台灣再生醫學學會

2015年度台灣再生醫學學會學術研討會暨會員大會
2015 Annual Meeting of Formosa Association of Regenerative Medicine

論文摘要 & 大會手冊

2015 年 03 月 07 日

台大醫學院 103 講堂

台北市仁愛路一段一號

目　錄

Consent

2015 年度台灣再生醫學學會學術研討會暨會員大會
2015 Annual Meeting of Formosa Association of Regenerative Medicine

Scientific Program

Time	Topic	Speakers & Authors	Institute	Moderator
08:30	**Registration 報 到** **開幕致詞**			
08:50~09:50 Oral Presentation Competition (Ⅰ)				
S-1 08:50~09:00	Using Bone Marrow Stromal Cells Modulated by Baculovirus-mediated MicroRNAs Sponge to Repair Osteoporotic Bone Defects	李奎璋 胡育誠	國立清華大學化學工程系	**張至宏** **方旭偉**
S-2 09:00~09:10	Stabilization of Collagen Nanofibers with L-Lysine Improves the Ability of Carbodiimide Cross-Linked Amniotic Membranes to Preserve Limbal Epithelial Progenitor Cells	林姿妤 賴瑞陽*	長庚大學生化與生醫工程研究所	
S-3 09:10~09:20	The Study of Cell and Bacteria Adherent Behavior on Different Functional Groups Surfaces	曹竣堰[1] 葉姝昀[2] 陳賢燁[2] 張志豪[3]	國立臺灣大學醫學系[1] 國立臺灣大學化學工程學系[2] 國立臺灣大學醫學院骨科[3]	
S-4 09:20~09:30	Reduction of Cardiomyocyte Apoptosis & Oxidative Stress by Human Placenta-Derived Multipotent Cells (PDMCs) is Enhanced by ECM Protein-Integrin Interactions	彭凱彥[1,2] 劉莞宏[3] 李柟葶[4] 嚴孟祿[4] 顏伶汝[2]	國立中央大學生命科學系[1] 國家衛生研究院細胞及系統醫學所再生醫學研究組[2] 亞東紀念醫院心臟血管醫學中心[3] 國立臺灣大學附設醫院婦產部及臺灣大學醫學院醫學系[4]	
S-5 09:30~09:40	Effective Transfer of Naked Plasmid Into Stem Cells Grown on Silica Nanosheets	黃念齊 徐善慧*	國立臺灣大學高分子科學與工程學研究所	
S-6 09:40~09:50	Study of Bone Cell Adhesion on Degradable Scaffold by $CaSiO_3$/HA 3D Printing	歐祖翔[1] 張志豪[2] 陳泓志[2] 劉福興[1]	龍華科技大學機械工程學系[1] 國立臺灣大學醫學院骨科[2]	
09:50~10:20 Coffee Break				

Time	Topic	Speakers & Authors	Institute	Moderator
10:20~12:10 Invited Lectures				
I-1 10:20~10:50	抗發炎及免疫療法調控再生醫學之應用	余家利 教授	台大醫院風濕免疫科	**劉華昌**
I-2 10:50~11:20	Unraveling Autoimmune Diabetes by Using Genetically Modified Mouse Models: From Mechanism Dissection to Clinical Application	司徒惠康 教授	國防醫學院微生物及免疫學科暨研究所	**侯連圍 嚴孟祿**
I-3 11:20~11:45	Cell Sheets Derived from Adipose-Derived Stem Cells Enhanced Wound Healing and Reduced Tissue Fibrosis in a Murine Wound Model	鄭乃禎 醫師	台大醫院外科部	
I-4 11:45~12:10	Mesenchymal Stem Cells in Heart Failure- Therapeutic Potential and Mechanism of Action	李啟明 教授	台大醫院內科部	
12:10 會 員 大 會				
12:10-13:30 Lunch Break				
13:30~15:15 Invited Lectures				
I-5 13:30~14:00	MAP4K Kinases and DUSP Phosphatases in Inflammation and T Cell-Mediated Diseases	譚澤華 教授	國家衛生研究院免疫醫學研究中心	**何美泠 林泰元**
I-6 14:00~14:25	Stem Cells and Immnuobiology	許素菁 博士	國家衛生研究院感染症與疫苗研究所	
I-7 14:25~14:50	Immunomodulatory Properties of Human Fetal-Stage Mesenchymal Stem Cells (MSCs): Mechanisms and Therapeutic Applications	顏伶汝 教授	國家衛生研究院細胞及系統醫學研究所	
I-8 14:50~15:15	Implantation of Olfactory Ensheathing Cells Promotes Neuroplasticity in Murine Models of Stroke	徐偉成 教授	中國醫藥大學免疫學研究所	
15:15-15:40 Coffee Break				

Time	Topic	Speakers & Authors	Institute	Moderator
15:40~16:20 Oral Presentation				
O-1 15:40~15:50	Molecular weight of hyaluronan alters its of chondrogenic effect on adipose derived stem cells via activation of MEK/ERK axis	吳順成[1] 張瑞根[1,3] 翼景城[1] 林怡珊[1,2] 陳振偉[1] 王國照[3] 何美泠[1,2]	高雄醫學大學骨科學研究中心[1] 高雄醫學大學生理學科[2] 高雄醫學大學附設醫院骨科[3]	**孫瑞昇 王至弘**
O-2 15:50~16:00	G protein-coupled estrogen receptor-1 (GPER-1) positively regulates cell proliferation in murine bone marrow mesenchymal stem cells	莊淑君[1] 蕭伯倫[1] 何美泠[1,2] 張瑞根[1,3,4]	高雄醫學大學骨科學研究中心[1] 高雄醫學大學生理學科[2] 高雄醫學大學附設中和紀念醫院骨科部[3] 高雄市立大同醫院[4]	
O-3 16:00~16:10	Comparison of Platelet-rich Plasma or Hyaluronic Acid with/without Bone Marrow Mesenchymal Stem Cells for the Treatment of Knee Osteoarthritis in a Rabbit Model	陳郁君[1] 許元銘[1] 陳瑾霈[1] 張至宏[1,2]	亞東紀念醫院外科部骨科[1] 元智大學生物科技與工程研究所[2]	
O-4 16:10~16:20	Bone Marrow Mesenchymal Stem Cells Reduced LPS-Induced Inflammatory Response in Chondrocytes	陳郁君[1] 陳瑾霈[1] 張至宏[1,2]	亞東紀念醫院外科部骨科[1] 元智大學生物科技與工程研究所[2]	
16:20~17:00 Oral Presentation Competition (Ⅱ)				
S-7 16:20~16:30	The study of matrix stiffness of hyaluronan(HA) hydrogel for cytocompatibility of human adipose derived stem cells	張倩玫[1,2] 吳順成[2,3] 張栩榮[2] 林侑陞[2] 張瑞根[2,3] 何美泠[1,2]	高雄醫學大學生理學科[1] 高雄醫學大學骨科學研究中心[2] 高雄醫學大學附設醫院骨科[3]	**張至宏 方旭偉**
S-8 16:30~16:40	Enhanced Proliferation and Life Span of Human Fibroblasts by the Formation of Cell Spheroids on Chitosan	蔡靜雯[1] 高于婷[1] 姜宜妮[1,2] 王至弘[3]* 楊台鴻[1]*	臺灣大學醫學工程研究所[1] 臺大醫院泌尿科[2] 臺大醫院骨科[3]	
S-9 16:40~16:50	Effect of Riboflavin Concentration on the Development of Photo-Crosslinked Amniotic Membranes for Cultivation of Limbal Epithelial Cells	羅麗娟 賴瑞陽*	長庚大學生化與生醫工程研究所	
S-10 16:50~17:00	Study of Bone Regeneration on Bio-ceramic Scaffolds by ZrO_2/HA 3D Printing	嚴勻謙[1] 張志豪[2] 陳泓志[2] 劉福興[1] 李瑞宗[1]	龍華科技大學機械工程學系[1] 國立臺灣大學醫學院骨科[2]	
論文比賽頒獎				
Close Remark				

Invited Lectures

10:20-10:50

I-1

抗發炎及免疫療法調控再生醫學之應用

余家利 台大醫院風濕免疫科

It is conceivable that mesenchymal stem cells (MSCs) interact with and modulate different lymphocyte subpopulations. By the production of TGF-β, normal MSCs exert immune suppressive effects via increase regulatory T (Treg) cell generation to inhibit the functions of CD_4^+T, CD_8^+T, γδ T and NK cells. However, in chronic inflammatory diseases such as rheumatoid arthritis (RA), the synovial MSCs conversely exhibit immune stimulation in the rheumatoid joints. The excessive production of the major pro-inflammatory cytokines including TNF-α and IFN-γ by rheumatoid synovial tissues are demonstrated to reduce MSC proliferation and osteogenic gene expression. These may result in osteoporosis and bone mass reduction in RA patients. In clinical practice, a combination of medications has been frequently applied in the treatment of RA including anti-inflammatory and different immunosuppressive agents. Aspirin and newly developed non-steroid anti- inflammatory drugs (NSAIDs) work as inhibitors of cycle-oxygenase (COX) pathways. NSAIDs can effectively suppress the synthesis of prostaglandins, TNF-α and IFN-γ syntheses by T-cells and monocytes/macrophages. Subsequently, the applications of ASA or NSAIDs in RA patients decrease apoptosis of MSCc. Glucocorticoids, another sort of potent anti-inflammatory (in small dose) as well as immune suppressive (in moderate and large doses) agents, have been confirmed in suppressing the functions of MSCs. As a basic therapeutic regimen for RA patients, disease-modifying anti-rheumatic drugs (DMARDs) belong to the immuno-suppressive agents that can impair the diverse functions of T cells, B cells, or monocytes/macrophages. Among these, cyclosporine, FK506 and other calcineurin inhibitors can decrease proliferation of embryonic stem cells. Accordingly, corticosteroids and DMARDs may prevent tissue healing by mesenchymal stem cells in chronic rheumatoid inflammation. These findings are consistent with the observations that joint destruction continues despite absence of inflammation in the patients with RA under DMARDs treatment. Conversely, TNF-α inhibitor including soluble TNF-α receptors (etanercept) and different kinds of monoclonal anti-TNF-α antibodies not only target inflammation but prevent MSC apoptosis and facilitate bone formation. In conclusion, it is quite important to find agents potentially impairing wound healing for avoiding these drugs in the field of regenerative medicine. However, the balance between inflammation-induced tissue destruction and repairment by stem cells in the treatment of chronic inflammatory diseases should be taken into consideration.

