone do not provide us with the information of biological functions of collagen. Biological activity as well as structural and mechanical function of collagen is conformation-dependent. Different forms of collagen could be useful in planning effective strategy with potential therapeutics of regenerative medicine.

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CONSTRUCTION OF DISCRETE MODEL OF HUMAN PLURIPOTENCY IN PREDICTING LINEAGE-SPECIFIC OUTCOMES AND TARGETED KNOCKDOWNS OF ESSENTIAL GENES

Priyanka Narad
Amity University, India

A network consisting of 45 core genes was developed for the genes/proteins responsible for loss/gain of function in human pluripotent stem cells. The nodes were included on the basis of literature curation. The initial network topology was further refined by constructing an inferred Boolean model from timeseries RNA-seq expression data. The final Boolean network was obtained by integration of the initial topology and the inferred topology into a refined model termed as the integrated model. Expression levels were observed to be bi-modular for most of the genes involved in the mechanism of human pluripotency. Thus, single and combinatorial perturbations/knockdowns were executed using an insilico approach. The model perturbations were validated with literature studies. A number of outcomes are predicted using the knockdowns of the core pluripotency circuit and we are able to establish the minimum requirement for maintenance of pluripotency in human. The network model is able to predict lineage-specific outcomes and targeted knockdowns of essential genes involved in human pluripotency which are challenging to perform due to ethical constraints surrounding human embryonic stem cells.

Note:

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THE ROLE OF THE FLOW RATE ON CELL DIFFERENTIATION DURING SEEDING OF THE STEM CELLS WITHIN THE MATRIX

Zahra Eslami
Shahid Beheshti University of Medical Sciences, Iran

Cardiovascular disease (involving narrowed or blocked blood vessels which can lead to heart attack or stroke) is a widespread disease throughout the world and conventional surgical implantation procedures such as coronary artery or peripheral by-pass require autologous vessels or synthetic grafts with diameters lower than 5 mm. In this case, tissue-engineered vascular grafts are considered as an appropriate alternative which are typically fabricated by seeding the stem cells into a porous tubular scaffold. However, the method by which the stem cells are seeded within a 3D tubular scaffold can be a dramatically decisive parameter to achieve a fully functional vascular graft. Spatially-uniform cellular distribution throughout the thickness of the tubular tissue-engineered graft is particularly of great importance since it provides the required conditions for uniform regeneration of the tissue. Also, the stem cell viability is often challenging because the mechanical driving forces used for seeding step can cause shear-mediated membrane lysis or may result in triggering of apoptotic pathways. To address these problems, simultaneous application of centrifugal force and the pumping flow offers a promising seeding procedure. As a particular item, various flow rates during incorporation of the stem cells within tubular scaffolds play a key role on cell differentiation in terms of cell number, distribution, viability and phenotype. The change in the applied volume of the flow as well as the change in the corresponding frequency leads to alterations in shear stress on protoplasm of the stem cells, which in turn results in differentiation of these cells to myocytes. The mentioned changes in the volume and frequency of the flow are applied by changing the diameter of the lumen.

Note:

J Genet Mol Biol 2019, Volume 3

METFORMIN EXHIBITED ANTICANCER ACTIVITY BY LOWERING CELLULAR CHOLESTEROL CONTENT IN BREAST CANCER CELLS

Ankit Sharma
Central university of Rajasthan, India

Literature suggested that Metformin, a well-known anti-diabetic drug, showed anticancer activity in various cancer types. Few clinical studies documented the low serum cholesterol and low TAG level in Metformin treated patients. Literature also indicated an existence of positive association between high cholesterol and cancer. This study aimed to find out a molecular mechanism involved in Metformin-inhibited cell growth and metastasis in breast cancer cells. Tumor sample based clinical study found the higher expressions of cellular cholesterol regulatory genes (e.g. HMGCoR, LDLR) in malignant breast cancer tissues as compared to benign tissues. Our cell culture study found that treatment of breast cancer MDA-MB-231 cells with Metformin decreased cellular cholesterol level with concomitant inhibition of various genes (e.g. SREBF1 and LDLR) which maintain the homeostasis of cellular cholesterol. Cell culture based experimental study documented that Metformin inhibited cell proliferation, migration, and colony and spheroid formation of metastatic breast cancer MDA-MB-231 cells. As a mechanism it was identified by RT-PCR that Metformin treatment inhibited anti-apoptotic markers (Bcl2, BCLxl) and mesenchymal marker genes (Vimentin, N-cadherin) transcript levels with simultaneous enhancement of apoptotic markers (Caspase3, Bax) and epithelial marker genes E-cadherin and Keratin19, indicated an inhibitory effect of Metformin in proliferation and EMT of breast cancer cells. Less number of colony and spheroid formation has been observed in Metformin treated breast cancer cells. RT-PCR analysis also found that Metformin treatment inhibited stemness marker CD44 and BMI-1 in metastatic breast cancer MDA-MB-231 cells. Our TRAP assay data showed that Metformin treatment inhibits breast cancer induced osteolytic activity, which further inhibits the osteolytic bone metastasis. Moreover, Metformin-inhibited cancer cell proliferation and migration was reversed by the exogenous treatment of cholesterol. Similarly, cholesterol treatment reversed the Metformin-inhibited Bcl2, Vimentin, BMI-1 expression. Moreover, zymography data documented that cholesterol treatment upregulated Metformin-inhibited MMP activity. Further, results showed that cholesterol depletion by MBCD (Methyl B- Cyclo Dextrin), a cholesterol depleting agent inhibited proliferation, migration and stemness in breast cancer cells. This study suggests a new molecular mechanism where Metformin inhibits proliferation, EMT, stemness and osteolytic bone metastasis of breast cancer cells presumably by lowering cellular cholesterol level".

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