10:50-11:20

I-2

Unraveling Autoimmune Diabetes by Using Genetically Modified Mouse Models: From Mechanism Dissection to Clinical Application

Huey-Kang Sytwu MD, PhD
Department of Microbiology and Immunology
National Defense Medical Center
Taipei, Taiwan, ROC

Insulin-dependent diabetes mellitus (IDDM) is a T cell-mediated autoimmune disease. To delineate the protective roles of some immune modulatory molecules, such as soluble decoy receptor 3 (DcR3), cytotoxic T lymphocyte antigen 4 (CTLA4), program death ligand 1 and 2 (PD-L1 and 2), heme oxygenase 1 (HO-1), and chemokine receptor D6 in the autoimmune process and to search for potential preventive and/or therapeutic targets in this disease, we generated (a) insulin promoter (pIns)-sDcR3 transgenic non-obese diabetic (NOD) mice, (b) pIns-single chain anti-CTLA4 transgenic NOD mice, (c) pIns-single chain anti-4-1BB transgenic NOD mice, (d) pIns-PD-L1 transgenic NOD mice, (e) pIns-HO-1 transgenic NOD mice, and (f) pIns-D6 transgenic NOD mice. Making full use of these unique mouse strains, we are quantitatively and qualitatively investigating the immunopathogenic mechanisms of autoimmune diabetes and providing valuable information for the development of novel immunotherapies.

11:20-11:45

I-3

Cell Sheets Derived from Adipose-Derived Stem Cells Enhanced Wound Healing and Reduced Tissue Fibrosis in a Murine Wound Model

Nai-Chen Cheng*, Jiashing Yu**
* Department of Surgery, National Taiwan University Hospital and College of Medicine, Taipei, Taiwan 100
** Department of Chemical Engineering, College of Engineering, National Taiwan University, Taipei, Taiwan 100

The abundance and easy accessibility of adipose-derived stem cell (ASC) have made it a promising candidate for stem cell therapies. However, transplantation of dissociated ASCs is frequently associated with early cell death with limited therapeutic effects. It has been proposed that the use of cell sheets is beneficial for cell transplantation, so we aimed to explore the regenerative capabilities of ASC sheets for cutaneous wound healing.

We stimulated extracellular matrix (ECM) secretion of ASCs and fabricated cell sheets by treatment with a stable form of ascorbic acid, ascorbate 2-phosphate (A2-P). Interestingly, we found enhanced expression of stemness markers (Nanog, Oct4 and Sox2) in ASCs within cell sheets. We further demonstrated that ASCs within cell sheets exhibited significantly enhanced neurogenic (ectoderm) and hepatogenic (endoderm) transdifferentiation capabilities comparing to monolayer ASCs, demonstrated by upregulated *Nestin* and *Albumin* genes when cultured in appropriate induction media. In the mean time, adipogenic and osteogenic (mesoderm) differentiation capacities of ASCs were still maintained after cell sheet formation. Our findings suggested that ascorbic acid enhances stemness and differentiation capabilities of ASCs through ECM synthesis, thus creating an *in vitro* ASC niche.

Using a murine model of healing-impaired full-thickness cutaneous wound, faster wound healing was noted in the group that received ASC sheet treatment, and we observed significantly less tissue fibrosis with more engrafted ASCs. Immunohistochemical staining of the wound sections further revealed less macrophage infiltration in the wounds treated with ASC sheets, suggesting a possible role of immunomodulation in the ASC sheet-mediated healing of cutaneous wounds. Therefore, A2-P-induced ASC sheet formation represents a promising approach of cutaneous wound healing resulting in less tissue fibrosis.

11:45-12:10

I-4

Mesenchymal Stem Cells in Heart Failure- Therapeutic Potential and Mechanism of Action

Chii-Ming Lee, M.D., Ph.D.,
Cardiology Division, Department of Medicine,
National Taiwan University College of Medicine

Heart failure (HF) is the final common pathway of multiple cardiovascular diseases and is an emerging burden on both medical and financial demands. Heart transplantation, a standard treatment for end-stage HF, is restrained not only by the availability of donor heart but also by transplant arteriosclerosis (TA), an immune-mediated vasculopathy that often results in cardiac allograft failure[1] [2] [3]. Recently, stem cell transfer has been actively investigated as the potential therapeutic modality for HF.

In the past five years, we focused on translational studies of bone marrow (BM)-derived mesenchymal stem cells (MSC) in porcine models of cardiovascular diseases and on the mechanisms underlying MSC-based cell therapy. We first demonstrated that, in a porcine model of femoral artery transplantation, MSCs prevent TA by enhancing local expression of interleukin (IL)-10, interferon (IFN)-γ, and indoleamine 2,3-dioxygenase (IDO), but not Foxp3. These results suggest that MSCs induce immune tolerance by activating the type 1 regulatory T (TR1)-like cells [4]. We further proved that, in a mixed lymphocyte reaction (MLR) system, MSCs-induced IL-10^{+}IFN-$\gamma^{+}$$CD4^{+}$ cells, which conferred resistance to allogeneic-antigen stimulated proliferation in an IL-10 dependent manner, resemble TR1-like cells. Prostaglandin E2 (PGE2) secreted by MSCs contributed to the induction of TR1-like cells. However, TR1-like cells were deficient in PGE2 and less potent than MSCs in suppressing MLR. PGE2 mimetic supplements can enhance the immune-suppressive potency of TR1-like cells. In a porcine model of allogeneic femoral artery transplantation, MSC-induced TR1-like cells combined with PGE2, but not either alone, significantly reduced TA. These findings indicate that PGE2 helps MSC-induced IL-10+IFN-γ+CD4+ TR1-like cells inhibit TA. PGE2 combined with MSC-induced TR1-like cells represent a new approach for achieving immune tolerance [5].

More recently, we demonstrated that, in a porcine model of myocardial infarction (MI), a CXCR4 antagonist TG-0054 mobilized CD271+ MSCs from BM into peripheral blood (PB), and prevented left ventricular (LV) dysfunction post-MI. The preserved cardiac function was accompanied by a significant decrease in the expression of TNF-α, IL-1β, and IL-6 in both myocardium and PB. We further documented that infusion of TG-0054-mobilized-CD271-MSCs reduced both myocardial and plasma cytokine levels in a pattern temporally correlated with TG-0054 treatment. Our findings indicate that TG-0054 preserves LV contractility following MI, at least in part, by mobilizing $CD271^{+}$ MSCs to attenuate the post-infarction inflammation.

In conclusion, MSCs, at least in part, through immunomodulation effectively prevent both post-MI LV dysfunction and TA. These findings in animal models will be translated into

clinical studies as the potential solutions for our unmet requirement in the treatment of HF.

1. Wu, Y.W., et al., PET assessment of myocardial perfusion reserve inversely correlates with intravascular ultrasound findings in angiographically normal cardiac transplant recipients. J Nucl Med, 2010. **51**(6): p. 906-12.
2. Lee, C.M., et al., Intravascular ultrasound correlates with coronary flow reserve and predicts the survival in angiographically normal cardiac transplant recipients. Cardiology, 2008. **109**(2): p. 93-8.
3. Lee, C.-M., et al., Intravascular ultrasound evidenc of angiograpfhically silent allograft vasculopathy inversely correlates with circulating level of hepatocyte growth factor. J Heart Lung Transplant, 2006. **25(12):1456-1461**.
4. Jui, H.Y., et al., Autologous Mesenchymal Stem Cells Prevent Transplant Arteriosclerosis by Enhancing Local Expression of Interleukin-10, Interferon-gamma, and Indoleamine 2,3-dioxygenase. Cell Transplant, 2012.
5. Hsu, W.T., et al., Prostaglandin E2 Potentiates Mesenchymal Stem Cell-Induced IL-10+IFN-gamma+CD4+ Regulatory T Cells To Control Transplant Arteriosclerosis. J Immunol, 2013.

13:30-14:00

I-5

MAP4K Kinases and DUSP Phosphatases in Inflammation and T cell-mediated Diseases

Tse-Hua Tan
Immunology Research Center, National Health Research Institutes,

The c-Jun N-terminal kinases (JNKs) belong to the mitogen-activated protein kinase (MAPK) superfamily. JNKs play crucial roles in cell proliferation, differentiation, stress responses, and apoptosis. JNK kinase activity can be activated by diverse stimuli, including growth factors, cytokines, environmental stresses, and apoptotic stimuli. MAPKs are regulated by various MAP kinase phosphatases (MKPs), which belong to a subfamily of dual-specificity phosphatases (DUSPs). We discovered that DUSP22/JKAP and DUSP14/MKP6 are negative regulators of T cell signaling by inhibiting Lck tyrosine kinase and TAB1/TAK1 kinase complex, respectively. DUSP22 knockout mice spontaneously develop systemic inflammation and autoimmunity.

MAP kinase kinase kinase kinases (MAP4Ks) are a subfamily of mammalian Ste20-like serine/threonine protein kinases that activate the JNK-MAPK kinase cascade. We have cloned and characterized the roles of three MAP4Ks, namely HPK1 (MAP4K1), GLK (MAP4K3) and HGK (MAP4K4) in T-cell signaling pathways and immune regulation. HPK1 (MAP4K1) is a negative regulator of T-cell signaling by phosphorylating the T-cell adaptor SLP-76, leading to SLP-76 degradation and attenuation of T-cell signaling. We also discovered that conditional knockout of HGK (MAP4K4) in T cells results in TRAF2 upregulation and subsequent induction of inflammatory IL-6/IL-17-producing T cells in adipose tissue, leading to type 2 diabetes and systemic inflammation. This reveals a novel pathogenesis mechanism of T helper 17 (Th17) cell-mediated type 2 diabetes.

GLK (MAP4K3) activates PKC-θ/IKK/NF-κB during TCR signaling. Autoimmune systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) patients show significantly increased GLK levels and PKC-θ/IKK/NF-κB activation in inflammatory Th17 cells, and the percentage of GLK-overexpressing T cells is correlated with autoimmune disease severity. We propose that GLK (MAP4K3) is a novel diagnostic biomarker and therapeutic target for various pro-inflammatory IL-17-mediated diseases.

1. Chuang HC et al. The kinase GLK controls autoimmunity and NF-κB signaling by activating the kinase PKC-θ in T cells. *Nature Immunology* (2011) 12:1113-1118.
2. Shui JW et al. HPK1 negatively regulates T cell receptor signaling and T cell-mediated immune responses. *Nature Immunology* (2007) 8:84-91.
3. Chuang HC et al. HGK/MAP4K4 deficiency induces TRAF2 stabilization and Th17 differentiation leading to insulin resistance. *Nature Communications* (2014) 5: 4602.
4. Li JP et al. The Phosphatase JKAP/DUSP22 inhibits T-cell receptor signaling and autoimmunity by inactivating Lck. *Nature Communications* (2014) 5:3618.
5. Yang CY et al. Dual-specificity phosphatase 14 negatively regulates T cell receptor signaling by inhibiting TAK1 activation. *Journal of Immunology* (2014) 192:1547-1557.

14:00-14:25

I-6

Stem Cells and Immnuobiology

Dr. Shu-Ching Hsu 許素菁博士
國家衛生研究院感染症與疫苗研究所

Abstract:

The reconstitution of blood/immune cells by hematopoietic stem cells (HSCs) is a potentially curative treatment for patients with various human malignant and non-malignant diseases, therefore, mechanisms underlying the efficacy of hematopoiesis and the interaction of HSCs and other cells within bone marrow niche remain poorly understood. In addition, the polymorphonuclear neutrophilic granulocytes, constitute the most abundant population of white blood cells, are the primary player at the front line of host defense against intruding microorganisms. However, the analysis of neutrophil generation and their gene function is complicated in a physiologically relevant situation because neutrophils are notoriously short-lived cells, thus acts for a significant problem in the field of studying neutophil biology and the setting strategy for microbial infection. In our preliminary studies, we have demonstrated that BM-MSCs regulate the functional activation of neutrophils via their role in modulating IL-17 from $CD4^+$ $CD45RO^+$ memory T cells. Besides, we have also developed the *ex vivo* HSCs-derived neutrophils platform to conduct the roles of MSCs in regulating neutrophils generation. Molecular mechanisms underlying the effects of T lymphocytes on hematopoietic and mesenchymal stem cells within bone marrow niche should be studied for further clinical treatment of inflammatory diseases and bone marrow transplantation.

14:25-14:50

I-7

Immunomodulatory Properties of Human Fetal-Stage Mesenchymal Stem Cells (MSCs): Mechanisms and Therapeutic Applications

B. Linju Yen, MD
Regenerative Medicine Research Group,
Institute of Cellular & System Medicine,
National Health Research Institutes (NHRI)
Zhunan, Taiwan

Abstract :

Despite the isolation of human embryonic stem cells (hESCs) and the more recently discovered induced pluripotent stem cells (iPSCs), many critical issues still surround these cells in terms of prevalent clinical use, the most important likely being the ethical concerns of hESC derivation, and tumorigenic potential of both these pluripotent stem cells. Increasing reports of plasticity for many adult stem cells (ASCs) have brought excitement and hope for broad therapeutic application, but these are rare cells and controversy still exists regarding ASC transdifferentiation capacity, especially to the extent of being clinically efficacious. Thus, the search continues for ethically conducive, easily accessible, and high-yielding source of stem cells. We have isolated and studied the immunobiology of novel sources of fetal-stage mesenchymal stem cells (MSCs), including placenta-derived multipotent cells (PDMCs) and hESC-derived mesenchymal progenitors. Fetal extraembryonic tissues are developmentally and immunologically more naïve than adult tissue, and often are discarded after the birth of the neonate, making this source ideal for isolation of progenitor cells for therapeutic use. Highly proliferative compared with adult BMMSCs, fetal-stage MSCs possess multilineage differentiation capacity, and are strongly immunomodulatory towards allogeneic leukocytes. Mechanistically, suppression of leukocyte reactivity by fetal-stage MSCs is largely due to secreted factors and can be surprisingly enhanced with interferon-**γ**, a proinflammatory cytokine. We have recently found that the high levels of hepatocyte growth factor (HGF), a non-immunological factor, secreted by these MSCs mediates immunosuppressive effects via expansion of myeloid-derived suppressor cells—a population of immunomodulatory leukocytes highly associated with human tumors—and modulation of CD14+ monocytes through divergent downstream pathways. Moreover, in recent translational studies, we demonstrate that application of fetal-stage MSCs can be effective treatments in diseases with ischemic and inflammatory components. With such broad immunosuppressive properties and multilineage differentiation capacity, fetal-stage MSCs may represent a potential cell source for therapeutic use.

14:50-15:15

I-8

Implantation of Olfactory Ensheathing Cells Promotes Neuroplasticity in Murine Models of Stroke

徐偉成
中國醫藥大學免疫學研究所

Abstract :

Murine olfactory ensheathing cells (OECs) promote central nervous system axonal regeneration in models of spinal cord injury. We investigated whether OECs could induce a neuroplastic effect to improve the neurological dysfunction caused by hypoxic/ischemic stress. In this study, human OECs/olfactory nerve fibroblasts (hOECs/ONFs) specifically secreted trophic factors including stromal cell–derived factor–1α (SDF-1α). Rats with intracerebral hOEC/ONF implantation showed more improvement on behavioral measures of neurological deficit following stroke than control rats. [18F]fluoro-2-deoxyglucose PET (FDG-PET) showed increased glucose metabolic activity in the hOEC/ONF-treated group compared with controls. In mice, transplanted hOECs/ONFs and endogenous homing stem cells including intrinsic neural progenitor cells and bone marrow stem cells colocalized with specific neural and vascular markers, indicating stem cell fusion. Both hOECs/ONFs and endogenous homing stem cells enhanced neuroplasticity in the rat and mouse ischemic brain. Upregulation of SDF-1α and CXCR4 in hOECs/ONFs promoted neurite outgrowth of cocultured primary cortical neurons under oxygen glucose deprivation conditions and in stroke animals through upregulation of cellular prion protein (PrPC) expression. Therefore, the upregulation of SDF-1α and the enhancement of CXCR4 and PrPC interaction induced by hOEC/ONF implantation mediated neuroplastic signals in response to hypoxia and ischemia.

Oral Presentations

15:40-15:50

O-1

Molecular Weight of Hyaluronan Alters its Of Chondrogenic Effect on Adipose Derived Stem Cells Via Activation of MEK/ERK Axis

吳順成[1] 張瑞根[1,3] 翼景城[1] 林怡珊[1,2] 陳振偉[1] 王國照[3] 何美泠[1,2]
高雄醫學大學骨科學研究中心[1] 高雄醫學大學生理學科[2] 高雄醫學大學附設醫院骨科[3]

Introduction: Due to the important role of hyaluronan(HA) in cartilage development and function, HA-based scaffolds are of interest for stem cells based articular cartilage tissue engineering. Cell-niche interaction may used for promoting chondrogenic differentiation of stem cells, and our previous finding indicates that hyaluronan(HA)-microenvironment initiates chondrogenesis of adipose derived stem cells (ADSCs). HA alters its biological effect through change its molecular weight (MW), however its molecular mechanism on chondrogenesis of ADSCs is unclear. This study was to search the possible molecular mechanism of MW of HA on chondrogenesis of ADSCs.
Materials and Methods: ADSCs were isolated from the adipose tissues of New Zealand white rabbit and human patients. The multilineage potential of the ADSCs was characterized. To investigate the chondrogenic effects of MW of HAs on ADSCs, the solutions of HA (The MWs of HAs are 80kDa, 600 kDa, and 2000 kDa) were coated on culture well, and ADSCs were seeded on the HA-coated wells. The cell aggregations of ADSCs were observed. The mRNA of expression of chondrogenic genes (collagen type II and aggrecan) was detected by real-time PCR. Sulfated glycosaminoglycan (sGAG) synthesis of ADSCs was determined by Alcian blue staining. The protein level of P-ERK, PI3K and phosphorylation of SOX-9 were determined by western blot analysis. For in vivo study, ADSCs mixed with HAs with different MWs were encapsulated in fibrin hydrogel and then implanted into the articular cavity of knee of rabbits.
Results: The ADSCs can be induced into multilineages. Cell aggregations, chondrogenic genes expression and sGAG synthesis were induced by HA with different MW. The in vivo study showed that the neo-cartilage formation of HA treated ADSCs increased along with the increase of MW of HA. The chondrogenic effect of HA on ADSCs increased along with the increase the MW of HA, and the 2000 kDa HA treated ADSCs showed the optimal effect on promoting chondrogenesis both in vitro and in vivo. The protein levels of P-ERK, and phosphorylation of SOX-9 of ADSCs also increased along with the increase of MW of HA, while that of PI3K were not changed.
Discussion: The chondrogenic effect of HA on ADSCs increased along with the increase the MW of HA. The protein levels of P-ERK, and phosphorylation of SOX-9 of ADSCs also increased along with the increase of MW of HA.
Conclusions: Our data suggest that M.W. of HA promotes chondrogenesis of ADSCs via activation of MEK/ERK axis thus phosporylation of SOX-9. This may be an important molecular mechanism of MW of HA on directing chondrogenic differentiation of ADSCs.

15:50-16:00

O-2

G-protein Couple Estrogen Receptor-1 (GPER-1)正向調控小鼠骨髓間葉幹細胞之增殖
G protein-coupled Estrogen Receptor-1 (GPER-1) Positively Regulates Cell Proliferation in Murine Bone Marrow Mesenchymal Stem Cells

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高雄醫學大學附設中和紀念醫院骨科部[3] 高雄市立大同醫院[4]

Introduction：Estrogens and estrogen receptors play important roles in physiological metabolism. Traditionally, the estrogen receptors are ascribed to two nuclear estrogen receptors (ERs), ERα and ERβ. Moreover, G protein-coupled estrogen receptor-1(GPER-1) was reported as a novel membrane receptor for estrogen in recent years. A report indicated that GPER-1-deficient male mice show increased femur size, BMD, trabecularization and cortical bone thickness. However, the detail molecular mechanism of osteogenesis regulated by GPER-1 was unclear. In our previous study, we have demonstrated GPER-1 negatively regulates osteogenic differentiation in murine bone marrow mesenchymal stem cells (BMSCs). In this study, we further study whether GPER-1 contributes to cell proliferation in BMSCs. The murine bone marrow mesenchymal stem cells (D1 cells) are used to examine the function of GPER-1 on cell proliferation.
Materials and Methods：D1 cells were cultured to 20% confluence in bone medium to evaluate cell proliferation. GPER-1 agonist, G1, and antagonist, G15, were used in the bone medium to enhance or block the function of GPER-1. The siRNA GPER-1 was used to block the gene expression of GPER-1. The cell proliferation marker genes, cyclin D1, D2, E1 and E2, were measured by real-time PCR. Cell proliferation was tested by MTT assay and BrdU proliferation assay. Cell cycle stage was evaluated by flow cytometry. Significant differences were tested by using ANOVA. The mean of different treatment groups was tested using Duncan's new multiple-range test. A p value < 0.05 was taken as significant.
Results：The GPER-1 gene expression was increased from 20% confluence (day 0) into confluence (day 3). The cell cycle gene expression of cyclin D1 and cyclin E1 were enhanced by agonist (G1) treatments, and inhibited by antagonist (G15) treatments ($p<0.05$ compared to the control group). The MTT assay and BrdU assay in D1 cells was enhanced in a time and dose dependent manner by G1 treatments ($p<0.05$ compared to the control group), but was inhibited by G15 treatments ($p<0.05$ compared to the control group). The flow cytometry data showed that agonist (G1) promoted G0/G1 to S phase, but antagonist (G15) arrested cell in G0/G1 phase. It showed that GPER-1 positively regulates cell proliferation on D1 cells. Moreover, the siRNA GPER-1 treatment also inhibited the cell cycle gene expression (cyclin D1 and cyclin E1) and cell proliferation ($p<0.05$ compared to the control group).
Discussion：According to a previous report, GPER-1 contributes to bone formation in in vivo study. However, the detail molecular mechanism is unclear. Osteogenic differentiation and cell proliferation in osteogenic cells contribute bone formation and osteogenesis. In our

previous study, we have demonstrated that GPER-1 may be a negatively regulator in osteo-differentiation. In this study, we further clarified that GPER-1 positively regulates cell proliferation in BMSCs. Our results showed that GPER-1 contribute to promote cell proliferation by enhancing cell cycle regulator, cyclin D1 and cylin E1. Moreover, GPER-1 enhanced G0/G1 phase to S phase in cell cycle. It showed that GPER-1 may contributed to cell proliferation in G0/G1 to S phase by enhancing cyclin D1 and cyclin E1 gene expression. The results showed that GPER-1 may play important roles in cell proliferation and osteogenic differentiation in BMSCs. The finding provides new information for the basic medical science in bone physiology and new drug development that act via GPER-1 function.

16:00-16:10

O-3

Comparison of Platelet-rich Plasma or Hyaluronic Acid with/without Bone Marrow Mesenchymal Stem Cells for the Treatment of Knee Osteoarthritis in a Rabbit Model

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Introduction：Articular cartilage lesions, with their inherent limited healing potential, are hard to treat. Numerous approaches have been proposed as noninvasive solutions for cartilage restoration treatment. However, none of the currently therapy can be considered an ideal procedure for the treatment of severe osteoarthritis(OA). Platelet-rich plasma (PRP) therapy is a convenient, non-expensive and minimally invasive method that provides a natural concentrate of autologous growth factors. Mesenchymal Stem Cells (MSCs) is an ideal cell source since it can repair/regenerate the diseased tissue. Thus, in the animal study, we try to compare the treatment effect of PRP, hyaluronic Acid (HA) with/without bone marrow MSC for OA therapy.
Materials and Methods：Eighteen New Zealand white rabbits were undergoing anterior cruciate ligament (ACL) transection to create knee osteoarthritis. Degenerated articular cartilage were then treated with PBS, PRP, HA, PRP+MSC and HA+MSC. Gross photograph exhibit the macroscopic changes in articular cartilage, while Safranin-O and immunohistochemistry (IHC) staining demonstrate the glycosaminoglycans (GAGs) and type II collagen content in articular cartilage.
Results：Compare with the results of HA group, the articular cartilage treated with PRP and PRP+MSC showed a more intact macro- and micro- morphology, and there is more GAG synthesis in these two groups. In the IHC staining results, the groups treated with PRP or PRP+MSC showed a type II collagen positive result.
Discussion：The degenerated cartilage by ACLT of different groups shown recovery of different levels, and Intra-articular injection of PRP+MSC possess the best stimulated healing ability compare with those of others and resulting the best condition in improved cartilage regeneration in rabbit's knee OA.
Conclusions：Based on the results of current study, PRP+MSC injection could be a promising method for the treatment of degenerated articular cartilage in the future.

16:10-16:20

O-4

Bone Marrow Mesenchymal Stem Cells Reduced LPS-Induced Inflammatory Response in Chondrocytes

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Introduction：Osteoarthritis (OA), a common forms of joint disease, affecting more than 80% of the population who reach the age of 70. It is related to focal and progressive hyaline articular cartilage loss, concomitant sclerotic changes in the subchondral bone and the development of osteophytes. Mesenchymal stromal cells injection therapy is a promising treatment for OA therapy since they are easily expanded in culture, generally none tumorigenic, and can readily obtain from patients. Most important, they express the immunosuppressive properties after exposure to an inflammatory environment by secretion of soluble factors.

Materials and Methods：Chondrocytes were isolated from porcine knee and then treated with different concentration of LPS to find the optimal concentration for inflammation induction. *In vitro* inflammatory chondrocytes were treated with three different ratio of MSC and gene expression for chondrocytes and was analyze by real-time PCR.

Results：Results showed that there were no significant difference in cell viability and cytotoxicity between untreated and LPS treated groups. But the cell morphology changed as the concentration of LPS increased, and the TNF-a, IL-6, iNOS and IL-1b mRNA of chondrocyte were up-regulated after 4 hours LPS incubation. Moreover, we found the MSC could down-regulated TNF-a, IL-6 and iNOS gene expression of chondrocyte after 24 hours incubation. The IL-1b mRNA of chondrocyte was also down-regulated after 72 hours incubation.

Discussion：*b*MSCs could retard the inflammatory response of chondrocyte, help the chondrocyte recover from LPS induce inflammation, and lead to the chondrocyte synthesis more ECM.

Conclusions：Based on the current results, MSC should possess an anti-inflammation ability, and it might be a promising cell source for OA therapy.

08:50-09:00

S-1

Using Bone Marrow Stromal Cells Modulated by Baculovirus-mediated MicroRNAs Sponge to Repair Osteoporotic Bone Defects

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Abstract：
Bone defects are common in elderly patients suffering from osteoporosis. Although many pharmacological agents prevent osteoporotic fractures, the repair of bone defects following fracture draws much less attention. MicroRNAs (miRNAs) are a class of small non-coding RNAs that play key regulatory roles in diverse biological processes, including osteoclastogenesis, osteoblast differentiation and bone formation. However, no study has attempted to utilize miRNAs for repairing osteoporotic bone defects. In this study, we found that bone marrow mesenchymal stem cells (BMSCs) harvested from rats with long-term estrogen deficiencies exhibited over-expression of microRNAs level (miR-30b, miR-138, miR-140 and miR-214) and decreased osteogenic capacity. To downregulate the miRNAs expression, we used baculovirus as a gene therapy vector to express microRNA sponges in BMSCs. We found that transduction of BMSCs with baculoviruses that over-expressed miR-140 or miR-214 sponges promoted osteogenesis, as evidenced by the elevated expression of runx2, alkaline phosphatase, osteocalcin, osteoprotegerin and matrix mineralization. Down-regulation of miR-140 or miR-214 expression in osteoporotic BMSCs by the baculovirus vectors also mitigated osteoclast maturation in a paracrine fashion. Whether the baculovirus-engineered osteoporotic BMSCs expressing microRNAs sponge were able to promote bone defect healing was evaluated by implanting the engineered cells into the critical-size defect at the femora of ovariectomised rats, followed by assessment of bone quality (volume, density, trabecular number, trabecular thickness and trabecular space) and bone healing by Skyscan μCT. These results collectively pave a new avenue to treatment of osteoporotic bone defects using miRNA-modulated BMSCs.

09:00-09:10

S-2

Stabilization of Collagen Nanofibers With L-Lysine Improves the Ability of Carbodiimide Cross-Linked Amniotic Membranes to Preserve Limbal Epithelial Progenitor Cells

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Introduction：To overcome the drawbacks associated with limited cross-linking efficiency of carbodiimide modified amniotic membrane (AM), this study aims to propose the use of L-lysine as an additional amino acid bridge to enhance the stability of nanofibrous tissue matrix for limbal epithelial cell (LEC) culture platform.
Materials and Methods：After further chemical modification of L-lysine-pretreated AM with carbodiimide, the collagen nanofiber stability against thermal denaturation and enzymatic digestion was determined. Quantitative real-time reverse transcription polymerase chain reaction and Western blot analyses were conducted on the LEC cultures to explore cell stemness associations with collagen nanofiber stability of L-lysine-pretreated and carbodiimide cross-linked AM substrates.
Results：Our data showed that the amount of positively charged amino acid residues incorporated into the tissue collagen chains is highly correlated with the L-lysine-pretreated concentration. In the range of 1 to 30 mM, the stemness gene and protein expressions in LECs were up-regulated with increasing amount of amino acid bridges in the chemically cross-linked AM scaffolds.
Discussion：Carbodiimide cross-linked AM can potentially serve as an artificial corneal epithelial stem cell nicheous matrix. The variations of resistances to thermal denaturation and enzymatic degradation are in accordance with the number of cross-links per unit mass of AM, indicating the L-lysine-modulated stabilization of collagen molecules. Cell culture studies suggest the crucial role of amino acid bridges in constructing suitable scaffolds to preserve limbal progenitor cells.
Conclusions：In summary, mild to moderate pretreatment conditions (i.e., 3-10 mM L-lysine) can provide a useful strategy to assist in the development of chemically cross-linked AM as a stable stem cell niche for corneal epithelial tissue engineering and regenerative medicine.

09:10-09:20

S-3

The Study of Cell and Bacteria Adherent Behavior on Different Functional Groups Surfaces

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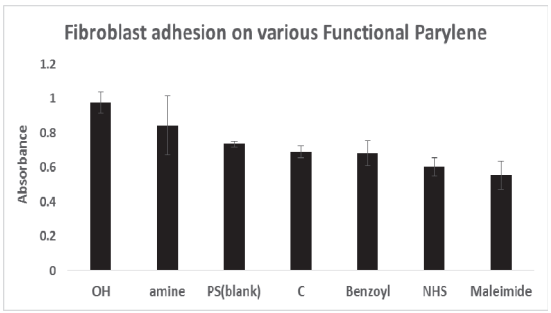
國立臺灣大學醫學系[1] 國立臺灣大學化學工程學系[2] 國立臺灣大學醫學院骨科[3]

Introduction：Cellular adhesion is a crucial cell function for establishing cell–cell contacts in tissues or cell-extracellular matrix contacts. Given the importance of cell adhesive surfaces in bioengineering and biotechnology, the development of in vivo biomimetic scaffold materials suitable for enabling cell attachment and for supporting a desirable architecture has been widely studied. In this study, we use functionalized-parylene with different kinds of functional groups on biomaterial surface via chemical vapor deposition (CVD), which is a facile, clean and substrate-independent approach for surface modification. Besides hydroxyl, carbonyl and amine groups, we have further studied on other functional groups commonly used for biomaterial modification, such as NHS ester, maleimide, benzoyl, and chloride (parylene C). To characterize these functionalized surfaces, we have analyzed the wettability, zeta potential, protein adsorption, and cell and bacteria adhesion of these modified surfaces.

Materials and methods：

1. Preparation for different functional group grafted parylene coating by CVD method.
2. Distinguish the surface characterization of functional parylene coating (QCM, FTIR, etc.).
3. Cell adhesion assay with NIH 3T3 mouse fibroblast cell and Escherichia Cloacae.



Results：Fibroblast cells showed different cell adhesion ability to functionalized-parylene coatings. The OH group grafted coating showed the highest adhesion ability, the absorbance was about 50% higher than maleimide grafted coating (the lowest adhesion surface).

Discussion：Micro-environmental cues direct a number of cell functions, such as adhesion, growth, survival, proliferation, migration, differentiation, and cell death. In the research, the hydrophobicity showed little effect on cell adhesion; on the other hand, the surface charge and the specific binding between the adsorbed protein and the cell membrane showed strong relation to cell and bacteria adhesion through changes in surface interaction.

Conclusions：Manipulating an incorporated scaffold to direct cell behaviors play a key role in tissue engineering. In this study, we have clarified the effect of cell adhesion on different functionalized-parylene coatings.

09:20-09:30

S-4

Reduction of Cardiomyocyte Apoptosis & Oxidative Stress by Human Placenta-Derived Multipotent Cells (PDMCs) is Enhanced by ECM Protein-Integrin Interactions

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亞東紀念醫院心臟血管醫學中心[3] 國立臺灣大學附設醫院婦產部及臺灣大學醫學院醫學系[4]

Introduction： In myocardial ischemia (MI), it has been identified that high levels of tumor necrosis factor-α (TNFα) in the infarct and border zones result in cardiomyocyte apoptosis and reactive oxygen species (ROS) production, which leads to further cell death. Thus, it is important for effective therapy of MI to diminish the degree of apoptosis and ROS. In recent years, stem cell therapy has been proposed as a novel therapy for MI. Placenta-derived multipotent stem cells (PDMCs) are multipotent cells expressing bone marrow mesenchymal stem cells (BMMSCs) and embryonic stem cell markers, and can differentiate into lineages of all three germ layers. Moreover, PDMCs are easily isolated and expanded *ex vivo,* and harbor strong immunomodulatory properties. We therefore were interested in exploring whether PDMCs are therapeutic for MI

Materials and Methods： Our animal model data demonstrate that injection of PDMCs in both a mouse and pig model of acute MI can significantly increase cardiac function. To assess the mechanisms involved in these effects, we co-cultured PDMCs with TNF -induced, apoptotic mouse cardiomyocytes

Results： We found that PDMCs can promote survival and retard apoptosis in cardiomyocytes through paracrine effects. Specifically, we found that cardiomyocyte apoptosis could be reduced by a profile of PDMC secreted factors. Moreover, these factors could also reduce intracellular ROS in cardiomyocytes with upregulation of antioxidant enzymes. Since PDMCs/MSCs are defined by *in vitro* culturing, we explored whether coating of various extracullar matrix (ECM) proteins could affect the secretory ability of PDMCs. We found that PDMCs cultured on ECM-coated cultured plates increased secretion of a number of paracrine factors, and decreased cardiomyocyte apoptosis more effectively. To elucidate how the ECM proteins mediate these functional changes in PDMCs, we silenced the expression of specific integrins—which are often receptors for various ECM proteins—expressed on PDMCs. We found that abrogation of specific integrin expression on PDMCs can affect paracrine factor secretion profile and decrease the anti-apoptotic effects of PDMCs on cardiomyocytes.

Discussion： It may a good strategy to improve the therapeutic arsenal against cell death by enhancing paracrine mechanisms.

Conclusions： Our study demonstrates the strong therapeutic applicability of PDMCs and the important role of the ECM on diverse MSC/PDMC function.

09:30-09:40

S-5

Effective Transfer of Naked Plasmid into Stem Cells Grown on Silica Nanosheets

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Introduction：Naked plasmid enters cells rather poorly. For this reason, there are two strategies for delivering plasmid into cells, forward and reverse transfection, but the efficiency of transfection without using any transfection agent (TA) is still very low. Our previous study has shown that a dense 3-aminopropyltriethoxysilane (APTES) modified silica upright nanosheets could enhance the transfer of plasmid/TA complexes into cells by the reverse transfection protocol. The current study focused on transferring a naked gene into human mesenchymal stem cells (hMSCs) by the silica upright nanosheets without the use of any vector.

Materials and Methods：The nanosheets with a wall depth of 5 nm covered on planar glass were directly used or modified with ATPES. Cells were cultured in planar silica (Planar), non-modified silica upright nanosheets (Nano), and APTES-modified silica upright nanosheets (Nano-NH_2) where they were concurrently exposed to the naked GATA binding protein 4 (GATA4) plasmids in serum-free medium for 12 h. The gene expression of GATA4 examined at 60 h and the immunostaining observed at 7 d.

Results：The gene expression of GATA4 for hMSCs on Nano-NH_2 and Nano reached to a similar level as that of the PolyFect-transfected group but had higher cell survival rate. The expression of the other two cardiac-associated genes, MEF2C and TBX5, was also upregulated in GATA4 transfected cells on Nano-NH_2. The immunofluorescence staining of cardiac-associated marker proteins were positive for cells plated on Nano (~70%) and Nano-NH_2 (~80%). On Planar, only GATA4 showed weak intensity (~3%). The expression of integrin 3 and v were upregulated on Nano and Nano-NH_2 at 12 h but did not endure at 60 h. The expressions of focal adhesion kinase (FAK) and Rho were upregulated on Nano and Nano-NH_2 at 60 h.

Discussion：The transfection efficiency and cell survival rate of Nano-NH_2 were close to those of Nano, suggesting that the positive charge on silica nanosheets and the electronic interaction between nanosheets and cells may not be the dominating mechanisms behind the enhanced gene delivery. The vector-free delivery of naked plasmid is achieved by the integrin-FAK-Rho signaling axis which can be activated by nanotopography in a limited time frame. hMSCs transfected with the naked GATA4 plasmid showed cardiac-associated markers, suggesting cardiac differentiation.

Conclusions：The silica upright nanosheets can promote the transfection of naked GATA4 plasmid into hMSCs without any transfection reagent. The vector-free silica nanosheet-induced transfection is simple and effective while faster and safer than the conventional technologies.

09:40-09:50

S-6

三維列印矽酸鈣/氫氧基磷灰石可降解支架之骨細胞貼附研究
Study of Bone Cell Adhesion on Degradable Scaffold by $CaSiO_3$/HA 3D Printing

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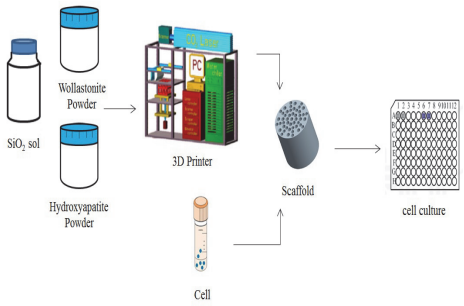
介紹：生物可降解材料可避免二次手術感染風險，而常用之生物可降解材料多為生物親合性高之高分子以及矽酸鈣陶瓷材料，但可降解性客製化之生醫陶瓷材料製作困難及機械強度不足，一直是研究者欲解決之問題。所以本研究之目標是為了同時解決上述兩項缺點，本研究使用矽酸鈣(Wollastonite, $CaSiO_3$)粉末、氫氧基磷灰石(Hydroxyapatite, HA)粉末及二氧化矽溶膠(SiO_2 sol)作為材料，採用三維列印技術將陶瓷材料建構成支架，藉由調整矽酸鈣以及氫氧基磷灰石不同的重量比進行 3D 列印成形，並測量支架之機械性質變化及探討 3D 支架對骨細胞的貼附及融合特性。



材料與方法：本研究使用矽酸鈣(Wollastonite, $CaSiO_3$)粉末、氫氧基磷灰石(Hydroxyapatite, HA)粉末及二氧化矽溶膠(SiO_2 sol)為材料，利用自製三維列印機透過選擇性雷射凝膠法(Selective laser gelling, SLG)並設計製作不同可降解性生醫陶瓷支架，進行材料毒性測試、骨細胞(MG63)之貼附、爬行及融合特性之研究。

結果：透過自製三維列印機以選擇性雷射凝膠法製作不同比例配方之 3D 生胚(Green part)，再將生胚進行熱處理，探討其機械性質，結果顯示添加 50%之矽酸鈣時可達到最高抗壓強度 34MPa。而 3D 陶瓷材料也無細胞毒性，且對骨細胞親合力高，本研究將探討此複合材料在長時間且不同培養液下之降解速率對骨細胞貼附及融合之影響。

討論：三維列印與傳統多孔性支架的製作方法不同，其優點在於不僅可製作出客製化內連通孔，也可依照支架所需要的結構強度做設計，以選擇性雷射凝膠法製作生醫陶瓷支架，將矽酸鈣(Wollastonite, $CaSiO_3$)、氫氧基磷灰石(Hydroxyapatite, HA)及二氧化矽溶膠(SiO_2 sol)以球磨方式混合為生醫陶瓷漿料，製作不同比例配方陶瓷支架。探討不同比例配方對於生醫陶瓷支架之機械強度影響及降解速率之影響同時分析 3D 植入物對骨細胞貼附性及生長狀況。

結論：本研究使用矽酸鈣、氫氧基磷灰石及二氧化矽溶膠混合之漿料，透過自製三維列印機以選擇性雷射凝膠法可客製出多孔可降解之生醫陶瓷支架，並獲得不同材料之生物降解率變化及對骨細胞貼附性、活性之影響，而這些生醫陶瓷三維支架，期能應用於未來臨床生醫可降解材料之選擇。

16:20-16:30

S-7

The Study of Matrix Stiffness of Hyaluronan(HA) Hydrogel for Cytocompatibility of Human Adipose Derived Stem Cells

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Introduction: Hyaluronan (HA) is one of the major extracellular matrix (ECM) components of articular cartilage, our previous study indicated that HA-microenvironment enhanced chondrogenesis of human adipose derived stem cells (hADSCs). The ECM stiffness has reported that influence stem cells fate, including proliferation and differentiation. However, little is known about the stiffness of HA enriched ECM on chondrogenesis of hADSCs. Based on this reason, the aim of this study is to develop HA based hydrogel with different matrix stiffness and test the cytocompatibility on hADSCs.
Materials and Methods: hADSCs are acquired from human patient undergoing orthopedic surgery. For the fabrication of HA based hydrogels, 1% methacrylated HA (MeHA) will be produced (The degrees of methacrylation will be measured by FT-IR). For UV cross-link reaction, 1% MeHA were dissolved 0.05% Irgacure 2959 (I2959) solution. The mixture was cross-linked with UV radiated for 10 min to obtain solid CL-MHs (These are HA based hydrogels with different matrix stiffness). The Young's moduli of CL-MHs will be evaluated under unconfined compression with a texture analyzer at a strain rate of 0.1 mm/sec until 10 % of gel thickness, and the stress will be recorded. The Young's moduli will be displayed as stress/strain. Cell survival will be assessed based on the integrity of the cellular membrane using a LIVE/DEAD® Viability/Cytotoxicity Kit**,** which contains calcein-AM (live dye, green) and ethidium homodimer-1 (dead dye, red). The ADSCs proliferation on CL-MHs was examined using MTS assay.
Results: The young's modulus of CL-MH showed that the matrix stiffness of CL-MH increased with changes methacrylation rate of HA. The young's modules of CL-MHs range from 2kPa to 10kPa. The Water uptake show that the CL-MH exhibited swollen with changes of methacrylation. Live/Dead stain also showed that the ADSCs remain survival on the surface of CL-MHs. The MTS assay found that hADSCs cultured in 65% and 85% CL-MH, the cell viability increased with culture days.
Conclusion: This study showed that increased the crosslinking degree of HA-MA hydrogel results in raising the construct elasticity and less cytotoxicity to hADSCs at day 1. Moreover, the more pronounced cell proliferation of hADSCs in 65%, 85% CL-MH then that in 15, 30 and 140% CL-MHs at day 5. In this study, we found that matrix stiffness of HA based hydrogel affects cytocompatibiliy of hADSCs in vitro.

16:30-16:40

S-8

Enhanced Proliferation and Life Span of Human Fibroblasts by the Formation of Cell Spheroids on Chitosan

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近年來，纖維母細胞已被廣泛地使用於細胞老化之研究。因此，本研究將使用人的初代包皮纖維母細胞 (Primary human foreskin fibroblasts)為模型，欲了解當初代纖維母細胞由市售之組織培養皿 (commercial tissue culture polystyrene, TCPS) 移種於生醫材料-幾丁聚醣 (Chitosan) -塗佈之基材時，細胞的增殖能力是否能有效地受到調控。首先，由實驗數據可以得知：分離所得之纖維母細胞體外培養 (*in vitro*)於 TCPS 上時，細胞繼代次數 (Population doubling times, PDs) 的最大值約為 55-60，隨著繼代次數的增加，細胞變得大且扁平，增殖速率下降，老化相關 β-半乳糖苷酶 (SA-β-gal)和分子蛋白 (p21, p53, pRB, 和 p16)的表現量也都顯著地累積。因此，當細胞到達複製性老化 (Replicative senescence)前 (PD48)，我們收集細胞移種於幾丁聚醣基材上三天，細胞於此基材上會懸浮聚集成直徑約 50-100 nm 之多細胞球。接著，我們將這群經過幾丁聚醣處理的細胞球再種回 TCPS 上，細胞會由細胞球逐漸爬出，且展現較佳的增殖能力和調降老化相關 β-半乳糖苷酶 (SA-β-gal)的表現活性。此外，經過幾丁聚醣處理之細胞其體外繼代次數甚至可延長至 70-75 代，但老化相關分子蛋白並不會因處理而受到調節。由初步實驗結果推測，幾丁聚醣處理使細胞聚集成球之過程，將導致細胞處於缺氧氣和養分之環境，此嚴苛環境能促使纖維母細胞提升增殖能力和降低老化表現。但幾丁聚醣的分子特性是否也是影響此現象的因素，而其影響作用之機制為何，將是我們未來欲深入探討和了解的。此研究初步證實人類的初代纖維母細胞經過幾丁聚醣的處理後能顯著地延遲複製性老化的發生，此實驗結果將有助於未來組織工程與細胞治療的發展。

Key words: 人類纖維母細胞, 老化, 幾丁聚醣, 細胞球

16:40-16:50

S-9

Effect of Riboflavin Concentration on The Development of Photo-Crosslinked Amniotic Membranes for Cultivation of Limbal Epithelial Cells

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Introduction：To overcome the limitations of possible toxicity of chemical cross-linkers, the amniotic membrane (AM) was photo-cross-linked by ultraviolet (UV) irradiation in the presence of riboflavin. This study aims to investigate the effect of photoinitiator concentration on the preparation of photo-cross-linked AM materials for cultivation of limbal epithelial cells (LECs)

Materials and Methods：Before exposure to UV for the duration of 20 min, the AM samples were treated with different concentrations of riboflavin. The cross-linking density and matrix stability of AM were determined. The photo-cross-linking-mediated alterations in AM ultrastructure and nanotopography were confirmed by transmission electron and atomic force microscopy studies. In vitro biocompatibility and cell culture performance of photo-cross-linked AM materials was also assessed.

Results：The number of cross-links per unit mass of collagen matrix was significantly increased with increasing riboflavin concentration from 0.1 to 10 mg/ml. In addition, the equilibrium water content, ultrastructure, nanotopography, and enzymatic degradability of AM were found to be associated with cross-linked structure of UV-irradiated biological tissues. Irrespective of riboflavin concentration, the test materials were biocompatible and retained anti-inflammatory activities.

Discussion：The physically cross-linked AM materials can be successfully prepared by the photo-cross-linking method with various amounts of UV-activated riboflavin. The absence of exogenous cross-linker molecules in the proteinaceous matrices following photo-cross-linking reaction is advantageous to maintain the biocompatibility of AM. Our findings suggest that both the cross-linking structure and surface roughness of AM have profound influences on the levels of enhanced cell stemness.

Conclusions：Here we report that the riboflavin concentration may play an important role in the modulation of properties of photo-cross-linked AM material as a new LEC carrier.

16:50-17:00

S-10

Study of Bone Regeneration on Bio-ceramic Scaffolds by ZrO_2/HA 3D Printing

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介紹：氫氧基磷灰石(Hydroxyapatite $Ca_{10}(PO_4)_6(OH)_2$, HA)為臨床上常用之生醫陶瓷材料，但其材料屬於硬脆特性，不易加工。因此，本研究用氫氧基磷灰石粉末透過選擇性雷射凝膠法(Selective laser gelling, SLG)製作生醫陶瓷支架。而此支架的抗壓強度僅 20 MPa，若要應用於臨床使用上稍顯不足，所以本研究即透過添加高機械強度之氧化鋯於氫氧基磷灰石中，藉此提升其機械強度做為骨再生醫學支架之可能性。

材料與方法：本研究採用成形方法為選擇性雷射凝膠法、材料為氧化鋯粉末(ZrO_2)、氫氧基磷灰石(Hydroxyapatite, HA)粉末與二氧化矽溶膠(SiO_2 Sol)之陶瓷漿料為主。

結果：本研究透過添加不同比例之氧化鋯於氫氧基磷灰石中，利用基層製造(Additive Manufacturing, AM)技術製作陶瓷生胚(Green part)。再將陶瓷生胚進行熱處理，藉由熔融原理可提高其機械強度。透過本研究之材料及方法製作而成之生醫陶瓷支架，其抗壓強度可達 100 MPa，最後再由體外細胞培養(cell culture)，觀察骨母細胞(osteoblast)在高硬度材質上之貼附情形以及細胞活性進行探討。

討論：本研究使用選擇性雷射凝膠法(SLG)，藉著溶膠-凝膠法使材質固化，係與市面上採用選擇性雷射燒結法直接將粉末熔融燒結的原理不同，本研究方法是較為環保且適用於綠色材料上。而本研究將氧化鋯粉末(ZrO_2)、氫氧基磷灰石(Hydroxyapatite, HA)粉末與二氧化矽溶膠(SiO_2 Sol)，依照不同的配方比例用球磨機混合成均勻分散的含水漿料，使用自行組裝的三維列印機(3D Printer)，可做出不同比例之三維高強度陶瓷支架，此一 3D 陶瓷支架因含有不同複合材質，所以與細胞之貼附性、細胞活性將呈現不同的影響。

結論：本研究使用選擇性雷射凝膠法，藉著溶膠-凝膠法使其固化，以 CO_2 雷射做為能量源並選用適當之雷射參數做為成形條件，使 3D 陶瓷支架擁有不同的機械性質，並闡述 3D 生醫陶瓷植入物與骨細胞之貼附性、骨細胞之鑲嵌性及骨細胞活性的影響，最後找出氧化鋯粉末(ZrO_2)、氫氧基磷灰石(Hydroxyapatite, HA)粉末與二氧化矽溶膠(SiO_2 Sol)最佳比例，作為植入物適用於臨床上骨再生之依據。

第六屆第二次會員大會

台灣再生醫學學會第六屆理、監事名單
(照筆劃順序排列)

理 事 長　楊台鴻

秘 書 長　洪士杰

常務理事　林峰輝、徐善慧、陳耀昌、嚴孟祿

理　　事　方旭偉、王兆麟、王至弘、江清泉、何美泠、林泰元、張至宏、陳敏慧、黃玲惠、蔡清霖

候補理事　林高田、陳文哲、陳志華、楊俊佑、楊榮森

常務監事　黃義侑

監　　事　侯連圍、孫瑞昇、張瑞根、鄭乃禎

候補監事　顏伶汝

第六屆第二次會員大會議程

時間：民國104年3月7日(星期六) 12:10

地點：臺大醫學院103講堂(台北市仁愛路一段一號)

主席：楊台鴻　理事長

一、大會開始

二、主席致詞

三、理、監事會工作報告

四、討論事項

1. 通過 103 年度工作報告、104 年度工作計劃
2. 通過 103 年度經費收支決算 、104 年度收支預算

五、臨時動議

六、散會

103年度工作報告

理事會報告

一、召開理監事會議計四次。

二、會員實際人數一百八十三人。

監事會報告

一、理事會處理會務均係依據大會章程辦理，遇有重大事項召開理監事聯席會議商討決定。

二、理事會所編財務報告，業經本監事會審核無誤。

三、本屆理事會竭盡全力推展會務。

104年工作計劃

一、招收會員

二、舉行三次理監事會議。

三、舉辦學術交流研討會。

四、隨時通知會員學會相關訊息。

台灣再生醫學學會

收支決算表

中華民國 103 年 1 月 1 日至 103 年 12 月 31 日止

科目				決算數	預算數	決算與預算比較數		說明
款	項	目	科目			增加	減少	
1			**本會收入**	**63,811**	**600,000**			
	1		會費收入	62,500	140,000		77,500	含入會費、常年會費
	2		捐款收入	0	450,000		450,000	廣告攤位收入及贊助會員捐款等
	3		利息收入	1,311	10,000		8,689	郵局、銀行利息
2			**本會支出**	**528,576**	**600,000**			
	1		人事費	244,000	180,000	64,000		員工及加班費
	2	1	文具	575	5,000		4,425	
		2	印刷費	4,319	50,000		45,681	
		3	郵電費	4,812	5,000		188	
		4	雜項	2,738	10,000		7,262	
	3	1	業務費	59,665	40,000	19,665		召開理監事會及辦理相關研討會所需之費用
		2	會議費	64,000	60,000	4,000		
		3	交通費	28,000	20,000	8,000		
			其他業務費	120,476	200,000		79,524	召開 2 月份年會
			提撥基金	0	30,000		30,000	
			本期結餘	-464,765				

理事長：　秘書長：　常務監事：　會計：　製表：

台灣再生醫學學會
收支預算表
中華民國 104 年 1 月 1 日至 104 年 12 月 31 日止

科目				預算數	上年度預算數	本年度與上年度預算比較數		說明
款	項	目	科目			增加	減少	
1			**本會收入**	**530,000**	**600,000**			
	1		會費收入	120,000	140,000		20,000	含入會、費常年會費
	2		捐款收入	400,000	450,000		50,000	廣告攤位收入及贊助會員捐款等
	3		利息收入	10,000	10,000			郵局銀行利息
2			**本會支出**	**530,000**	**600,000**			
	1		人事費	180,000	180,000			員工薪資及加班費
	2	1	文具	3,500	5,000		1,500	
		2	印刷費	12,000	50,000		38,000	
		3	郵電費	5,000	5,000			
		4	雜項	5,000	10,000		5,000	
	3	1	業務費	60,000	40,000	20,000		召開理監事會及辦理相關研討會所需之費用
		2	會議費	60,000	60,000			
		3	交通費	28,000	20,000	8,000		
			其他業務費	150,000	200,000		50,000	
			提撥基金	26,500	30,000		3,500	依收入總額提列 5%作為準備基金

理事長：　　秘書長：　　常務監事：　　會計：　　製表：

台灣再生醫學學會
現金出納表
中華民國 103 年 1 月 1 日至 103 年 12 月 31 日止

收入		支出	
科目名稱	金額	科目名稱	金額
上期結餘	$1,696,754	本期支出	$528,576
本期收入	$63,811	本期結餘	$1,231,989
合計	$1,760,565	合計	$1,760,565

理事長： 秘書長： 常務監事： 會計： 製表：

台灣再生醫學學會
資產負債表
中華民國 103 年 1 月 1 日至 103 年 12 月 31 日止

資產		負債	
科目	金額	科目	金額
庫存現金	$231,989	累計基金	$104,000
定期存款	$1,000,000	本期結餘	$1,127,989
合計	$1,231,989	合計	$1,231,989

理事長： 秘書長： 常務監事： 會計： 製表：

台灣再生醫學學會章程

第一章 總 則

第 一 條 本會名稱為 **台灣再生醫學學會**(以下簡稱本會)。

第 二 條 本會以國內外人士共同發揚再生醫學之研究、教學及應用為宗旨。

第 三 條 本會以全國行政區域為組織區域。

第 四 條 本會會址設於主管機關所在地區。本會得視會員人數及分配與會務進行之需要設分會與各種委員會，其組織簡則由理事會擬訂，報請主管機關核准後實施，變更時亦同。

第 五 條 本會之任務如左：

一、提倡再生醫學之研究並發揚醫學倫理道德。

二、調查國內外再生醫學之發展，徵集有關圖書資訊以供各學術團體之參考及交流。

三、舉辦學術演講及討論會。

四、出版會誌及有關書刊。

五、獎助再生醫學及組織工程學人才及舉辦其他有關事宜。

六、與有關公司、廠商及機構合作，共求再生醫學及組織工程學之發展與應用。

第 六 條 本會之主管機關為內政部。

本會之目的事業應受各該事業主管機關之指導、監督。

第二章 會 員

第 七 條 本會會員申請資格如下：

一、個人會員：凡贊同本會宗旨、取得中華民國醫師執照者或取得與再生醫學、組織工程學相關博士學位者並經理事會通過後得申請為本會個人會員。

二、贊助會員：贊助本會工作之團體或個人。 申請時應填具入會申請書，經理事會通過，並繳納會費後，始得為本會贊助會員。

三、準 會 員：凡贊同本會宗旨的碩、博士班學生、博士後研究員、 住院醫師、研究助理或等同資格者，由會員二人推薦，經理監事會審查通過，得為本會準會員。

第 八 條 會員(會員代表)有表決權、選舉權、被選舉權與罷免權。每一會員(會員代表)為一權。 贊助會員、準會員無前項權利。

個人會員另享有 1. 參加本會年會及本會所舉辦之其他集會之權利。

2. 參加本會所舉辦各種活動或事業之權利。

3. 本會各種書刊訂閱優待之權利。

贊助會員享有 1. 參加本會年會及本會所舉辦之其他集會之權利。

2. 本會出版之資訊及刊物贈閱之權利。

準會員享有　　1. 參加本會年會及本會所舉辦之其他集會之權利。
2. 本會出版之資訊及刊物贈閱之權利。

第　九　條　會員有遵守本會章程、決議及繳納會費之義務。
會員每年年初需繳納會費，以利本會之運作。未繳納會費者，不得享有會員權利；連續二年未繳納會費者，視為自動退會。會員經出會、退會或停權處分，如欲申請復會或復權時，除有正當理由經理事會審核通過者外，應繳清前所積欠之會費。

第　十　條　會員(會員代表)有違反法令，章程或不遵守會員大會決議時，得經理事會決議，予以警告或停權處分，其危害團體情節重大者，得經會員(會員代表)大會決議予以除名。

第 十一 條　會員喪失會員資格或經會員大會決議除名者，即為出會。

第 十二 條　會員得以書面敘明理由向本會聲明退會。

第三章　組織及職權

第 十三 條　本會以會員大會為最高權力機構。
會員人數超過三百人以上時得分區比例選出會員代表，再召開會員代表大會，行使會員大會職權。會員代表任期二年，其名額及選舉辦法由理事會擬訂，報請主管機關核備後行之。

第 十四 條　會員大會之職權如左：
一、訂定與變更章程。
二、選舉及罷免理事、監事。
三、議決入會費、常年會費、事業費及會員捐款之數額及方式。
四、議決年度工作計畫、報告及預算、決算。
五、議決會員(會員代表)之除名處分。
六、議決財產之處分。
七、議決本會之解散。
八、議決與會員權利義務有關之其他重大事項。前項第八款重大事項之範圍由理事會定之。

第 十五 條　本會置理事十五人、監事五人，由會員(會員代表)選舉之，分別成立理事會、監事會。 選舉前項理事、監事時，依計票情形得同時選出候補理事五人，候補監事一人，遇理事、監事出缺時，分別依序遞補之。本屆理事會得提出下屆理事、監事候選人參考名單。

理事、監事得採用通訊選舉，但不得連續辦理。通訊選舉辦法由理事會通過，報請主管機關核備後行之。

第 十六 條　理事會之職權如左：
一、審定會員(會員代表)之資格。
二、選舉及罷免常務理事、理事長。
三、議決理事、常務理事及理事長之辭職。
四、聘免工作人員。
五、擬訂年度工作計畫、報告及預算、決算。
六、其他應執行事項。

第 十七 條　理事會置常務理事五人，由理事互選之，並由理事就常務理事中選舉一人為理事長。理事長對內綜理督導會務，對外代表本會，並擔任會員大會、理事會主席。理事長因事不能執行職務時，應指定常務理事一人代理之，未指定或不能指定時，由常務理事互推一人代理之。理事長、常務理事出缺時，應於一個月內補選之。

第 十八 條　監事會之職權如左：
一、監察理事會工作之執行。
二、審核年度決算。
三、選舉及罷免常務監事。
四、議決監事及常務監事之辭職。
五、其他應監察事項。

第 十九 條　監事會置常務監事一人，由監事互選之，監察日常會務，並擔任監事會主席。常務監事因事不能執行職務時，應指定監事一人代理之，未指定或不能指定時，由監事互推一人代理之。
監事會主席(常務監事)出缺時，應於一個月內補選之。

第 二十 條　理事、監事均為無給職，任期二年，連選得連任。理事長之連任，以一次為限。

第二十一條　理事、監事有左列情事之一者，應即解任：
一、喪失會員(會員代表)資格者。
二、因故辭職經理事會或監事會決議通過者。
三、被罷免或撤免者。
四、受停權處分期間逾任期二分之一者。

第二十二條　本會置秘書長一人，承理事長之命處理本會事務，其他工作人員若干人，由理事長提名經理事會通過聘免之，並報主管機關備查。但秘書長之解聘應先報主管機關核備。前項工作人員不得由選任之職員擔任。工作人員權責及分層負責

事項由理事會另定之。

第二十三條　本會得設各種委員會、小組或其他內部作業組織，其組織簡則經理事會通過後施行，變更時亦同。

第二十四條　本會得由理事會聘請名譽理事長一人，名譽理事、顧問各若干人，其聘期與理事、監事之任期同。

第四章　會　　議

第二十五條　會員大會分定期會議與臨時會議二種，由理事長召集，召集時除緊急事故之臨時會議外應於十五日前以書面通知之。定期會議每年召開一次，臨時會議於理事會認為必要，或經會員(會員代表)五分之一以上之請求，或監事會函請召集時召開之。本會辦理法人登記後，臨時會議經會員(會員代表)十分之一以上之請求召開之。

第二十六條　會員(會員代表)不能親自出席會員大會時，得以書面委託其他會員(會員代表)代理，每一會員(會員代表)以代理一人為限。

第二十七條　會員(會員代表)大會之決議，以會員(會員代表)過半數之出席，出席人數較多數之同意行之。但章程之訂定與變更、會員(會員代表)之除名、理事及監事之罷免、財產之處分、本會之解散及其他與會員權利義務有關之重大事項應有出席人數三分之二以上同意。

本會辦理法人登記後，章程之變更以出席人數四分之三以上之同意或全體會員三分之二以上書面之同意行之。本會之解散，得隨時以全體會員三分之二以上之可決解散之。

第二十八條　理事會、監事會至少每六個月各舉行會議一次，必要時得召開聯席會議或臨時會議。前項會議召集時除臨時會議外，應於七日前以書面通知，會議之決議，各以理事、監事過半數之出席，出席人數較多數之同意行之。

第二十九條　理事應出席理事會議，監事應出席監事會議，不得委託出席。 理事、監事連續二次無故缺席理事會、監事會者，視同辭職。

第五章　經費及會計

第 三十 條　本會經費來源如左：

一、入會費：個人會員新台幣壹仟元，於會員入會時繳納。

贊助會員新台幣壹仟元，於會員入會時繳納。

準會員新台幣五百元，於會員入會時繳納。

二、常年會費：個人會員新台幣壹仟元。

贊助會員新台幣貳仟元。

準會員新台幣五百元。

三、事業費。

四、會員捐款。

五、委託收益。

六、基金及其孳息。

七、其他收入。

第三十一條　本會會計年度以曆年為準，自每年一月一日起至十二月三十一日止。

第三十二條　本會每年於會計年度開始前二個月由理事會編造年度工作計畫、收支預算表、員工待遇表，提會員大會通過(會員大會因故未能如期召開者，先提理監事聯席會議通過)，於會計年度開始前報主管機關核備。並於會計年度終了後二個月內由理事會編造年度工作報告、收支決算表、現金出納表、資產負債表、財產目錄及基金收支表，送監事會審核後，造具審核意見書送還理事會，提會員大會通過，於三月底前報主管機關核備(會員大會未能如期召開者，先報主管機關。)

第三十三條　本會解散後，剩餘財產歸屬所在地之地方自治團體或主管機關指定之機關團體所有。

第六章　附　　則

第三十四條　本章程未規定事項，悉依有關法令規定辦理。

第三十五條　本章程經會員(會員代表)大會通過，報經主管機關核備後施行，變更時亦同。

第三十六條　本章程經本會93年2月7日第一屆第一次會員大會通過。
報經內政部93年5月14日台內社字第0930018951號函准予備查。

台灣再生醫學學會會員名單：

編號	姓　名	編號	姓　名	編號	姓　名	編號	姓　名
001	劉華昌	029	林瑞模	061	鍾瑞嶂	091	李宣書
002	侯勝茂	030	徐郭堯	062	范揚峰	092	楊長彬
003	陳耀昌	032	吳輝傑	063	戴浩志	093	王貞棣
004	楊台鴻	033	蕭逸民	064	洪士杰	095	楊曙華
005	楊榮森	034	李建和	066	劉有漢	096	邱錦輝
006	楊俊佑	036	黃振勳	067	許致榮	097	郭兆塋
007	林峰輝	037	施庭芳	068	黃國淵	098	陳學明
008	林文澧	038	侯連圍	069	李裕滄	099	林柳池
009	王清貞	039	陳志華	070	陳沛裕	100	潘如瑜
010	黃義侑	040	李炫昇	072	林頌然	101	楊維宏
011	王兆麟	041	張瑞根	073	游敬倫	102	劉明偉
012	江清泉	044	李敏旭	075	陳吳坤	103	王文志
013	石朝康	045	江鴻生	076	鄧文炳	104	方旭偉
014	蔡清霖	046	陳昭宇	077	鄭耀山	105	陳敏慧
015	張恆雄	047	張宗訓	079	簡松雄	106	張明熙
017	蘇芳慶	048	釋高上	080	郭繼陽	107	陳興源
018	陳瑞明	050	張至宏	081	王世杰	108	蔡文龍
019	陳全木	051	蔡慶豐	082	蔡友士	109	郭宗甫
020	童瑞年	054	楊治雄	083	王至弘	110	王禎麒
021	殷金儉	055	蔡文基	084	張志豪	111	湯月碧
022	何始生	056	林高田	085	趙建銘	112	黃玲惠
023	孫瑞昇	057	古鳴洲	087	曾鵬文	113	王佩華
026	陳文哲	058	宋信文	088	徐明洸	114	郭源松
027	周正義	059	姚俊旭	089	詹益聖	115	翁文能
028	陳英和	060	呂紹睿	090	吳錫銘	116	徐善慧

編號	姓　名	編號	姓　名	編號	姓　名	編號	姓　名
117	蘇正堯	138	許文明	159	王德原	180	陳宣佑
118	楊世偉	139	黃鶴翔	160	賴志毅	181	邵宏仁
119	林偉彭	140	陳偉勵	161	吳佳慶	182	徐永康
120	謝豐舟	141	劉席瑋	162	沈延盛	183	賴瑞陽
121	方紀宇	142	李冠瑢	163	李一麟	184	薛元毓
122	蘇慶華	143	胡育誠	164	何美泠	185	施明光
123	曾育弘	144	黃維超	165	楊宗霖	186	曾效參
124	林佐文	145	陳安泰	166	吳坤佶	187	趙崧筌
125	史　中	146	謝清河	167	趙本秀	188	黃柏誠
126	鄭乃禎	147	彭慶安	168	鄭明德	189	彭凱彥
127	謝式洲	148	劉滄梧	169	李源芳	190	李奎璋
128	蘇鴻麟	149	薛敬和	170	嚴孟祿	191	陳崇桓
129	曾清秀	150	林毅誠	171	顏伶汝	192	朱恆毅
130	劉百栓	151	顏君哲	172	林泰元	193	傅尹志
131	唐逸文	152	陳江山	173	陳尹愷	194	陳達慶
132	王清正	153	侯君翰	174	許元銘	195	吳順成
133	王盈錦	154	吳俊昇	175	鄭有仁	196	陳郁君
134	吳信志	155	廖振焜	176	侯添財	197	黃惠君
135	簡雄飛	156	傅再生	177	賴文福	198	洪堃哲
136	高國慶	157	蔡宗廷	178	施子弼	199	曾庭箴
137	徐新生	158	羅文政	179	黃鼎鈞		

台灣再生醫學學會　個人會員入會申請書

姓　　名		性別		出　生 年月日		出生地		身分證 號　　碼	
學　　歷	民國　　年　　月畢業於								
戶籍住址									
現任職務	醫院或單位：			科部：		職稱：			
服務單位 地　　址						專科醫師證書字號： (無者免填)			
電　　話	(公)			(宅)		傳真：			
其他連絡 方　　式	電子信箱(e-mail)：					行動電話：			
審查結果 (由學會填寫)		會員類別 (由學會填寫)			會員證號碼 (由學會填寫)				

本人贊同貴會宗旨，擬加入為會員，嗣後並願意遵守會章，共圖發展

此致　　台灣再生醫學學會　　申請人：　　(簽章)

中　華　民　國　　年　　月　　日

會員資料異動申請書

本單填妥後請回傳至台灣再生醫學學會

Fax: 02-8921-3969

會員姓名：

變 更 為：

郵遞區號：

通訊地址：

服務單位：

聯絡電話：

傳　　真：

e-mail：